Anesthesiology
V 41, No 2, Aug 1974

General Principles and Determinants of Circulatory Transport

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THE HIGHLY DEVELOPED homeostatic relationship of the complex mammalian organism to the environment depends on the system of circulatory transport of materials to and from each organ and tissue of the body. Although the widely distributed capillary (exchange) vessels constitute the only site at which transfer of these materials between the tissues and the blood occurs, the ultimate responsibility for transport to each region of the body rests with all divisions of the circulatory system. Unless the heart pumps the blood to the filtering and diffusion membranes of the capillary beds, exchange cannot take place. Yet to pump blood into the aorta does not, *per se*, distribute the blood through the complex system of vascular conduits to the capillary beds in the various tissues according to their ever-changing needs. Also, in a very real sense, the transport of materials between the environment and the tissues is not the concern solely of the circulatory system. All organ systems contribute to this physiologic goal to some extent, the neuroendocrine systems particularly. The physical characteristics of the fluid being transported also influence the delivery to the terminal exchange vessel pathways.

The overall mission of the circulation is ensured by the structural organization and specialized function of each of its major components acting in concert: the heart, the large vascular conduits (arteries and veins), and the capillary exchange vessels. Each has unique features and each is concerned with separate operational activities.

General Principles of Transport

The heart, as a bicameral pump, serves as the pressure generator of the system and is concerned with providing a cardiac output sufficient to maintain the blood in continuous circuit at relatively constant pressure. The large arteries divide blood flow into appropriate fractions by their architectural arrangement and branchings and the provision of variable resistances to flow in their parallel pathways. They also serve as elastic conduits concerned with maintaining the blood pressure. Their compliant–elastic walls aid the forward movement of the blood and convert the pulsatile flow to relatively continuous flow at constant pressure. The veins and venules can collect and store the blood because they have a large physical capacity. Their compliant walls also regulate the return of blood to the heart. Since most of the blood volume is in the veins and venules, it requires only small changes in their capacity to significantly affect venous outflow volume. In the capillaries the blood–parenchymal barrier is reduced to a single endothelial layer to mediate exchange. These small-caliber vessels serve, therefore, as the nutritive channels, and are concerned with providing adequate tissue blood flow. Their functional behavior and immense capacity also influence the volume of blood emptying into the collecting venules, hence they contribute to the return of blood to the heart via the low pressure venous system.

Vascular Control Systems

While each major circulatory component incorporates a well-developed intrinsic (auto-) regulatory capacity,* the cardiovascular system as a whole requires a highly integrated system of control mechanisms. This regulatory

Received from the Department of Anesthesiology, Albert Einstein College of Medicine, New York, New York. Author's original research referred to in this report was supported in part by USPHS grant HL-11391-07.

* Autoregulation of local tissue blood flow and local tissue homeostasis are not identical physiologically. Not all of the blood flow to a tissue can exchange solutes with tissue fluid—the essence of homeostasis. Autoregulation refers to the maintenance of constant total blood flow locally, even though perfusion pressure may vary. Constant total flow does not ensure constant nutritional supply.

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system operates through a battery of closely coordinated, functionally interconnected sensing devices, chemical transmitters, and neural pathways which activate a series of positive and negative feedback flow-control circuits. The master controls are housed primarily in the vasomotor center of the brain. Interestingly, the supervisory function of this elegant regulatory system complex is largely limited to the systemic circulation, the operation of which primarily guarantees the hydrodynamic efficiency of the circulation as a whole. It exercises little direct control over the functional behavior of the terminal-exchange–vascular bed, which operates principally to ensure the adequacy of local tissue blood flow. The activity of this terminal vascular component is essentially self-regulated by locally generated vasoactive mediators, most of which are products of local metabolism. The role of many of these local mediators in autoregulation is to modulate the tonic action of blood-borne vasotropic humoral materials. Nevertheless, events in the systemic circulation influence the function of the exchange vessels through the ability to limit the amount of blood it delivers to them. And the terminal bed affects the functioning of the systemic vasculature by release in the tissues of chemicals which spill over into the systemic circulation to reach the regulatory systems of the brain and specialized sensing devices in the large vessels. In abnormal situations, particularly low-flow states, the autonomous, purposive behavior of the terminal bed may be diametrically opposite to that of the centrally controlled systemic circulation. A competition develops for the limited available cardiac output, initially resolved in favor of the systemic circulation in its attempt to safeguard the integrity of the whole organism. Ultimately, local regulatory factors, acting to capture the scarce supply of blood for the needs of the local tissues, become dominant. Much of the limited blood supply is sequestered in the microcirculation, an eventuality which results in circulatory collapse. In this sense it can be said that the tissue nutritive (microcirculatory) system provides the ultimate regulatory control of the cardiovascular system, a thesis which is phylogenetically well-based.

Peripheral Circulation and Regional Vascular Beds

Although the delineation is somewhat arbitrary, regional blood flow is usually considered to be the province of the peripheral circulation. The latter, by definition, includes all of the blood vessels outside of the heart, comprising both the systemic circulation and the microcirculation—that portion of the terminal vascular bed distal to the major resistance arterioles. Except for the very large arteries and veins (major distributing vessels) the peripheral circulation, as an entity, is made up from the collective regional vascular beds contained within the various tissues and organs. Each of these beds, although separate, is comparably constituted both in basic design and by vessel types. Regional beds are characterized by a set of parallel inflow- and outflow-coupled circuits, sections of which incorporate variable numbers of series-coupled circuits of different layouts and functions according to the nature of the tissues they serve.

Vascular Differentiation

The architecture of the vessels in each peripheral bed provides for three general patterns of regional blood flow which subserve three basic homeostatic requirements: 1) Flow which provides the direct or metabolic needs of the local tissue cells *per se*. 2) Flow which serves the needs of the specialized activity of the tissue (secretion, contraction, synthesis, etc.)—functions essentially synonymous with serving the needs of the organism as a whole. 3) Flow which traffics blood through thoroughfare pathways without any local exchange to move it in continuous circuit and make it available to other tissues downstream. (This is not a true shunting pathway, which implies a direct, non-exchanging, arteriovenous channel.) In keeping with the prudent economy of the body, each of these flow patterns, depending on the particular tissue, may subserve additional functions: *e.g.*, blood flow to the skin to regulate heat loss is adjusted via the thoroughfare channels to maintain

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body temperature within a narrow range. Also, blood flow through the purely arterial capillaries of the renal glomeruli (although they "exchange" unidirectionally) is nonnutritional. But localization of their filtrate within Bowman's capsule increases the efficiency of the tubular reabsorptive function of the vascular counter-current system.

A more common description of the makeup of the peripheral vascular system classifies the different types of blood vessels more precisely by differentiation of their function, subdividing them as "distribution," "shunt," "resistance," "exchange," and "capacitance" vessels. Folkow and his associates have proposed a widely accepted functional description of the peripheral vascular bed based on the relation of consecutive sections of the cardiovascular circuit to blood pressure levels. This concept is physiologically rational since the major operational response of the peripheral systemic bed is the defense of the blood pressure. There are still other functional classifications of the peripheral circulation. It is of less importance whether one or another classification is more correct, but rather that there is general agreement that a functional basis is more useful than a morphologic basis to characterize the sequential vascular pathways in each regional vascular bed.

Functional Determinants of Transport

Hemodynamics and Hydrodynamics

The dynamic functions of the regional vascular beds obey certain physical laws in the movement of blood through their vessels. This blood flow closely resembles the flow of an ideal (Newtonian) fluid in uniform, rigid tubes under constant pressure; the laws of hydrodynamics. But blood flow is more complex because blood is a heterogeneous fluid and the blood vessels are distensible, branched and non-uniform in dimension. The driving force is not constant in pressure, but is pulsatile. Hemodynamics and hydrodynamics, therefore, are neither interchangeable terms nor identical dynamic activities. Yet the latter provides much of the basis for our understanding of the former when some of the significant deviations are taken into account.

Most dynamic physical entities can be described in terms of their dimensions, which combine measurable units of mass, length and time, so-called "dimensional analysis." The dimensions of flow are volume per unit of time, *i.e.*, milliliters per second or liters per hour. In this physical context, the dimensions of pressure and resistance can also be described in combinations of these measurable units—pressure equals force (mass times acceleration) per unit of area. Resistance equals pressure divided by flow. The volume of blood in any tissue at a given time, although frequently measured, is an unreliable index of flow. Similarly, the velocity with which blood moves through a vessel is not a good estimate of flow. Large volumes may move slowly, small volumes rapidly, and *vice versa*.

The essential content of the subject of hydrodynamics concerns the relationship between the force driving fluid through a system of tubes, the resulting flow, and the effects on flow due to the nature of the fluid and the tube. This relationship is analogous to *Ohm's law*—the flow (of current) is directly proportional to the pressure gradient (voltage drop) and inversely proportional to resistance—which incorporates the above dimensions. The dimension of resistance is a complex entity in that it is obviously influenced by the geometry and physical characteristics of the tube and by the nature of the fluid. The longer or narrower the tube, the greater the resistance; the greater the viscosity of the fluid, the greater the resistance. This more complex relationship, which includes the dimensions of resistance and viscosity, is incorporated in *Poiseuille's law*—flow is directly proportional to the pressure drop, to the fourth power of the radius, and inversely proportional to the tube length and the viscosity of the fluid. Poiseuille's relatively simple equation holds rigidly only for laminar, nonpulsatile flow of perfect (Newtonian) fluids in rigid tubes under constant pressure conditions. But only minor errors are involved when this law is applied to blood, an imperfect mixture, flowing through many parts

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of the circulation. Its use in most clinical applications is acceptable. Poiseuille's equation has been extended to include the influence of turbulent flow by accounting for velocity, tube diameter, fluid density and viscosity to derive an index termed "Reynolds number." When this number exceeds a critical value, flow changes from laminar to turbulent. The Reynolds number for blood in circulation is probably lower than for ideal fluids. Except in certain pathologic conditions such as stenosis, clot formation, or aneurysm, turbulent flow is not a normal feature of peripheral vessels. Flow is laminar in all the smaller vessels comprising the resistance beds. Since vessel radius is the most important factor in determining resistance, the Poiseuille equation applies equally to both hemodynamics and hydrodynamics.

Pressure

Except for the true endothelial capillaries, all blood vessels are elastic; they can be deformed by the forces acting on their walls to vary their dimensions. *Elasticity* defines the physical property of reversible deformation. *Distensibility* refers to the degree of deformation from an applied stress and is the preferable term to describe the effect of pressure on the blood vessel wall. For the vessel *in vivo*, the deforming force is the *transmural pressure*., the difference between the pressures in the vessel and surrounding tissue; the resulting increase in volume capacity is what is measured. The greater the amount of elastic tissue in the wall, the less distensible the vessel. Distensibility is an important functional property of vascular beds; it adjusts the reservoir function of the veins and determines static filling pressures and the range of pulse pressure.

Pressure exerted perpendicular to the wall of a hollow circular tube is directly related to the stretch placed on a circular section of the wall. This relationship is expressed by LaPlace's law—tension equals pressure times radius; the greater the pressure, the greater the tension. Extrapolated to blood vessels to take wall thickness into account, this law correctly assumes that wall thickness decreases in proportion to decrease in vessel radius; thus the ability of the small, thin-walled vessel to withstand pressure is not decreased. Stated otherwise, at a given transmural pressure, wall tension increases as the vessel radius increases. In small blood vessels, with a decrease in local pressure or an increase in constrictor activity, the radius becomes smaller, leading to reduced tension, with further decrease in radius resulting ultimately in complete closure of the vessel. The minimum pressure needed to prevent this spontaneous closure is the *critical closing pressure*. Whatever the functional role of this factor may be, its physiologic significance has not been determined.

The rate at which the *pressure wave* is transmitted through the system of blood-filled vessels is determined by many factors including their distensibility, the viscosity of the fluid (blood), and the density of their walls. With laminar flow, pressure imparted to the fluid creates perpendicular to the surface a velocity gradient, the *rate of shear*, essentially a viscous force resembling friction which retards the motion of the fluid, more marked in the peripheral than in the axial stream. A homogeneous (Newtonian) fluid like water has an *absolute viscosity* independent of the rate of shear. Non-Newtonian fluids (emulsions, suspensions, etc.) show *anomalous viscosity*—increased viscosity with decrease in flow velocity. Although blood, a suspension, is a heterogeneous fluid, it behaves, rheologically, very like a homogeneous fluid, except that it does show anomalous viscosity, especially at very low shear rates. This is apparently related to the confluence of erythrocytes in low-velocity flow, but it is probably not of serious significance in active, *in-vivo*, circumstances. The relative viscosity of blood, although it increases markedly at low shear rates when measured *in vitro*, increases only moderately, even at low flow rates, when measured *in vivo*, provided the composition of the blood remains normal.† The term "effective" viscosity is

† In a sense, the contribution of blood viscosity to flow resistance in the narrow, rigid-walled, endothelial capillaries is minimized by a functional modification of the blood which enters them. Capillaries uniformly arise at right angles

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often used to characterize the viscous properties of blood in the active, *in-vivo*, circulation, to differentiate it from viscosity determined *in vitro*. In the latter situation whole blood has a relative viscosity four to five times that of saline solution when measured at high shear rates. *In vivo*, whole blood has a viscosity close to that of saline solution and only twice that of plasma, even at moderate or relatively low shear rates. The significance of blood viscosity as a resistance to blood flow is, therefore, minimal when blood flow is brisk.

The *density* of the vessel wall, in contributing to the rate of propagation of the pressure wave, relates principally to the structural makeup of successive sections of the arterial system. Peripherally, from the heart, the proportion of elastic fibers in the walls decreases and the relative amount of vascular smooth muscle increases. The large vessels are distensible primarily because of the elastic fibers in their walls; hence, their function is determined almost entirely by physical laws. Small arteries and arterioles, with heavy sheaths of vascular muscle and very little elastic tissue, are distensible largely by virtue of the tone of the muscular elements in their walls. The large arteries, therefore, participate in pressure propagation, the smaller arteries and arterioles are more important in determining vascular resistance and the blood pressure through regulatory adjustment of their vascular tone.

The driving force which keeps the blood in continuous circuit is the *pressure gradient* across the consecutive sections of the vascular channels. Pressures in both arterial and venous vessels are normally subject to considerable fluctuations, but these are held to within rather narrow limits. Arterial pressure, in simplistic terms, is determined by the ratio between the rate at which the heart delivers blood to the arterial system and the rate of blood run-off from the arteries. Run-off is conditioned by the resistance between the heart and the artery in which the pressure is measured. Venous pressure depends largely on the vertical distance between the vein in which the pressure is measured and the "level" of the heart. Venous pressure is also conditioned by other factors, such as the pumping action of the valves in the veins of the extremities and the negative intrathoracic pressure.

There are several elements which determine the inflow and outflow pressures (pressure gradient) within a given tissue or organ. Increase or decrease in cardiac output in response to neurohumoral influences or drugs will tend to modify arterial pressure, to increase or decrease it, but only transiently. Major fluctuations in mean arterial pressure are rapidly attenuated by cardiovascular readjustments initiated via the exquisitely sensitive and balanced baroreceptor mechanisms. The hemodynamic resistance is negligible between the aorta and the large tissue-inflow arteries; hence little drop in pressure occurs. It is beyond this point that local resistance to flow may be markedly increased, accompanied by a significant decrease in pressure in the small arteries and arterioles, the extent of the pressure drop depending on the *vascular tone* in these vessels. Run-off into the capillary bed (or beyond the point at which the arterial pressure is measured) will also affect intra-arterial pressure upstream. But run-off from a single vessel (or from the arteries in one series-coupled circuit) does not describe the run-off effect on blood pressure in that vessel. Pressure in a peripheral artery lying within a tissue or organ is determined by the total of all arterial outflows from all parallel alternative pathways in that tissue. It is important to point out that although changes in pressure gradient modify tissue perfusion, the effect of pressure change, *per se*, on flow is actually unselective through all tributaries,

from their parent arterioles. (Their site of origin is marked by a single, circumferential band of one or two vascular smooth muscle cells, identifying this junctional portion of the capillary as the "precapillary sphincter.") This structural feature leads to a degree of plasma skimming of the blood entering the capillaries at the precapillary orifices. (Arteriolar flow is laminar with axial erythrocyte streaming.) Turbulence created at this site causes the introduction of additional erythrocytes from the axial stream into the capillary inflow, but in lower proportion than in the blood of the parent vessels. Capillary blood, therefore, has a lower hematocrit than its parent arteriolar vessels, hence a lower relative and effective viscosity.

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hence unrelated to the state of tissue activity or needs. Pressure gradient is, therefore, not the major determinant of tissue blood flow, but other factors such as vascular tone and resistance exercise major influence.

Vascular Tone

Vascular Smooth Muscle. The enormous capacity of the circulatory system is clearly such that the heart does not have the capacity to deliver an output equal to the combined regional blood flows at constant pressure during circumstances in which all blood vessels are maximally dilated. A degree of what is generically referred to as vascular tone must be maintained above a given level in the resistance vessels to ensure efficient vascular function. Vascular tone is a reflection of smooth muscle activity representing, in fact, a basal state of partial contraction of the vascular musculature in the regional beds. *Basal vascular tone* is an inherent, locally regulated myogenic activity in the principal resistance vessels (arterioles and precapillary sphincters) and is evident even in the absence of extrinsic neural or blood-borne influences. This basal level of tone, on-line, controls (autoregulates) local blood flow and pressure by the device of counterbalancing the constrictor effects of intrinsic mechanical, myogenic factors with the dilator, chemical effects of local metabolites. This local mechanism is important in maintaining capillary blood flow and pressure at normal levels in the face of wide swings of systemic pressure.

In addition to the local factors which determine basal vascular tone, there are other factors, primarily neurogenic, which may increase or decrease basal tone. These remote, extrinsic control mechanisms operate in stress states to distribute the circulating blood volume so as to adapt the overall circulation to meet the needs of the body as a whole. Various humoral materials, principally the catecholamines, also participate significantly in this extrinsic control of vascular caliber. Many drugs, of course, profoundly influence vascular smooth muscle.

In the overall regulation of the tone and caliber of peripheral vessels, the vascular smooth muscle cell provides the ultimate effector mechanism. Although vascular smooth muscle cells from blood vessels in various tissues are morphologically apparently identical, they do not respond identically to the same stimuli. These differences in responsiveness are usually quantitative, but are also sometimes qualitative, as demonstrated by the diametrically opposite effects of epinephrine, which dilates the arterioles in skeletal muscle and constricts those in the skin. There are other situations of functional differentiation of vascular smooth muscle in the arteries and veins throughout the body, as determined by factors such as automaticity, electrical activity, and even the contractile process itself. But all the characteristics of "single-unit" visceral smooth muscle cells (such as occur in the ureter and intestine) are found in the strategic resistance arterioles and precapillary sphincters in all major vascular beds. These small vessels demonstrate a relatively high level of intrinsic basal tone and a relatively high sensitivity to neurohumoral vasoactive stimuli. However, generalizations about vascular smooth muscle behavior in specific tissues can be misleading. Each tissue and each vessel type must be individually considered. The activity of vascular smooth muscle exercises the most important influence on regional blood flow, since changes in the radius of these resistance vessels are normally the main determinant of peripheral resistance, hence tissue blood supply.

Resistance. Resistance in each regional vascular bed constitutes the hindrance to the passage of blood in the complex network of channels between its inflow arterial supply and the veins which drain it. It is composed of all of the parallel and alternative connecting arteriovenous pathways, each of which is made up of a small artery, arteriole, terminal arteriole, capillary unit, venules, and small veins; vessels whose caliber can be varied by activity of vascular smooth muscle to affect total resistance to flow. Most of the total resistance to flow is contributed by the small arteries and larger arterioles. The precapillary sphincters within the capillary unit, although part of the resistance bed, function entirely to determine the number of capillaries perfused at any one time. Their activity does

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not impede the entry of blood from the capillary bed into the collecting venules. Capillaries, of themselves, as rigid tubes of uniform radius, exert no active, variable influence on resistance, blood flow rate or exchange. The small veins and venules contribute little to total peripheral resistance and blood flow. Whatever resistance is present determines the pressure in the postcapillary venules and contributes principally to capillary hydrostatic pressure (fluid exchange) and blood volume.

Peripheral resistance is controlled primarily through two mechanisms, by the central nervous system and by autoregulation. Autoregulation, as previously indicated, is a local mechanism which adjusts vascular caliber to meet local blood flow requirements. Our understanding of the specific machinery and materials of local control mechanisms is incomplete, except that the autoregulatory features of various segments of the circulation are different. The principal remote mechanism, the central nervous system, modifies peripheral resistance by inducing vasoconstriction or vasodilation in response to a decrease or an increase in arterial blood pressure. In this mediation the baroreceptors constitute the main input for arterial pressure regulation by means of changes of peripheral resistance. Various blood-borne vasoactive humoral systems also contribute to the remote regulation of peripheral resistance, but are not considered here. Unlike local autoregulation, remote control systems subserve general circulatory homeostasis of the entire organism so as to maintain normal blood pressure and blood volume. Remote systems also mediate a variety of specialized systemic circulatory adjustments such as stress reactions, thermoregulation, responses to vigorous exercise, etc. Within the central nervous system, vascular resistance is modulated by the sympathetic adrenergic vasoconstrictor fibers. These nerves innervate blood vessels throughout the body (except precapillary sphincters and capillaries), but regional differences exist, as in the density of nerve supply and the depth of penetration of the mural vascular layers. Along the course of these nerves, complex synaptic junctions occur in autonomic ganglia with multiple cholinergic‡ as well as adrenergic mediation and interaction. In the vessels the nerves terminate in so-called "receptors" at which neural transmission is translated to smooth muscle response. The types and density of receptors also differ regionally; hence, receptor populations may be considered part of the remote, resistance control system. Activation of alpha receptors induces vasoconstriction; activation of beta receptors induces vasodilation. The concept of the alpha and beta receptor mechanisms characterizes the responses of smooth muscle to the various sympathetic adrenergic transmitters, as well as to the many adrenergic or sympathomimetic drugs.

Summary and Conclusions

The cardiovascular system is charged with the responsibility of meeting the wide-ranging circulatory requirements of the different tissues, yet operating within the hemodynamic–hydrodynamic limits of the heart-pump and blood volume. The specialized structure and function of each major cardiovascular component make it possible for the cardiovascular system as a whole to satisfy this responsibility. Coordinated control systems which integrate intravascular pressure, resistance, and blood flow in the various peripheral beds distribute the cardiac output so as to ensure regional blood supplies appropriate to the metabolic needs and operational priorities of the different tissues and organs. These control systems may be intrinsic to the local tissue or may exercise their regulation by remote neurohumoral mechanisms. Acting in concert, the control systems are exquisitely counterbalanced to adjust blood flow from moment to moment in both normal and emergency states to meet the hemodynamic needs of the organism as a whole or of any given tissue, whichever is more critical for survival.

In general, the operational activities of the

‡ A sympathetic cholinergic, true vasodilator system with fibers distributed probably only to vessels of skeletal muscle has been demonstrated, but the mechanism whereby the cholinergic transmitter inhibits vascular muscle tone is not established.

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circulatory system largely obey the physical laws of hydrodynamics, modified to take into account the significant deviations which hemodynamically distinguish it from physical hydraulic systems. Blood supply to regional beds, thereby, is accomplished via adjustments of the various physical determinants of blood flow from within a given tissue and from without, by remote controls, primarily neurohumoral. These adjustments are largely effected by the induction of excitatory and inhibitory changes in vascular tone, which ration the cardiac output according to needs. Within this framework of the general organization of blood transport and distribution, physical factors which relate, hemodynamically, to pressure as a driving force and to resistance as an impedence to flow, are considered in the foregoing discussion in some detail, since these are the basic determinants of regional blood flow. The intent is to provide some background to the more thorough consideration these factors are given in the articles in this Symposium which follow.

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