

TEACHING THE PRINCIPLES OF HEMODYNAMICS

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Knowledge of hemodynamic principles is crucial to an understanding of cardiovascular physiology. This topic can be effectively taught by discussing simple physical principles and basic algebraic equations. A variety of examples from everyday observations can be used to illustrate the physical principles underlying the flow of blood through the circulation, thereby giving the student an experiential feel for the topic in addition to an understanding of theory. Moreover, opportunities abound for showing how each hemodynamic principle can explain one or another functional feature of the cardiovascular system or a cardiovascular pathophysiological state. Thus hemodynamics can be used as an organizational thread to tie together other aspects of cardiovascular physiology.

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Hemodynamics is concerned with the physical and physiological principles governing the movement of blood through the circulatory system. There can be no true understanding of the circulation without an appreciation and understanding of some basic hemodynamic principles. It is an essential part of the infrastructure of the working mental model of the circulation that we want our students to acquire during their introduction to cardiovascular physiology.

At my institution, we offer introductory physiology courses to a variety of students: first-year medical students; first- and second-year graduate students (both MS and PhD students) in physiology, pharmacology, and other basic medical sciences; and first-year students in our physical therapy MS program. Thus all our introductory courses focus on mammalian physiology and, in particular, human physiology. These courses are traditional, discipline-based, lecture format courses. In our medical physiology class, the cardiovascular block is the largest single block, occupying nearly one month of the course. This allows us

the luxury of incorporating problem sessions, case conferences, wet labs, and clinical correlation lectures into the curriculum. These afford the students many different opportunities to apply the basic knowledge garnered from lectures and reading to “real-life” situations. These activities greatly aid the development of the student’s understanding of the subject.

This article is written from the educational perspective outlined in the previous paragraph. That is, it is oriented toward human physiology, and is presented in the context of a traditional lecture-format class. If your classes are conducted in problem-based or some other format, you will need to adopt whatever you might learn from this article into your particular format. In fact, although my hemodynamics lectures contain most of the information outlined in this article, most (or all) of these concepts, examples, and lines of logic are also reiterated and emphasized in the various case conferences, wet labs, and individual student sessions that accompany our courses. Whatever the format, these are the principles that must be learned, and I have noted some of the most common

difficulties that students encounter as they try to learn them.

BASIC VARIABLES AND THEIR UNITS

The basic variables that are used in analyzing the circulation of blood are volume, flow, pressure, resistance (or conductance), velocity, and compliance. However students are exposed to hemodynamics—whether by lecture or by directed reading and self-study—they must come to understand these basic variables and how they interact. Be sure to identify the units of each variable, as it is introduced, and insist that the students use appropriate units in their answers and questions. Precision of expression will help the students develop clarity of understanding.

As we introduce these variables and their relationships, we can begin to integrate the course content that is to follow. For example, most students will know that the “normal” blood volume (for man, the species) is 5 liters and that a “normal” cardiac output (CO) is 5 l/min.¹ Although these are two different variables, despite sharing the value “5,” their “typical” baseline values do indicate that the cardiovascular system circulates the equivalent of our entire blood volume each minute. As the students will come to appreciate over the course of the next several lectures, this economy of blood utilization has important consequences for cardiovascular control and homeostasis.

BULK FLOW LAW/POISEUILLE’S LAW: PRESSURE AND RESISTANCE

The basic law of hemodynamics is the Bulk Flow Law

$$Q = \frac{\Delta P}{R} \quad (1)$$

where ΔP is the intravascular pressure gradient (measured in mmHg) between the upstream (larger pressure) and downstream (smaller pressure) ends of the blood vessel. Blood will flow from upstream to downstream at a rate Q (measured in ml/min or l/min) that

¹ For a variety of personal reasons, I no longer consider the 70-kg, 21-year-old male, on which these “normal” figures are based, to be emblematic of the human ideal. I tell the students this and note that blood volume and CO are very dependent on body size.

TABLE 1
Definitions and units for abbreviations used in this article

Abbreviation	Variable	Most common units
A	Cross-sectional area of blood vessel(s)	cm ²
CO	Cardiac output	l/min
d	Vessel diameter (inner diameter)	cm
G	Gravitational potential energy	mmHg dyn/cm ²
g	Gravitational acceleration constant	980 cm/s ²
h	Height of fluid column	cm
K	Kinetic energy	mmHg dyn/cm ²
MAP	Mean arterial pressure	mmHg kPa
N_R	Reynolds number	Dimensionless
P	Pressure	mmHg kPa
Q	Blood flow	ml/min l/min
R	Resistance	mmHg·min·ml ⁻¹ mmHg·min·l ⁻¹ mmHg·s·ml ⁻¹ (PRU)
RAP	Right atrial pressure	mmHg
TFE	Total fluid energy	mmHg dyn/cm ²
TPR	Total peripheral resistance	mmHg·min·ml ⁻¹ mmHg·min·l ⁻¹ mmHg·s·ml ⁻¹ (PRU)
v	Velocity	cm/s
ΔP	Pressure gradient	mmHg
η	Viscosity	poise
ρ	Density	g/cm ³

PRU, peripheral resistance unit.

is proportional to ΔP and inversely proportional to the resistance to flow R (expressed in mmHg·min·ml⁻¹ or mmHg·min·l⁻¹, depending on the units of Q). (See Table 1; also see *problem 1* in APPENDIX.)

Comparing the Bulk Flow Law to Ohm’s Law of electricity (flow vs. current, pressure gradient vs. voltage gradient, hemodynamic resistance vs. electrical resistance) helps to orient students to something they already know. We can immediately demonstrate the utility of the Bulk Flow Law by giving an example

$$CO = \frac{MAP - RAP}{TPR} \approx \frac{MAP}{TPR} \quad (2)$$

where CO is cardiac output, MAP is mean arterial

pressure, RAP is right atrial pressure, and TPR is total peripheral resistance. Because RAP is usually negligibly small in comparison to MAP, Eq. 2 can be approximated by a simple relationship (Eq. 3) among three variables: CO, MAP, and TPR.

$$\text{MAP} \approx \text{CO} \times \text{TPR} \quad (3)$$

Even at this early point, we can emphasize how important this relationship will be to an understanding of cardiovascular control.

A century and a half ago, Poiseuille conducted a series of experiments with tubes of various sizes and fluids of different viscosities.² He found that resistance (R) was a function of viscosity (η), tube length (l), and tube radius (r), as described in Eq. 4.³

$$R = \frac{8\eta l}{N\pi r^4} \quad (4)$$

The additional factor N in Eq. 4 refers to the number of identical tubes (of radius r and length l) that are arranged in parallel (see below). If we substitute Eq. 4 for R in the Bulk Flow Law (Eq. 1), we arrive at Poiseuille's Law

$$Q = \frac{N\pi r^4}{8\eta l} \Delta P \quad (5)$$

Although the equation will initially “look like Greek” to the students, most of its content is intuitively obvious. By asking the students to draw on their everyday experiences with hoses, household plumbing, the flow properties of different common fluids (e.g., water vs. corn syrup), and so on, we can guide them toward understanding or even “predicting” much of this equation.

² Jean Louis Poiseuille (1799–1869). These experiments were conducted between 1842 and 1846. For medical students, I point out that Poiseuille was a physician-scientist. As a medical student, he investigated the use of the mercury manometer as a device for measuring blood pressure.

³ For Eq. 4, r and l are expressed in cm, and N , π , and 8 are dimensionless. η is expressed in poise (to honor Poiseuille), where 1 poise = 1 dyn·s·cm⁻². Therefore, R is expressed as dyn·s·cm⁻⁵, which is equivalent to (dyn·cm⁻²)/(cm³·s⁻¹), or (force/area) divided by flow. Because the value of Eqs. 4 and 5 is mostly to delineate the important variables that determine flow, rather than to perform specific practical calculations, I do not emphasize the unit analysis for these particular equations.

It is important to note that Poiseuille's Law is only an approximation of reality. Perhaps it would be better to call it Poiseuille's equation, rather than a law. As I explain to my students, Poiseuille imposed several empirical constraints on his investigations (1842–1846) into the flow of fluids through tubes. Even the theoretical derivation of the Poiseuille equation from Newton's definition of viscosity required the equivalent mathematical assumptions.⁴ The important empirical and theoretical assumptions were as follows: 1) flow is steady and streamlined; 2) the vessels are rigid, cylindrical, straight tubes that are uniform in shape and are long in comparison to their diameter; and 3) the fluid flowing through the tubes is a Newtonian fluid with constant viscosity.

In fact, most of these assumptions are violated in the cardiovascular system, at least in certain parts of the system. Flow in the arteries is decidedly pulsatile, although it is more steady in the microcirculation. Flow can be turbulent (the alternative to streamlined) in the large arteries and is neither turbulent nor streamlined in the capillaries. Our vessels are elastic and compliant, not rigid. They do tend to be long in relation to their diameter, but they have branches, arches, bends, and other interesting anatomic features. Blood is not a homogeneous fluid but is a suspension of cells in plasma. As a result, it has some very curious viscous properties. Thus the Poiseuille equation fails to predict flow exactly, even when the values of the other variables are known.

What, then, is the value of the Poiseuille equation? Well, first, it enumerates the factors that are important in determining flow. Second, when combined with some knowledge of circulatory physiology, it tells us which factors are the most important in determining flow. If we are interested in acute changes in flow, we should recognize that viscosity and vessel length change very little from moment to moment. Moreover, arterial pressure is tightly regulated. If arterial pressure shows a sustained rise of only 20%, we regard it as a disease—hypertension. Yet cardiac output (a flow) can increase four- or fivefold in ordinary individuals during intense exercise. What is left to change, according to Poiseuille? Vessel radius. Moreover, any

⁴ I refer to the theoretical derivation as provided by Wiedemann (1856) and Hagenbach (1860).

change in vessel radius is magnified by a power factor of 4. Thus a 20% increase in vessel radius would allow flow through that vessel to double.

There is another major benefit of having vessel radius be the key variable in changing blood flow, which can be gleaned from the Poiseuille equation. This arrangement allows the cardiovascular system to vary blood flow selectively to different organs, e.g., raising flow to skeletal muscle during exercise but to the gastrointestinal tract during digestion of a meal. If blood flow were controlled primarily by changes in arterial pressure or blood viscosity, then cardiac output would have to be raised to all organs in equal proportion. This would be a very inefficient strategy indeed.

SERIES AND PARALLEL RESISTANCES

Poiseuille's equation is written for a single vessel or a series of identical vessels arranged in parallel. Complexity is added by the elaborate circuitry of the cardiovascular system, which incorporates both series and parallel resistances of greatly different magnitudes. Students should understand the basic arborizing structure of the systemic circulation and the essential anatomic features (internal diameter and the endothelial, smooth muscle, and elastic components of the vessel wall) of the various vascular segments: aorta, elastic arteries, arterioles, capillaries, venules, veins, and venae cavae. Also, they should understand the contributions of serial and parallel resistors to overall network resistance. The equation for series resistances is

$$R_{\text{total}} = R_1 + R_2 + \dots + R_n \quad (6)$$

and that for parallel resistances is

$$\frac{1}{R_{\text{total}}} = \frac{1}{R_1} + \frac{1}{R_2} + \dots + \frac{1}{R_n} \quad (7)$$

Any initial intimidation felt by students with these equations can be overcome by solving simple network problems (see APPENDIX). Conceptual problems are also helpful. For example, to explain how the number of parallel vessels affects resistance, I ask my students to imagine trying to escape Manhattan island on a Friday afternoon in July. What would happen to their weekend plans if 3 of the island's 20-odd existing bridge and tunnel crossings were taken out of service

because of repaving, protest demonstrations, or an overturned tractor trailer? The concept that more parallel vessels results in less resistance to flow is thus internalized. For a physiological example, consider what happens to CO and MAP in an individual after amputation of a limb. The students will probably conclude that CO will be reduced, and some will reason, using Eq. 3, that the smaller CO will be accompanied by a lower MAP. In actuality, however, MAP is a regulated variable and is maintained relatively unchanged, even in our amputee. Thus TPR must have been increased to maintain MAP in the face of a reduced CO. Equation 7 tells us that a higher TPR would have resulted simply because of the surgeon's removal of one of the parallel resistance elements in the systemic circulation. Maintenance of MAP in this instance, therefore, does not require active vasoconstriction in any of the remaining vessels, thereby avoiding the undesirable reductions in flow to those remaining tissues that would have resulted from such vasoconstriction.

The "N" in Poiseuille's equation (Eq. 5) represents a special case of parallel resistors, namely, one in which all of the parallel vessels have the same individual resistance (R_i). If there are N such identical vessels arranged in parallel, then

$$\frac{1}{R_{\text{total}}} = \sum_{i=1}^N \frac{1}{R_i} = \frac{N}{R_i} \quad (8a)$$

$$R_{\text{total}} = \frac{R_i}{N} \quad (8b)$$

HEMODYNAMICS IN THE MAMMALIAN CARDIOVASCULAR SYSTEM

At this point, students must come to understand what happens to intravascular pressure as blood travels through the consecutive vascular segments (aorta, elastic arteries, arterioles, capillaries, etc.) of the systemic circulation (Fig. 1). Note that Fig. 1 represents a "typical" systemic vascular bed, such as resting skeletal muscle, and not the entire systemic circulation. The profiles of other vascular beds, such as the kidneys or the intestines, would be very different.

The major messages of Fig. 1 are that 1) energy (pressure) is lost as flowing blood overcomes resistance, and 2) the greatest pressure drop occurs as blood flows

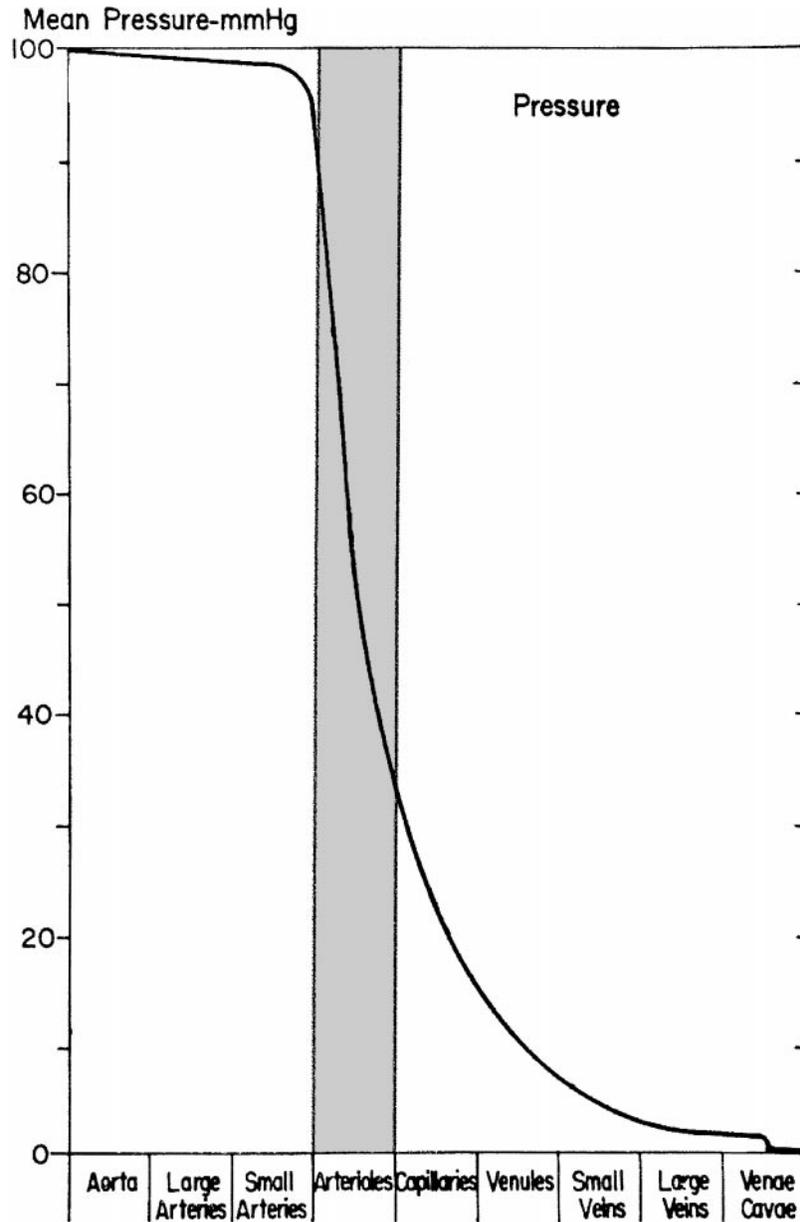


FIG. 1.

Pressure profile in “typical” systemic vascular bed. Note that the greatest fraction of the pressure is dissipated as blood flows through the arterioles (shaded area). [Modified from Berne and Levy (2) with permission.]

through the arterioles. Thus it is the systemic arterioles that provide the greatest resistance to flow.⁵ This is a

point that merits emphatic articulation and reiteration.

⁵ The smallest arteries also contribute significantly to resistance, but this detail can be omitted or deemphasized in an introductory course.

Why do the arterioles present the greatest resistance? After all, the capillaries are much smaller. The Poiseuille equation can be used to resolve this paradox.

Although larger in radius than capillaries, there are far fewer arterioles in parallel (N in Eq. 4 or 5) than there are capillaries. In contrast, although there is only one aorta, and only relatively few elastic arteries, the radii of these vessels are so large in comparison to those of arterioles that they offer a relatively insignificant portion of the overall systemic vascular resistance (TPR in Eq. 2). Most standard textbooks provide data on vessel diameter or radius and the number of vessels found in the circulatory system of a human or some animal subject. We can plug these values into Eq. 4 to illustrate the point.⁶

We should also bring the students to understand that, in addition to having the largest structural resistance, the arterioles also have the greatest capacity to alter their resistance. Because the arterioles possess muscular walls and because this smooth muscle is so responsive to neurotransmitters, hormones, locally released metabolic signals, and physical factors (pressure, shear), the radius of arterioles can become substantially larger or smaller, resulting in significant changes in vascular resistance and blood flow. Capillaries lack smooth muscle, so their radius can change only slightly and passively. Elastic arteries and veins do have smooth muscle, and their resistance can change. Their basal contribution to the TPR is so small, however, that increases or decreases in the resistance of these components have relatively minor effects on TPR, in contrast to the effects of changes in arteriolar resistance. Students tend to have difficulty incorporating this concept into their thinking. Having them work problems with appropriately weighted series resistors can help them understand the mathematics of the idea (see APPENDIX, problems 6–8).

Students need to appreciate that changes in segmental vascular resistance also affect the pressures in the cardiovascular system as well as the blood flow. In Fig. 2, curve B illustrates the effects of arteriolar dilatation, relative to the control state (curve A), on the segmen-

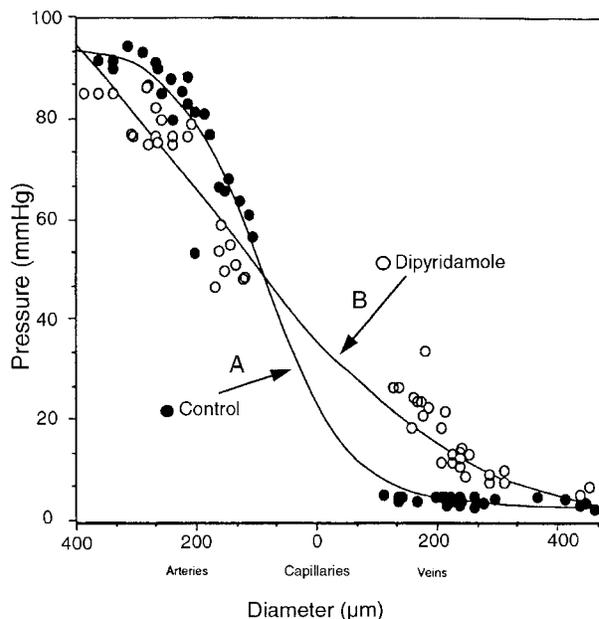


FIG. 2. Pressures measured in microvessels of cat left ventricle during control conditions (curve A, ●) and during infusion of dipyridamole, a vasodilator (curve B, ○). Blood flow to the left ventricle was increased during the vasodilator infusion to ~3.5 times baseline. [Modified from Chilian et al. (4) with permission.]

tal vascular pressure profile. The general principle to be remembered is that vasodilatation (Fig. 2, compare curve B with curve A) reduces pressure upstream from the point of dilatation but increases the pressure downstream. Conversely, vasoconstriction (Fig. 2, compare curve A with curve B) increases upstream pressure but decreases downstream pressure. The consequences of this may be very significant. Intracapillary pressure is an important factor in determining the rate of filtration or reabsorption of fluid across the capillary wall. Pressures in the venules and small veins will affect the volume of blood residing in these compliant vessels. Although these physiological concepts (fluid filtration, venous blood pooling, or mobilization) are usually explored as separate topics, I think it is helpful to foreshadow them by pointing out the hemodynamic principle at this first exposure.

When students are first exposed to Fig. 2 or to the equivalent concepts presented in other ways, they almost always become confused on a particular point. If arterioles constrict, why does arterial pressure not

⁶ Be careful to ensure that the values work out before assigning such a problem for calculation. Most books will list a value for the number of arterioles but neglect to partition them into the various branching orders that characterize the arteriolar network. Thus not all the arterioles included in the typical textbook value for N are arranged in parallel; some are in series. Neglecting this makes the “calculated” arteriolar resistance underestimate the actual case.

become elevated to the point where flow remains constant? After all, *Eq. 3* predicts that for a given CO, an elevated TPR will raise the MAP. To resolve this paradox, the students need to see that there are two possibilities for a closed circulatory system, i.e., one in which a constant volume is being circulated. The system can operate as either a constant flow system, in which CO remains constant, or as a constant pressure system, in which MAP remains constant. Challenge the students to decide which design would be more appropriate. In fact, the mammalian circulatory system functions very nearly as a constant pressure system. Under normal circumstances, MAP remains within $\pm 20\%$ of its normal value, whereas CO can increase to 300–400% of baseline even in those of us who live an unapologetically sedentary life style by choice. Therefore, from the idealized standpoint of an individual regional circulation, as represented in Fig. 2, local arteriolar dilatation or constriction will alter blood flow through that organ, and alter the distribution of pressures within that organ, without affecting the original input pressure, MAP.

Reality does not quite meet this ideal, however. When a sufficient number of different regional vascular beds undergo vasodilatation or vasoconstriction, a fall or an increase, respectively, in arterial pressure may accompany the changes in flow and distribution of pressure indicated in Fig. 2. These changes in arterial pressure are usually minimized, after perhaps a more noticeable transitory change, by the various homeostatic mechanisms that regulate blood pressure. Other factors, such as mobilization of blood from venous pools, may further complicate the arterial pressure response. Thus systemic injection of a vasodilator will lower both TPR and MAP. Dynamic exercise, in contrast, will lower TPR but raise MAP (albeit only modestly). This is the result of various other physiological mechanisms that support a larger increase in CO during exercise than occurs simply because of arteriolar dilatation. I try to get the student to understand the basic hemodynamic relationships and mechanisms at this early stage in the course. As new elements are added to the student's knowledge, she or he can begin to use them to understand increasingly complex situations, such as the apparent contradictory changes in MAP with vasodilator injection and exercise.

DRIVING FORCES

Total Fluid Energy

Up to this point, we have used pressure as the sole driving force for moving blood through the circulatory system. In fact, the energy available for moving blood may be present in one of three forms. The arterial blood pressure, as commonly measured, is a form of potential energy. It results from the pumping action of the heart. Energy may also be present as another form of potential energy (namely, gravitational potential energy) or as kinetic energy. A. C. Burton used the term total fluid energy (TFE) to include all three forms of energy, where P is pressure, G is the gravitational potential energy, and K is the kinetic energy (3).

$$\text{TFE} = P + G + K = P + \rho gh + \frac{1}{2}\rho v^2 \quad (9)$$

Pressure. Pressures in the cardiovascular system are commonly expressed in units of millimeters of mercury (mmHg), because the mercury manometer is the standard pressure measurement device in human medicine.⁷ If you attach a pressure source (e.g., a catheter inserted into an artery, facing into the direction of arterial flow) to one arm of a U tube filled with fluid, the fluid will be pushed upward into the other arm until an equilibrium is reached (Fig. 3). The equilibrium results from the equality of two pressures. The first is the source pressure (arterial pressure in our example) pressing on the fluid surface in the left arm of the U tube in Fig. 3. The second pressure is exerted on a virtual surface that lies in the reference plane, which is defined by the top surface of the fluid in the arm exposed to the external source pressure (see Fig. 3). That second pressure results from the column of fluid lying above this reference plane, and it exactly matches the external source pressure. It can be quantified as the product ρgh , where ρ is the density (in g/cm^3) of the fluid in the U tube, g is the gravitational acceleration constant ($980 \text{ cm}/\text{s}^2$), and h

⁷ Other common pressure units include the tor (plural torr), in honor of Toricelli, the inventor of the mercury barometer (in 1644). With the *Système International de Unités*, the pascal ($1 \text{ Pa} = 1 \text{ N}/\text{m}^2 = 10 \text{ dyn}/\text{cm}^2$) is the preferred pressure unit. Blood pressure is typically expressed in kPa, where $1 \text{ kPa} = 7.5 \text{ mmHg}$. Thus an MAP of 100 mmHg could also be expressed as 13.3 kPa. The kPa unit is not widely used in the USA, which is going metric only at a slow, inch-by-inch pace.

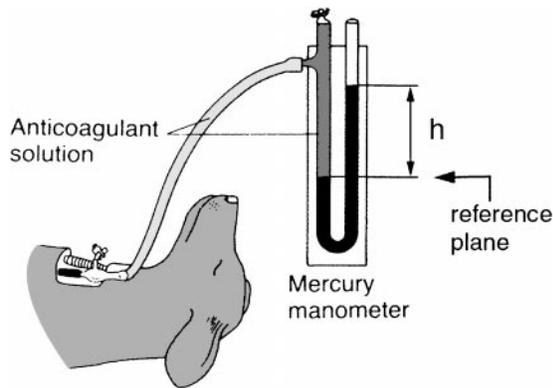


FIG. 3.

A simple mercury manometer. The height (h) of the mercury column in the right arm of the U tube is proportional to the vascular pressure pushing on the mercury in the left arm. The pressure is equal to ρgh , where ρ is the density of the fluid in the U tube and g is the gravitational acceleration constant. [Modified from Guyton and Hall (5) with permission.]

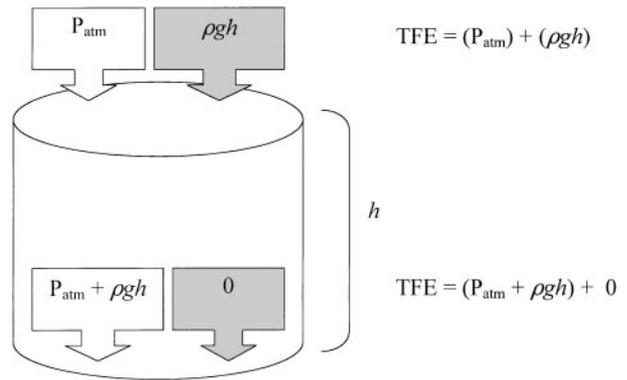


FIG. 4.

Pressures and gravitational forces in a fluid-filled container. Pressures are given in open boxes and gravitational forces in shaded boxes. Although there is a pressure gradient between the bottom and top of the container, there is no gradient in total fluid energy (TFE). P_{atm} , atmospheric pressure.

is the height (in cm) of the fluid in the right arm above the reference plane.

With the use of the U tube model and the formula $P = \rho gh$, it easily can be shown that $1 \text{ mmHg} = 1,333 \text{ dyn/cm}^2$.⁸ I work through this calculation in class to make three points. One, it shows students that “mmHg” is a legitimate unit with which to measure pressure, which is a force (dynes) per unit area (cm^2). Two, it gives the students a handy conversion factor ($1 \text{ mmHg} = 1,333 \text{ dyn/cm}^2$), which I tell them to write in their notebooks or memorize. Three, it shows students that the hydrostatic force of a column of fluid is equivalent to a pressure, like blood pressure.⁹ This last point becomes critical for achieving a true understanding of the driving forces in the circulation.

⁸ The density of mercury is 13.6 g/cm^3 . One dyne = $1 \text{ g} \cdot \text{cm} \cdot \text{s}^{-2}$. Thus, for a column of mercury 1 mm high, $\rho gh = (13.6 \text{ g/cm}^3) \times (980 \text{ cm/s}^2) \times (0.1 \text{ cm}) = 1,333 \text{ g} \cdot \text{cm} \cdot \text{s}^{-2}/\text{cm}^2 = 1,333 \text{ dyn/cm}^2$.

⁹ In 1733, the Rev. Stephen Hales became the first person to measure blood pressure in a living animal using an approach even simpler than the mercury manometer. He inserted a long glass tube (with a bend near the proximal end to allow the tube to rise up perpendicular to the ground) into the carotid artery of a horse. The horse's blood rose to a height of 8 ft, 3 in. At this point the hydrostatic pressure of the blood column itself was equal to the horse's arterial pressure, so the blood stopped rising. What was the arterial pressure in mmHg? Why was it so high?

Gravitational potential energy. The Bulk Flow Law (Eq. 1) says that flow will occur from a higher pressure to a lower pressure. The pressure at the bottom of the ocean is clearly greater than it is at the surface, where only atmospheric pressure (P_{atm}) applies (Fig. 4). Pressure is greater at the ocean floor because of the hydrostatic pressure exerted by the “column” of water pressing on the ocean floor. This extra pressure is equal exactly to ρgh , where h is the height (depth) of the ocean, making the floor pressure equal to $(P_{atm} + \rho gh)$. Why, then, does water not flow upward from the ocean floor? The answer, of course, is that the water at the ocean's surface has a greater potential to do work because of its gravitational position relative to the earth's center than does water at the bottom of the ocean. This potential energy (G) is equal to the very same ρgh . If we use the ocean floor as the gravitational reference point, then TFE at the ocean surface is equal to

$$(P_{atm}) + (\rho gh)$$

and TFE at the ocean floor is equal to

$$(P_{atm} + \rho gh) + 0$$

Therefore, although there is a pressure gradient, there is no gradient in TFE.

The reference level chosen for gravitational potential energy is arbitrary, but the same reference must be used throughout the calculation. In the example shown in Fig. 4, the ocean floor is the reference point at which G is set equal to zero. If the ocean surface had been the reference, G at that point would have been zero, whereas G at the floor would have been $-\rho gh$. For physiological measurements, the tricuspid valve is the normal reference point. In addition to convenient access, there is a reason why the brachial artery is the usual site for measurement of arterial blood pressure by sphygmomanometry: the pressure cuff on the upper arm is located at heart level.

The physiological effect of this interconversion of pressure and gravitational potential energy can be visualized by looking at the veins on the inner surface of the wrist. When the wrist is held at heart level (at the reference level of the tricuspid valve), the pressure within the veins is $\sim 15\text{--}20$ mmHg, the normal peripheral venous pressure. If the wrist is raised above the heart, however, pressure will fall while gravitational potential energy is raised by increments of ρgh . As the students can easily calculate, $15\text{--}20$ mmHg is equivalent to $\sim 20\text{--}27$ cmH₂O, or 8–11 in. of H₂O. Thus, as the wrist is raised $\sim 8\text{--}11$ in. above the heart, the pressure inside the vein will fall to zero and the vein will collapse.

The physiological consequences of a vein collapsing in this way are not great. Because veins have such thin walls, the cross section of a collapsed vein is not occlusive but assumes the “dog bone” shape that we can see when we try to collapse a rubber band. Therefore, flow continues through the flattened, but not occluded, vein. What happens in the reverse conversion, however, is physiologically significant. When gravitational potential energy is converted to pressure in vessels below the heart, the vessels will tend to expand as the intravascular pressure pushes out on the stretchable walls. This expansion is minor in the case of arteries but can be very significant in the case of the more highly compliant veins. In fact, a significant volume of blood can accumulate in the veins in the lower portions of the body, particularly in the long veins in the legs, over the several seconds following an abrupt change from a horizontal to a vertical position. As this blood accumulates, venous return to the heart (right atrium) is correspondingly

reduced, as is ventricular stroke volume, cardiac output, and arterial blood pressure. The orthostatic hypotension that ensues can be enough to cause syncope (fainting) as a result of reduced blood flow to the brain. This is a good example because virtually everyone has experienced lightheadedness upon standing suddenly at least once in their life.

Why do we not faint every time we stand up? Well, the presence of valves in the long veins is a major reason. By interrupting the column of blood rising from the lower leg to the right atrium, the full hydrostatic force (ρgh) of the blood is attenuated. If the vein segments below the valves become too engorged with blood, the valve leaflets may no longer overlap as required for their effective function. The pumping action of the leg muscles, which helps push blood back toward the heart, prevents this from happening. Moreover, a variety of reflex control mechanisms, such as the arterial baroreflexes, act to counteract any reduction in blood pressure that does occur. Orthostatic hypotension is more likely to occur when these defense mechanisms are impaired, as in certain autonomic neuropathies. In otherwise healthy people, orthostatic hypotension is more likely when blood volume has been reduced, e.g., by hemorrhage caused by trauma or donating blood, or following diuresis triggered by prolonged (days) bed rest or weightlessness. With less blood volume available to buffer the effects of venous pooling, cardiac output is more easily impaired by standing.

Students often have the misimpression that it is harder to pump blood uphill than it is to pump blood downhill. In a closed system like the cardiovascular system, this is not correct. The interconversion of pressure and G render the concepts of “uphill” and “downhill” meaningless in a closed system. It is, rather, the compliance of our blood vessels that creates circulatory challenges as a result of postural changes, because the vessels change their shape (collapse, expand) in response to pressure but are insensitive to G per se.

Kinetic energy. The third term contributing to TFE (Eq. 9) is kinetic energy, the energy present in the blood’s movement. It appears in Eq. 9 as $\frac{1}{2}\rho v^2$ because the usual kinetic energy definition ($\frac{1}{2}mv^2$) has been normalized to (divided by) a unit volume. If we put in

centimeter-gram-second (cgs) units for all the variables (ρ = density in g/cm^3 , v = velocity of blood in cm/s), we will see that each of the three energy terms is expressed in dyn/cm^2 .

Blood flowing in a tube will travel at a certain velocity (v), given by the relation between flow (Q , measured in ml/s or cm^3/s) and the cross-sectional area of the blood vessel (A , expressed in cm^2)

$$v = \frac{Q}{A} = \frac{\text{cm}^3/\text{s}}{\text{cm}^2} = \frac{\text{cm}}{\text{s}} \quad (9)$$

A simple physical model of the circulation (1), which I use as a demonstration in my lectures, offers an excellent opportunity to demonstrate how velocity is dependent (inversely) on tube diameter. A squeeze bulb with one-way inlet and outlet valves represents the heart, whereas tubing segments of different diameters and compliance model the veins on the inlet side of the “heart” and the elastic arteries and resistance vessels on the outlet side. Fluid (water) squirted through a narrow tube will leave the tube with a greater exit velocity and, consequently, a greater trajectory distance than fluid pushed through a larger diameter tube.

The Bernoulli Principle is often evoked to demonstrate how kinetic energy and pressure are interchangeable. We can physically demonstrate the principle in lecture by blowing gently downward between two parallel sheets of paper held about 1 in. apart. Contrary to most students’ predictions, the sheets will move closer together when the air velocity between them is increased. A conversion of static pressure to kinetic energy has occurred.

Kinetic energy in most parts of the mammalian cardiovascular system is normally rather small. Because the aorta has the smallest aggregate cross-sectional area of all the systemic vascular segments, the blood velocity and, hence, kinetic energy will be greatest there (Fig. 5). For a CO of 5 l/min through a human aorta with a diameter of 2.5 cm, the mean blood velocity will be 17 cm/s . Kinetic energy will be $\sim 144 \text{ dyn}/\text{cm}^2$ or 0.1 mmHg. The CO is ejected in spurts, and peak velocity might easily reach a value threefold greater than the average velocity. Still, this would represent a

kinetic energy of only $\sim 1,300 \text{ dyn}/\text{cm}^2$ or 1 mmHg. These values are small in comparison to the total fluid energy at systole of $\sim 120 \text{ mmHg}$. When cardiac output is elevated 4- or 5-fold, however, peak aortic flow velocity will be 4 or 5 times greater than at its baseline and kinetic energy will be 16 or 25 times greater. Therefore, kinetic energy can at times represent a significant fraction of the total fluid energy.

An interesting clinical example is afforded by aortic stenosis. Here, the narrowed aortic valve results in an ejection velocity that can be substantially greater than normal. In the root of the aorta, then, the kinetic energy will be increased at the expense of a reduced systolic blood pressure. This is a classic Bernoulli effect. However, because the ostia of the main coronary arteries are located in the root of the aorta, right behind the cusps of the aortic valve, there is a potentially significant physiological consequence. Coronary blood flow may be adversely affected by the reduced arterial pressure available for perfusing the coronary arteries, at least during systole.

Arterial Pressure

When physiologists refer to arterial pressure, they may focus on any one of several aspects: mean arterial pressure, systolic and/or diastolic pressure, or the arterial pulse pressure. Students do not come to their initial study of mammalian physiology with a clear understanding of these various terms or with their implications.

Systolic and diastolic arterial pressure can best be understood in the context of the cardiac cycle. In the classic “Wiggers diagram” used to illustrate the events of the cardiac cycle, aortic pressure sits atop the left ventricular pressure curve, and students can see that ventricular ejection of a stroke volume is the cause of aortic pressure rising from its diastolic low to its systolic high value. It is more difficult, I think, for students to see why arterial pressure falls so slowly after the aortic valve closes, in contrast to the rapid fall in left ventricular pressure. Two vascular elements are needed to explain this: elastic arteries and downstream resistance vessels. The absence of either element would cause the arterial pressure cycle to closely resemble the left ventricular pressure cycle. With the use of a physical model of the circulation, in which a compliant latex rubber segment (finger of a

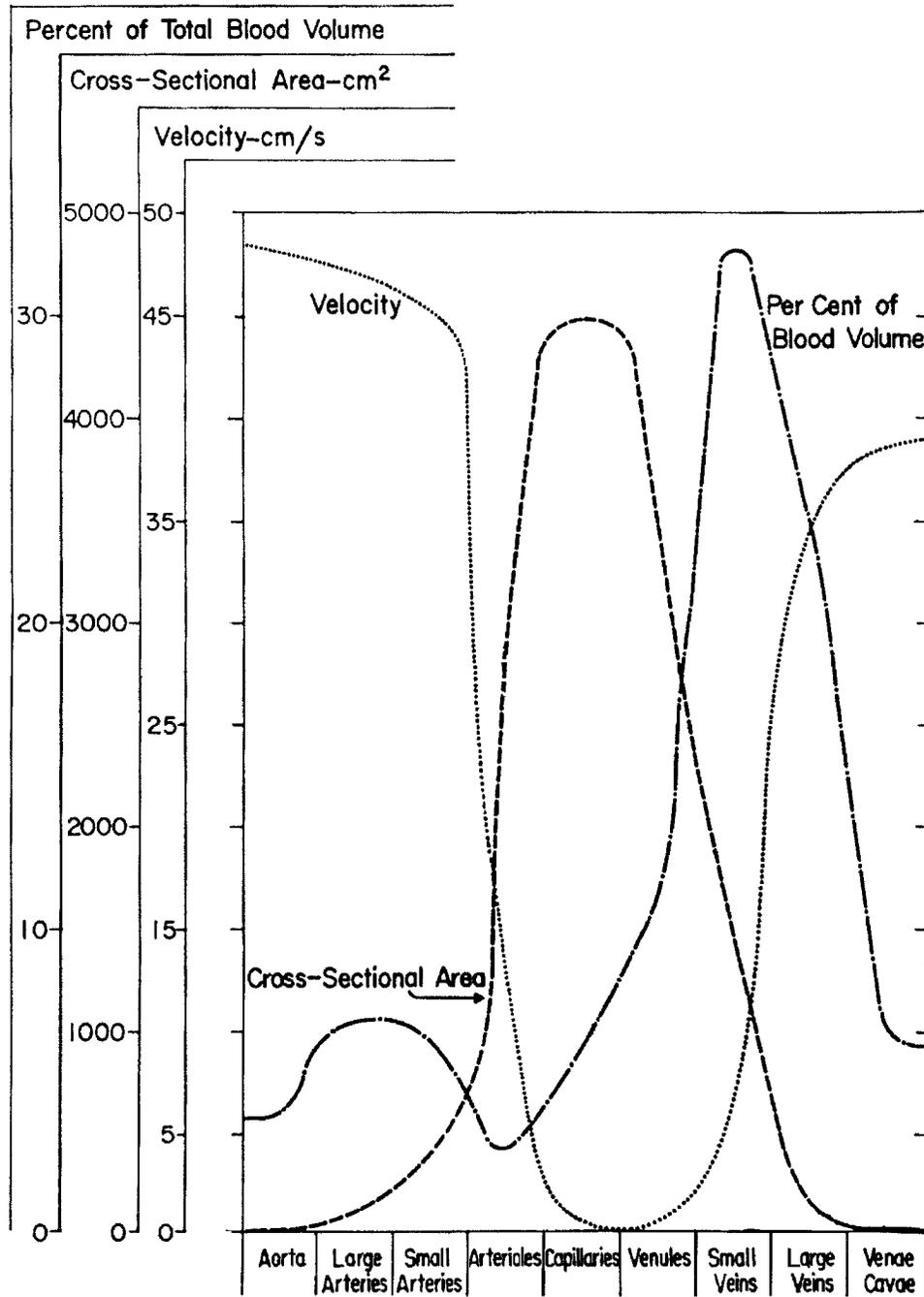


FIG. 5.

Profiles of aggregate cross-sectional area, velocity, and blood volume throughout the systemic circulation. Note that blood velocity is highest in the aorta, the capillaries have the greatest vascular cross-sectional area, and most of the blood volume is located in veins. [Modified from Berne and Levy (2) with permission.]

surgical glove) is coupled in series with a narrow resistance element (plastic pipette tip), I can convert the pulsatile output from the squeeze bulb “heart” into a sustained, pulseless efflux from the resistance element.

I stress to the students that the arterial pulse pressure (systolic pressure minus diastolic pressure) is determined primarily by two physiological variables: stroke volume and arterial compliance. If a larger stroke volume flows into and out of the elastic artery segment during each cardiac cycle, the arterial pressure will rise and fall by a greater amount. In similar fashion, the same stroke volume flowing into and out of a more rigid (less compliant) system of elastic arteries will result in a larger arterial pulse pressure. Arterial compliance tends to be reduced as we age, resulting in higher arterial pulse pressures. Because arterial compliance normally does not change over the short term, however, acute changes in arterial pulse pressure can be viewed as reflecting changes in stroke volume. This is a powerful insight toward understanding some common clinical signs (e.g., a weak or a strong radial pulse) and basic cardiovascular data.

Of course, reality is a little more complex than the simple picture outlined in the previous paragraph. The rate at which the stroke volume is ejected can affect pulse pressure. The diastolic-to-systolic rise in arterial pressure is caused by the increase in arterial volume resulting from the addition of the stroke volume less any blood volume that runs off from the elastic arteries into the microcirculation during the ejection of the stroke volume from the left ventricle. If ejection is slow (as in aortic stenosis), the net increase in arterial volume from each ventricular ejection may be reduced. Stroke volume is itself influenced by a variety of factors, including the afterload, which is related to arterial pressure. Arterial compliance is also affected by arterial pressure. Compliance is reduced at higher pressures, just as a rubber band becomes harder to stretch farther once one has already stretched it. If you are going to use case study examples, or if your course has a cardiovascular laboratory associated with it, you should be sure that your students are aware of these additional factors. However, it is important, I think, to stress that acute changes in pulse pressure usually reflect changes in stroke volume.

Some textbooks, and some cardiologists and physiologists, provide the student with the rule of thumb that diastolic arterial pressure is an index of TPR. For example, patients with essential hypertension have elevated diastolic arterial pressure. Reductions in their diastolic pressure with diet or pharmacological treatment is taken as reflecting clinical improvement (reduced TPR). I find this rule of thumb not to be helpful at all, because it is often incorrect. Most subjects will find that their diastolic arterial pressure during dynamic exercise changes very little from that at rest, or that it even rises by a couple of millimeters of mercury. Yet TPR is significantly reduced in dynamic exercise. A hemorrhagic shock victim may have blood pressures such as 80/60, but his TPR is elevated. After the injection of a systemic vasodilator, a subject’s blood pressure may be 120/60—the same diastolic pressure as our hemorrhage patient—but now this does reflect a reduced TPR. Perhaps the diastolic pressure rule of thumb can be a handy reminder to someone who already knows what is happening to a patient or experimental subject. For students trying for the first time to understand what is going on, however, I find that the “rule” does more harm than good, by leading them to think that a few quick rules will provide them with insight. I prefer to provide the students with the mechanistic building blocks for an accurate mental model of the circulation. This allows them to consider the entire cardiovascular profile (MAP, stroke volume, heart rate, CO, TPR, etc.) to evaluate the homeostatic mechanisms at work in any given situation.

Compliance

Compliance is a fairly straightforward concept. If volume is added to a blood vessel or network of blood vessels, compliance is the ratio between the change in volume (ΔV ; numerator) to the accompanying change in pressure (ΔP ; denominator). Graphically, volume and pressure can be plotted on an X - Y plot, with either variable on the X -axis. If pressure is the abscissa, then the slope of the curve is equal to the compliance. If volume is on the abscissa, compliance is the reciprocal of the slope.¹⁰

Instructors sometimes confuse students by trying to draw distinctions among several related terms: compli-

¹⁰ In this case, the slope ($\Delta P/\Delta V$) is equal to the elastance.

ance, capacitance, capacity, distensibility, and elasticity. There are distinctions among these variables, but those distinctions are probably beyond what a new student needs to know to construct a mental model of the circulation. I believe that compliance is all that is needed for that purpose and that the other terms should be omitted from an introduction to cardiovascular physiology. If you find it important to use them, however, be sure to use them correctly. For example, do not succumb to the temptation to say that veins are more elastic than arteries, and do not allow students to say it. Elasticity is the property that allows a body to resist and recover from deformation. Young's modulus of elasticity is the ratio of stress to strain and, in effect, is $\Delta P/\% \Delta l$. This is roughly the inverse of compliance ($\Delta V/\Delta P$). In fact, because arteries are more able to resist stretch than are veins, arteries are more elastic. This is an example of common usage differing from precise scientific usage. In the classroom, precise language should prevail.¹¹

Venous compliance is significantly greater (by ~20-fold) than arterial compliance, which we have already used to explain why gravity has greater effects on the volume in veins than in arteries. As another consequence of its large compliance, the venous compartment contains the largest fraction of the blood volume (Fig. 5). It is even more significant, however, that this large blood reservoir is variable in magnitude. We have already seen that an increase in venous blood volume (termed venous pooling) can reduce cardiac output. If blood is pooling in peripheral veins, it is not returning to the heart. Ventricular filling is thereby reduced and, as a consequence of the preload-dependent behavior of cardiac muscle (Starling's Law of the heart), stroke volume and CO are reduced. Conversely, venous blood pools can be mobilized, i.e., shifted back toward the heart. In that case, ventricular filling, stroke volume, and CO are increased.

How is blood mobilized or pooled in these venous reservoirs? We have already discussed the effects of gravity on venous pooling in the lower portions of the

¹¹ Our colleagues who teach respiratory physiology use elasticity in the appropriate way. We should be careful not to use the term sloppily, which would only confuse the students when they get to that section of the course. Besides, who enjoys having their colleagues shout corrections (or worse) at them from the back of the lecture hall?

body, particularly in the long veins of the legs. There are three additional major mechanisms by which the size of the venous blood pool can be adjusted to support (or restrict) venous return and CO. First, venous smooth muscle can contract or relax. In fact, the effect of venoconstriction and venodilatation on venous volume is far more important physiologically than is the effect of venomotor tone on the TPR. A second major mobilizing mechanism is the "muscle pump." Rhythmic contraction of the skeletal muscle, particularly that surrounding the long veins in the legs, serves as an auxiliary circulatory pump that, in essence, mobilizes blood from these storage sites and makes it available for ventricular filling. This is an important component of the total cardiovascular response to exercise (7). Finally, venous blood pools may be mobilized as a result of arteriolar constriction. The arteriolar constriction causes pressures downstream to fall (Fig. 2), which is associated with a reduction in venous volume governed by the compliance of that venous bed. Such "passive" mobilization of blood pools can be significant in splanchnic organs during moderate to severe exercise or in various organs in response to hemorrhage (6, 7).

Streamlined and Turbulent Flow: Reynolds Number

We saw earlier that the Poiseuille equation makes the assumption that flow is streamlined (also termed laminar flow). This is a very efficient pattern of flow in which each layer (or lamina) of fluid flows smoothly in parallel with its neighboring layers, without mixing across layers. In a cylindrical tube, this translates into a series of concentric cylindrical lamina of fluid. The lamina do not all travel at the same velocity. The innermost stream, at the longitudinal axis of the vessel, travels the fastest. The outermost layer, which touches the inner surface of the vessel wall, does not travel at all, i.e., its velocity is zero. The lamina between these two extremes travel at progressively slower velocities as the vessel wall is approached. In fact, the axial velocity gradient is a function of the fluid's viscosity. The greater the viscosity, the more slowly the velocity falls off, i.e., the velocity profile across the vessel diameter is more uniform or "flat."

The "opposite" of streamlined flow is turbulent flow. Here, the flowing fluid from adjacent lamina mix with each other to such an extent that defined lamina

cannot be identified. This flow pattern is not as efficient a laminar flow, because energy is lost as a result of the increased internal friction within the flowing fluid. In other words, if all the other variables on the right side of the Poiseuille equation were equal, there would be less flow in a turbulent setting than in a streamlined setting.

For a medical student, the real importance of turbulent flow is that there is often an audible noise associated with it. Cardiovascular noises are important diagnostically. For example, the Korotkoff sounds of auscultatory manometry are the result of turbulent flow, as are most common heart murmurs. It is useful for such students, therefore, to have an idea of when flow will be streamlined and when it will be turbulent. For that, we use the Reynolds number (N_R)¹²

$$N_R = \frac{\rho v d}{\eta} \quad (10)$$

When values for the variables on the right of the equation (ρ = density, v = velocity, d = diameter, η = viscosity) are given in cgs units, N_R is a dimensionless number whose value predicts the flow pattern. Flow is streamlined for $N_R < 2,000$ and turbulent for $N_R > 2,000$. When N_R is just a little above or below 2,000, flow might be generally streamlined with local regions of turbulence, especially around branches or irregularities on the surface of the vessel wall.

It is appealing to use the analogies of a gentle stream and white water rapids as illustrative examples of streamlined and turbulent flow. This is an important visual aid, especially because the white water image gives a feel for what turbulent flow is like, namely, churning and noisy. Unfortunately, it is a false analogy because all rivers and streams exhibit turbulent flow. Pick out your favorite stream and plug values for velocity and diameter into *Eq. 10*. (For water, $\rho = 1 \text{ g/cm}^3$ and $\eta = 0.01 \text{ poise}$.) If the water is travelling at the leisurely pace of 50 cm/s (~1 mile/h), any stream wider than 4 mm (0.4 cm) will contain turbulent flow. I give the students the visual analogy but tell them the truth about the misleadingly gentle stream.

¹² Osborne Reynolds (1842–1916).

Flow is never turbulent outside the largest vessels. Remember that velocity decreases as blood passes through the microcirculation (Fig. 5) and that individual vessel diameters are also smaller than in the aorta.¹³ The threshold N_R value is never approached. In the aorta, however, flow can be streamlined, turbulent, or locally turbulent ($N_R < 2,000$, $N_R > 2,000$, or $N_R \approx 2,000$, respectively). For example, for a 2.5-cm aorta with a flow velocity of 50 cm/s and blood viscosity of 0.04 poise, $N_R = (1 \text{ g/cm}^3) \times (50 \text{ cm/s}) \times (2.5 \text{ cm}) / (0.04 \text{ poise}) = 3,125$. Flow will be turbulent.

Flow through the brachial artery is usually streamlined. When a pressure cuff placed over the upper arm is inflated, however, the brachial artery can be compressed. When the applied cuff pressure is between the diastolic and systolic arterial pressures, then the brachial artery is only partially occluded during part of the cardiac cycle. During this time, vessel diameter is reduced. This has little effect on flow, however, because the major resistance still lies downstream in the small arteries and arterioles of the muscular and cutaneous circulatory beds of the arm. Because flow is approximately normal but vessel diameter and vascular cross-sectional area are reduced, the blood velocity is increased. Moreover, the velocity is increased by a power of two compared with the reduction in diameter. That is, if diameter is one-half normal, then velocity will be four times normal. This will increase N_R to the point where the flow becomes turbulent, and therefore noisy, during the period of the cardiac cycle when blood is flowing through the partially occluded artery. This is the basis of the Korotkoff sounds used to measure blood pressure by auscultatory sphygmomanometry. By similar reasoning, it is easy to appreciate that blood also flows through stenotic cardiac valves at a high velocity and, therefore, in a noisy, turbulent way. Even retrograde flow through incompetent cardiac valves can be turbulent because the diameter of the leak path is small compared with the normal diameter of the open valve. This is the basis of valvular heart murmurs.

In conclusion, hemodynamics is an important building block in the student's mental model of the

¹³ The Reynolds number is calculated for individual vessels, not aggregate vascular networks.

mammalian circulation. Because of its dependence on physical principles and mathematical relationships, albeit rather elementary ones, it is a topic that is challenging for some students. On the other hand, it is also a topic that is amenable to different learning styles and pedagogical approaches. Whereas the physical principles and mathematical relationships must be presented to the student, the instructor must be certain to emphasize the physiological principles that govern the behavior of the circulatory system. Although hemodynamics most often represents only a small portion of the time in a curriculum allocated for the cardiovascular system, it can be effectively used to introduce most of the larger physiological concepts that will follow. Hemodynamics, therefore, can serve as a framework for the student's conceptual understanding of cardiovascular physiology.

APPENDIX

Sample Problems

The following are examples of simple problems that can be used to reinforce some of the hemodynamic principles discussed in this article. They are presented here as simple questions plus the answers and sample calculations. They can easily be converted into multiple-choice questions. Note also that there are several ways to calculate the correct answers, not just the approaches shown here.

1) Calculate the TPR for an individual with MAP = 95 mmHg, RAP = 5 mmHg, and CO = 5 l/min.

Answer:

$$CO = 5 \text{ l/min} = 5,000 \text{ ml/min} = 83.3 \text{ ml/s}$$

$$CO = (MAP - RAP)/TPR$$

$$TPR = (MAP - RAP)/CO$$

$$= (95 - 5)/5 = 18 \text{ mmHg}\cdot\text{min}\cdot\text{l}^{-1}$$

$$= (95 - 5)/5,000 = 0.018 \text{ mmHg}\cdot\text{min}\cdot\text{ml}^{-1}$$

$$= (95 - 5)/83.3 = 1.08 \text{ mmHg}\cdot\text{s}\cdot\text{ml}^{-1} = 1.08 \text{ PRU}$$

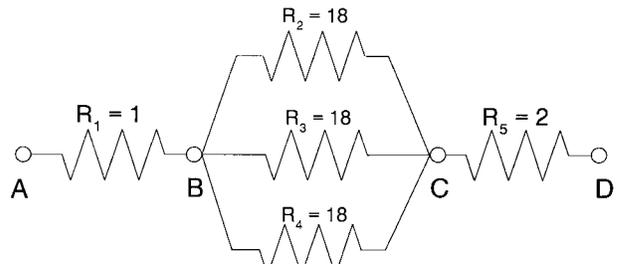


FIG. 6.

Schematic representation of a vascular network. Each vessel has a resistance (R_1 - R_5) whose value is given in units of mmHg·min·ml⁻¹. Pressure at point A (P_A) is 90 mmHg; pressure at point D (P_D) is 0 mmHg.

Use Fig. 6 to solve *problems 2-5*, given that $P_A = 90$ mmHg and $P_D = 0$ mmHg. Resistances for R_1 - R_5 are given beside each vessel (in mmHg·min·ml⁻¹).

2) What is the flow through this network?

Answer: 10 ml/min

$$R_{\text{total}} = 1 + (18/3) + 2 = 9 \text{ mmHg}\cdot\text{min}\cdot\text{ml}^{-1}$$

$$Q = \Delta P/R = (90 - 0)/9 = 10 \text{ ml/min}$$

3) What are the pressures at points B and C?

Answer: $P_B = 80$ mmHg, $P_C = 20$ mmHg

$$Q = (P_A - P_B)/R_1 \text{ or } Q = (P_C - P_D)/R_5$$

$$P_B = P_A - (Q \cdot R_1) = 90 - (10 \times 1) = 80 \text{ mmHg}$$

$$P_C = P_D + (Q \cdot R_5) = 0 + (10 \times 2) = 20 \text{ mmHg}$$

4) If R_4 is made infinite (total occlusion), what is the flow?

Answer: 7.5 ml/min

$$R_{\text{total}} = 1 + (18/2) + 2 = 12 \text{ mmHg}\cdot\text{min}\cdot\text{ml}^{-1}$$

$$Q = \Delta P/R = (90 - 0)/12 = 7.5 \text{ ml/min}$$

5) If another vessel with a resistance $R_6 = 18$ mmHg·min·ml⁻¹ is added in parallel to vessels R_2 , R_3 , and R_4 in the original network, what is the flow?

Answer: 12 ml/min

$$R_{\text{total}} = 1 + (18/4) + 2 = 7.5 \text{ mmHg}\cdot\text{min}\cdot\text{ml}^{-1}$$

$$Q = \Delta P/R = (90 - 0)/7.5 = 12 \text{ ml/min}$$

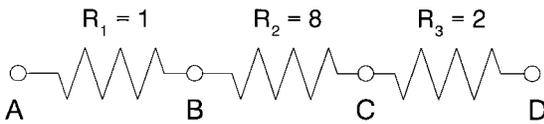


FIG. 7.

Schematic representation of a vascular network. Each vessel has a resistance (R_1 – R_3) whose value is given in units of mmHg·min·ml⁻¹. $P_A = 100$ mmHg; $P_D = 0$ mmHg.

Use Fig. 7 to solve *problems 6–8*, given that $P_A = 100$ mmHg and $P_D = 0$ mmHg. Resistances for vessels R_1 – R_3 are given (in mmHg·min·ml⁻¹).

6) What is the flow through this network?

Answer: 9.1 ml/min

$$R_{\text{total}} = 1 + 8 + 2 = 11 \text{ mmHg}\cdot\text{min}\cdot\text{ml}^{-1}$$

$$Q = \Delta P/R = (100 - 0)/11 = 9.1 \text{ ml/min}$$

7) If R_2 is halved ($R_2 = 4$), what is the flow?

Answer: 14.3 ml/min (a 57% increase from the original flow)

$$R_{\text{total}} = 1 + 4 + 2 = 7 \text{ mmHg}\cdot\text{min}\cdot\text{ml}^{-1}$$

$$Q = \Delta P/R = (100 - 0)/7 = 14.3 \text{ ml/min}$$

8) If R_2 is restored to its original value ($R_2 = 8$), but R_3 is halved ($R_3 = 1$), what is the flow?

Answer: 10 ml/min (only a 10% increase from the original flow)

$$R_{\text{total}} = 1 + 8 + 1 = 10 \text{ mmHg}\cdot\text{min}\cdot\text{ml}^{-1}$$

$$Q = \Delta P/R = (100 - 0)/10 = 10 \text{ ml/min}$$

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