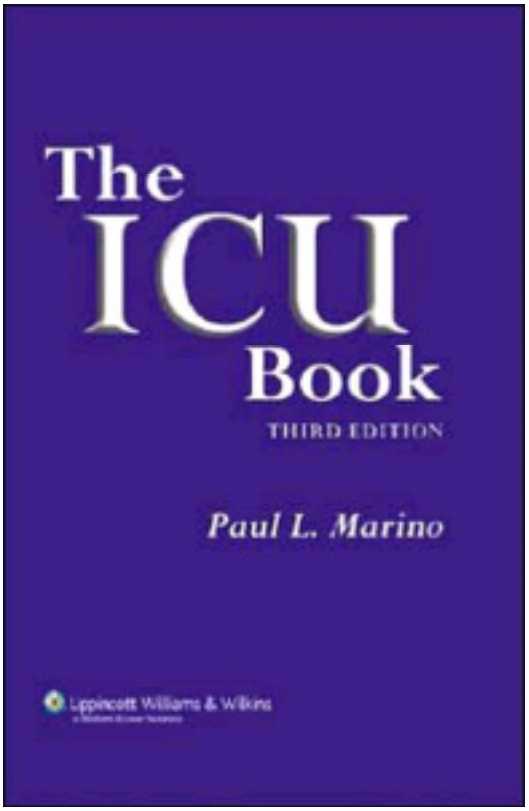

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Dedication

To Daniel Joseph Marino, My 18-year-old son. No longer a boy, And not yet a man, But always terrific.

Quote

I would especially commend the physician who, in acute diseases, by which the bulk of mankind are cutoff, conducts the treatment better than others.

—HIPPOCRATES

Preface to Third Edition

The third edition of *The ICU Book* marks its 15th year as a fundamental sourcebook in critical care. This edition continues the original intent to provide a generic textbook that presents fundamental concepts and patient care practices that can be used in any intensive care unit, regardless of the specialty focus of the unit. Highly specialized areas, such as obstetrical emergencies, thermal injury, and neurocritical care, are left to more qualified authors and their specialty textbooks.

Most of the chapters in this edition have been completely rewritten (including 198 new illustrations and 178 new tables), and there are two new chapters on infection control in the ICU (Chapter 3) and disorders of temperature regulation (Chapter 38). Most chapters also include a final section (called A Final Word) that contains an important take-home message from the chapter. The references have been extensively updated, with emphasis on recent reviews and clinical practice guidelines.

The ICU Book has been unique in that it reflects the voice of one author. This edition welcomes the voice of another, Dr. Kenneth Sutin, who added his expertise to the final 13 chapters of the book. Ken and I are old friends who share the same view of critical care medicine, and his contributions add a robust quality to the material without changing the basic personality of the work.

Preface to First Edition

In recent years, the trend has been away from a unified approach to critical illness, as the specialty of critical care becomes a hyphenated attachment for other specialties to use as a territorial signpost. The landlord system has created a disorganized array of intensive care units (10 different varieties at last count), each acting with little communion. However, the daily concerns in each intensive care unit are remarkably similar because serious illness has no landlord. The purpose of *The ICU Book* is to present this common ground in critical care and to focus on the fundamental principles of critical illness rather than the specific interests for each intensive care unit. As the title indicates, this is a 'generic' text for all intensive care units, regardless of the name on the door.

The present text differs from others in the field in that it is neither panoramic in scope nor overly indulgent in any one area. Much of the information originates from a decade of practice in intensive care units, the last three years in both a Medical ICU and a Surgical ICU. Daily rounds with both surgical and medical housestaff have provided the foundation for the concept of generic critical care that is the theme of this book.

As indicated in the chapter headings, this text is problem-oriented rather than disease-oriented, and each problem is presented through the eyes of the ICU physician. Instead of a chapter on GI bleeding, there is a chapter of the principles of volume resuscitation and two others on resuscitation fluids. This mimics the actual role of the ICU physician in GI bleeding, which is to manage the hemorrhage. The other features of the problem such as locating the bleeding site, are the tasks of other specialists. This is how the ICU operates and this is the specialty of critical care. Highly specialized topics such as burns, head trauma, and obstetric emergencies are not covered in this text. These are distinct subspecialties with their own texts and their own experts, and devoting a few pages to each would merely complete and outline rather than instruct.

The emphasis on fundamentals in *The ICU Book* is meant not only as a foundation for patient care but also to develop a strong base in clinical problem solving for any area of medicine. There is a tendency to rush past the basics in the stampede to finish formal training, and this leads to empiricism and irrational practice habits. Why a fever should or should not be treated, or whether a blood pressure cuff provides accurate readings, are questions that must be dissected carefully in the early stages of training, to develop the reasoning skills needed to be effective in clinical problems solving. This inquisitive stare must replace the knee-jerk approach to clinical problems if medicine is to advance. *The ICU Book* helps to develop this stare.

Wisely or not, the use of a single author was guided by the desire to present a uniform view. Much of the information is accompanied by published works listed at the end of each chapter and anecdotal tales are held to a minimum. Within an endeavor such as this, several shortcomings are inevitable, some omissions are likely and bias may occasionally replace sound judgment. The hope is that these deficiencies are few.

Acknowledgments

Acknowledgements are few but well deserved. First to Patricia Gast, the illustrator for this edition, who was involved in every facet of this work, and who added an energy and intelligence that goes well beyond the contributions of medical illustrators. Also to Tanya Lazar and Nicole Dernoski, my editors, for understanding the enormous time committment required to complete a work of this kind. And finally to the members of the executive and medical staff of my hospital, as well as my personal staff, who allowed me the time and intellectual space to complete this work unencumbered by the daily (and sometimes hourly) tasks involved in keeping the doors of a hospital open.

Basic Science Review

The first step in applying the scientific method consists in being curious about the world.

--Linus Pauling

Chapter 1

Circulatory Blood Flow

> *When is a piece of matter said to be alive? When it goes on "doing something," moving, exchanging material with its environment*.
>
> *--Erwin Schrodinger*

The human organism has an estimated 100 trillion cells that must go on exchanging material with the external environment to stay alive. This exchange is made possible by a circulatory system that uses a muscular pump (the heart), an exchange fluid (blood), and a network of conduits (blood vessels). Each day, the human heart pumps about 8,000 liters of blood through a vascular network that stretches more than 60,000 miles (more than twice the circumference of the Earth!) to maintain cellular exchange (1).

This chapter describes the forces responsible for the flow of blood though the human circulatory system. The first half is devoted to the determinants of cardiac output, and the second half describes the forces that influence peripheral blood flow. Most of the concepts in this chapter are old friends from the physiology classroom.

Cardiac Output

Circulatory flow originates in the muscular contractions of the heart. Since blood is an incompressible fluid that flows through a closed hydraulic loop, the volume of blood ejected by the left side of the heart must equal the volume of blood returning to the right side of the heart (over a given time period). This conservation of mass (volume) in a closed hydraulic system is known as the *principle of continuity* (2), and it indicates that the stroke output of the heart is the principal determinant of circulatory blood flow. The forces that govern cardiac stroke output are identified in Table 1.1.

TABLE 1.1 The Forces that Determine Cardiac Stroke Output

Force	Definition	Clinical Parameters
Preload	The load imposed on resting muscle that stretches the muscle to a new length	End-diastolic pressure
Contractility	The velocity of muscle contraction when muscle load is fixed	Cardiac stroke volume when preload and afterload are constant
Afterload	The total load that must be moved by a muscle when it contracts	Pulmonary and systemic vascular resistances

Preload

If one end of a muscle fiber is suspended from a rigid strut and a weight is attached to the other free end, the added weight will stretch the muscle to a new length. The added weight in this situation represents a force called the *preloa*, which is a force imposed on a resting muscle (prior to the onset of muscle contraction) that stretches the muscle to a new length. According to the length–tension relationship of muscle, an increase in the length of a resting (unstimulated) muscle will increase the force of contraction when the muscle is stimulated to contract. Therefore **the preload force acts to augment the force of muscle contraction**.

In the intact heart, the stretch imposed on the cardiac muscle prior to the onset of muscle contraction is a function of the volume in the ventricles at the end of diastole. Therefore the end-diastolic volume of the ventricles is the preload force of the intact heart (3).

Preload and Systolic Performance

The pressure-volume curves in Figure 1.1 show the influence of diastolic volume on the systolic performance of the heart. As the ventricle fills during diastole, there is an increase in both diastolic and systolic pressures. The increase in diastolic pressure is a reflection of the passive stretch imposed on the ventricle, while the difference between diastolic and systolic pressures is a reflection of the strength of ventricular contraction. Note that as diastolic volume increases, there is an increase in the difference between diastolic and systolic pressures, indicating that the strength of ventricular contraction is increasing. The importance of preload in augmenting cardiac contraction was discovered independently by Otto Frank (a German engineer) and Ernest Starling (a British physiologist), and their discovery is commonly referred to as the *Frank-Starling relationship of the heart* (3). This relationship can be stated as follows: **In the normal heart, diastolic volume is the principal force that governs the strength of**

ventricular contraction (3).

Clinical Monitoring

In the clinical setting, the relationship between preload and systolic performance is monitored with *ventricular function curves* like the ones

shown in Figure 1.2. End-diastolic pressure (EDP) is used as the clinical measure of preload because end-diastolic volume is not easily measured (the measurement of EDP is described in Chapter 10). The normal ventricular function curve has a steep ascent, indicating that changes in preload have a marked influence on systolic performance in the normal heart (i.e., the Frank-Starling relationship). When myocardial contractility is reduced, there is a decrease in the slope of the curve, resulting in an increase in end-diastolic pressure and a decrease in stroke volume. This is the hemodynamic pattern seen in patients with heart failure.

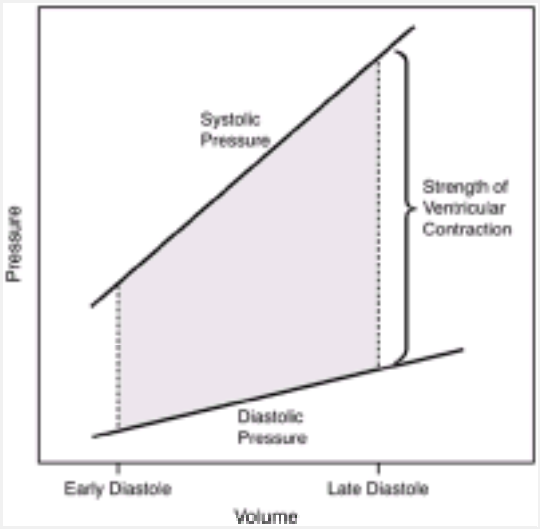

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Figure 1.1 Pressure-volume curves showing the influence of diastolic volume on the strength of ventricular contraction.

Ventricular function curves are used frequently in the intensive care unit (ICU) to evaluate patients who are hemodynamically unstable. However, these curves can be misleading. The major problem is that conditions other than myocardial contractility can influence the slope of these curves. These conditions (i.e., ventricular compliance and ventricular afterload) are described next.

Preload and Ventricular Compliance

The stretch imposed on cardiac muscle is determined not only by the volume of blood in the ventricles, but also by the tendency of the ventricular wall to distend or stretch in response to ventricular filling.

The distensibility of the ventricles is referred to as *compliance* and can be derived using the following relationship between changes in end-diastolic pressure (EDP) and end-diastolic volume (EDV) (5):

$$\text{Compliance} = \Delta EDV / \Delta EDP \qquad (1.1)$$

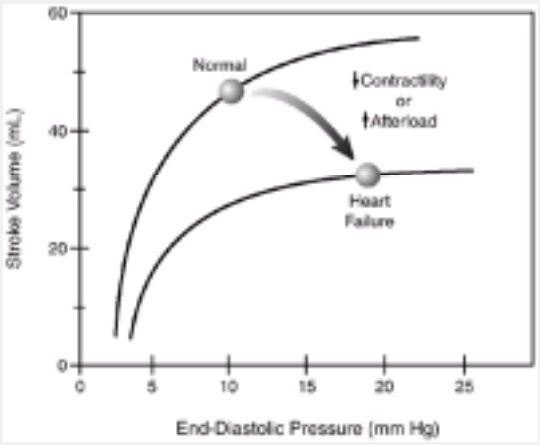

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Figure 1.2 Ventricular function curves used to describe the relationship between preload (end-diastolic pressure) and systolic performance (stroke volume).

The pressure-volume curves in Figure 1.3 illustrate the influence of ventricular compliance on the relationship between ?EDP and ?EDV. As compliance decreases (i.e., as the ventricle becomes stiff), the slope of the curve decreases, resulting in a decrease in EDV at any given EDP. In this situation, the EDP will overestimate the actual preload (EDV). This illustrates how changes in ventricular compliance will influence the reliability of EDP as a reflection of preload. The following statements highlight the importance of ventricular compliance in the interpretation of the EDP measurement.

1. End-diastolic pressure is an accurate reflection of preload only when ventricular compliance is normal.
2. Changes in end-diastolic pressure accurately reflect changes in preload only when ventricular compliance is constant.

Several conditions can produce a decrease in ventricular compliance. The most common are left ventricular hypertrophy and ischemic heart

disease. Since these conditions are also commonplace in ICU patients, the reliability of the EDP measurement is a frequent concern.

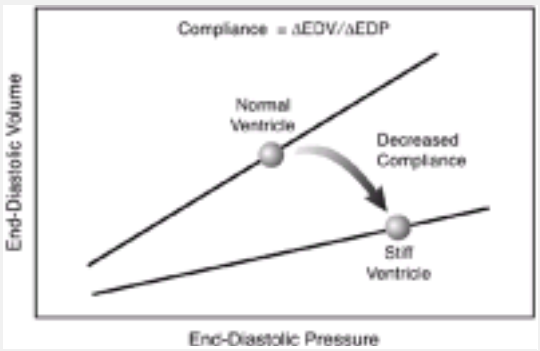

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Figure 1.3 Diastolic pressure-volume curves in the normal and noncompliant (stiff) ventricle.

Diastolic Heart Failure

As ventricular compliance begins to decrease (e.g., in the early stages of ventricular hypertrophy), the EDP rises, but the EDV remains unchanged. The increase in EDP reduces the pressure gradient for venous inflow into the heart, and this eventually leads to a decrease in EDV and a resultant decrease in cardiac output (via the Frank-Starling mechanism). This condition is depicted by the point on the lower graph in Figure 1.3, and is called *diastolic heart failure* (6). Systolic function (contractile strength) is preserved in this type of heart failure.

Diastolic heart failure should be distinguished from conventional (systolic) heart failure because the management of the two conditions differs markedly. For example, since ventricular filling volumes are reduced in diastolic heart failure, diuretic therapy can be counterproductive. Unfortunately, it is not possible to distinguish between the two types of heart failure when the EDP is used as a measure of preload because the EDP is elevated in both conditions. The ventricular function curves in Figure 1.3 illustrate this problem. The point on the lower curve identifies a condition where EDP is elevated and stroke volume is reduced. This condition is often assumed to represent heart failure due to systolic dysfunction, but diastolic dysfunction would also produce the same changes. This inability to distinguish between systolic and diastolic heart failure is one of the major shortcomings of ventricular function curves. (See Chapter 14 for a more detailed discussion of systolic and diastolic heart failure.)

Afterload

When a weight is attached to one end of a contracting muscle, the force of muscle contraction must overcome the opposing force of the weight before the muscle begins to shorten. The weight in this situation represents a force called the *afterload*, which is defined as the load imposed on a muscle *after* the onset of muscle contraction. Unlike the preload force, which facilitates muscle contraction, **the afterload force opposes muscle contraction** (i.e., as the afterload increases, the muscle must develop more tension to move the load). In the intact heart, **the afterload force is equivalent to the peak tension developed across the wall of the ventricles during systole** (3).

The determinants of ventricular wall tension (afterload) were derived from observations on soap bubbles made by the Marquis de Laplace in 1820. His observations are

expressed in the Law of Laplace, which states that the tension (T) in a thin-walled sphere is directly related to the chamber pressure (P) and radius (r) of the sphere: T = Pr. When the LaPlace relationship is applied to the heart, T represents the peak systolic transmural wall tension of the ventricle, P represents the transmural pressure across the ventricle at the end of systole, and r represents the chamber radius at the end of diastole (5).

The forces that contribute to ventricular afterload can be identified using the components of the Laplace relationship, as shown in Figure 1.4. There are three major contributing forces: pleural pressure, arterial impedance, and end-diastolic volume (preload). Preload is a component of afterload because it is a volume load that must be moved by the ventricle during systole.

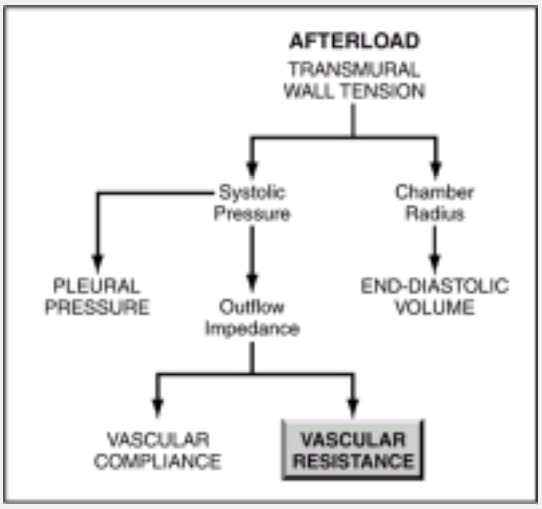

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Figure 1.4 The forces that contribute to ventricular afterload.

Pleural Pressure

Since afterload is a transmural force, it is determined in part by the pleural pressure on the outer surface of the heart. Negative pleural pressures will increase transmural pressure and increase ventricular afterload, while positive pleural pressures will have the opposite effect. Negative pressures surrounding the heart can impede ventricular emptying by opposing the inward displacement of the ventricular wall during systole (7,8). This effect is responsible for the transient decrease in systolic blood pressure (reflecting a decrease in cardiac stroke volume) that normally occurs during the inspiratory phase of spontaneous breathing. When the inspiratory drop in systolic pressure is greater than 15 mm Hg, the condition is called “pulsus paradoxus” (which is a misnomer, since the response is not paradoxical, but is an exaggeration of the normal response).

Positive pleural pressures can promote ventricular emptying by facilitating the inward movement of the ventricular wall during systole (7,9). This effect is illustrated in Figure 1.5. The tracings in this figure show the effect of positive-pressure mechanical ventilation on the arterial blood pressure. When intrathoracic pressure rises during a positive-pressure breath, there is a transient rise in systolic blood pressure (reflecting an increase in the stroke volume output of the heart). This response indicates that positive intrathoracic pressure can provide

cardiac support by “unloading” the left ventricle. Although this effect is probably of minor significance, positive-pressure mechanical ventilation has been proposed as a possible therapeutic modality in patients with cardiogenic shock (10). The hemodynamic effects of mechanical ventilation are discussed in more detail in Chapter 24.

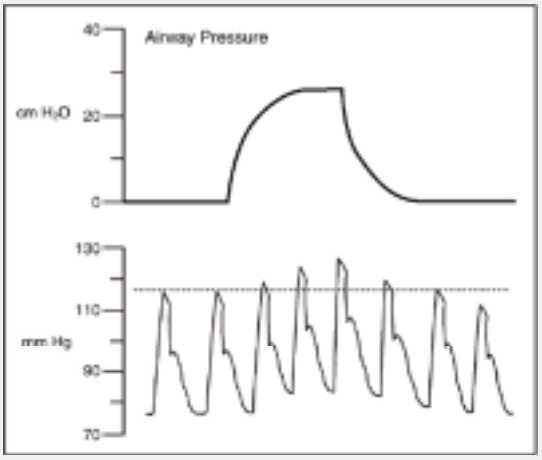

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Figure 1.5 Respiratory variations in blood pressure during positive-pressure mechanical ventilation.

Impedance

The principal determinant of ventricular afterload is a hydraulic force known as *impedance* that opposes phasic changes in pressure and flow. This force is most prominent in the large arteries close to the heart, where it acts to oppose the pulsatile output of the ventricles. **Aortic impedance is the major afterload force for the left ventricle**, and pulmonary artery impedance serves the same role for the right ventricle. Impedance is influenced by two other forces: (a) a force that opposes the rate of change in flow, known as *compliance*, and (b) a force that opposes steady flow, called *resistance*. Arterial compliance is expressed primarily in the large, elastic arteries, where it plays a major role in determining vascular impedance. Arterial resistance is expressed primarily in the smaller peripheral arteries, where the flow is steady and nonpulsatile. Since resistance is a force that opposes nonpulsatile flow, while impedance opposes pulsatile flow, **arterial resistance may play a minor role in the impedance to ventricular emptying**. Arterial resistance can, however, influence pressure and flow events in the large, proximal arteries (where impedance is prominent) because it acts as a downstream resistance for these arteries.

Vascular impedance and compliance are complex, dynamic forces that are not easily measured (12,13). Vascular resistance, however, can be calculated as described next.

Vascular Resistance

The resistance (R) to flow in a hydraulic circuit is expressed by the relationship between the pressure gradient across the circuit (?P) and the rate of flow (Q) through the circuit:

$$R = \Delta P/Q \qquad (1.2)$$

Applying this relationship to the systemic and pulmonary circulations yields the following equations for systemic vascular resistance (SVR) and pulmonary vascular resistance

(PVR):

$$SVR = SAP - RAP/CO \quad (1.3)$$
$$PVR = PAP - LAP/CO \quad (1.4)$$

SAP is the mean systemic arterial pressure, RAP is the mean right atrial pressure, PAP is mean pulmonary artery pressure, LAP is the mean left atrial pressure, and CO is the cardiac output. The SAP is measured with an arterial catheter (see Chapter 8), and the rest of the measurements are obtained with a pulmonary artery catheter (see Chapter 9).

Clinical Monitoring

There are no accurate measures of ventricular afterload in the clinical setting. The SVR and PVR are used as clinical measures of afterload, but they are unreliable (14,15). There are two problems with the use of vascular resistance calculations as a reflection of ventricular afterload. First, arterial resistance may contribute little to ventricular afterload because it is a force that opposes nonpulsatile flow, while afterload (impedance) is a force that opposes pulsatile flow. Second, the SVR and PVR are measures of total vascular resistance (arterial and venous), which is even less likely to contribute to ventricular afterload than arterial resistance. These limitations have led to the recommendation that PVR and SVR be abandoned as clinical measures of afterload (15).

Since afterload can influence the slope of ventricular function curves (see Figure 1.2), changes in the slope of these curves are used as indirect evidence of changes in afterload. However, other forces, such as ventricular compliance and myocardial contractility, can also influence the slope of ventricular function curves, so unless these other forces are held constant, a change in the slope of a ventricular function curve cannot be used as evidence of a change in afterload.

Contractility

The contraction of striated muscle is attributed to interactions between contractile proteins arranged in parallel rows in the sarcomere. The number of bridges formed between adjacent rows of contractile elements determines the contractile state or *contractility* of the muscle fiber. The contractile state of a muscle is reflected by the force and velocity of muscle contraction when loading conditions (i.e., preload and afterload) are held constant (3). The standard measure of contractility is the acceleration rate of ventricular pressure (dP/dt) during isovolumic contraction (the time from the onset of systole to the opening of the aortic valve, when preload and afterload are constant). This can be measured during cardiac catheterization.

Clinical Monitoring

There are no reliable measures of myocardial contractility in the clinical setting. The relationship between end-diastolic pressure and stroke volume (see Figure 1.2) is often used as a reflection of contractility; however, other conditions (i.e., ventricular compliance and afterload) can influence this relationship. There are echocardiography techniques for evaluating contractility (15,16), but these are very specialized and not used routinely.

Peripheral Blood Flow

As mentioned in the introduction to this chapter, there are over 60,000 miles of blood vessels in the human body! Even if this estimate is off by 10,000 or 20,000 miles, it still points to the incomprehensible vastness of the human circulatory system. The remainder of this chapter will describe the forces that govern flow through this vast network of blood vessels.

A Note of Caution: The forces that govern peripheral blood flow are derived from observations on idealized hydraulic circuits where the flow is steady and laminar (streamlined), and the conducting tubes are rigid. These conditions bear little resemblance to the human circulatory system, where the flow is often pulsatile and turbulent, and the blood vessels are compressible and not rigid. Because of these differences, the description of blood flow that follows should be viewed as a very schematic representation of what really happens in the circulatory system.

Flow in Rigid Tubes

Steady flow (Q) through a hollow, rigid tube is proportional to the pressure gradient along the length of the tube (?P), and the constant of proportionality is the hydraulic resistance to flow (R):

$$Q = \Delta P \times 1/R \qquad (1.5)$$

The resistance to flow in small tubes was described independently by a German physiologist (G. Hagen) and a French physician (J. Poisseuille). They found that resistance to flow is a function of the inner radius of the tube (r), the length of the tube (L), and the viscosity of the fluid (m). Their observations are expressed in the following equation, known as the Hagen-Poisseuille equation (18):

$$Q = \Delta P \times (\pi r^4/8\mu L) \qquad (1.6)$$

The final term in the equation is the reciprocal of resistance (1/R), so resistance can be described as

$$R = 8\mu L/\pi r^4 \qquad (1.7)$$

The Hagen-Poisseuille equation is illustrated in Figure 1.6. Note that flow varies according to the fourth power of the inner radius of the tube. This means that **a two-fold increase in the radius of the tube will result in a sixteen-fold increase in flow**: $(2r)^4 = 16r$. The other components of resistance (i.e., tube length and fluid viscosity) exert a much smaller influence on flow.

Since the Hagen-Poisseuille equation describes steady flow through rigid tubes, it may not accurately describe the behavior of the circulatory system (where flow is not steady and the tubes are not rigid). However, there are several useful applications of this equation. In Chapter 6, it will be used to describe flow through vascular catheters (see Figure 6.1). In Chapter 12, it will be used to describe the flow characteristics of different resuscitation fluids, and in Chapter 36, it will be used to describe the hemodynamic effects of anemia and blood transfusions.

Flow in Tubes of Varying Diameter

As blood moves away from the heart and encounters vessels of decreasing diameter, the resistance to flow should increase and the flow should decrease. This is not possible because (according to the principle of

continuity) blood flow must be the same at all points along the circulatory system. This discrepancy can be resolved by considering the influence of tube narrowing on flow velocity. For a rigid tube of varying diameter, the velocity of flow (v) at any point along the tube is directly proportional to the bulk flow (Q), and inversely proportional to the cross-sectional area of the tube: v = Q/A (2). Rearranging terms (and using $A = p^2$) yields the following:

$$Q = v \times (\pi r^2) \qquad (1.8)$$

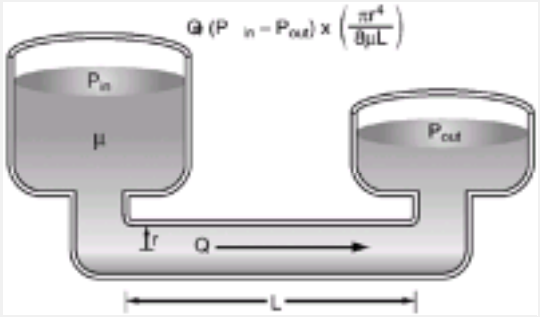

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Figure 1.6 The forces that influence steady flow in rigid tubes. Q = flow rate, P_{in} = inlet pressure, P_{out} = outlet pressure, μ = viscosity, r = inner radius, L = length.

This shows that bulk flow can remain unchanged when a tube narrows if there is an appropriate increase in the velocity of flow. This is how the nozzle on a garden hose works and is how blood flow remains constant as the blood vessels narrow.

Flow in Compressible Tubes

Flow through compressible tubes (like blood vessels) is influenced by the external pressure surrounding the tube. This is illustrated in Figure 1.7, which shows a compressible tube running through a fluid reservoir. The height of the fluid in the reservoir can be adjusted to vary the external pressure on the tube. When there is no fluid in the reservoir and the external pressure is zero, the driving force for flow through the tube will be the pressure gradient between the two ends of the tube (P_{in} - P_{out}). When the reservoir fills and the external pressure exceeds the lowest pressure in the tube (P_{ext} – P_{out}), the tube will be compressed. In this situation, the driving force for flow is the pressure gradient between the inlet pressure and the external pressure (P_{in} - P_{ext}). Therefore **when a tube is compressed by external pressure, the driving force for flow is independent of the pressure gradient along the tube** (20).

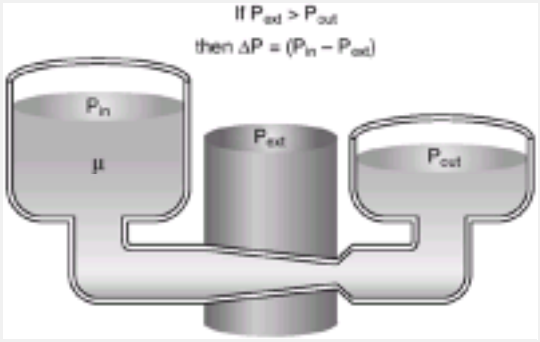

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Figure 1.7 The influence of external pressure on flow through compressible tubes. P_{in} = inlet pressure, P_{out} = outlet pressure, P_{ext} = external pressure.

P.14

The Pulmonary Circulation

Vascular compression has been demonstrated in the cerebral, pulmonary, and systemic circulations. It can be particularly prominent in the pulmonary circulation during positive-pressure mechanical ventilation, when alveolar pressure exceeds the hydrostatic pressure in the pulmonary capillaries (20). When this occurs, the driving force for flow through the lungs is no longer the pressure gradient from the main pulmonary arteries to the left atrium (PAP - LAP), but instead is the pressure difference between the pulmonary artery pressure and the alveolar pressure (PAP - Palv). This change in driving pressure not only contributes to a reduction in pulmonary blood flow, but it also affects the pulmonary vascular resistance (PVR) calculation as follows:

$$\text{Normal: } PVR = PAP - LAP/CO \quad (1.9)$$
$$\text{When } Palv > LAP: \quad PVR = PAP - Palv/CO \quad (1.10)$$

Vascular compression in the lungs is discussed again in Chapter 10 (the measurement of vascular pressures in the thorax) and in Chapter 24 (the hemodynamic effects of mechanical ventilation).

Blood Viscosity

A solid will resist being deformed (changing shape), while a fluid will deform continuously (flow) but will resist changes in the rate of deformation (i.e., changes in flow rate) (21). The resistance of a fluid to

P.15

changes in flow rate is a property known as *viscosity* (21,22,23). Viscosity has also been referred to as the “gooiness” of a fluid (21). When the viscosity of a fluid increases, a greater force must be applied to the fluid to initiate a change in flow rate. The influence of viscosity on flow rate is apparent to anyone who has poured molasses (high viscosity) and water (low viscosity) from a container.

Hematocrit

The viscosity of whole blood is almost entirely due to cross-linking of circulating erythrocytes by plasma fibrinogen (22,23). **The principal determinant of whole blood**