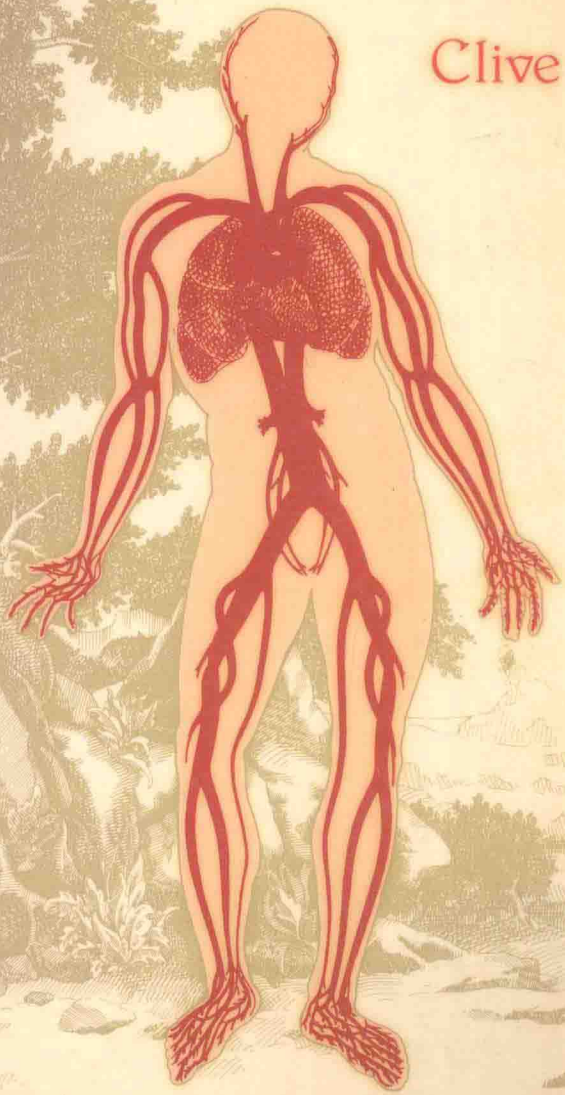
Clinical Cardiovascular and Pulmonary Physiology

Clive Rosendorff



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To my family, with love.

Preface

This book was created in response to the need for a concise but comprehensive outline of the clinical physiology of the heart, circulation, and lungs. The concept evolved from the frustration of my students at trying to relate what they had read in textbooks of physiology to what they were seeing in patients. The content was developed from courses in cardiovascular and pulmonary physiology for preclinical medical students and from bedside teaching in cardiovascular and pulmonary diseases to students in their years of clinical study. The cardiovascular and pulmonary systems are described in such a way that the clinical relevance of the physiologic effects is always stressed. The main emphasis of this book is on the disordered function that is produced by diseases of the cardiovascular and respiratory systems including complications, end-stage effects, and pathophysiologic consequences of disease. There is no attempt made to describe the molecular mechanisms of the disease processes as this would require another book of at least the same size.

The emphasis on clinical and applied aspects of cardiovascular and pulmonary physiology not only responds to the somewhat fashionable demand for relevance, but also comes out of a deep personal conviction that medical students should be taught human, applied, and clinical physiology: "The proper study of mankind is man." Of the many ways of thinking about the heart and lungs, this book describes those that the student or physician will encounter in the wards and clinics, and the instrumentation and tests described are those that will be used in everyday practice or in specialized laboratories.

The practical focus of this volume makes it a suitable textbook for medical, paramedical and nursing students, and for interns, residents, and physicians preparing for postgraduate examinations, as well as for others in cardiovascular and pulmonary sections of courses in physiology that have a clinical bias.

Clive Rosendorff

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Less tangible, but just as important, is the great privilege of having been able to talk with so many friends and outstanding leaders in cardiovascular and respiratory physiology and medicine about the current concepts and future trends, and to read, with pleasure and admiration, their work. In this regard I am particularly grateful to Arthur Guyton, Fran Ganong, Richard Gorlin, Jeremy Swan, Eliot Corday, John Ross, Jr., Bill Parmley, Julien Hoffman, John Laragh, John West, Ed Frohlich, John Shepherd, Attilio Maseri, and Lionel Opie. To all of these, and to many others too numerous to mention, I owe a debt of gratitude.

Section 1

The Cardiovascular System

The Master said, To learn and at due times to repeat what one has learnt, is that not after all a pleasure?

—The Analects (Lun Yü) of Confucius

Contents

1

Cardiovascular Dynamics

The cardiovascular system (CVS), by means of blood flow, conveys nutrients from one part of the body to another: oxygen from the lungs to the tissues, carbon dioxide from the tissues to the lungs, and waste products to excretory organs, such as the liver and kidney. It is responsible for the transfer of information from one part of the body to another in the form of hormones and for the dissipation of heat from the skin.

FLOW, PRESSURE, AND RESISTANCE IN A RIGID TUBE

All functions of the CVS depend on relatively simple hemodynamic principles, which describe the relationship between flow, perfusion pressure (i.e., the pressure that drives the fluid along the vessel), and resistance. Let us first

consider a plumbing analogy, the situation that applies when a Newtonian liquid, such as water, flows along a rigid tube, and the flow is laminar. A Newtonian liquid is one in which the viscosity is independent of flow rate. Laminar flow (streamline or nonturbulent flow) is flow in which the particles of liquid move parallel to the long axis of the tube; there is no component of motion across the tube (see Fig. 1.20). We shall see later that blood is not a Newtonian liquid, that blood flow is not always laminar, and obviously, that blood vessels do not have a constant caliber. However, the plumbing analogy provides a basis on which to consider the more complicated case of blood flow in distensible vessels.

The relationship between flow, perfusion pressure, and resistance for a rigid tube and a Newtonian liquid with laminar flow is given by:

$$F \alpha \frac{P}{R},$$

where F = flow (in ml/min), P = perfusion pressure [i.e., the pressure at the beginning of the tube minus the pressure at the end of the tube (in mm Hg, dyne/cm^2, or N/m^2); this should be distinguished from the transmural pressure, i.e., the pressure within the tube minus the pressure outside the tube], and R = resistance.

This equation is fundamental in cardiovascular physiology and is analogous to the more familiar Ohm's law which states that I = E/R, where I = current (in amperes), E = electromotive force (in volts), and R = resistance (in ohms). The implications of the relationship are described in Table 1.1.

Corollaries of the F α P/R relationship are P α FR and R α P/F. The graphic representation of the F α P/R relationship is known as the "passive pressure flow relationship." If the resistance is constant (R_1), then F α P and the graph is a straight line (Fig. 1.1).

TABLE 1.1. *Factors causing increasing or decreasing flow*

Flow increases	Flow decreases
Perfusion pressure increases with resistance constant	Perfusion pressure decreases with resistance constant
Both perfusion pressure and resistance increase, but perfusion pressure increases more than resistance	Both perfusion pressure and resistance decrease, but perfusion pressure falls more than resistance
Perfusion pressure remains constant and resistance falls (e.g., by an increase in diameter of the pipe, or vasodilatation in the CVS)	Perfusion pressure remains constant and resistance rises (e.g., by a decrease in diameter of the pipe, or vasoconstriction in the CVS)
Both perfusion pressure and resistance fall, but resistance falls more than perfusion pressure	Both perfusion pressure and resistance increase, but resistance increases more than perfusion pressure
Perfusion pressure increases and resistance falls	Perfusion pressure falls and resistance rises

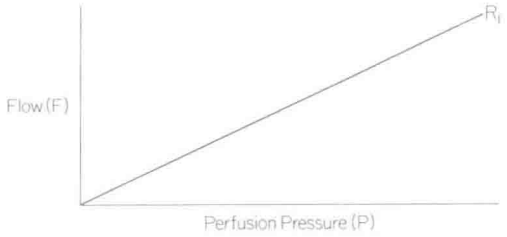

FIG. 1.1. Relationship between perfusion pressure, flow, and resistance: the passive system (see text).

FLOW, PRESSURE, AND RESISTANCE IN THE CARDIOVASCULAR SYSTEM

The first respect in which blood vessels differ from lead pipes is that they have a critical closing pressure (CCP) (Fig. 1.2). The CCP in blood vessels is the smallest pressure that will generate flow. Alternatively, it is the pressure below which no flow takes place, i.e., the vessel is collapsed or closed. In the case of capillaries, this may be due to the fact that it takes some pressure to force red blood cells (mean diameter, ~7.5 μm) through capillaries (mean diameter, ~6.0 μm). Also, the vessels are surrounded by tissues that exert a small but definite pressure on them; when the intraluminal pressure falls below the tissue pressure, the vessels collapse. In inactive tissues, for example, the pressure within capillaries may be below the CCP because the precapillary sphincters and metarterioles are constricted; therefore, many of these capillaries are collapsed.

Second, blood vessels can change their diameter. This is particularly true of those vessels that have a lot of smooth muscle in the medial layer of the vessel wall, such as arterioles. Since arterioles, by changing their diameter, can control the resistance to blood flow through organs, they are often referred to as "resistance vessels." In contrast, large capacity vessels with a poorly developed smooth muscle layer, such as the great veins, are less capable of constricting or dilating and therefore contribute much less to the total peripheral vascular resistance than arterioles. Also, because of their large size and the large volume of blood contained within them, the great veins are sometimes referred to as "capacitance vessels." However, even the limited ability of the great veins to constrict or dilate is important in control of cardiac output and in overall cardiovascular control.

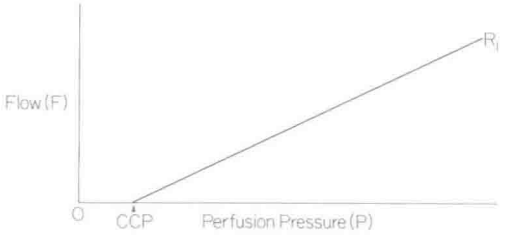

FIG. 1.2. Relationship between perfusion pressure, flow, and resistance: the critical closing pressure (CCP).

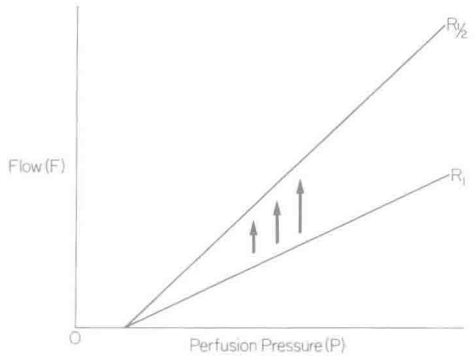

FIG. 1.3. Relationship between perfusion pressure, flow, and resistance: effect of decreasing resistance.

If the diameter of a vessel is increased (vasodilatation) such that the resistance is reduced from R_1 to $R_{1/2}$, the pressure-flow relationship is altered, as shown in Fig. 1.3. If the resistance is decreased, the flow is increased for any given perfusion pressure; this is also obvious from the $F \alpha P/R$ equation.

If resistance is increased from R_1 to R_2 (vasoconstriction), then the pressure-flow relationship is altered, as shown in Fig. 1.4. Here, flow is decreased for any given perfusion pressure, i.e., in the equation $F \alpha P/R$, an increase in R causes a fall in F, if P is constant.

If the resistance increases or decreases continuously and at the same rate as the perfusion pressure, then flow will be constant (Fig. 1.5). That is, if in the relationship $F \alpha P/R$, P and R change at the same rate, then F is constant.

Constant blood flow is a common situation in the vascular beds of many organs, such as the brain, myocardium, and kidneys; i.e., the flow is relatively constant over a wide range of perfusion pressures because of a continuous adjustment, or regulation, of the resistance of the system. Even if the autonomic nerves supplying the arterioles are cut or blocked, the resistance vessels keep flow constant by adjusting their diameter independent of the nerve supply. This phenomenon is known as autoregulation. Most organs autoregulate within a range of perfusion pressures from about 60 to about 120

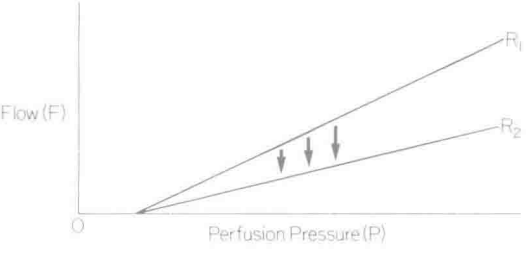

FIG. 1.4. Relationship between perfusion pressure, flow, and resistance: effect of increasing resistance.

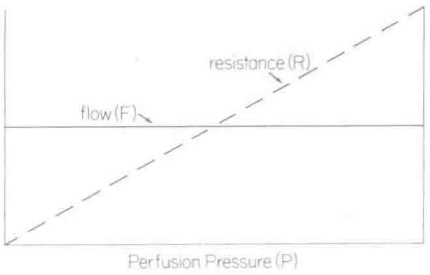

FIG. 1.5. Relationship between perfusion pressure, flow, and resistance: principle of autoregulation (see text).

mm Hg. The perfusion pressure of the organ is the pressure in the artery or arteries supplying that organ minus the pressure in the vein or veins draining the organ. Venous blood pressure is small as compared with arterial blood pressure, so that as an approximation, the perfusion pressure can be considered to be the same as the arterial blood pressure. The arterial blood pressure, which is usually referred to in this context, is the mean arterial blood pressure. If the mean arterial pressure (P) falls from its normal value of about 90 mm Hg (e.g., during hemorrhage), resistance vessels dilate, causing a fall in vascular resistance (R), so that flow ($F \alpha P/R$) remains constant (Fig. 1.6). Conversely, if P rises, resistance vessels constrict, causing an increase in R, so that flow ($F \alpha P/R$) remains constant.

At the lower limit of autoregulation (about 60 mm Hg) vessels are maximally dilated, i.e., resistance is at the lowest possible value. Any further decrease in perfusion pressure results in a decrease in flow. Between 60 and 120 mm Hg, there is a progressive increase in vascular resistance (vasoconstriction), which parallels the increase in arterial pressure; thus flow remains constant. At about 120 mm Hg, the vessels are maximally constricted and unable to increase their resistance. Further increase in perfusion pressure causes a corresponding increase in flow (Fig. 1.6).

Most organs are capable of surviving a modest fall in blood pressure, providing that the vessels are capable of dilating normally. An inability to

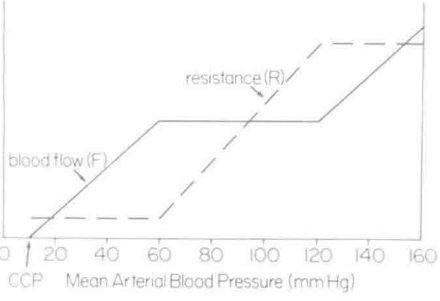

FIG. 1.6. Relationship between perfusion pressure, flow, and resistance: the autoregulated system (see text).

dilate normally in response to a fall in perfusion pressure (i.e., failure of autoregulation) occurs if the vessels are diseased, as in atheroma or arteriosclerosis, or if blood flow is obstructed, causing poor oxygenation, as in stroke. Even modest falls in blood pressure may seriously compromise organ blood flow in elderly patients, especially those with diseased arteries. Mean arterial pressures below 50 to 60 mm Hg, especially if prolonged, are dangerous in patients of all ages.

PRESSURE

Units of Pressure

In the centimeter-gram-second (cgs) system of units, pressure is measured as dyne/cm^2. In the Système International d'Unités (SI) system, where the basic units are the meter, the kilogram, and the second, the unit of force is the newton (N), which is the force required to accelerate a mass of 1 kg at 1 m/sec^2. Pressures are measured as N/m^2 [1 N/m^2 = 1 pascal (Pa)]. Physiologic pressure-measuring devices are usually calibrated against mercury, water, or saline columns; hence, it is customary to use mm Hg, cm H_2O, or cm saline.

If a column of liquid has a height h, cross-sectional area A and density ρ, then the volume of the column is hA and its mass is ρhA. The downward force is mass times acceleration, $g\rho$hA newtons, where g is the acceleration due to gravity, 9.8 m/sec^2. This force divided by the cross-sectional area is pressure, $g\rho$h N/m^2, (i.e., $g\rho$h Pa). Therefore, the pressure exerted by a column of mercury 1 mm high is 9.8 m/sec^2 × 13,600 kg/m^3 × 0.001 m = 133 N/m^2 or 133 Pa. The pressure exerted by a column of saline 1 cm high (density 1.04 × 10^3 kg/m^3) is 9.8 × 1,040 × 0.01 = 102 Pa. Because 1 mm Hg = 133 Pa, a normal arterial blood pressure of 120/80 mm Hg is approximately 16,000/10,600 Pa, or 16.0/10.6 kPa. The reaction of a patient to being told that his blood pressure is 16,000/10,600 can only be imagined. It will be some time before the SI system becomes popular among physiologists, clinicians, or patients for the measurement of blood pressure.

Arterial pressure is usually measured as mm Hg, whereas venous pressure is usually measured as cm H_2O or cm saline. The two are readily interconvertible; mm Hg may be converted to cm saline by multiplying by the factor 133/102 = 1.3; cm saline may be converted to mm Hg by multiplying by the factor 102/133 = 0.77. High pressures, such as those in the ventricles of the heart or the arterial system, are measured as mm Hg, whereas low pressures, as in the venous system, the right atrium, and the cerebral ventricles, may be expressed as mm Hg or as cm H_2O, (or as cm blood or cm cerebrospinal fluid).

Occasionally, blood pressures are expressed in torr (after Torricelli). A torr is 1/760 of a "standard atmosphere"; a standard atmosphere is 1.013 × 10^5 Pa; therefore, 1 torr = 133.19 Pa, which is identical, for all practical purposes, to 1 mm Hg.

Measurement of Blood Pressure

U-Tube Manometer

U-tube manometers (Fig. 1.7) are not commonly used to measure blood pressure, but it is helpful to understand their mechanism because the commonly used sphygmomanometer is based on the same principle. A needle, cannula (a blunt, rigid short tube), or catheter (a flexible tube) is placed in the artery or vein of interest. The needle (or cannula or catheter) and the tubing to which it is attached are filled with sterile saline (0.9 g sodium chloride/100 ml water) and the other end of the tubing is attached to one arm of a U-tube partially filled with mercury (for arterial pressures) or saline (for venous pressures). In Fig. 1.7, the pressure at the open meniscus (a) is atmospheric, and the pressure at b, h mm below a, is h mm Hg. This is the usual way of expressing pressures, but pressure could also be shown as 133 h Pa. The arterial blood-saline interface should be at the same horizontal level as the saline-mercury interface. For the measurement of relatively low pressures (e.g., venous blood pressure or cerebrospinal fluid pressure), saline solution instead of mercury is used in the manometer; care should be taken to ensure that the zero of the saline scale is at the same horizontal level as the body zero reference level, usually taken as the level of the right atrium when venous pressures are being measured. All these pressures can be recorded by a kymograph arranged to write directly on a moving strip of paper.

Sphygmomanometer

In the commonly used sphygmomanometer (Figs. 1.8 and 1.9), a direct arterial needle or catheter is not used. Instead, the U-tube manometer is connected, via an air-filled tube, to an inflation cuff. The cuff is wrapped around the arm, over the brachial artery, and inflated to a level above the expected systolic pressure (peak arterial pressure during ventricular contrac-

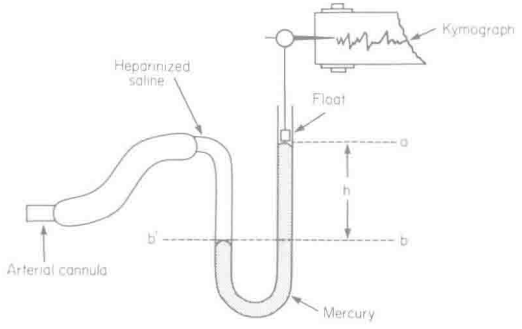

FIG. 1.7. U-tube manometer.

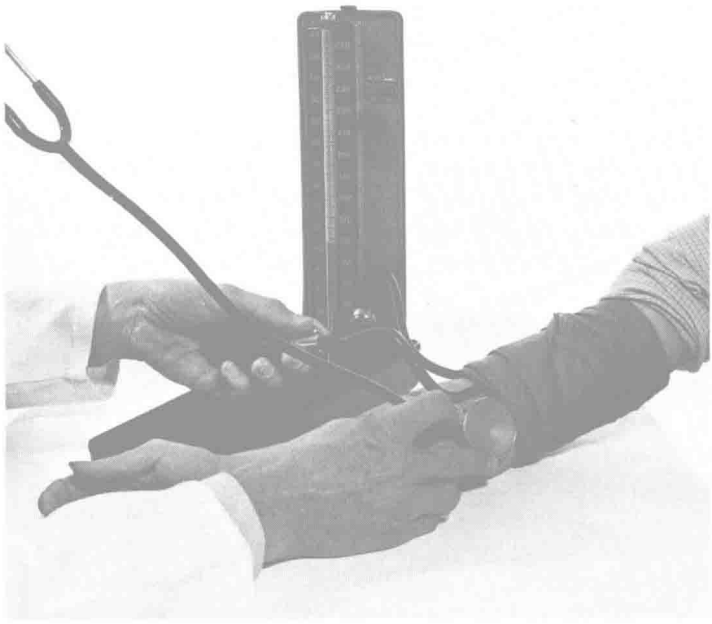

FIG. 1.8. Sphygmomanometer.

tion), thereby occluding the underlying artery and interrupting pulse transmission. A stethoscope is placed over the brachial artery, and the cuff is then slowly deflated at a rate of 2 to 3 mm Hg per beat. Nothing will be heard until the cuff pressure falls just below the systolic arterial pressure. At this point, a spurt of blood will pass through with each heart beat; since the artery is still partially occluded, turbulence is generated, and the arterial wall oscillates,

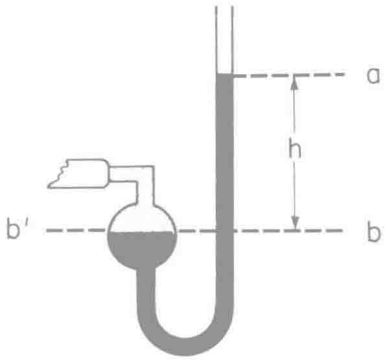

FIG. 1.9. Sphygmomanometer (diagrammatic). The level b–b' is constant; i.e., the reservoir is so large that the level hardly alters as the column is driven up the narrow tube.

producing the characteristic sound changes (Korotkow sounds). The first appearance of the sound at the systolic pressure is phase I. As the cuff pressure is lowered further, the sound assumes a blowing or swishing quality (phase II), then becomes a soft thud (phase III), and suddenly becomes softer and muffled (phase IV). The disappearance of the sound is referred to as Korotkow phase V.

Systolic blood pressure is assigned to the point at which the first sound is heard. There is some controversy as to when to assign the point of diastolic blood pressure (pressure within the arterial system during ventricular relaxation). The point of muffling (phase IV) is favored by some, whereas the point of disappearance of the sound (phase V) is preferred by others. Generally, the point of muffling produces a slight overestimate, and the point of disappearance a slight underestimate, of the true intravascular diastolic pressure. Sometimes, to avoid confusion, the diastolic blood pressure is read at both points. Thus, a normal auscultatory pressure reading would be 120/80 mm Hg if the point of muffling is used, 120/70 mm Hg if the point of disappearance is used, or 120/80/70 mm Hg if both are used. In this case, the true intravascular blood pressure might be about 120/75 mm Hg.

There are several pitfalls to be avoided in measuring blood pressure with a sphygmomanometer:

1. The mercury column in the sphygmomanometer must be at 0 before the cuff is inflated.
2. The right size cuff must be used. The blood pressure is usually measured in the arm with a standard cuff. Blood pressure in the leg can be measured with a cuff around the thigh and the stethoscope over the popliteal artery. Since there is more tissue between the cuff and the underlying artery in the leg than in the arm, more of the cuff pressure is dissipated, and the standard arm cuff gives falsely high pressures from the leg. Also, when brachial arterial pressures are measured in individuals with obese arms, the blanket of fat dissipates some of the cuff pressure, producing a reading that is higher than the true arterial pressure. In both situations, accurate pressure can be obtained by using a cuff that is wider than the standard arm cuff. Conversely, narrower cuffs should be used in very thin or small arms, e.g., in children.
3. The cuff must be level with the heart to obtain a pressure that is uninfluenced by gravity.
4. If the cuff is left inflated for too long, the discomfort felt by the patient may cause generalized reflex vasoconstriction, thus raising the blood pressure.
5. If the cuff is left inflated for some time, reactive hyperemia, i.e., vasodilatation produced when the cuff is released, may produce a spuriously low reading for the diastolic pressure.