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Clinical Management of Shock

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Preface

Coronary artery disease causes about 675,000 deaths per year. Most of these patients die from cardiogenic shock. In 1980 trauma claimed 164,000 lives in the United States and was the third leading cause of death. Furthermore, there are between 100,000 and 300,000 cases of Gram-negative bacteremia per year in the United States. About 20% of these patients develop septic shock, which carries a greater than 50% mortality risk.

It is clear that shock is a common clinical problem. It is important therefore for all physicians to understand the mechanisms of shock and the rationale for effective treatment.

This book starts with a review of the normal circulatory system and its control mechanisms. This background information is necessary so that the pathophysiologic alterations in shock will be understood. An introductory chapter on shock is next, followed by separate chapters on hemorrhagic (trauma), cardiogenic, septic, and neurogenic shock. Finally, a chapter on multiple organ system failure is included.

This book is intended for generalists. In particular, primary care physicians, emergency room physicians, internists, junior surgeons, house staff, and medical students should find it worthwhile reading. For the intended audience this book should bridge the gap between the pathogenesis of shock and its appropriate clinical management.

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Notice

The author and the publisher have exercised great care to ensure that the drug dosages, formulas, and other information presented in this book are accurate and in accord with the professional standards in effect at the time of publication. Readers are, however, advised to always check the manufacturer's product information sheet that is packaged with the respective products to be fully informed of changes in recommended dosages, contraindications, and the like before prescribing or administering any drug.

Contents

1 The Normal Cardiovascular System and Its Control

Overview

This chapter focuses on the normal cardiovascular system and its control mechanisms. The heart will be discussed first. Consideration will be given to the mechanisms of cardiac muscle contraction, hemodynamics, myocardial energetics, and coronary blood flow. The vascular system will then be discussed. This will include a description of the different types of blood vessels, the determinants of vascular control, and the mechanisms for control of blood flow. Cellular energetics, oxygen consumption and delivery, blood volume, and blood pressure will be discussed. Finally, an integration of the cardiac system and the vascular system will be presented so that overall circulatory function, including homeostatic mechanisms, may be understood. This is an important prerequisite in the understanding of the pathophysiology of shock.

Introduction

Shock is a common clinical problem, particularly nowadays. With improvements in paramedic and emergency medical services many acutely ill patients and victims of trauma are being successfully treated at the scene and transported alive to the hospital. Trauma and heart disease are common causes of shock. In 1980 trauma caused over 160,000 deaths in the United States. It is the third leading cause of death. Coronary heart disease causes 675,000 deaths per year and is the leading cause of death.

Trauma and cardiac disease are common causes of shock.

It is important for all physicians to understand the pathophysiology of shock and the rationale behind proper treatment. The major pathophysiologic event in shock is diminished tissue perfusion. It is this deficit in tissue perfusion and not hypotension per se that is the essence of shock. Shock causes

cellular metabolic failure, organ dysfunction, and death. To adequately treat the patient in shock the inciting event must be controlled and tissue perfusion must be improved.

Shock results from circulatory failure.

Shock is the clinical manifestation of a failing circulation. The normal circulatory system should be viewed as a closed loop consisting of three interdependent components (Figure 1.1): 1) the heart (the pump), 2) the vessels (the conduits), and 3) the blood volume (the intravascular volume). Shock may be caused by a defect in any or all of these components. The target organ is the capillary bed, across which vital gases, metabolic nutrients, and waste products exchange.

One must have knowledge of the normal circulation and its control in order to understand the homeostatic mechanisms of the cardiovascular system during shock. This involves an integration of the cardiac system with the vascular system.

The Heart

Cardiac muscle is composed of myocardial cells or fibers about 40–100 μm (micrometers) long and 10–20 μm in diameter (Figure 1.2). These cells are composed of parallel arrangements of myofibrils, which are in turn made up of numerous serially repeating units called sarcomeres. Sarcomeres are the basic contractile units of muscle and are composed of the contractile proteins actin and myosin. Sarcomeres occupy about 50% of the mass of the cardiac muscle cell and about 90% of the mass of the skeletal muscle cell. Mitochondria, the intracellular organelles responsible for generating the energy substrate required for protein contraction, occupy spaces between the myofibrils and account for 20–30% of the cardiac cell mass.

Sarcomeres are the basic contractile units of the myocardial cell.

Myosin is composed of light and heavy meromyosin. The light meromyosin forms the linear backbone of the total myosin protein molecule, whereas the heavy meromyosin protrudes away from the light meromyosin. The globular head of heavy meromyosin forms cross-links or bridges with actin

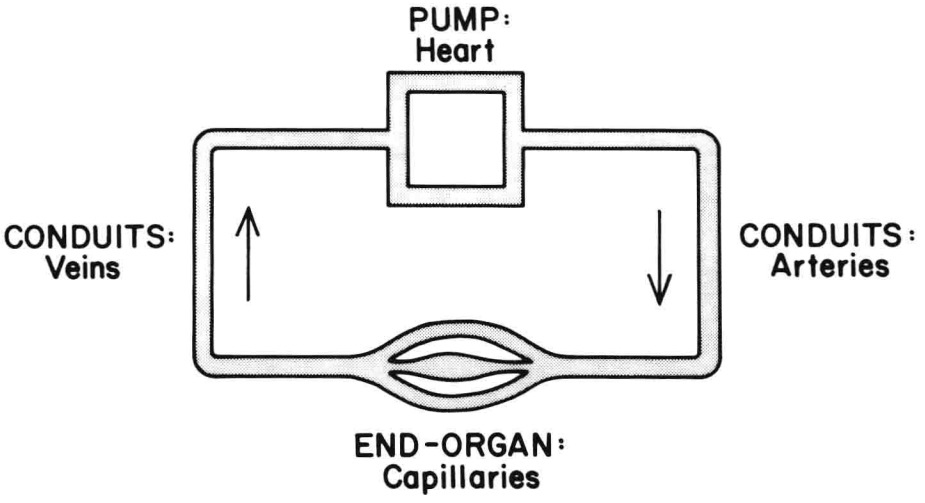

FIGURE 1.1 The circulatory system can be viewed as a closed loop made up of three parts: 1) the heart (the pump), 2) the blood vessels (the conduits), and 3) the blood volume (the intravascular volume).

during muscle contraction. Two additional proteins, tropomyosin and troponin, are associated with actin and are necessary for normal contraction. Troponin normally inhibits the interaction of actin and myosin.

Depolarization of the muscle cell membrane initiates the contraction process. When the muscle cell membrane is depolarized, calcium is released by the sarcoplasmic reticulum. The sarcoplasmic reticulum is a membranous tubular network which intimately surrounds the myofibrils. The calcium released by this system during depolarization binds to the troponin protein and removes troponin's inhibitory function. Actin and myosin can then form cross-bridges. These cross-bridges can be viewed as hinges, so that the actin molecule can slide on the myosin molecule within the sarcomere unit. The length of the sarcomere diminishes and the myocardial cell contracts. The energy necessary for this process comes from the hydrolysis of adenosine triphosphate (ATP) by the enzyme adenosine triphosphatase (ATPase), which is associated with the myosin molecule.

The energy necessary for muscle contraction comes from ATP.

The actin and myosin proteins overlap each

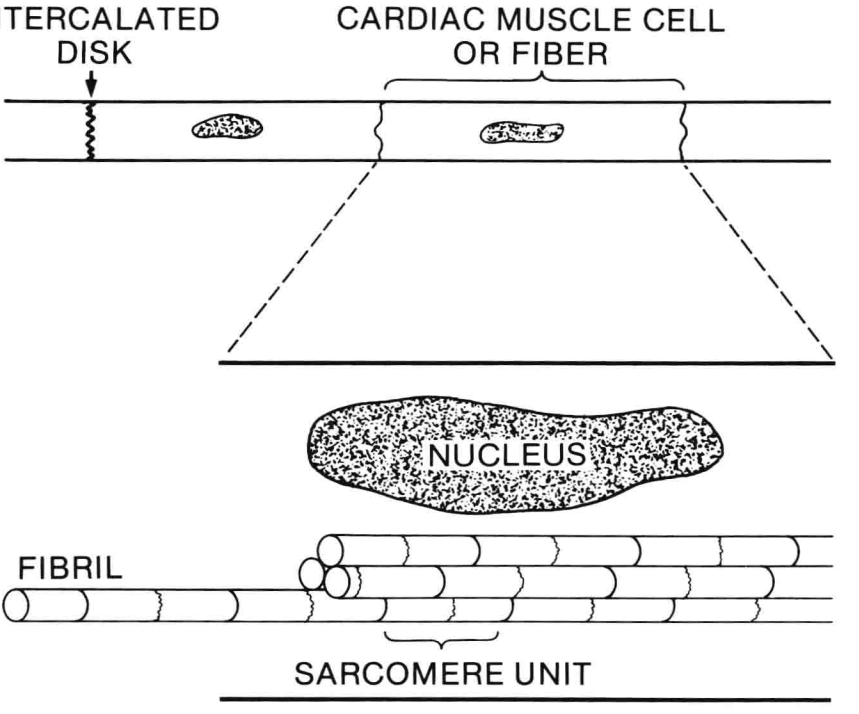

FIGURE 1.2 Sarcomeres are the basic contractile units of the myocardial cell. They occupy about 50% of the mass of the cardiac muscle cell. Adapted from Braunwald E, Ross J Jr, Sonnenblick EH. Mechanisms of Contraction of the Normal and Failing Heart. Little, Brown, Boston, 1976, p 3.

Sarcomere length is related to ventricular volume and pressure.

other within the sarcomere units so that the number of potential coupling sites between the two proteins varies with sarcomere length. The optimal sarcomere length required for maximal muscle tension development is 2.2 μm. In the heart, sarcomere length is related to ventricular volume and pressure. When the left ventricle is empty of blood volume, the sarcomere length is about 1.9 μm. By steadily increasing left ventricular filling volume and, subsequently, left ventricular filling pressure, the sarcomere length can be stretched so that the maximal number of potential coupling sites between the actin and myosin molecules is attained. The ideal sarcomere length of 2.2 μm is attained when the left ventricular diastolic

pressure reaches 12–15 mm Hg and the right ventricular diastolic pressure reaches 8–10 mm Hg.

Cardiac Hemodynamics

The primary function of the heart is to effect adequate perfusion of the capillaries. The physiologic expression of this effected blood flow is called the cardiac output. It is the quantity of blood moved per minute by the heart from the venous system to the arterial system. In the average supine adult it measures about 5.6 liters/min or 3.0 ± 0.5 liters/min/m^2 of body surface area (cardiac index). Cardiac output can vary normally from a value during standing position that is 25% below the output during supine position, up to a value during severe exercise that is 8 times the normal resting supine value.

Cardiac output = 5.6 liters/min
Cardiac index = 3.0 ± 0.5 liters/min/m^2

Cardiac output (CO) equals the product of heart rate (HR) times stroke volume (SV):

$$CO = HR \times SV$$

Cardiac output equals stroke volume times heart rate:
$CO = HR \times SV$

Stroke volume is the volume of blood ejected by the heart per cardiac contraction. It is measured by subtracting the volume of blood in the ventricle at the end of systole from the volume of blood in the ventricle at the end of diastole. In the average supine resting adult stroke volume measures about 70 ml or 40 ± 7 ml/m^2 of body surface area (stroke index). The fraction of ventricular end-diastolic blood volume ejected per contraction, called the ejection fraction, is at least 50% in the normal resting adult.

Stroke volume = 70 ml
Stroke index = 40 ± 7 ml/m^2

If the stroke volume is held constant, then cardiac output varies as a linear function of heart rate. Although the sinoatrial node receives sympathetic and parasympathetic innervation via the cardiac and vagus nerves, respectively, under usual physiologic conditions heart rate is determined by the intrinsic rhythmicity of the sinoatrial node pacemaker cells. This intrinsic regulation is a function of the metabolic rate of the sinoatrial node itself.

Varying the heart rate per se usually does not alter cardiac output significantly due to concomitant changes in stroke volume. Those conditions that usually cause a sinus tachycardia (fear, exercise, etc.) also usually cause an activation of the entire cardio-

vascular system such that stroke volume is increased as well.

Cardiac output is regulated mainly by stroke volume. Stroke volume depends on cardiac preload, afterload, and contractility.

The regulation of cardiac output and cardiac performance is, therefore, mainly achieved via regulation of stroke volume. The magnitude of stroke volume is determined by the degree of myocardial cell shortening during contraction. This is controlled by three factors: 1) the preload, 2) the afterload, and 3) the contractile state or contractility of the myocardium.

Preload

Preload is the amount of stretch put on the cardiac muscle cell prior to contraction. This stretch will determine the degree of shortening of the cardiac muscle during contraction and the strength of muscle contraction. Increasing the preload up to the optimal sarcomere length of 2.2 μm will increase the

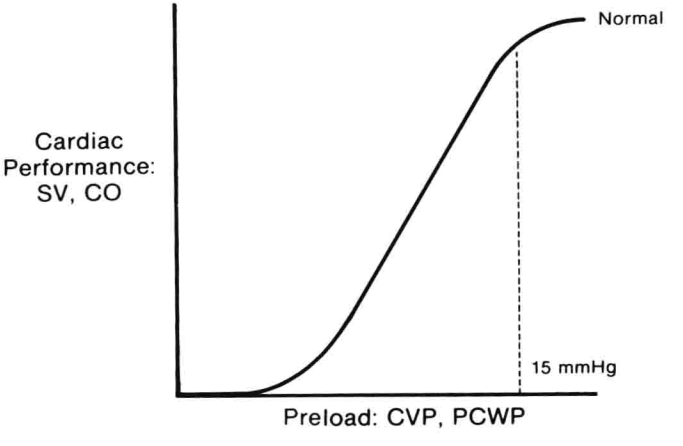

FIGURE 1.3 The ventricular function curve is a graphic way to portray cardiac performance as a function of preload. Preload and cardiac performance reach a plateau stage at a PCWP of 15 mm Hg. Numerous factors can affect preload, including blood volume, body position, intrathoracic and intrapericardial pressure, venous tone, atrial contraction, and the pumping action of skeletal muscle. Reprinted with permission from Kreis DJ Jr. Shock (Part I): Pathophysiology. Curr Rev Respir Ther 6:60, 1983.

stroke volume. Beyond this level of stretch the potential coupling sites between actin and myosin decrease so that there is less shortening and less contraction strength. The relationship between preload (measured as diastolic muscle length) and strength of contraction (measured as systolic muscle tension) is known as the Frank-Starling relationship or Starling's law of the heart.

The Frank-Starling relationship relates diastolic length to systolic tension.

Preload is usually expressed as the pressure in the ventricle at the end of diastole, or the end-diastolic filling pressure. This pressure is a direct function of the volume of blood in the ventricle at the end of diastole, or the end-diastolic filling volume. Preload is thus proportional to the end-diastolic filling volume. Normally, an increase in end-diastolic filling volume is accompanied by an increase in end-diastolic filling pressure. Since the average resting stroke volume in the supine adult is about 70 ml, about 70 ml of venous blood must be delivered to the heart prior to each contraction. If cardiac output is to be maintained at a specific level, then the amount of venous return must equal the amount of stroke volume per contraction.

To maintain cardiac output, venous return must equal the amount of stroke volume per contraction.

Various factors affect the preload. These include body position, blood volume, intrathoracic and intrapericardial pressure, venous tone, pumping action of skeletal muscle, and the efficiency of atrial contraction. As examples, in pericardial tamponade the intrapericardial pressure increases so that the heart cannot distend in order to receive venous return. In tension pneumothorax the elevated positive intrathoracic pressure compresses the great veins of the thorax and inhibits ventricular filling. In atrial fibrillation the contribution of the atria to the ventricular filling is lost.

Factors that influence preload

The Frank-Starling relationship is best expressed as a ventricular function curve relating preload to cardiac performance (Figure 1.3). Clinically, preload of the right ventricle is obtained by measuring the right atrial pressure or the central venous pressure (CVP). The CVP is the pressure measured within the superior vena cava and is normally less than 10 cm H_2O. The preload of the left ventricle, or the left ventricular end-diastolic pressure (LVEDP), is obtained by measuring the mean pulmonary capillary wedge pressure (PCWP) or the pulmonary artery diastolic pressure (PADP) using a Swan-Ganz

The ventricular function curve relates preload to cardiac performance.

Normal CVP is below 10 cm H_2O.

balloon flotation catheter. The mean PCWP is the mean pressure within a small distal branch of one of the pulmonary arteries. It reflects directly the mean pressure within the pulmonary capillary and pulmonary venous systems. It measures 6–12 mm Hg normally. The PADP is the pressure within the pulmonary artery during diastole. It usually is only slightly higher than the mean PCWP, by 1–3 mm Hg. In most cases the PADP can be used to approximate the PCWP. There are some important exceptions, however, as in pulmonary hypertension. A detailed discussion of CVP, PCWP, and PADP and their clinical applications is found in the section on "Patient Monitoring" in Chapter 2. Cardiac performance is measured as either cardiac output or cardiac index, stroke volume or stroke index, or stroke work, which is the product of stroke volume and mean aortic pressure.

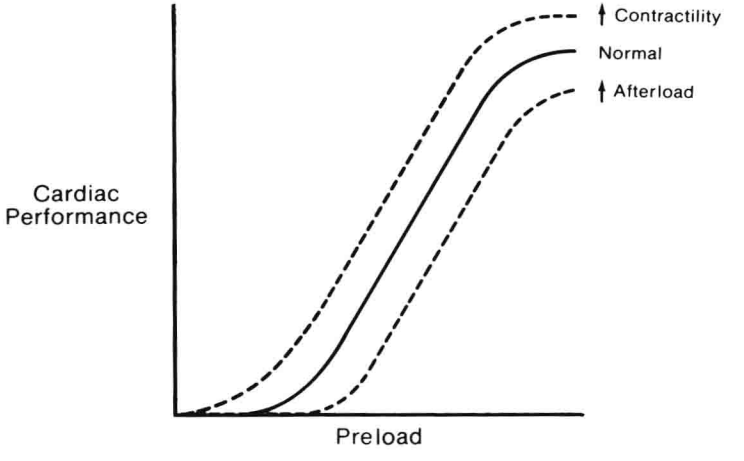

FIGURE 1.4 Changes in afterload and myocardial contractility affect the position of the ventricular function curve. An increase in afterload causes the function curve to shift downward and to the right, so that for each preload there is a decrease in cardiac performance. An increase in myocardial contractility has the opposite effect. Several factors influence the inotropic state of the heart, including autonomic nervous system influences, endogenous catecholamines, and inotropic drugs. Reprinted with permission from Kreis DJ Jr. Shock (Part I): Pathophysiology. Curr Rev Respir Ther 6:60, 1983.