The ICU Book

THIRD EDITION

Paul L. Marino

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

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.

Contents

Section I

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 *preload*, 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

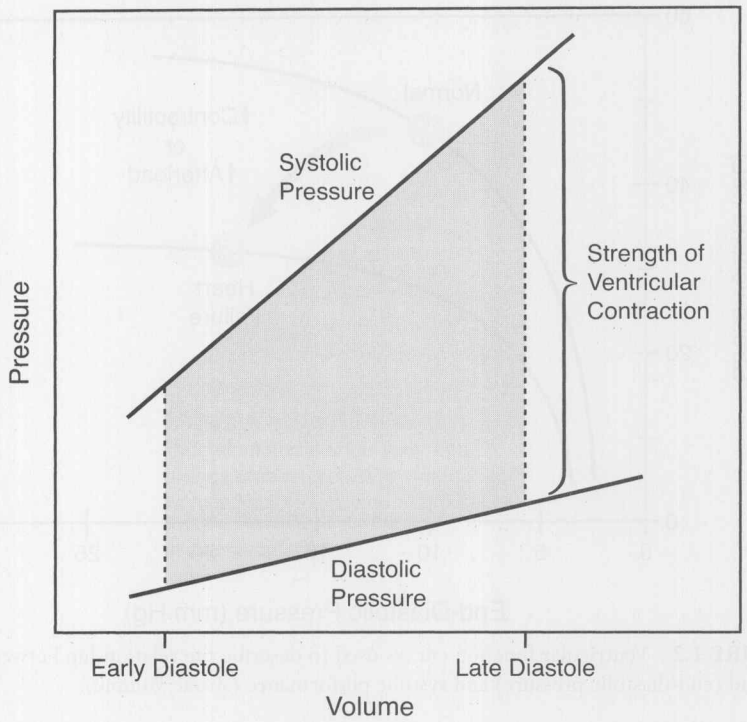

FIGURE 1.1 Pressure-volume curves showing the influence of diastolic volume on the strength of ventricular contraction.

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.

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.