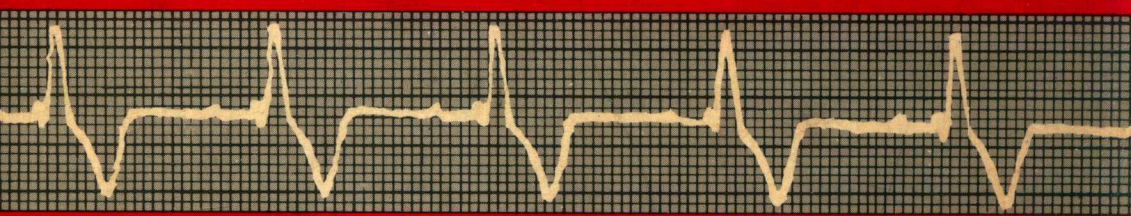
MANUAL of CARDIAC ANESTHESIA

EDITED BY

Stephen J. Thomas



CHURCHILL LIVINGSTONE

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PREFACE

I. Why another book about cardiac anesthesia? Given the ever-increasing deluge of articles, reviews, and books on this subject, it is incumbent upon any author or editor to justify adding even one more title.

II. This book is different from other currently available treatises about anesthesia in patients with heart disease both in format and scope.

   A. Format. This is a manual. Pertinent physiologic, pharmacologic, and anesthetic information is presented briefly and succinctly in outline form. Tables and diagrams have been used whenever possible. This should aid the first-time reader in organizing and understanding the material as well as facilitating future reference, since specific topics can be reviewed quickly and conveniently. I had originally hoped that an outline would also help control the total length of the book. Whether due to the loquaciousness of the authors, insufficient pruning by the editor, or the inherent nature of the information, the book is a bit longer than projected. Nonetheless, most chapters can be easily perused at one sitting and the book in its entirety can be read and studied during a two month cardiac rotation.

   B. Scope of information. My coauthors and I emphasize the pathophysiologic profiles of a wide variety of cardiac lesions. This knowledge is crucial for rational anesthetic planning, which centers on selection of specific drug(s) and techniques that will produce and/or maintain hemodynamic conditions most appropriate for the patients's specific disease. Stated somewhat differently, we first describe the "why," then follow with a discussion of "how" and "when." We also detail the use of nonanesthetic drugs and techniques (vasodilators, inotropes, antiarrhythmics, pacing), which form an integral part of cardiovascular therapeutics. These principles are relevant to specific cardiac lesions and are applicable to all types of surgery.

      1. The text is not meant to be all inclusive. Topics with a very limited audience (e.g., cardiac transplantation, left heart assist devices) are omitted.
      2. The virtues of multi-authorship exact an inevitable price in terms of repetition. Although every effort has been made to minimize this effect, some retracing of previously covered ground was accepted so that each chapter could stand independently.
      3. Reference lists are current but vary considerably in length depending on the author's preferences.

   C. Intended audience

      1. Anesthesia residents—especially those rotating on cardiac anesthesia.

2. Medical students who desire a brief and current review of cardiac pathophysiology, pharmacology, and anesthetic management of specific cardiac diseases.
3. Anesthesiologists or nurse anesthetists who are infrequently confronted with patients with heart disease. These practitioners might readily appreciate a reference source to refresh their basic knowledge when such cardiac patients require anesthesia and surgery.
4. This is not intended as a comprehensive text on cardiac anesthesia for specialists in the field.

III. The material is presented in 17 chapters which can be grouped into five subject areas.
   A. Chapters 1 through 5 describe how the heart works, how to measure and monitor that work, and how to alter cardiac function—pharmacologically, electrically, and anesthetically. Treatment of low cardiac output and cardiac arrhythmias is also discussed.
   B. Chapter 6 covers the preoperative evaluation of the adult with cardiac disease, emphasizing assessment of risk.
   C. Chapters 7 through 11 characterize specific cardiac lesions—hypertensive, ischemic, valvular, and congenital, as well as some of the less common heart diseases.
   D. Chapter 12 describes anesthetic management of the cardiac patient undergoing vascular surgery, while Chapters 13 through 16 discuss topics relevant to open heart surgery (how the pump works, how to manage the patient for cardiopulmonary bypass, how to stop the bleeding and how best to keep the heart alive while the surgeon is fixing it).
   E. Chapter 17 deals with postoperative care.

IV. Thanks, appreciation, kudos, and the like are due:
   A. My coauthors, who gave generously of their expertise, time, equanimity, and patience.
   B. My associates in cardiac anesthesia at New York University, who provided insights about how to manage cardiac patients, gave me the time needed to edit, and were always willing and eager to edit the editor.
   C. Herman Turndorf, Chairman of the Anesthesiology Department at New York University, for continuous encouragement and support.
   D. My cardiac surgical colleagues at NYU for providing the perspective from their side of the ether screen and who, nights and weekends nothwithstanding, work ceaselessly to provide new cases to refine and hone the techniques herein described.
   E. Mary Helen Purcell and Lauren Eichelbaum, who managed to keep both me and the book organized and Meryl Blocker, Sisi Alailima, and Patrice Heinz for help with the manuscript.
   F. The publisher Lew Reines, who originated the idea for this book

and who alternately cajoled, coaxed, pleaded, and finally ordered it to completion. A promise of dinner at Lutece was the consummate and ultimately successful bribe. And to Peggy Brigg, who graciously put up with innumerable indecipherable scribbles and last-minute corrections.

G. Finally, and most importantly, to Ellen, Kira, and Craig for accepting this book as a house guest rather than as an unwanted and uninvited intruder.

V. Dedicated to the memory of my father, Lawrence J. Thomas.

Stephen J. Thomas

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1

Cardiac Pump Function and How to Monitor It

Paul G. Barash
Charles J. Kopriva

INTRODUCTION

In the perioperative period, cardiovascular performance can be evaluated using both invasive and noninvasive methods. Each method can supply important information required for clinical decision making. A thorough knowledge of both the advantages and limitations of each of these measurements is essential. This chapter will focus on the physiologic basis for evaluating cardiac performance and the clinical application of commonly employed indices to assess left ventricular function. Cardiac performance can be described in terms of *myocardial mechanics* (muscle function) and *pump function*.

I. Myocardial mechanics

By studying the response of the isolated papillary muscle preparation, it is possible to simplify the complex muscle function of the heart. Four variables are employed to experimentally evaluate mechanical function: (1) exerted *force* (tension), (2) *velocity* of shortening (rate), (3) instantaneous *length*, (4) *time* after activation.

For each experiment two curves can be generated. One describes the relationship between length (shortening) and time, the other between tension (force) and time.

A. Experimental model

1. Preload

One end of a papillary muscle is attached to a tension transducer and the other end to a lever (Fig. 1.1). Suspended from the opposite end of the lever is a weight which stretches the muscle to the desired length. This stretching force is the *preload*. For a given muscle, an increase in preload (initial fiber length) results in:

a) Increased resting tension
b) An increase in the initial rate of rise of tension (velocity)
c) An increase in peak tension
d) No change in time to peak tension.

2. Afterload

Once the desired length is reached a stop is placed on the lever. When additional weight, *afterload*, is then added to pre-

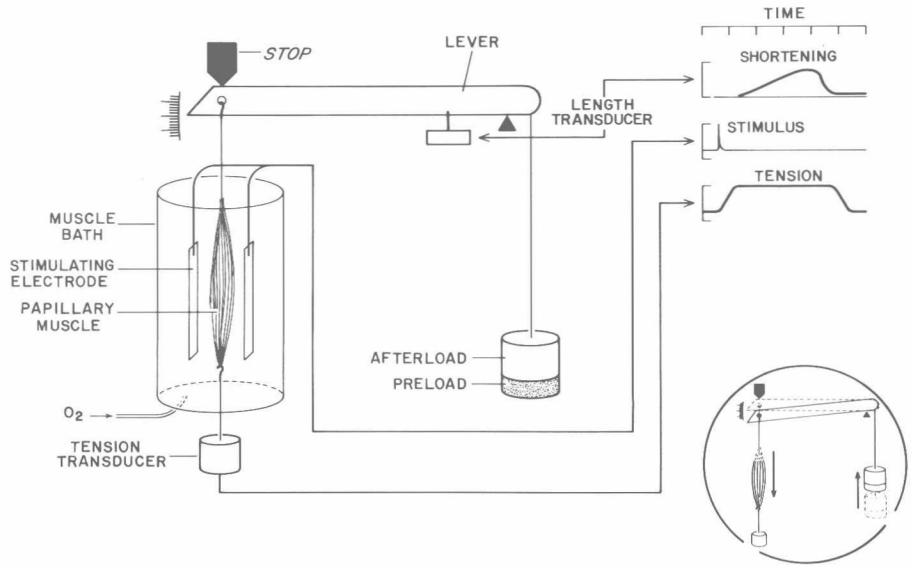

Fig. 1.1. A schematic drawing of a typical isolated papillary muscle experiment. Due to the stop, the afterload is only "seen" by the muscle after the start of contraction (inset).

load, the stop prevents further stretch of the muscle (Fig. 1.1, inset). Thus, the afterload will not influence the muscle, until contraction has started. For a given muscle, an increase in afterload results in.

a) Increased active tension
b) Increased time to peak tension
c) Decreased extent of shortening
d) Decreased velocity of shortening

3. Contractile state

There is no one universally accepted definition of *contractility*. Contractility has been defined as the degree of inotropic state independent of changes induced by presystolic fiber length (preload) or muscle loading (afterload) which best reflects the active state of the myocardium. It is a unique *time-independent* relationship among *force, velocity,* and *length.* A change in contractility implies that for a given load and instantaneous length, the velocity of shortening has been altered.

B. Phases of contraction

1. Resting phase

Before stimulation, the muscle is in a "*resting phase,*" and there is a small resting tension the magnitude of which is determined by the preload (Fig. 1.2). Muscle stimulation results in a two-phase response.

2. Isometric contraction

   In the *isometric phase* of contraction (Fig. 1.2). An increase in tension is seen without change in muscle length (iso = same, metric = length).

3. Isotonic contraction

   In the *isotonic phase* (Fig. 1.2), muscle shortening takes

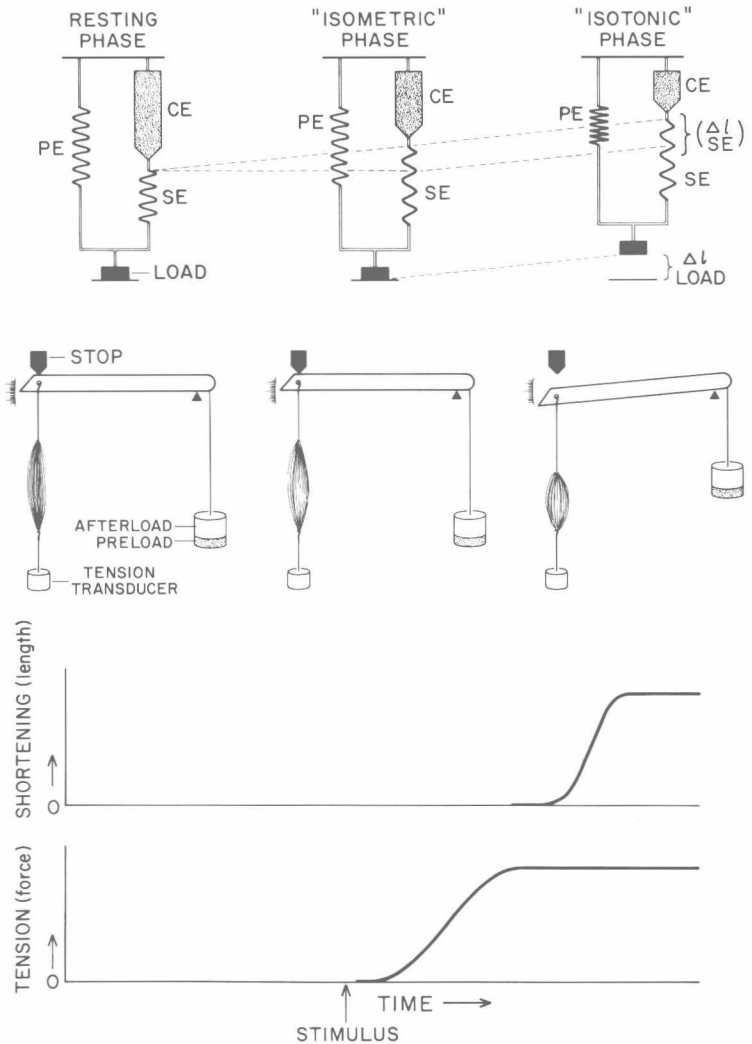

Fig. 1.2. The three phases of cardiac muscle in the isolated papillary muscle preparation. This muscle model, based on the work of A. V. Hill, is composed of three elements. The contractile element (CE) is the major site of conversion of chemical to mechanical energy. The series elastic element (SE) acts like an undamped spring. The parallel elastics element (PE) maintains resting tension and is thought to reside in the connective tissue (see text).

place when the contractile element of the muscle has developed enough tension to exceed the total load (afterload plus preload). After this point is reached, tension remains constant (iso = same, tonic = tension) although shortening continues (Table 1.1).

Extrapolation of the isolated muscle preparation to clinical care represents a double-edged sword. It is obviously very useful in studying the effects of physiologic and pharmacologic interventions on the properties of cardiac muscle. But a price is paid, because these findings may not be parallel to those in the intact heart. Despite the degree of quantification, the study of muscle mechanics has failed to yield an absolute index that will separate normal from abnormal myocardium.

II. Pump function

A. Frank–Starling mechanism

Some of these limitations are solved by the use of both the heart-lung preparation and studies involving the "intact" circulation. The merging of muscle function to pump function of the heart has been credited both to Otto Frank (1895) and to Ernest Starling (1914). Starling viewed the heart as a pump and concentrated on the pressure–volume relationships, employing such displacement terms as *cardiac output, stroke volume,* and *stroke work.*

Starling made several important observations: (1) cardiac output is determined by venous return; (2) stroke volume is determined by venous return (if heart rate is contant); (3) stroke volume is dependent on end-diastolic volume.

From these observations Starling synthesized the "*Law of the Heart*":

> The law of the Heart is therefore the same as that of skeletal muscle namely that the mechanical energy set free on passage from the resting to contracted state depends on the area of 'chemically active surfaces' i.e., on the length of the muscle fibres.

The four major determinants of pump function are:

1. *Preload* or end-diastolic fiber length is best measured clinically by assessing end-diastolic volume (contrast angiography, echo-

TABLE 1.1. EFFECTS OF CHANGES IN LOADING CONDITIONS ON MUSCLE PERFORMANCE

	Increased preload	Increased afterload	Increased contractility	Increased heart rate
Extent of shortening (stroke volume)	Increase	Decrease	Increase	Increase
Velocity of shortening	Increase	Decrease	Increase	Increase
Exerted force (tension)	Increase	Increase	Increase	Increase

cardiography, or radionuclear angiography). Ventricular end-diastolic pressure, mean atrial pressure or mean pulmonary capillary wedge pressure is only an approximation of end-diastolic fiber length. Increases in preload increase:

a) End-diastolic volume
b) Stroke volume
c) Wall tension

2. *Afterload* is the force resisting muscle shortening during contraction. The tension in the left ventricular (LV) wall during systole, as defined by Laplace's Law (T = Pr/2h where T = tension, P = pressure, r = radius, h = wall thickness), is an estimate of the resistance to ventricular ejection. Impedance (ratio of the rate of change of mean arterial pressure to mean aortic flow) may be assessed by systemic vascular resistance (SVR) since 93 percent of impedance to LV ejection is due to SVR. Other correlates of afterload include LV systolic pressure and aortic pressure. Acute increases in afterload result in:
   a) Decreased extent of wall shortening
   b) Decreased velocity of shortening
   c) Increased ventricular end-diastolic volume
   d) Decreased end-systolic volume
   e) Decreased stroke volume
   f) Increased ventricular radius
   g) Increased wall tension
3. *Contractility* is the ability of the myocardium to develop tension from a given end-diastolic fiber length. To document a change of contractility in the intact heart, preload, afterload, and heart rate must remain constant while the intensity of the active state of cardiac muscle is altered.
4. *Heart Rate* is considered the intrinsic rhythmicity of sinoatrial node firing with a coordinated and conducted pattern to the

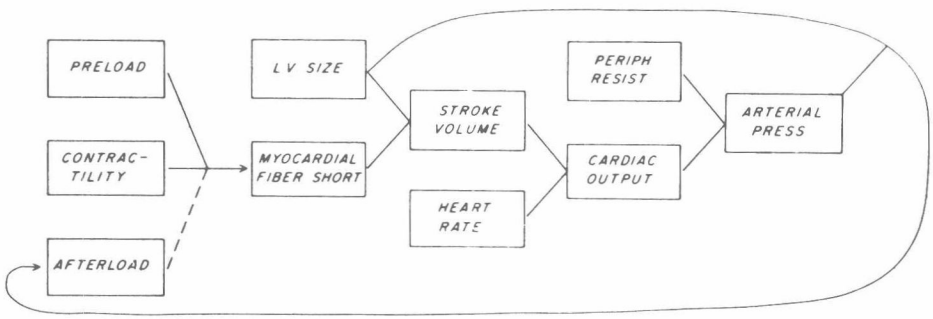

Fig. 1.3. Determinants of cardiac performance. (Reproduced with permission of the New England Journal of Medicine from E Brunwald: Regulation of the circulation. N Engl J Med 290:1124, 1974.)

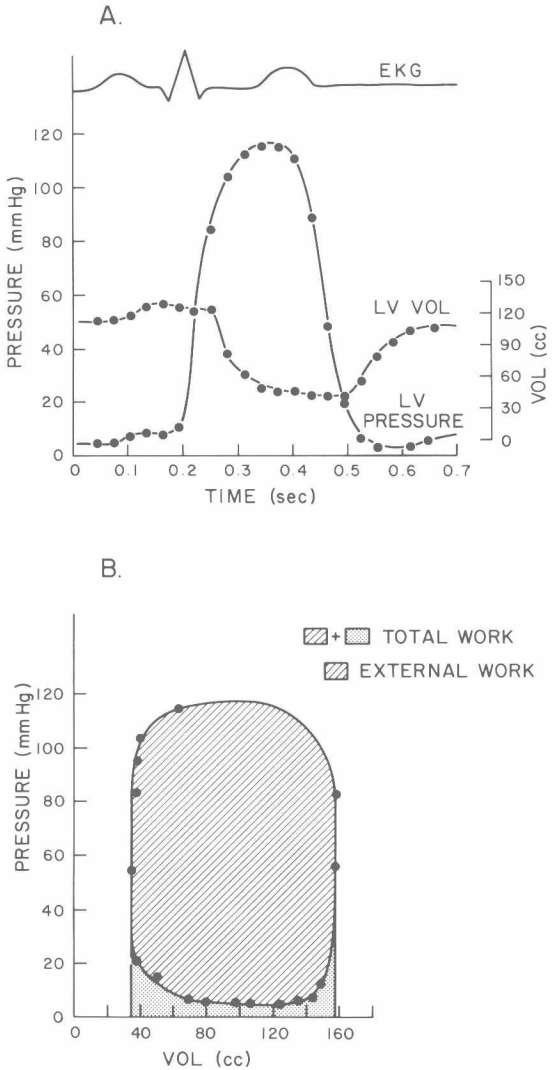

Fig. 1.4. Simultaneous measurements of left ventricular pressure (LV) and volume obtained during cardiac cycle (A) are replotted as a ventricular pressure-volume loop (B).

ventricle. Loss of the "P" wave (nodal rhythm) causes loss of "booster pump" action of atrium. Increase in heart rate impacts upon duration of diastole. Although the actual duration of systole is unaffected, the *relative* duration of systole increases. Thus, myocardial oxygen supply decreases as the demand requirements increase. Stroke volume is maintained constant at heart rate less than 140 beats in normal man.

To summarize, see Fig. 1.3 (block diagram).

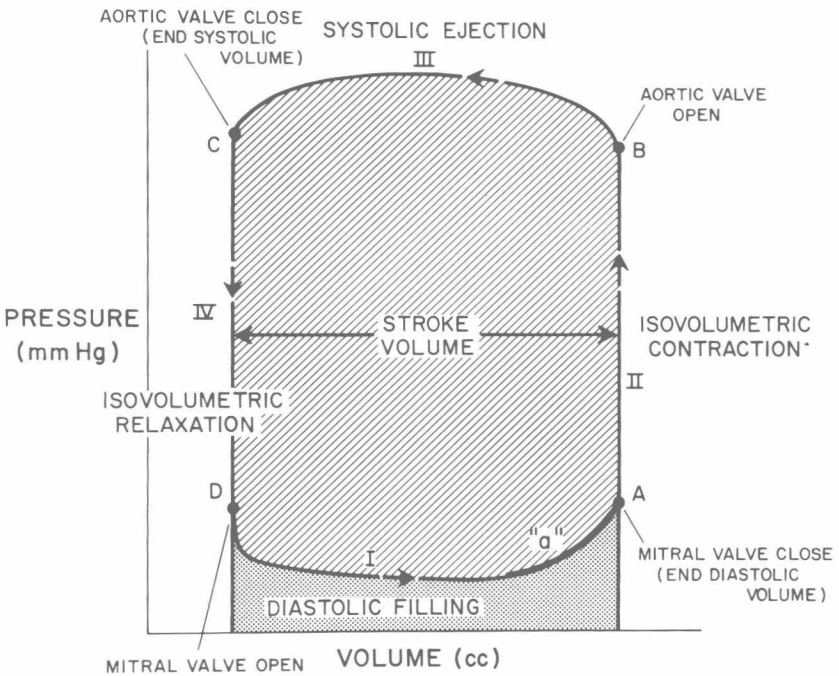

Fig. 1.5. An idealized pressure-volume loop with corresponding events of the cardiac cycle. The stroke volume is the difference between ventricular volume at end-diastole and end-systole.

B. Pressure-volume loop

Analysis of pump function can be simplified by simultaneous measurements of chamber size and pressure obtained during the entire cardiac cycle. This relationship can be plotted as ventricular volume versus ventricular pressure (Fig. 1.4A), or the *pressure-volume loop* (Fig. 1.4B).

1. Phase I: D → A (Fig. 1.5)

   During early and mid-ventricular diastole, filling of the ventricle is passive. In late diastole the atrium contracts (''a'' wave), which results in the final end-diastolic volume (LVEDV) and pressure (LVEDP).

2. Phase II: A → B

   Isovolumic (isometric) contraction results in progressive rise in pressure with little change in volume. This corresponds to isometric contraction of the isolated muscle preparation.

3. Phase III (B → C)

   When intraventricular pressure exceeds aortic pressure the aortic valve opens (B) and ejection begins. Although similar to the isotonic contraction of the isolated muscle preparation, it is