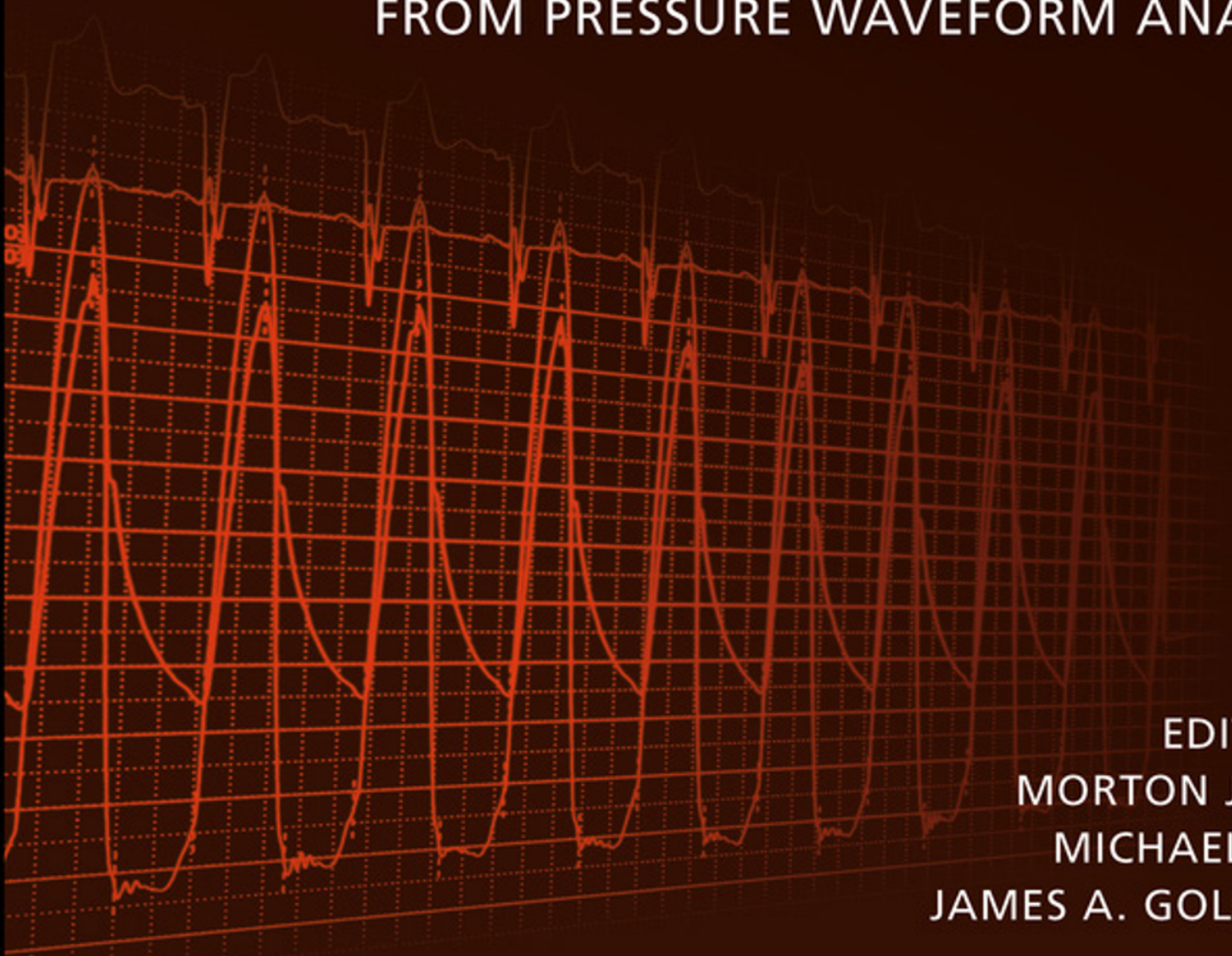


FOURTH EDITION

# HEMODYNAMIC ROUNDS

INTERPRETATION OF CARDIAC PATHOPHYSIOLOGY  
FROM PRESSURE WAVEFORM ANALYSIS



EDITED BY  
MORTON J. KERN  
MICHAEL J. LIM  
JAMES A. GOLDSTEIN

WILEY Blackwell



## Hemodynamic Rounds



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Interpretation of Cardiac Pathophysiology from  
Pressure Waveform Analysis

Fourth Edition

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*JAG—To my wife Cindy, who keeps life fun while I am working.*



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## Preface to the Fourth Edition

Although superseded by modern echocardiography, invasive hemodynamic data continue to be an integral part of cardiology training and comprise validation for much of the pathophysiology obtained from clinical examination, echocardiography, and new imaging modalities. With the advances in imaging technology, the continued reliance on the graphics of hemodynamics has been in decline. However, hemodynamics remain useful for diagnosis and treatment of the multitude of cardiovascular conditions. In the care of the cardiac patient, a critical integration of clinical symptoms, anatomical disorders, and the physiologic underpinnings of these disorders often leads to the best diagnosis and treatment. The understanding of hemodynamic waveforms and the insights provided into the patient's pathophysiology remain the cornerstone for this text.

The first edition of *Hemodynamic Rounds* emphasized the interpretation of hemodynamic waveforms for clinical decision-making as presented from a series of cases published in *Catheterization and Cardiovascular Diagnosis*, now known as *Catheterization and Cardiovascular Interventions*. The case-based format limited itself to description of individual hemodynamic tracings, but was not presented in a formalized textbook fashion. The subsequent two editions of *Hemodynamic Rounds* extended this work, enlarged it, and reorganized it into new sections, providing a more logical approach to the study of pressure waveforms and the associated pathology.

This fourth edition further expands a more thematic approach to the understanding of pathophysiologic waveforms. Since the last edition new procedures such as TAVR (transaortic valve replacement) have provided unique insights into intraprocedural hemodynamics as guides or warning signs of impending complications. The text has been divided into six major parts, logically arranging the previously material and adding new and dynamic tracings, incorporating some of the latest publications on novel hemodynamic topics as they continue to evolve and move into our modern practice.

Part One describes normal and pathophysiologic hemodynamic waveforms and is organized according to the study of pressure wave measurement systems, artifacts, and normal waveforms. The hemodynamics of the tricuspid valve, the mitral valve, and left-sided V waves are reviewed. Left ventricular end diastolic pressure, simultaneous right and left heart pressures, and effects of nitroglycerin and pulsus alternans are also discussed.

Parts Two, Three, and Four cover valvular, constrictive, and restrictive physiology and structural heart disease hemodynamics, respectively. In Part Three, constrictive, restrictive, and tamponade physiologic waveforms are described in detail. Among the topics in valvular heart disease in Part Two, hypertrophic obstructive cardiomyopathy is included in an expanded presentation on the history of the TASH methodology and its outcomes. More uncommon hemodynamics are provided again in Part Five, which covers several topics including coronary and renal hemodynamic assessment along with congenital heart disease, and a unique chapter on left ventricular support devices and "extra" hearts, both transplanted and mechanical. The material on coronary hemodynamics has been expanded given a decade of new studies demonstrating better outcomes using interventions guided by FFR (fractional flow reserve) and the emergence of basal indices to assess coronary stenoses. Of course, after two decades of study and publications, coronary hemodynamics can be used for better decision-making during coronary angiography in daily practice.

The concluding Part Six on clinical-pathophysiologic correlations is dedicated to a discussion of crucial clinical and bedside correlations of hemodynamics, describing the anatomic and pathophysiologic presentations of dyspnea, edema and anasarca, syncope, hypotension, and low cardiac output in four distinct blocks, presenting correlative findings between anatomy, hemodynamics, and clinical manifestations.

It is the hope of the authors that this work will be of lasting value to students, trainees, practicing physicians,

and all related health-care personnel dealing with the important subject of cardiac hemodynamics. We thank Dr. Frank Hildner, first editor and founder of *Catheterization and Cardiovascular Interventions*, formerly *Catheterization and Cardiovascular Diagnosis*, for his encouragement and involvement with this work,

without which this book would never have been published.

*Morton J. Kern, MD*  
*Michael J. Lim, MD*  
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## Introduction

Morton J. Kern and James A. Goldstein<sup>1</sup>

### Historical Review

On February 28, 1733, the president of the Council of the Royal Society, Sir Hans Sloane, requested that Stephen Hales, one of the counselors, present his information on the mechanics of blood circulation from a previous presentation of a series of hemodynamic experiments reported in his book *Haemastaticks* [1]. Hales took his place in medical history next to William Harvey in studies of the human and animal circulation. *De Motu Cordis* [2] and *Haemastaticks* stimulated scientists' interest in the newly developed principles and mathematical computations of fluid mechanics as applied to circulatory physiologic events. The simple measurement of blood pressure now became a subject of great scientific concern.

From such basic interests, experimental physiologists at Oxford University in the 1800s, investigating the physiology of the circulation, began estimating the output of ventricular contraction and velocity of blood flow in the aorta, based on relatively primitive measurements of cardiovascular structures. These data remain valid today and correspond to those currently accepted and obtained by modern quantitative techniques. Cardiologists interested in hemodynamics should continue to emulate Stephen Hales, who relied on direct measurements and observations repeatedly checked and applied on simple but confirmed computations. Hales's numerous original achievements in hemodynamics are remarkable even by today's standards and include the first direct and accurate measurement of blood pressure in different animals (see Figure I.1) under different physiologic conditions such as hemorrhage and respiration; cardiac output estimated by left ventricular systolic stroke volume measured from the diastolic volume after death of the animal; calculations of pressure measured on the internal surface of the left ventricular at the beginning of systole; and

determination of blood flow velocity in the aorta approximating 0.5 m/sec. Hales introduced the concept of the wind castle or capacitance effect in the transformation of pulsatile flow in large vessels to continuous flow in smaller vessels. He also made the first direct measurement of venous blood pressure and correct interpretation of venous return on cardiac output in relation to contraction and respiration. Since recording equipment documenting Hales's observations was unavailable, understanding the unique collection of data depends on interpreting descriptive material.

Our current appreciation of hemodynamics, hopefully enhanced in this book, comes to us from a small group of modern physiologists active in the 1920s, among whom Dr. Carl Wiggers, from Western Reserve University in Ohio, emerges as a major figure. Advances in hemodynamic research arose from the development of recording instruments with fidelity, able to capture and reproduce the waveforms of rapidly changing pressures during the various phases of cardiac contractions. Importantly, Dr. Wiggers and colleagues also employed the newly developed electrocardiogram to obtain simultaneous pressure waveforms and electrical activity and, thus, establish the fundamental electrical-mechanical intervals. These relationships are the benchmark against which the observations of the pressure tracings of classical diseased conditions can be compared [4]. Almost a hundred years separate Wiggers and other originators of clinical cardiovascular physiology from today's cardiologists.

From the time Claude Bernard (1840) coined the phrase "cardiac catheterization" [5], laboratories of that type and name had been examining human physiology, ultimately incorporating radiologically determined anatomic information during the development of cardiac angiography in the 1960s. In 1929, Werner Forssman performed the first documented human

<sup>1</sup> With special acknowledgments to Frank Hildner, MD for his contribution.



**Figure I.1** Drawing depicting Dr. Stephen Hales (seated) directing and observing the measurement of arterial pressure in a sedated horse circa 1730. *Source:* Lyon 1987 [3]. Reproduced with permission of John Wiley & Sons.

cardiac catheterization—on himself [6]—changing the nature of the work from exclusively animal to human subjects. In the late 1930s, Cournand and Ranges [7] used the new right-heart catheterization technique to investigate pulmonary physiology. World War II expanded the scope and direction of their work to include hemorrhagic shock and drug effects on the circulation. However, in those days the most serious problems patients presented related to congenital and rheumatic heart disease. Accordingly, laboratories around the world began publishing data on the hemodynamics and physiology of atrial septal defects [8], ventricular septal defects [9], stenotic and insufficient mitral and aortic valves, and ventricular function. The beginning of invasive cardiology had now evolved into a distinct field of study that would produce valuable diagnostic and therapeutic results.

Without doubt, the most crucial development needed for the advancement of the field was the cathode ray tube, a direct result of the war. Before the image intensifier [10, 11], cardiac fluoroscopy utilized high-dose radiation and required physicians to accommodate their eyes to a green fluorescent screen by wearing red goggles for 15–20 minutes before starting. Indeed, the faintly glowing image in a completely dark room frequently failed to

reveal even the position of the catheter [12]. Without the additional light provided by the image intensifier, “angiocardiology” was nothing more than a simple flat-plate radiograph, or perhaps a sequence of cut films obtained on the newly developed serial film changer [13]. Cineangiography was developed in the late 1950s through the persistent efforts of Lanker (1954) [14] and Sones (1958) [15]. Advanced radiographic imaging spurred the development of catheter invasive techniques, permitting the investigation of heretofore unapproachable anatomical sites, clinical conditions, and disease entities. The findings in turn resulted in more effective and novel cardiac surgical techniques.

After the basic mechanics of congenital anomalies and rheumatic abnormalities were confirmed, expanded study was undertaken of conditions related to occlusive coronary artery disease such as myocardial infarction, left ventricular aneurysms, mitral chordal, and septal rupture. The concepts of systolic and diastolic myocardial mechanical function, hypertrophic obstructive and nonobstructive cardiomyopathy, electrophysiologic relations, and other previously unappreciated conditions came under scrutiny. The result was a new body of knowledge leading to the development and use of remarkable noninvasive techniques, including phonocardiographs, ballistocardiographs, exercise stress testing, radionuclide imaging, and echocardiography. While echocardiography and other important imaging techniques have superseded invasive approaches to some diagnoses, the acquisition and interpretation of hemodynamics remain critical to a proper understanding and appreciation of all cardiovascular conditions and situations.

## Approach to Hemodynamic Waveform Interpretation

In the first edition of *Hemodynamic Rounds*, each chapter had been published in the journal *Catheterization and Cardiovascular Diagnosis*, now known as *Catheterization and Cardiovascular Interventions*, in a case-based format. The material was intended to provide both novice and advanced cardiologists with classical and, at times, unique pressure tracings to emphasize the value of careful waveform observation as it relates to different cardiac pathophysiologies. This fourth edition of the book carries this format forward and expands and updates the discussions to those areas where new information has been acquired, such as transaortic valve replacement (TAVR) procedures.

High-quality hemodynamic data are required for accurate hemodynamic determinations. As in the days of Stephen Hales, some hemodynamic data are

extraordinarily simple, such as using a sphygmomanometer for indirect assessment of systemic arterial pressure. Some hemodynamic data are complex, requiring placement of multiple and specialized catheters within various locations and heart chambers to determine valvular gradients, myocardial contraction, relaxation, compliance, impedance, and work [16–18]. Percutaneous coronary and structural heart interventions prompted the development of the study of human coronary hemodynamics, now available easily to all on a daily basis. For coronary stenosis assessment, intracoronary pressure and flow measures are indispensable for accurate diagnosis beyond angiography alone. Intravascular ultrasound and optical coherence tomographic imaging catheters provide unique complementary anatomic information only dreamed about by the founders of our field.

As with all laboratory data, the significance of various hemodynamic findings should be placed in context of the ancillary historical, clinical, echocardiographic, roentgenographic, and electrocardiographic data. Acting on isolated laboratory values is dangerous and continues to be the nemesis of all technical innovations in medicine.

## Methodologies Involved in Hemodynamic Data Collection

Each laboratory, and preferably all physicians, should establish protocols for right- and left-heart catheterization. A uniform and consistent approach to data collection insures complete, accurate, and reliable data for the majority of clinical problems. The standardized routine also obviates easily overlooked data collection steps being missed. Further, time is saved during procedure setup and data recording. The technical staff do not have to rethink what will happen for the unique and personal hemodynamic protocol of each different operator. Right-heart catheterization, sometimes performed sequentially with left-heart catheterization, may often be combined with it simultaneously to provide the most complete data. In most academic laboratories, a combined methodology is preferred.

The methodology for performing right-heart catheterization has been reviewed previously [19], but the indications have changed [19,20]. In general, routine right-heart catheterization is not indicated, but certainly should be liberally employed when patient care demands it. Shanes *et al.* [21] and Barron *et al.* [22], though arriving at opposite opinions, agree that right-heart catheterization is critical to evaluate patients with previous congenital heart disease, valvular heart disease, left- or right-heart failure, cardiomyopathy, or any unexplained significant clinical historical or physical findings.

Left-heart hemodynamic measurements most often use a single pressure transducer which screens for LV–aortic gradients. For accuracy, simultaneous left ventricular (LV) and central aortic pressure can easily be obtained using a dual-lumen pigtail catheter with two transducers. Measurements of cardiac work, calculation of flow resistance, valve areas, and shunt calculations require accurate hemodynamic data, arterial and venous blood oxygen saturations, and cardiac output determinations.

If hemodynamic information is considered important, the operators should take the time to obtain reliable and unequivocal pressure waveforms, separating artifact from true pathology. To achieve this goal, operators must be familiar with the equipment producing the waveforms and the sources of error found in recording techniques, tubing, transducers, and catheters. A complete description of the mechanics, techniques, pitfalls, and errors of hemodynamic data recording is provided in the *Cardiac Catheterization Handbook*, sixth edition, 2015 [23].

## Initiating the Study of Pressure Waveforms

Pressure waveforms may be confusing for both the cardiovascular fellow-in-training and the clinician trying to understand the results of the procedure. After an intense training period in which the components of all pressure waves found in cardiovascular structures are reviewed and discussed, the young physician must be encouraged to continue practicing pattern recognition, deductive analysis, and a systematic approach to understanding the full meaning of the complete pressure data obtained. This systematic waveform interpretation includes consideration of the following key points:

- 1) Identify the cardiac rhythm. Most cardiac events can be identified by their timing from within the R–R cycle. Hemodynamic data obtained during arrhythmias may be confusing, since the various irregular contraction sequences distort pressure waves.
- 2) Determine the pressure scale on which the waveform is recorded and verify the pressure per division to be certain there is no recording artifact. Also, note the recording speed to assess the appropriate cardiac rhythm and timing of events occurring within one cardiac cycle. The comparison of waveforms for the chamber of interest should be made against known waveforms of normal physiology. The type of artifacts due to catheter fling or over- or underdamping will be discussed in the initial chapters.
- 3) Interpret the waveforms in conjunction with the clinical presentation and suspected diseased conditions

of the patient. A large V wave does not always represent valvular regurgitation. The equilibration of right and left ventricular diastolic pressures may be hypovolemia rather than pericardial constriction. Consider alternative clinical and physiologic explanations.

The examination, consideration of possible mechanisms, and clinical interpretation of the various waveform phenomena form the rationale for this book. We hope that this approach will enhance accuracy and lead to the best decisions for your patients' clinical care.

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## Part One

### Normal Waveforms



## Principles of Normal Physiology and Pathophysiology

James A. Goldstein and Morton J. Kern

Pathophysiologic derangements of cardiac anatomic components and mechanics manifest as “cardinal” cardiovascular symptoms, most of which are reflected in distinct hemodynamic disturbances. These symptomatic–hemodynamic constellations include (i) dyspnea, reflecting pulmonary venous congestion; (ii) fatigue, attributable to inadequate cardiac output; (iii) syncope, resulting from transient profound hypotension; and (iv) peripheral edema, related to systemic venous congestion. Chest pain typically suggesting ischemia does not usually result directly from primary hemodynamic derangements, does not lend itself to this anatomic–pathophysiologic hemodynamic approach, and will not be addressed in these discussions.

It is important to emphasize that these symptom groups in isolation are nonspecific. Identical complaints reflecting disparate pathophysiologic processes can occur due to a variety of mechanisms. For example, dyspnea is an expected symptomatic manifestation of pulmonary venous hypertension attributable to a spectrum of left-heart derangements, the underlying mechanisms of which vary greatly (e.g., mitral stenosis, mitral regurgitation, left ventricular cardiomyopathy, etc.). The treatments and prognoses also vary greatly. Dyspnea is also commonly of pulmonary origin, with circumstances in which the heart may be completely normal or impacted only as an innocent bystander (e.g., cor pulmonale).

Similarly, peripheral edema and ascites reflect systemic venous congestion resulting from a spectrum of right-heart failure mechanisms (e.g., tricuspid valve disease, right ventricular cardiomyopathy, pericardial disorders, etc.). However, edema may also develop under conditions with normal systemic venous pressures, as may occur in patients with cirrhotic liver disease, nephrotic syndrome, inferior vena cava (IVC) compression, and so on. Thus, for cardiovascular assessment, symptoms and signs must be characterized according to the underlying anatomic–pathophysiologic mechanisms.

To establish an anatomic–pathophysiologic differential diagnosis, first consider the anatomic cardiac components (myocardium, valves, arteries, pericardium, and conduction tissue) that may be involved and then focus on the fundamental mechanisms that impact each anatomic component, finally asking how such anatomic–pathophysiologic derangements and hemodynamic perturbations are reflected in the symptoms, physical signs, and invasive waveforms.

### Cardiac Mechanical Function and Hemodynamics

Hemodynamic assessment is an integral part of the anatomic–physiologic approach to circulatory pathophysiology, employing bedside examination with confirmatory or complementary invasive and noninvasive (echo-Doppler data) hemodynamic information.

The purpose of the cardiovascular system is to generate cardiac output to perfuse the body. However, although perfusion is the heart’s “bottom line,” perfusion depends on pressure to drive the blood through the tissues. Organ perfusion is determined by arterial driving pressure modulated by vascular bed resistances. The regulation of the circulation (pressure and flow) can be understood by the application of Ohm’s law–related resistance to pressure and flow. In classical physics applied to an electrical circuit, Ohm’s law is expressed as:

$$\Delta V = I \times R$$

where  $\Delta V$  is the driving voltage potential difference across the circuit,  $I$  is the current flow, and  $R$  is the circuit resistance. Thus, circuit output or current flow is a function of the “driving” voltage divided by circuit resistance, or  $I = \Delta V / R$ . Translating Ohm’s law to the cardiac circulation, blood pressure (dV) = cardiac output

(I)  $\times$  systemic vascular resistance (R) and can be applied to the systemic circulation or to individual organ beds.

The key components of blood pressure can be further considered. Thus, cardiac output (CO) = heart rate (HR)  $\times$  stroke volume (SV). Furthermore, SV is a function of three cardiac mechanisms: preload, afterload, and contractility. Systemic vascular resistance is determined by total blood volume and vascular tone (a function of intrinsic vessel contraction or relaxation interacting with systemic and local neuro-hormonal influences, metabolic factors, other vasomotor mediators, etc.).

## Fundamentals of Hemodynamic Waveforms: The Wiggers Diagram

All pressure waves of the cardiac cycle can be understood by reviewing and knowing how electrical and mechanical activity of the heart's contraction and relaxation are related. Every electrical activity is followed normally by a mechanical function (either contraction or relaxation), resulting in a pressure wave. The timing of mechanical events can be obtained by looking at the electrocardiogram (ECG) and corresponding pressure tracing (Figure 1.1) [1].

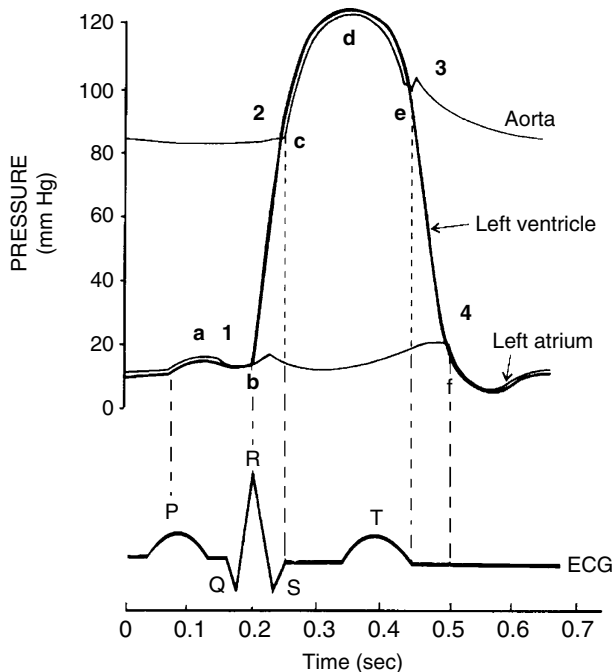
The ECG P wave is responsible for atrial contraction, the QRS for ventricular activation, and the T wave for ventricular relaxation. The periods between electrical activation reflect impulse transmission times to different

areas of the heart. These time delays permit the mechanical functions to be in synchrony and generate efficient cardiac output and pressure. When the normal sequence of contraction and relaxation of the heart muscle is disturbed by arrhythmia, cardiac function is inefficient or ineffective, as demonstrated on the various pressure waveforms associated with the arrhythmia.

The cardiac cycle begins with the P wave. This is the electrical signal for atrial contraction. The atrial pressure wave (A wave; Figure 1.1, #1) follows the P wave by 30–50 msec. Following the A wave peak, the atrium relaxes and pressure falls, generating the X descent (point b). The next event is the depolarization of the ventricles with the QRS (point b). The left ventricular (LV) pressure after the A wave is called the end-diastolic pressure (LVEDP). It can be denoted by a vertical line dropped from the R wave to the intersection of the LV pressure (point b). About 15–30 msec after the QRS, the ventricles contract and the LV (and right ventricular, RV) pressure increases rapidly. This period of rise in LV pressure without change in LV volume is called the isovolumetric contraction period (interval b–c). When LV pressure rises above the pressure in the aorta, the aortic valve opens and blood is ejected into the circulation (point c). This point is the beginning of systole. Some hemodynamicists include isovolumetric contraction as part of systole.

About 200–250 msec after the QRS, at the T wave, repolarization starts and the heart begins relaxing. By the end of the T wave (point e), the LV contraction has ended and LV relaxation produces a fall in the LV (and aortic pressure). When the LV pressure falls below the aortic pressure, the aortic valve closes (point e). Systole concludes and diastole begins. After aortic valve closure, the ventricular pressure continues to fall. When the LV pressure falls below the left atrial (LA) pressure, the mitral valve opens and the LA empties into the LV (point f). The period from aortic valve closure to mitral valve opening is called the isovolumetric relaxation period (interval e–f). Diastole is the period from mitral valve opening to mitral valve closing.

Observing the atrial pressure wave across the cardiac cycle, it should be noted that after the A wave, pressure slowly rises across systole, continuing to increase until the end of systole when the pressure and volume of the LA are nearly maximal, producing a ventricular filling wave (V wave). The V wave (point f, #4) peak is followed by a rapid fall when the mitral valve opens. This V wave pressure descent is labeled the Y descent and usually parallels LV pressure. After the V wave, the LV is filled by the small pressure gradient assisting blood flow from the atria into the ventricles over the diastolic period (called diastasis), until the cycle begins again with atrial pressure building, until again atrial activation and contraction



**Figure 1.1** The Wiggers diagram. Source: Opie 2015 [1]. Reproduced with permission of Elsevier.

generate the A wave, ejecting atrial blood into the LV. The peaks and descents of the atrial pressure waves are changed by pathologic conditions and are used to support the diagnosis of these pathologies, as will be seen in the examples dealing with heart failure, constrictive physiology, and RV infarction.

## Valve Hemodynamics

To appreciate hemodynamic valve dysfunction, consider when cardiac pressure normally opens and closes the valves. The aortic and pulmonary valves open in systole, when ventricular pressure exceeds aortic pressure (and RV exceeds pulmonary artery or PA pressure). Stenosis of these valves produces systolic pressure gradients and characteristic high-velocity heart murmurs. The mitral and tricuspid valves are closed in systole when LV pressure is greater than atrial pressure. A mitral or tricuspid regurgitant valve that fails to close is characterized by a low-velocity systolic murmur with a rumbling quality. Conversely, incompetent aortic valves fail to seal and let blood continue to rush backward into the LV in diastole. The blood rushes into the LV with a diastolic murmur. At the beginning of diastole, LA pressure is at its highest. If the mitral valve is stenotic, the high LA pressure emptying into the LV produces a diastolic rumble. When reviewing the cardiac hemodynamics, we can always refer to the Wiggers diagram for what the expected normal hemodynamic responses should be.

## Systolic and Diastolic Performance

The hemodynamic evaluation of the circulation may be considered as two sides of a single coin of cardiac function: (i) systolic function, the ability of the heart to pump, generate pressure, and perfuse the body; and (ii) diastolic performance, the ability of the chambers to fill at physiologic pressures with the preload necessary to generate SV.

### Systolic Function

Systolic function reflects the ability of the ventricle to contract and generate output or stroke work, a function determined by its loading conditions, including both preload (determined by venous return and end-diastolic volume), afterload (related to aortic impedance and wall stress), and the contractile state (the force generated at any given end-diastolic volume).

The Frank–Starling mechanism established the relationship between end-diastolic volume (preload) and

ventricular performance (stroke volume, cardiac output, and/or stroke work), wherein isovolumetric force at any given contractile state is a function of the degree of end-diastolic fiber stretch (also known as a force–length relationship; Figure 1.2). Thus, the normal LV functions are on the ascending limb of this force–length relationship. Afterload, the impedance during ejection, is defined as the force per unit area acting upon myocardial fibers, a force resulting in wall stress, which is expressed by the Law of Laplace ( $\text{Wall stress} = \text{Radius}/2 \times \text{Thickness}$ ). Afterload is influenced by changes in ventricular volume and wall thickness, as well as aortic pressure or aortic impedance.

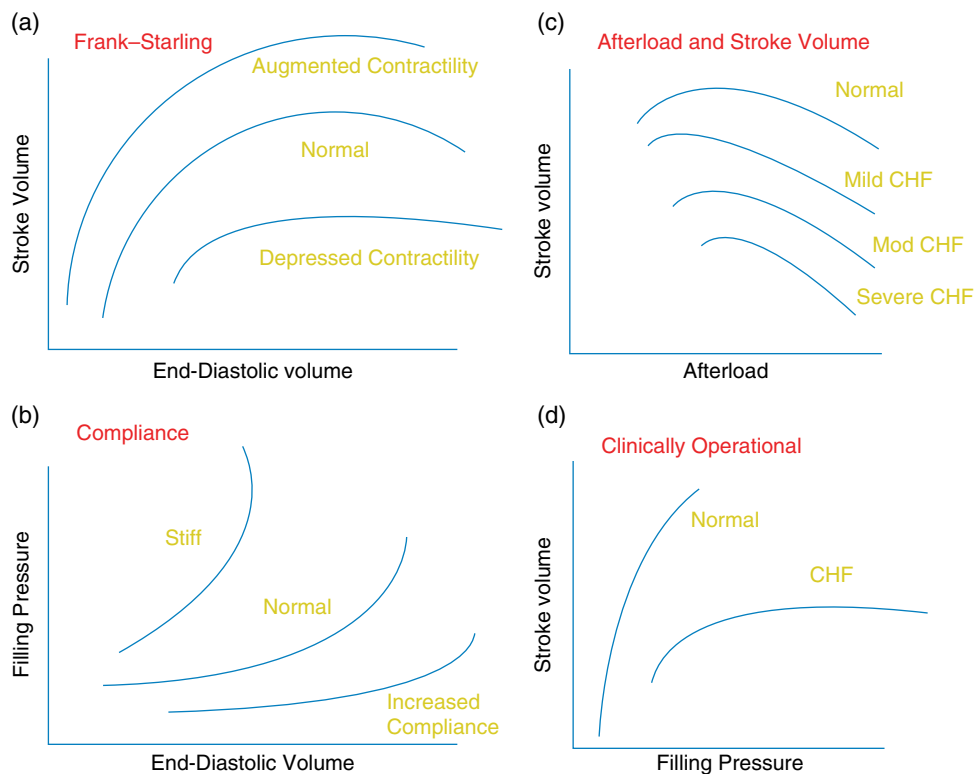
### Frank–Starling and Ventricular Waveforms

Ventricular waveforms reflect both systolic and diastolic function and include the effects of chamber preload, contractility, and afterload. The upstroke of RV or LV pressure (+  $dP/dt$ ) is influenced by preload and contractility, but is a poor measure of either. A brisk upstroke suggests reasonable function versus a sluggish or delayed pressure rise of depressed performance. The peak amplitude reflects both contractility and afterload.

In diastole, ventricular relaxation ( $-dP/dt$ ) is an active energy-requiring process and reflects intrinsic aspects of myocardial contractility as the ventricle actively “relaxes.” The pressure wave of the downstroke relaxation phase is an active process requiring adenosine triphosphate (ATP) and closely mirrors systolic function. The pressure downstroke can also be used to assess cardiac dysfunction. A slurred or retarded negative  $dP/dt$  (also known as tau, a LV relaxation measurement) may indicate cardiomyopathy and adversely influenced diastolic properties.

### Arterial Waveforms

Arterial waveforms reflect the ejection of blood from the LV (and therefore its preload, contractility, and afterload), together with the intrinsic resistance and compliance of the pulmonary or systemic circuit. Filling pressures in the ventricles reflect diastolic properties, influenced by intrinsic chamber factors (e.g., pressure overload hypertrophy, volume overload, ischemia, infiltration, inflammation), as well as extrinsic effects from the pericardium or contralateral ventricle through diastolic ventricular interactions. The arterial waveform reflects dynamic interactions between SV and the capacitance (distensibility) of the peripheral arterial tree (which determines the rate at which the ejected volume of blood flows from the proximal arterial compartment into the peripheral tissues). The first peak of the arterial pressure waveform is the percussion wave, which reflects



CHF = congestive heart failure.

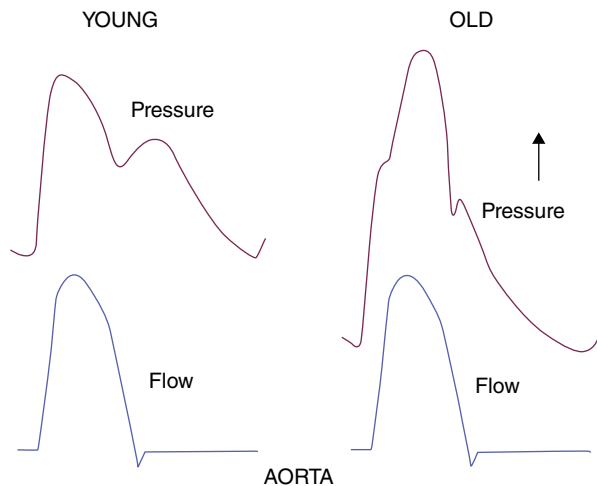
**Figure 1.2** Family of cardiac function curves illustrating and combining principles of systolic function, diastolic compliance, effects of excess afterload, and a symphysis pertinent to clinical day-to-day life. (a) Classic Frank–Starling relationship of true end-diastolic volume output. (b) Compliance curves illustrating rapid rise in pressure attributable to impaired ventricular compliance. (c) Adverse effects of increasing afterload on ventricular stroke volume, particularly deleterious under conditions of depressed LV systolic function. (d) “Clinically operational” graph illustrating hemodynamic impact of mechanical abnormalities on patients who normally generate optimal stroke volume with low filling pressures, compared to dysfunctional ventricles (from both systolic and diastolic abnormalities) in which despite high filling pressures stroke volume is low.

the impulse of the LV stroke modulated by the reflected pressure from the vascular tree (and therefore its compliance); therefore, the arterial upstroke to its peak reflects LV preload, contractility, and afterload (both that imposed by the aortic valve and the stroke volume ratio or SVR). A secondary tidal wave follows, reflecting primarily the returning pulse wave from the upper body (peripheral tone), which then smoothly falls to the dichrotic notch (incisura) which corresponds to aortic valve closure. The subsequent decline in aortic pressure represents pure diastolic runoff. In early diastole, a small positive wave may be seen, the dichrotic wave, most likely an effect of reflected pulse from the lower body.

### Pulse Amplification

As the pulse wave travels distally through the arterial circulation, the waveform may increase, a phenomenon termed peripheral amplification [2]. Amplification is characterized by a taller systolic peak, delayed dichrotic notch, lower end-diastolic pressure, and later pulse

arrival. The systolic peak is steeper going to the periphery, attributable to summated reflected waves which develop as the narrowing and branching of blood vessels reflect some of the pulse back toward the aortic valve (Figure 1.3). As the resistance of the branching arterial tree increases, the more of the pressure wave is reflected. The more resistant the tree (i.e., the more atheromatous, hypertrophic, and calcified the arteries), the greater the magnitude of reflection. This is particularly relevant in those with stiff, noncompliant vessels (e.g., the elderly or hypertensive patients), in whom the pulse wave velocity is rapid and reflected waves from both upper and lower body return quickly during late systole, causing a more prominent tidal wave, which may even exceed the percussion wave. This condition may explain the absence of *pulsus parvus et tardus* in very elderly aortic stenosis patients in whom the carotid pulse is preserved and reflects an exaggerated peripheral amplification from noncompliant vessels). However, there is little change in mean arterial pressure (MAP) because there is little change in the resistance to flow from aorta to radial



**Figure 1.3** Arterial pressure wave reflection comparing manifestations in young versus elderly patients. The pressure wave reflection arises from the junction of conduit and low-resistance arteries with high-resistance arteries. Given that there are firmer junctions in the lower versus upper body, most reflection returning from the periphery comes from the lower body up into the proximal thoracic aorta. This reflected wave injects stroke volume, giving rise to the peak systolic pressure in the contour of the tidal wave. In older patients with nondistensible vasculature, the augmented reflected wave results in higher peak systolic pressure for any given stroke volume. Source: O'Rourke 2006 [2]. Reproduced with permission of Elsevier.

artery; rather, MAP changes more dramatically at the arteriolar level.

Aortic diastolic pressure reflects the aggregate resistance of the systemic arterial tree back upon the aortic valve. Noncompliant vessels similarly cause this pressure to be raised. In contrast, the soft vasoplegic (dilated or relaxed) vessels of a septic patient will offer little resistance, and the diastolic pressure will be lower. A regurgitant aortic valve will also cause this pressure to be lower than normal, because the pressure wave travels all the way through to the ventricle manifested as the regurgitant jet.

Pulse pressure is the difference between peak systolic and end-diastolic aortic pressures. A widened pulse pressure suggests aortic regurgitation, because in diastole the arterial pressure drops to fill the left ventricle through the regurgitating aortic valve, and at the same time forward runoff is great, since peripheral resistance is also reduced. In contrast, a narrow pulse pressure may occur in conditions such as cardiac tamponade, or any other low-output state (e.g., severe cardiogenic shock, massive pulmonary embolism or tension pneumothorax).

### Pressure–Volume Loops

In aggregate, the relationships between preload, afterload, and contractility are illustrated in ventricular pressure–volume (PV) loops which plot the changes of these

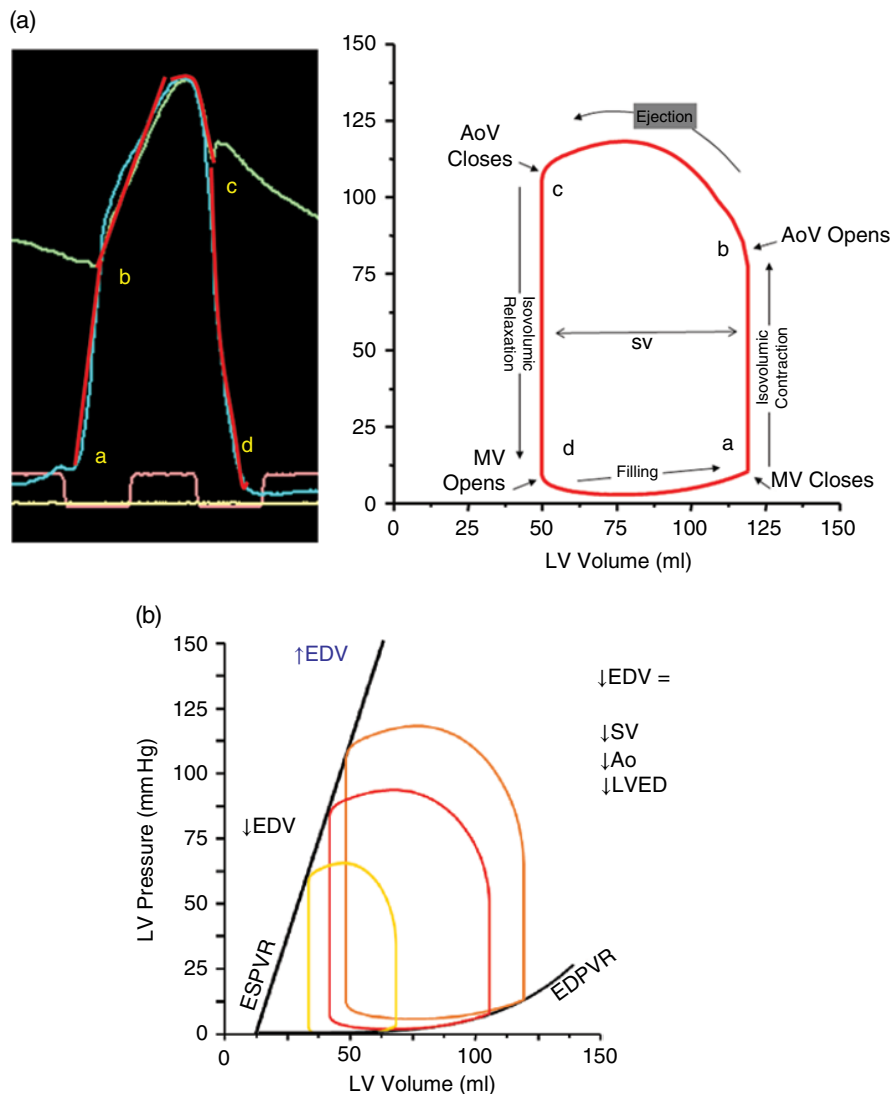
variables over a cardiac cycle [3]. Each PV loop (Figure 1.4) represents one cardiac cycle. Beginning at end diastole (point a), LV volume has received the atrial contribution and is maximal. Isovolumic contraction (a to b) increases LV pressure with no change in volume. At the end of isovolumic contraction, LV pressure exceeds aortic pressure, the aortic valve opens, and blood is ejected from the LV into the aorta (point b). Over the systolic ejection phase, LV volume decreases and, as ventricular repolarization occurs, LV ejection ceases and relaxation begins. When LV pressure falls below aortic pressure, the aortic valve closes, a point also known as the end-systolic pressure–volume (ESPV) point (c). Isovolumic relaxation occurs until LV pressure decreases below the atrial pressure, opening the mitral valve (point d).

The stroke volume is represented by the width of the PV loop, the difference between end-systolic and end-diastolic volumes. The area within the loop represents stroke work. Load-independent LV contractility, also known as  $E_{max}$ , is defined as the maximal slope of the ESPV points under various loading conditions, and the line of these points is the ESPV relationship (ESPVR). Effective arterial elastance ( $E_a$ ), a measure of LV afterload, is defined as the ratio of end-systolic pressure to stroke volume. Under steady-state conditions, optimal LV contractile efficiency occurs when the ratio of  $E_a:E_{max}$  approaches 1.

The PV loop describes contractile function, relaxation properties, SV, cardiac work, and myocardial oxygen consumption. Hemodynamic alterations and interventions change the PV relationship in predictable ways and comparisons of various hemodynamic interventions can be made more precisely by examining the PV loop (Figures 1.5 and 1.6).

Acute changes in cardiac function such as might occur with acute myocardial infarction (AMI) are also easily demonstrated. In AMI, LV contractility ( $E_{max}$ ) is reduced; LV pressure, SV, and LV stroke work may be unchanged or reduced, and LVEDP is increased. In cardiogenic shock,  $E_{max}$  is severely reduced, LV afterload ( $E_a$ ) may be increased, LVEDV and LVEDP are increased, and SV is reduced, findings easily seen to display reduced LV contractile function, acute diastolic dysfunction, elevated LVEDV and LVEDP, and increased LV work (oxygen demand). In more severe cases of myocardial infarction that evolve into cardiogenic shock, LV contractile function is more severely reduced, with associated significant increases in end-diastolic pressure and volume. The LV impairment results in a markedly reduced SV, with an increased myocardial oxygen demand.

The most common applications of PV loops characterize only left ventricular hemodynamics. For research into right ventricular function or extracardiac problems,



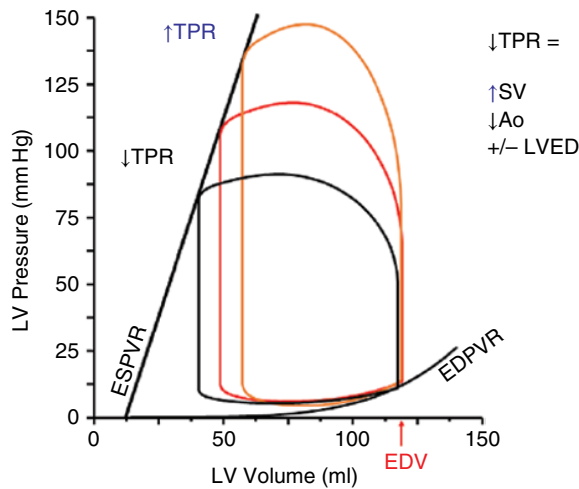
**Figure 1.4** (a) The pressure–volume (PV) loop characterizes the changes in pressure flow over the course of one cardiac cycle. Left panel, the left ventricular (LV) and aortic pressure as measured during cardiac catheterization. Right panel, the PV loop derived from the hemodynamics of LV pressure and volume. Point a, LV end-diastolic pressure is followed by isovolumetric contraction ending at point b, the aortic valve (AoV) opening. Ejection continues until the repolarization of the LV produces a fall in LV ejection. LV pressure falls past point c, aortic valve closure, and continues to fall along the line of isovolumetric relaxation to point d, mitral valve (MV) opening. Changes in the shape of the PV loop demonstrate changes in contractility and cardiac output (stroke volume, SV). Load-independent LV contractility, also known as  $E_{max}$ , is defined as the maximal slope of the end-systolic pressure–volume (ESPV) point under various loading conditions, known as the ESPV relationship (ESPVR). Effective arterial elastance ( $E_a$ ) is a measure of LV afterload and is defined as the ratio of end-systolic pressure and stroke volume. (b) The effect of changes in LV preload. Increasing the left ventricular end-diastolic pressure (LVEDP) along the line of the end-diastolic pressure–volume relationship (EDPVR). As volume is increased, LVEDP, SV, and aortic pressure increase. (See insert for color representation of the figure.)

the standard PV loops become complex and affected by additional factors, altering the PV loop configuration and interpretation.

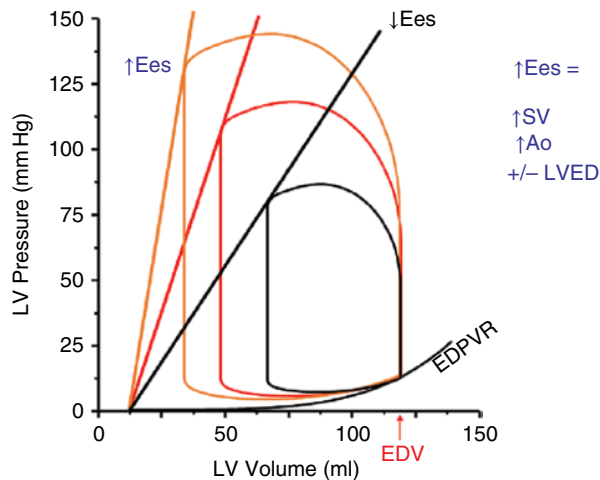
#### Left Ventricular Rotational Mechanics: Systolic Twist and Diastolic Suction

Due to the spiral architecture of its myofibers, the LV twists or rotates from apex to base in a systolic “wringing” motion, generating the SV pathway through the LV

outflow tract, an action that contributes significantly to LV systolic performance [4]. The LV twist also stores potential energy during the systolic phase. During subsequent isovolumetric relaxation (an active ATP requiring process to re-sequester  $Ca^{++}$  into the sarcoplasmic reticulum), the “untwisting” or recoil of stored energy contributes to the diastolic “suction” that opens the mitral valve and accelerates atrial emptying along the LV inflow path. These important mechanics may be deranged under a wide variety of pathologic conditions,



**Figure 1.5** The effect of increasing afterload or total peripheral vascular resistance (TPR) decreases SV, increases aortic pressure, and minimally modifies LVEDP. (See insert for color representation of the figure.)



**Figure 1.6** The effect of increasing contractility: the increasing slope of the line of end-systolic elastance (Ees) increases SV and aortic pressure, with minimal effect on LVEDP. (See insert for color representation of the figure.)

may be evident in hemodynamic traces, and contribute to clinical hemodynamic compromise.

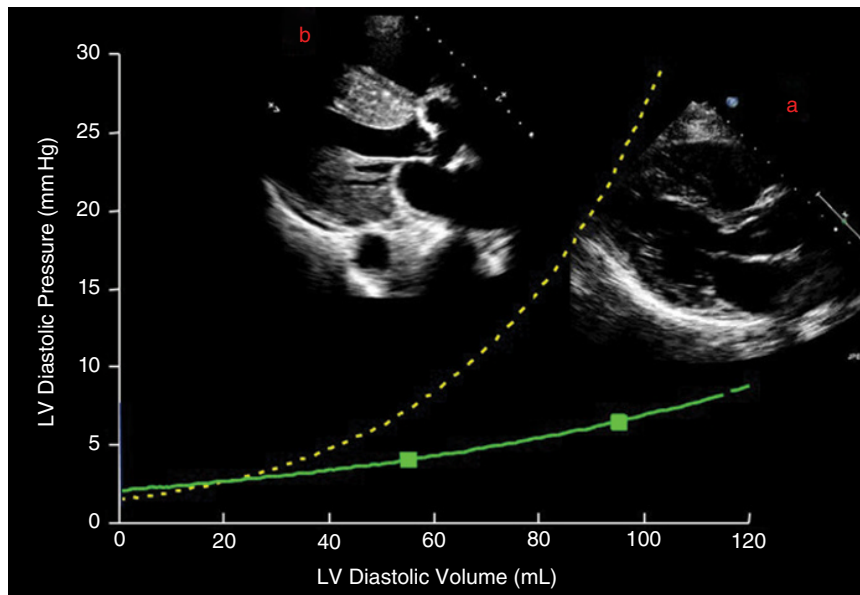
### Diastolic Performance and Cardiac Compliance

Diastolic function is the ability of a chamber to obtain its necessary preload at physiological filling pressures to generate CO under a variety of physiologic conditions, both at rest and during stress (exercise and metabolic stress such as infection, surgery, etc.). Diastole is not a passive process and is fundamentally influenced by various active factors. Diastole can be considered in four phases: isovolumic relaxation, early filling, diastasis, and atrial contraction. Isovolumic relaxation (lusitropic function) is a bit of

a misnomer, for this is an active ATP-requiring process that untwists the LV, rapidly reducing ventricular pressure and through suction opening the mitral valve and initiating the rapid early filling phase. The majority of LV filling occurs here, through ventricular suction; this is followed by equilibration of LA and LV pressures and temporary cessation of flow (diastasis). Finally, active atrial contraction contributes the booster pump function, which delivers additional ventricular preload. This booster optimizes ventricular filling at a lower mean atrial pressure, and the end-diastolic “kick” elevates ventricular end-diastolic pressure (EDP) as the atria actively relax (X descent), thereby facilitating ventricular–atrial pressure reversal which initiates AV valve closure; in aggregate, these effects optimize LV preload while concomitantly minimizing the effects of ventricular diastolic pressure on the back tributaries of filling; that is, the lungs. These diastolic patterns are best illustrated not by invasive catheter interrogation, but rather by Doppler echocardiography under physiologic conditions. LV inflow velocity across the mitral valve is most rapid early, reflected as a predominant E wave on the transmitral Doppler echocardiogram. In normal anatomy, the preload contributed by atrial contraction is relatively small (in contrast to when the ventricle is stiff or the AV valve is obstructed), and therefore the velocity imparted by atrial contraction (the transmitral inflow A wave) is relatively low, thus the normal E/A wave ratio is greater than 1 but less than 2.

Functional preload is the amount of blood actually distending the cardiac chamber. This volume is reflected in filling pressure according to chamber compliance, the relationship between diastolic pressure and volume in any anatomic chamber (ventricle, atrium, pericardium, cranium, etc.). Cardiac chamber diastolic pressure is determined by the volume of blood in the chamber and its distensibility (compliance). In normal anatomy, optimal filling occurs at low filling pressures (Figures 1.2 and 1.7).

During diastole, the LV, left atrium, and pulmonary veins form a “common chamber,” which is continuous with the pulmonary capillary bed; in the right heart a similar relationship exists. Diastolic dysfunction is defined as a functional abnormality of diastolic relaxation, filling, or distensibility, in which filling is limited by abnormal chamber stiffness (hypertrophy, ischemia, fibrosis, infiltration, extrinsic pericardial resistance). Increased stiffness dictates that at any given level of chamber filling, the filling pressure is disproportionately elevated (Figure 1.7). Diastolic dysfunction may occur in association with chamber dilation and related systolic dysfunction (e.g., ischemic cardiomyopathy), or with a small stiff chamber with an intact ejection fraction (e.g., hypertensive cardiomyopathy). Figure 1.2a is the classic Frank–Starling curve wherein end-diastolic volume (true preload) generates output (SV) dependent on the inotropic state. Pure diastolic pressure–volume relationships are illustrated in



**Figure 1.7** Left ventricular pressure–volume relationships demonstrating compliance curves in a normal LV (b) versus a thick stiff LV (a). (See insert for color representation of the figure.)

Figure 1.2b, wherein chamber compliance determines the actual distending pressure (and thus back pressure) for any given level of true preload (Figure 1.2c). What matters clinically is the diastolic pressure needed to generate a given SV. The compliant and contractile ventricle can accommodate a dramatically increased preload (i.e., stress or exercise) and generate high output at low filling pressures. In contrast, both types of heart failure, those with stiff hearts and preserved ejection fraction (HFPEF) and those with dilated hearts and reduced ejection fraction (HFREF), suffer elevated filling pressure and low output syndromes. Finally, consideration of these principles must also take into account the profound influence of excess afterload on ventricular performance (Figure 1.2c), which has disproportionate effects on SV in those with depressed ejection fraction (EF).

#### **Differentiation of Cardiac Preload and Filling Pressures: Left Ventricular End-Diastolic Pressure Does Not Necessarily Reflect Left Ventricular Filling**

Cardiac performance is optimal when SV is generated at low filling pressures. However, diastolic pressure generated by any given degree of filling (true preload) is a function of the compliance of the chamber, and therefore filling pressure reasonably reflects preload *only* if chamber compliance is normal.

Thus, impaired compliance attributable to intrinsic factors (hypertrophy, infiltration or ischemia, or primary pressure and volume overload) or extrinsic constraint (pericardial disease or ventricular interactions) distorts the relationship between filling pressure and true preload. This distortion may confound clinical and invasive

hemodynamic assessment. Measurement of intracardiac filling pressures (for example, LVEDP) is used for two basic purposes: (i) to determine whether preload is adequate to generate SV (i.e., whether the patient is volume depleted); and (ii) to determine whether there is elevated pressure exerting adverse “backward” congestive effects.

With respect to assessing true preload in a patient with clinical low-output hypoperfusion, pulmonary capillary wedge pressure (PCWP) or LVEDP is a convenient surrogate for left-heart preload, although under noncompliant conditions (e.g., severe LV hypertrophy or cardiac tamponade) LV preload may be markedly reduced, but intracardiac pressures may be strikingly elevated. In fact, in some cases patients may be in pulmonary edema despite an LV with small cavity and intact contractility (e.g., restrictive cardiomyopathy). Conversely, chronic volume overload lesion such as aortic regurgitation may result in dramatically increased chamber volumes, but in those who are well compensated, intracardiac pressures are relatively normal as the chamber and pericardium dilate and become more compliant.

#### **Cardiac Mechanics, Atrial Waveforms, and the Venous Circulations**

A critical relationship exists among cardiac mechanics and atrial waveforms, the physiology of the venous circulations, and the dynamic effects of intrathoracic pressure (ITP) and respiratory motion on cardiovascular physiology, permitting a better interpretation of the waveforms to reflect pathophysiology.

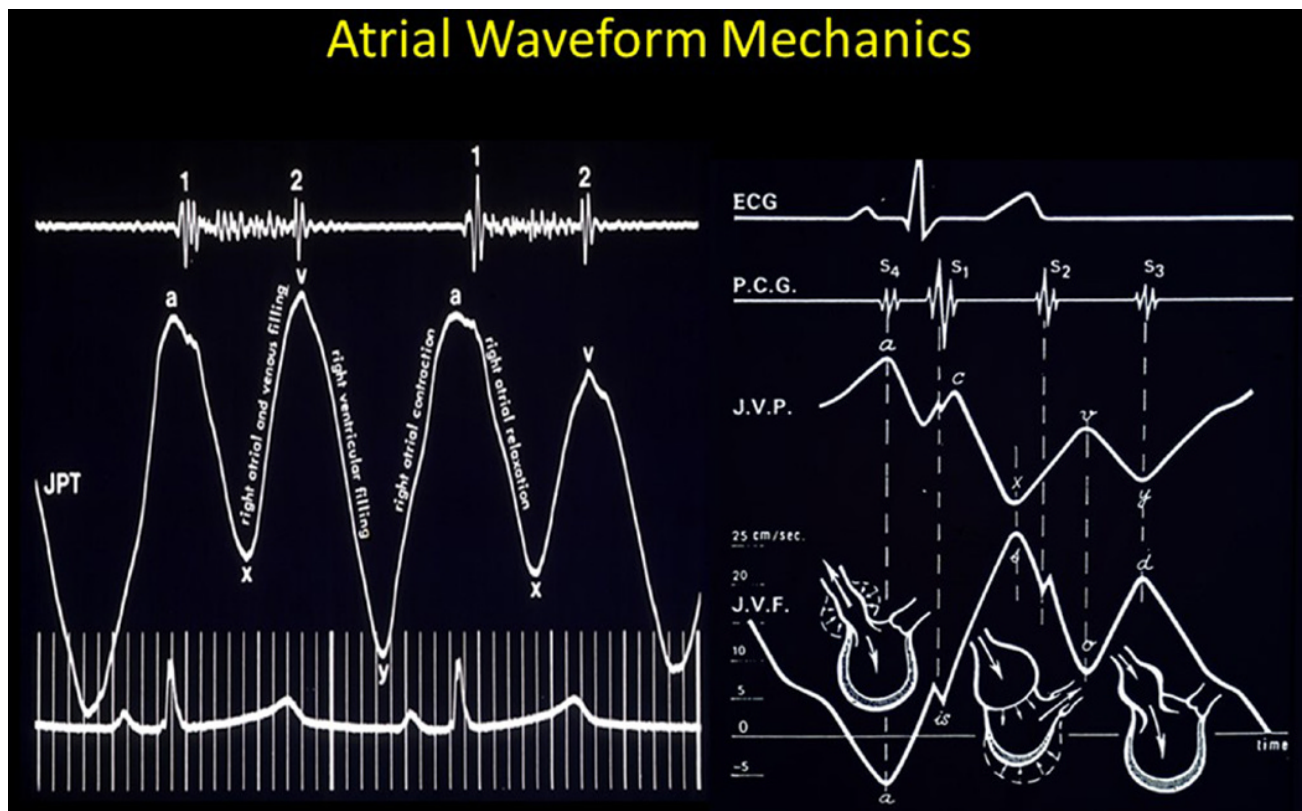
## Atrial Waveforms

Analysis of the atrial waveforms (Figure 1.8) yields insight into cardiac chamber and pericardial compliance. The atrial waveforms are constituted by two positive waves (A and V peaks) and two collapsing waves (X and Y descents). The atrial A wave is generated by atrial systole following the P wave on ECG. Atrial mechanics behave in a manner similar to that of ventricular muscle. The strength of atrial contraction is reflected in the rapidity of the A wave upstroke and peak amplitude. The X descent follows the A wave and is generated by two events: the initial decline in pressure reflecting active atrial relaxation, with a latter descent component reflecting pericardial emptying during ventricular systole (also called systolic intrapericardial depressurization, a condition that is exaggerated when pericardial space is compromised). The X descent's second component is affected by the pericardial space and changes when the ventricles are maximally emptied, therefore pericardial volume and intrapericardial pressure (IPP) are at their nadir.

During ventricular systole, venous return results in atrial filling and pressure which peak with the V wave, whose height reflects the atrial pressure–volume compliance characteristics. The subsequent diastolic Y descent represents atrial emptying and depressurization. The steepness of the Y descent is influenced by the volume and pressure in the atrium just prior to atrioventricular (AV) valve opening (height of the V wave) and resistance to atrial emptying (AV valve resistance and ventricular–pericardial compliance).

## Venous Circulations and Respiratory Oscillations

Venous return to both atria is inversely proportional to the instantaneous atrial pressure, which is itself dependent on compliance. The lowest return occurs when each pressure is highest. Under physiologic conditions, venous return to both atria is biphasic, with a systolic peak determined by atrial relaxation (corresponding to



**Figure 1.8** Atrial mechanical cycle. The upstroke and amplitude of the A wave reflect atrial contraction and the initial portion of the extra set reflects atrial relaxation, with the latter portion due to systolic intrapericardial pressurization. From the downslope of the latter portion of the extra set to the height of the V wave represents the peak period of atrial venous return filling (or regurgitant filling if the atrioventricular valve is incompetent), and therefore is a reflection of atrial compliance. The widest set reflects ventricular relaxation, opening of the AV valve, and subsequent atrial emptying. Source: Kalmanson 1971 [5]. Reproduced with permission of Oxford University Press.

the X descent of the atrial and jugular venous pressure [JVP] waveforms) and a diastolic peak determined by tricuspid valve (TV) resistance and RV compliance (corresponding to the Y descent of the atrial and JVP waveforms). It is essential to consider the relationship of IPP to atrial pressures and flows. Normal IPP is subatmospheric, and both approximates and varies with pleural pressure, decreasing dramatically during inspiration. IPP also tracks right atrial (RA) pressure and shows fluctuations that are associated with the cardiac cycle. In general, IPP increases when cardiac volumes are increased (peak ventricular filling, the V wave) and is lowest at peak ventricular emptying (the later portion of the X descent). It follows that inspiratory decrement in pleural pressure normally reduces pericardial, RA, RV, wedge, and systemic arterial pressures slightly. However, IPP decreases somewhat more than RA pressure, thereby augmenting right-heart filling and output.

Under physiologic conditions, respiratory oscillations exert complex effects on cardiac filling and dynamics since the respiratory effects on the right and the left heart are disparate, owing to differences in the venous return systems and the intrapleural space. The left heart and its tributary pulmonary veins are entirely within the intrathoracic space. In contrast, although both right-heart chambers are intrathoracic, the tributary systemic venous system is extrapleural. Normally, inspiration-induced decrements in ITP (from expiratory 5 to end-inspiratory 25–30 mm Hg) are transmitted through the pericardium to the cardiac chambers. In the right heart, these decrements in ITP enhance the filling gradient from the extrathoracic systemic veins to the right atrium, thereby increasing the caval–right atrial gradient and augmenting venous return flow by 50–60%, increasing right-heart filling and output.

In contrast, the left heart and its tributary pulmonary veins are entirely intrathoracic. Therefore, since pleural pressure changes are evenly distributed to the left heart and pulmonary veins, the pressure gradient from the pulmonary veins to the left ventricle shows minimal change with respiration. However, left-heart filling, stroke volume, and aortic systolic pressure normally decrease with inspiration (up to 10–12 mm Hg). The mechanisms responsible for this normal inspiratory oscillation in aortic pressure include variable ventricular volumes as each ventricle competes for its part of the entire cardiac volume constrained by the pericardium. This competition leads to leftward septal displacement due to augmented right-heart filling, increased LV “afterload,” and inspiratory delay of augmented RV output through the lungs. This physiologic respiratory blood pressure oscillation phenomenon has somewhat confusingly been termed paradoxical pulse, but is normal when <10–12 mm Hg. The moniker *pulsus paradoxus* was

bestowed by W. Kussmaul in 1898, describing the findings of cardiac tamponade in a patient who was tachycardic by auscultation but manifested “paradoxical” phasic dropout of radial pulse on palpation. Paradoxical pulse >12–15 mm Hg is abnormal and may reflect cardiac tamponade and other conditions of enhanced ventricular interaction with intact inspiratory venous return.

## Hemodynamics and Exercise/Stress

Cardiac output increases to meet peripheral demands during exercise or metabolic stress (e.g., infection, surgery). Under physiologic conditions, increased CO is mediated by neuro-hormonal stimulated tachycardia together with increased stroke volume achieved by augmented increased contractility, as well as by peripheral vasodilation (primarily of skeletal muscle). The increased heart rate is associated with enhanced contractility (the systolic “force–frequency relationship”). In addition, no increase in CO can be achieved without a proportional increase in venous return to both sides of the heart. During exercise, venous return is enhanced by the pumping action of skeletal muscle, venous valves, inspiratory suction induced by enhanced respiratory effort which augments right-heart filling, and ventricular suction during diastole. The LV “suction” effect related to active relaxation and “untwisting” further facilitates an increased diastolic filling rate during exercise by rapidly and markedly decreasing LV pressure during early diastole. Normal LV distensibility allows increased end-diastolic volume with minimal change in mean filling pressure. These mechanisms are frequently deranged in various pathologic conditions.

## Ventricular Interactions

The right and left hearts are connected “in series” across the lungs. The right heart is designed to pump blood through the lungs to deliver oxygenated preload to the left heart. (This observation was first appreciated by Sir William Harvey, who stated that “The purpose of the right heart is to pump blood through the lungs, not to nourish them” [6]). Optimal in-series performance is essential to maintain adequate CO at rest and increased CO under conditions of exercise or stress. This requires (i) adequate RV preload (inflow; i.e., systemic venous return); and (ii) optimal pulmonary blood flow through the lungs, which is influenced by RV contractility, the pulmonary valve, and pulmonary vascular resistance (pre-, intra-, and postcapillary). It therefore follows that derangements of any component may lead to in-series failure, which may be subcategorized as “forward failure”

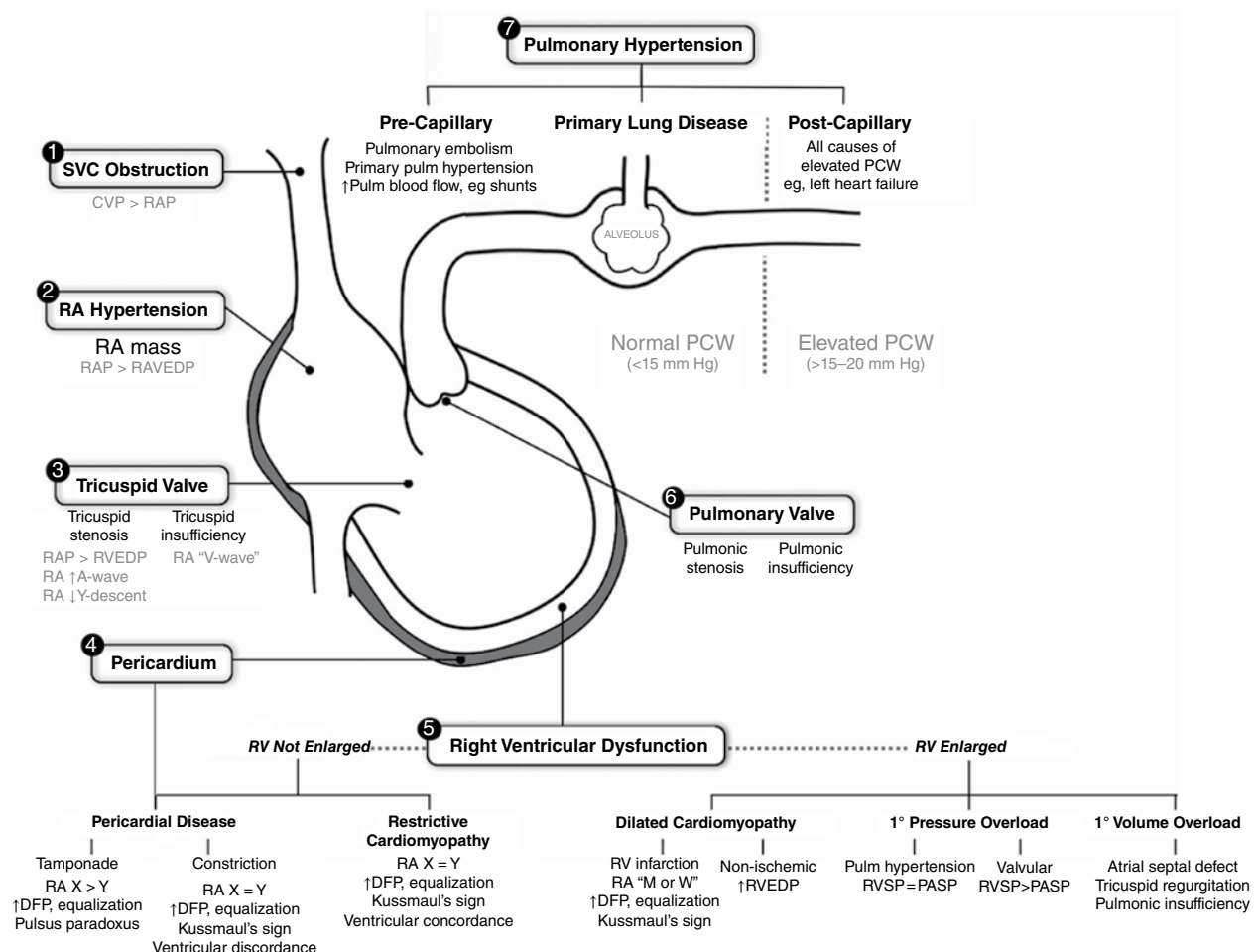
due to primary derangements of RV preload and contractility, or RV afterload proximal to the pulmonary venous circulation, or “backward failure,” in which the primary inciting pathophysiologic mechanism is elevated pulmonary capillary pressure attributable to distal downstream elevation of LA pressure, leading to elevated pulmonary vascular resistance.

Impairment of RV forward failure may result from primary impaired contractility (e.g., acute RV infarction or nonischemic cardiomyopathy), increased RV afterload, or both. RV afterload excess may result from obstruction at any site along the RV flow path (pulmonic stenosis, pulmonary emboli, or pulmonary hypertension of any cause). In the absence of pure obstruction, right-heart afterload failure is due to pulmonary hypertension, which may be differentiated based on anatomic-pathophysiologic hemodynamic considerations as pre-capillary, intracapillary (pulmonary), or postcapillary (Figure 1.9). Precapillary pulmonary hypertension reflects primary abnormalities of the pulmonary arterial

bed resulting from thromboembolic disease, primary pulmonary hypertension, or occasionally extrinsic mass obstruction of the major pulmonary arteries from mediastinal tumors. Primary intrapulmonary processes include the broad range of primary obstructive or restrictive lung diseases.

### Acute RV Failure

Acute increases in afterload or acute ischemia may induce abrupt RV failure. Acutely increased pulmonary resistance imposed by massive and submassive acute pulmonary emboli commonly precipitate RV forward failure. In this setting, it is important to emphasize that the RV (a volume pump by design) is acutely ill equipped and therefore unable to adjust to abrupt increases in afterload imposed by abrupt increments in pulmonary vascular resistance. Acute RV failure is also commonly seen in patients with acute inferior ST elevation myocardial infarction, 50% of whom manifest with concomitant RV infarction. Acute ischemic depression of RV



**Figure 1.9** Post-capillary pulmonary hypertension is attributable to increased pulmonary capillary wedge pressure which, in the absence of pulmonary veno-occlusive disease, reflects elevated left atrial pressure.

contractility diminishes transpulmonary delivery of LV preload, leading to decreased cardiac output despite intact LV contractility. Biventricular diastolic dysfunction contributes to hemodynamic compromise in 25% of cases overall. Abrupt RV dilatation within the noncompliant pericardium elevates intrapericardial pressure, the resultant constraint further impairing RV and LV compliance and filling, and therefore forward output.

#### Chronic RV Failure

Under chronic conditions, the RV can hypertrophy to an extent in response to increased afterload (but not as well or as long as the more muscular LV), thus RV forward (and backward) failure ensues. This is the case in any patient with significant pulmonary hypertension, regardless of the cause. It is now well recognized that depressed RV systolic function is common in patients with dilated LV cardiomyopathies (either as part of the intrinsic depression of contractility in nonischemic patients, or in those with ischemic cardiomyopathy largely a result of pulmonary venous congestion leading to pulmonary hypertension. Regardless of the clinical congestive heart failure setting, chronic depression of RV output contributes to morbidity and mortality.

#### Backward Heart Failure

Elevated pulmonary capillary pressure leads to elevated pulmonary vascular resistance and ultimately pulmonary congestion. Such “post-capillary pulmonary hypertension” is attributable to increased pulmonary capillary wedge pressure which, in the absence of pulmonary veno-occlusive disease, reflects elevated LA pressure (Figure 1.9). Pathologic LA pressure increases are attributable to mitral valve disease or LV impairment (pressure overload, volume overload, or cardiomyopathy), or rarely LA myxomas.

Resting elevations of PCWP, particularly >25 mm Hg, lead to pulmonary hypertension that is tripartite in its “protective” pathophysiology, including vasoconstriction together with thickening of the pulmonary capillaries, pulmonary arteriolar intimal thickening, and medial hypertrophy. It is important to note that these protective mechanisms, including increased capillary lymphatic drainage, are designed to keep the lungs dry, but at the cost of reduced CO. These adaptive mechanisms may allow chronic resting elevations of PCWP even >25 mm Hg to be tolerated without resting dyspnea, rales or congestion seen on chest radiogram. A regurgitant mitral valve exerts disproportionate back pressure for any given mean elevation of PCWP (and thus greater magnitudes of pulmonary hypertension), a phenomenon undoubtedly attributable to the systolic pulsation of blood deep into the pulmonary venous system.

#### Parallel Ventricular Interactions: Septal-Mediated Diastolic Ventricular Interactions

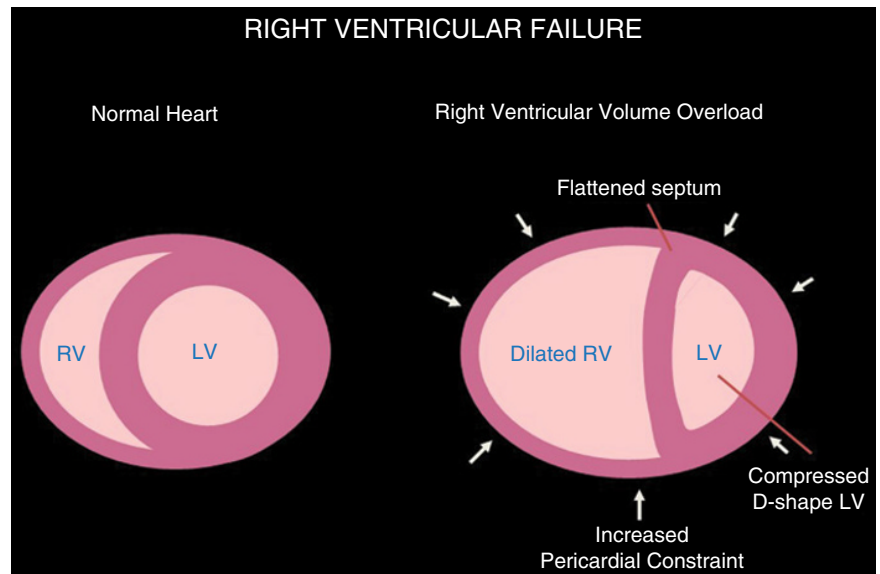
Pressure or volume overload of one ventricle influences the compliance and filling of the contralateral ventricle by septal-mediated diastolic interactions. The normal pericardium more tightly constrains the ventricles and therefore enhances such interactions. Even in the absence of the pericardium, pressure or volume overload of one ventricle influences the compliance and filling of the contralateral ventricle. The pericardium envelopes the cardiac chambers and under physiological conditions exerts mechanical effects that enhance normal ventricular interactions, balancing left and right cardiac outputs. Because the pericardium is noncompliant, conditions that cause intrapericardial crowding (e.g., blood or masses) elevate intrapericardial pressure, which may be the mediator of adverse cardiac compressive effects.

Elevated IPP may result from primary disease of the pericardium itself (tamponade or constriction) or from abrupt chamber dilatation (e.g., right ventricular infarction [RVI] or abrupt RV dilation after major pulmonary embolus). Acute RV dilatation within the noncompliant pericardium elevates IPP, the resultant constraint further intensifying septal-mediated diastolic ventricular interactions and thereby impairing both RV and LV compliance and filling (Figure 1.10). These effects contribute to the pattern of equalized diastolic pressures and RV “dip-and-plateau” characteristic of both conditions. In patients with acute RVI, ischemic right ventricular free wall (RVFW) dysfunction diminishes transpulmonary delivery of LV preload, leading to decreased cardiac output despite intact LV contractility. Biventricular diastolic dysfunction contributes to hemodynamic compromise. The ischemic RV is stiff and dilated early in diastole, which impedes inflow, leading to rapid diastolic pressure elevation. Acute RV dilatation and elevated diastolic pressure shift the interventricular septum toward the volume-deprived left ventricle, further impairing LV compliance and filling.

#### Septal-Mediated Systolic Ventricular Interactions

Under conditions of acute ischemic RVFW dysfunction, RV performance is dependent on LV-septal contractile contributions transmitted via systolic ventricular interactions, mediated by the septum through both paradoxical septal motion and primary septal contributions. In early isovolumic systole, unopposed LV-septal pressure generation creates a left-to-right transseptal pressure gradient, resulting in early systolic septal bulging into the RV cavity. This paradoxical motion not only contributes to early generation of RV systolic pressure,

**Figure 1.10** Pericardial mediated diastolic interactions. Under normal conditions, the interventricular septum is concave left to right, reflecting greater LV versus RV stiffness. RV dilatation and diastolic pressure elevation shift the septum right to left, altering LV filling and compliance. Abrupt RV dilatation (e.g., acute pulmonary embolus or RV infarction) also occurs across the pericardium, increasing pericardial pressure and exacerbating this adverse diastolic interaction. *Source:* Haddad 2008 [6]. Reproduced with permission of Wolters Kluwer Health, Inc.



but also helps stretch the dyskinetic RVFW, a prerequisite to providing a stable buttress upon which later LV-septal thickening and shortening can generate peak RV pressure and effective pulmonary flow.

## Fundamentals of Right-Heart Hemodynamics

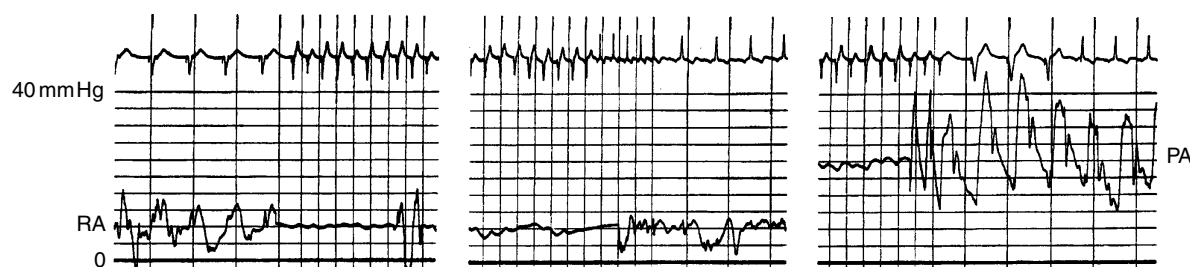
### Hemodynamics of Abnormal Cardiac Rhythms

One of the most common pitfalls in the interpretation of hemodynamic data is the failure to appreciate abnormalities in cardiac rhythms, which often account for the alteration and, at times, misinterpretation of pressure waveforms [8, 9]. Let us examine the right-heart hemodynamics in a patient undergoing evaluation for shortness of breath. The right atrial pressure (Figure 1.11, left and middle) demonstrates an alteration in the phasic waveform. At left, the V wave or S wave suggests tricuspid regurgitation. However, this waveform is significantly different during the measurements made only a

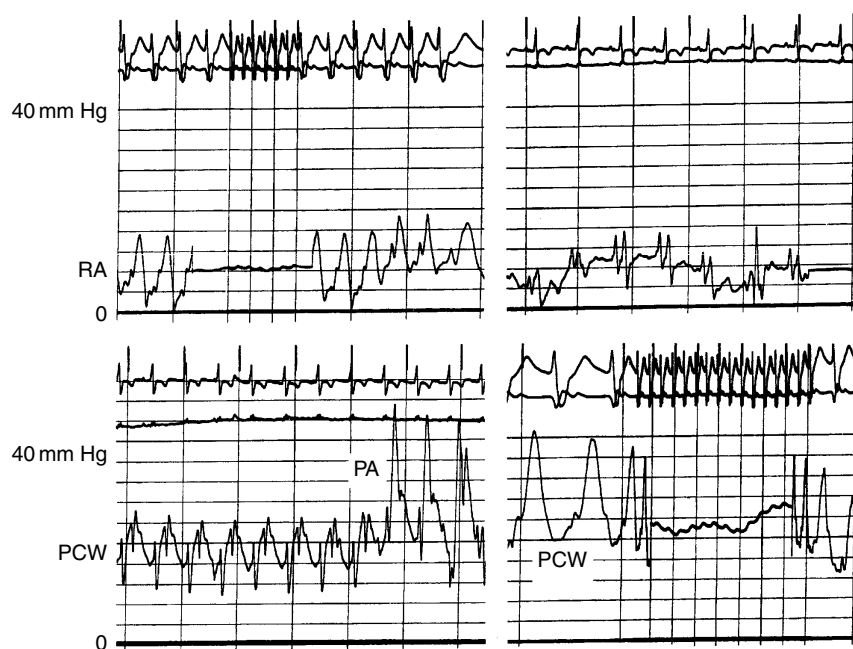
few minutes later (Figure 1.11, middle). How has the physiology been affected?

Examining the ECG, a paced rhythm is responsible for the initially regurgitant wave in the right atrial pressure. The peak of the pressure wave corresponds to the QRS (the tracing lines align the specific wave or segment of the ECG to the pressure wave). As the rhythm changes to a sinus mechanism (Figure 1.11, center of middle panel), the normal right atrial waveform is restored and the regurgitant waveform is eliminated. The changing rhythm also affects the determination of maximal pulmonary artery pressures (Figure 1.11, right). During pacing, the pulmonary artery pressure has a larger respiratory variation and is higher than pressures obtained during normal sinus rhythm.

Observe the right-heart hemodynamics obtained in a patient undergoing evaluation for ischemia-induced ventricular dysrhythmia and congestive heart failure (Figure 1.12). The mean right atrial pressure (Figure 1.12, top left) is 9 mm Hg with large V waves. Why is the right atrial waveform different between the left and right panels of Figure 1.12? Consider the ECG. When sinus rhythm



**Figure 1.11** (Left) Right atrial (RA) pressure (0–40 mmHg scale) during alterations in cardiac rhythm (middle). (Far right) Change in pulmonary artery (PA) pressure.



**Figure 1.12** Right-heart hemodynamics during changes in cardiac rhythm. (Top left and right) RA, right atrial; (bottom left and right) PCWP, pulmonary capillary wedge pressure; PA, pulmonary artery.



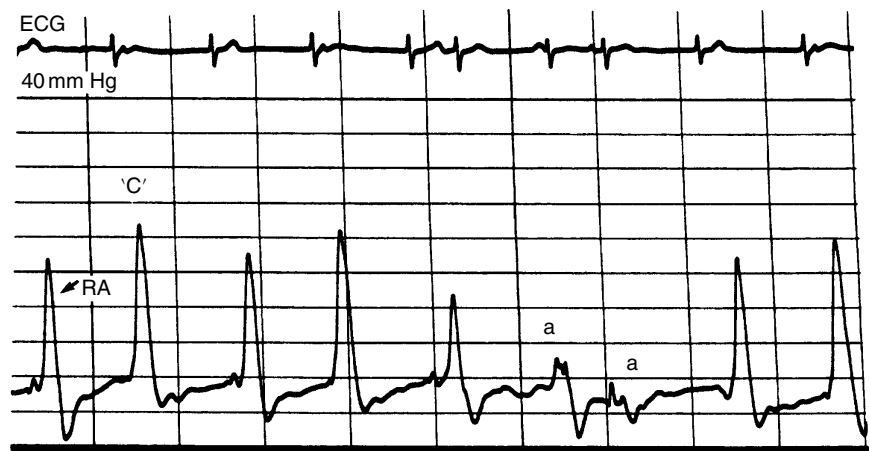
**Figure 1.13** (Top right) Right atrial (RA) and (top left and lower right) right ventricular (RV) pressures (0–40 mm Hg scale) during alterations in cardiac rhythm. (Lower right) RV pressure scale change from 0–40 mm Hg to 0–100 mm Hg (far right).

is restored (note the P waves), the V wave is attenuated and a normal right atrial pressure waveform can be seen, with a reduction in right atrial pressure to a mean value of 7 mm Hg (Figure 1.12, top right). A similar response can be appreciated in the pulmonary capillary wedge pressure (Figure 1.12, lower left). During sinus rhythm, PCWP averages 18 mm Hg, with a V wave up to 24 mm Hg. However, during ventricular tachycardia, the mean PCWP markedly increases to 24 mm Hg, with V waves

up to 40 mm Hg (Figure 1.12, lower right). Interpretation of the hemodynamic waveform is thus critically dependent on the particular cardiac rhythm which reflects the physiologic responses generating the cardiac pressure (Figures 1.13 and 1.14).

The influence of cardiac rhythm on right-heart hemodynamics can be appreciated in a 39-year-old man with congestive heart failure and cardiomyopathy undergoes hemodynamic evaluation (Figure 1.13). Examine the

**Figure 1.14** Right atrial (RA) pressure (0–40 mm Hg scale) in a patient after myocardial infarction. See text for details.



right atrial waveform at the top right (Figure 1.13). Note the alteration of the V wave configuration. How does one explain the large pointed waveforms at the left side in contrast to the smaller, broader waveforms demonstrated at the right? While considering the possibilities, examine the differences in right ventricular pressures obtained during this study (Figure 1.13, upper right and lower left panels). Compare the effects on the cardiac rhythm of the generation of right ventricular pressure.

The right atrial pressure V waves (Figure 1.13, top left) occur during junctional rhythm with atrial superimposition on the QRS and third-degree heart block. As the atrial time delay permits normal sinus mechanisms to intervene, the waveform changes into a sinus-type rhythm with a larger A wave and a small V wave. Note the decrease in mean right atrial pressure from 10–12 mm Hg to <8 mm Hg when sinus rhythm is in play.

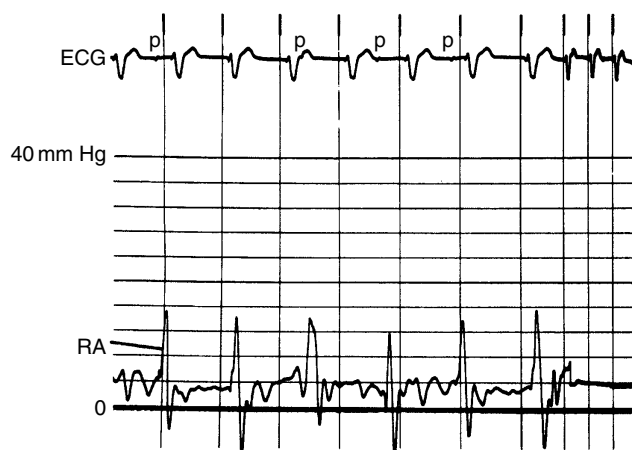
The evaluation of right ventricular pressure is also interesting, in that the accelerated junctional rhythm during complete heart block has a lower peak systolic pressure of approximately 38 mm Hg (Figure 1.13, top right, right side). When the rhythm changes, the peak systolic right ventricular pressure increases to approximately 50 mm Hg and the initiation of the atrial wave can be seen as a deflection on the QRS (Figure 1.13, mid-portion at upper right). As the sinus mechanism contributes to the filling of the ventricle and precedes QRS activation of ventricular contraction, the systolic pressure increases and remains in excess of 50 mm Hg. The rhythm changes explain the wide variations in right atrial and ventricular pressure waveforms, and should be important functional clues in the evaluation of hemodynamics for such individuals.

Consider the right atrial pressure wave obtained in a 66-year-old man following myocardial infarction (Figure 1.14). Cardiac rhythm disturbances were noted on the resting electrocardiogram on day 3. The right

atrial pressure wave was recorded using a fluid-filled catheter. Examine the pressure waves and consider the following: What is the etiology of the large, spiked waves (C)? Is the tricuspid valve normal? What accounts for the C spike variation from 16 to 24 mm Hg? What is responsible for the change in waveform on beats #6 and 7?

These large, spiked waves are C waves or (giant) cannon waves. This occurs when atrial contraction falls out of sequence with normal ventricular systole and the atria contract against a tricuspid valve closed by the increased right ventricular pressure during ejection. The size of the C wave is dependent on the timing of atrial contraction relative to ventricular filling (and the position of the tricuspid valve). When the atrial contraction precedes ventricular contraction in its normal synchronous mode, normal A waves are generated (beats #6 and 7). When atrial synchrony is lost, the cannon waves return. These cannon waves can be observed on bedside physical examination in the jugular venous pulse and should be differentiated from systolic tricuspid regurgitant waves (to be identified below). Iatrogenic induced (pacemaker) or spontaneous abnormalities of conduction also can produce similar types of cannon waves of atrial activity out of synchrony with ventricular contraction.

Large spiked pressure waves appeared in the right atrial tracing of a 78-year-old woman with a ventricular pacemaker (Figure 1.15). Examine the rhythm first. The pressure tracing demonstrates brief, sharp peaked waves, less prominent than the atrial contraction waves of the previous tracing. The dyssynchronous ventricular pacemaker timing relative to atrial dissociated contraction appears to be responsible. Note the wider C-type wave of beat #3 with the P wave falling on the QRS. The high-pressure spike of very narrow width also suggests artifact from catheter impaction, but the timing sequence also is highly consistent with “cannon”-type waves.



**Figure 1.15** Right atrial (RA) pressure (0–40 mm Hg scale) in a patient with compensated heart failure. See text for details.

### Vena Caval Pressure Waveforms in Tricuspid Regurgitation

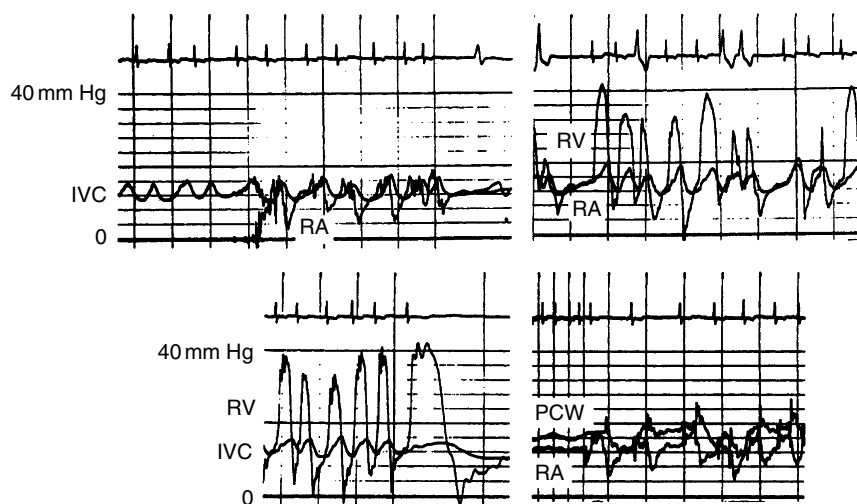
Tricuspid regurgitation often produces distinct waveforms in the right atrial pressure [10–13]. Sometimes, transmission of the regurgitant wave can be detected beyond the vena cava (i.e., transmitted to the jugular veins) [14]. However, it is uncommon to observe changing waveforms in the inferior vena cava compared to those in the right atrium. For purposes of demonstration, we recorded the inferior vena caval pressure and compared the waveforms to those of the right atrial and ventricular pressures in a patient with modest tricuspid regurgitation (Figure 1.16).

A 61-year-old woman had tricuspid regurgitation of unknown etiology which was associated with mild pulmonary hypertension and dyspnea. Coronary arteriography was normal. Left ventriculography demonstrated mild global hypokinesis with an ejection fraction of 55%.

Aortic pressure was 125/70 mm Hg, left ventricular pressure was 125/12 mm Hg, and pulmonary capillary wedge pressure was 20 mm Hg with normal A and V wave configurations. The effects of tricuspid regurgitation on the right atrium and inferior vena cava were assessed by measuring two pressures simultaneously, one from each of the two lumens of a balloon-tipped pulmonary artery catheter. The systolic wave of regurgitation could be appreciated in both the right atrium and inferior vena cava. The right atrial pressure was measured with the tip of the balloon-tipped catheter looped within the right atrium (Figure 1.16, upper left). The inferior vena cava pressure was measured approximately 10 cm below the inferior border of the right atrium. The pressure wave fidelity between the two systems was demonstrated to be equivalent by comparing the right ventricular and right atrial pressures measured with the same transducers on advancement of the catheter into the right ventricular apex (Figure 1.16, top right).

The waveform of the inferior vena cava is slower in upstroke and downstroke, with reduced velocity. The blunted waveform is due, in part, to the considerably higher capacity and compliance of the vena cava compared to those of the right atrium. It is also important to note that the right atrial pressure mean, as expected, is lower than that of the vena cava, with a 2–4 mm Hg pressure gradient required for maintenance of normal blood flow. The pressure gradient between the inferior vena cava and right atrium occurs predominantly during the end of atrial diastole (Figure 1.16, top left).

The severity of tricuspid regurgitation can be appreciated when comparing the right ventricular and right atrial pressures (Figure 1.16, top right), showing the regurgitant S wave and large V wave of tricuspid regurgitation. Tricuspid regurgitation is, by waveform characteristics, most severe during the ventricular couplet



**Figure 1.16** (Top left) Right atrial (RA) and inferior vena cava (IVC) pressures in a patient with tricuspid regurgitation. (Top right) RA and RV pressures. (Lower left) RV and IVC pressures. (Lower right) Pulmonary capillary wedge (PCW) and RA pressures.

appearing in the mid portion of this tracing, in which the pressure during systole is achieved for both beats. When comparing the vena cava and right ventricular pressures (Figure 1.16, lower left), the delay in waveform and transmission of the vena cava can be appreciated. Of interest is that the differences between the mean pulmonary capillary wedge (16 mm Hg) and mean right atrial pressures (12 mm Hg; Figure 1.16, lower right) demonstrate the larger Y descent in right atrial pressure (Figure 1.16, upper left) which drives the inferior vena cava flow. Inferior vena cava flow in most cases parallels that of superior vena cava flow and thus large V waves in the jugular vein on clinical examination can reflect either significant tricuspid regurgitation or functional regurgitation during cardiac arrhythmias [14].

The jugular pulse closely reflects events in right atrial pressure and also parallels changes observed in the vena cava [15, 16]. However, one must remember that the change in pressure within the right atrium is reflected principally by a change in volume for the venous system. It is thought that the pulse wave transmission from the right atrium to the jugular veins has the least disparity for pressure waves which are positive and are thought to be conducted more rapidly compared to negative pressure waves [17]. The venous pulse A and C waves have an average delay from right atrial pressures of approximately 60 msec, the V wave 80 msec, the Y trough 90 msec, and the X trough 110 msec [17]. It requires 60 msec for the right atrial A wave to reach the right ventricle and cause a positive deflection in this chamber. These delays should be considered when examining the jugular venous pulse, as well as inferior vena caval pressure waves as a reflection of right atrial pressure [11].

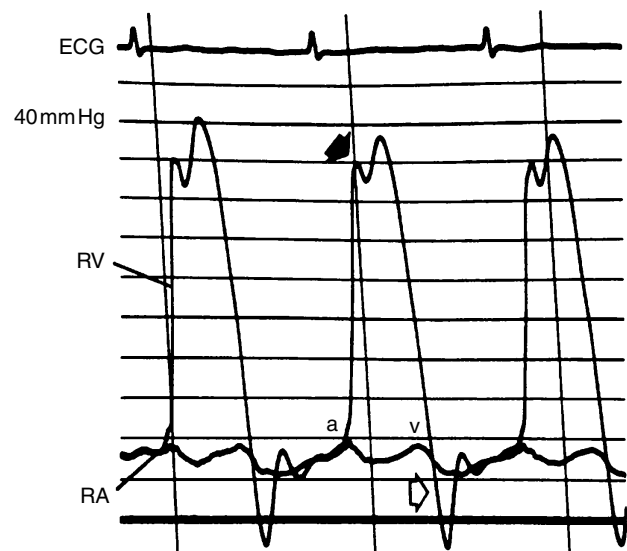
Jugular vein pulsations may occur from an induced artifact of transmitted carotid arterial pressure waves. This artifact can be recognized by an irregular pulsation often obscuring the X descent. The irregular wave shows a carotid-like contour, with the dichrotic notch recognized in the middle of the X descent [11]. Tavel [14] notes that tricuspid insufficiency often produces a prominent V wave, beginning early and tending to obliterate the X descent. In severe tricuspid regurgitation, the V wave corresponds and begins with the C wave and shows a broad plateau terminating in a steep Y descent. This wave has been termed the regurgitant or S wave. In the setting of atrial fibrillation, nearly complete obliteration of the X descent is required before making the diagnosis of tricuspid insufficiency from a venous pulse wave. In patients with normal sinus rhythm, changes in the venous pulse suggesting tricuspid regurgitation may demonstrate only slight decrease in the X descent equal to or above the level of the Y trough. In some patients, a separate systolic wave may appear on the V wave ascent and may be an obscured clue to the presence of tricuspid

regurgitation [13]. In addition, in tricuspid regurgitation there may be a relatively normal venous pulse wave; hence the diagnostic accuracy of the waveform is helpful, in that a normal curve cannot be used to exclude tricuspid disease. The characteristic pulse waves may be absent at rest but brought on by inspiratory maneuvers or increasing heart rate [13].

### Normal Tricuspid Valve Function

Simultaneous right ventricular and right atrial pressure waves using fluid-filled transducer systems were measured in a 40-year-old woman with a history of dyspnea and chronic obstructive pulmonary disease (COPD; Figure 1.17). Identical matching of the right atrial and right ventricular diastolic pressures is the norm. The A wave (atrial contraction) of the right atrial pressure corresponds to the A wave of the right ventricle. The V wave corresponds to the passive increase in right atrial pressure from venous return along with the rapid rise in right ventricle pressure during RV systole. The V wave peak precedes the tricuspid valve opening. Note that pressure immediately after the A wave, called the X descent, falls and does not begin to increase until late in systole. As right ventricular pressure falls below right atrial pressure, the tricuspid valve opens, releasing atrial pressure (the Y descent of the V wave). Not shown on this tracing is a C wave of ventricular contraction apparent on some beats as a “notch” immediately after an A wave or the initial upstroke of ventricular pressure.

The higher ejection velocity and faster development of the right ventricular pressure cause more oscillation of



**Figure 1.17** Simultaneous right ventricular (RV) and right atrial (RA) pressures measured through two fluid-filled catheters (0–40 mm Hg scale). See text for details.

the transducer system than those of the right atrial pressure, and therefore a high-frequency “ringing” is observed as a notch on the upstroke before the systolic peak of the right ventricle (Figure 1.17, black arrow) and a rapid dip (negative overshoot) in early diastole (open arrow). This common artifact using fluid-filled systems is important to consider when assessing tricuspid and pulmonary gradients.

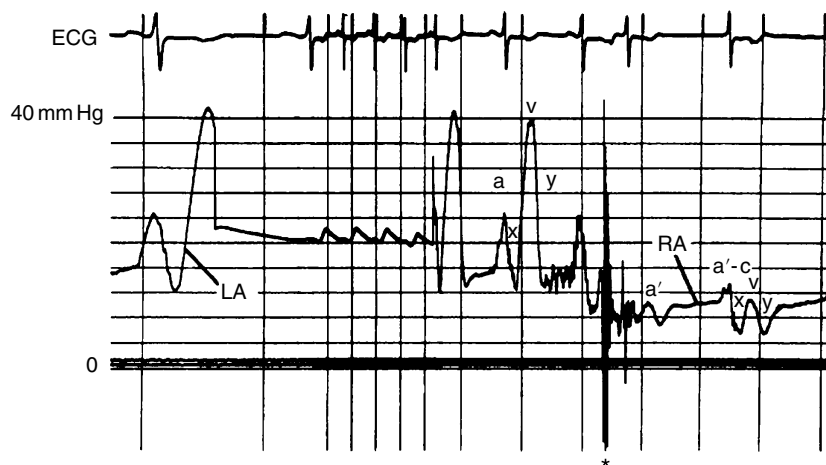
### Atrial Pressure X and Y Troughs

Compare the typical hemodynamic normal example (Figure 1.17) of tricuspid valve function to right and left atrial pressure waveforms in a 62-year-old man with aortic stenosis (Figure 1.18). These pressures were measured continuously on pullback of a fluid-filled transeptal Brockenbrough catheter. The mean left atrial pressure is elevated approximately 22 mm Hg with striking A and V waves. There are two principal negative or downward motions of the right (and left) atrial pressure waves. The X trough results from movement of the tricuspid (or mitral) valve away from the atrium when intrapericardial pressure is decreasing immediately after ventricular contraction begins and left ventricular volume falls. The Y trough occurs with opening of the tricuspid valve [18]. There is a reciprocal relationship between pressure and right atrial or venous flow. Flow is virtually absent at times of peak positive A and V waves [15, 19]. The left atrial V wave is giant (i.e., twice the mean pressure) and occurs, in this particular patient, in the absence of mitral regurgitation. The V wave reflects the pressure–volume relationship (also known as compliance of the atrium) and will be discussed in the review of the V wave below. A noncompliant or stiff ventricle often is associated with large A and V waves. The differences between left and right atrial pressures are easily appreciated during the

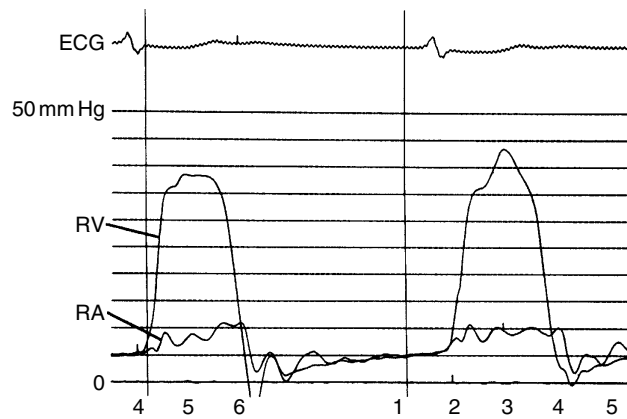
pullback from left to right atrium (Figure 1.18). The left atrial V waves are generally greater than V waves on the right atrial pressure where the A wave predominates. The C waves, again, are not evident. Also note that atrial arrhythmias may significantly alter the waveforms (Figure 1.18, first right atrial beat after \*, a1).

### Systolic Regurgitant Right Atrial Waves

In contrast to large cannon-type waves, positive systolic pressure waves on the right atrial tracing may also be due to an incompetent or occasionally a stenotic tricuspid valve. A 50-year-old woman with atrial fibrillation and a history of rheumatic fever has increasing pedal edema and dyspnea. Simultaneous right ventricular and right atrial pressures were measured with two fluid-filled catheters (Figure 1.19). The right atrial pressure matches the right ventricular pressure in diastole, and rises across the systolic period of right ventricular ejection, which is the most common pressure wave pattern of tricuspid regurgitation [12]. As anticipated in atrial fibrillation, the right atrial and ventricular A waves are absent. A prominent Y descent occurs after the point of the maximal right atrial pressure (V wave) and falls sharply with the drop in right ventricular pressure. Although the slope of the atrial pressure during right ventricular ejection is generally proportional to the severity of tricuspid regurgitation [Figure 1.15], the compliance or pressure–volume relationship of the atrium will determine the size and character of the pressure wave. Note that the diastolic pressures of the right atrial and right ventricular tracings are nearly identical throughout the majority of diastole. If the catheters are zeroed, calibrated properly, and the resonant features and sensitivity of the two fluid-filled systems are matched, small diastolic gradients of tricuspid stenosis can be reliably determined.



**Figure 1.18** Continuous pressure recording from left atrium (LA) to right atrium (RA) on pullback (\*) across the intra-atrial septum, demonstrating phasic waveforms A and V and X and Y descents, respectively, for the two atria. See text for details.



**Figure 1.19** Right atrial (RA) pressure (0–40 mm Hg scale) with a varying murmur along the right sternal border. See text for details. Source: Lingamneni 1979 [12]. Reproduced with permission of John Wiley & Sons.

### Pulsatile Venous Waves

A 39-year-old woman with severe ascites and dyspnea at rest has large V waves during jugular vein examination and a pulsatile liver. The simultaneous right ventricular and right atrial pressures (Figure 1.20) show the marked and more striking upslope of right atrial pressure during right ventricular ejection with a V or S (systolic) wave to 32 mm Hg. The more rapid rise of right atrial pressure indicates severe tricuspid regurgitation (compared with Figure 1.19). Early diastolic right ventricular pressure drop is associated with an early right atrial–right ventricular pressure gradient which equilibrates before the first one-third of diastole following a rapid decline, reflecting mostly high flow and not necessarily significant tricuspid valvular stenosis. The faster heart rate (compared with the previous patient in Figure 1.19) may also contribute to the early right atrial–right ventricular diastolic gradient. The regurgitant S wave, occurring slightly earlier than the V wave, is very prominent on physical examination and can be seen in the neck and even transmitted down to the femoral vein (Figure 1.20, lower panel). The marked regurgitant waves seen on the lower panel are measured in the femoral vein. Femoral vein pressure may be as high as 40 mm Hg. Thus, on puncture of the femoral vein, a “venous” pressure pulse may be observed. The timing of the V wave is coincident with the electrocardiographic T wave, but may be easily confused with an arterial pulse of low amplitude.

### Tricuspid Valve Dysfunction: Right Atrial–Right Ventricular Gradients

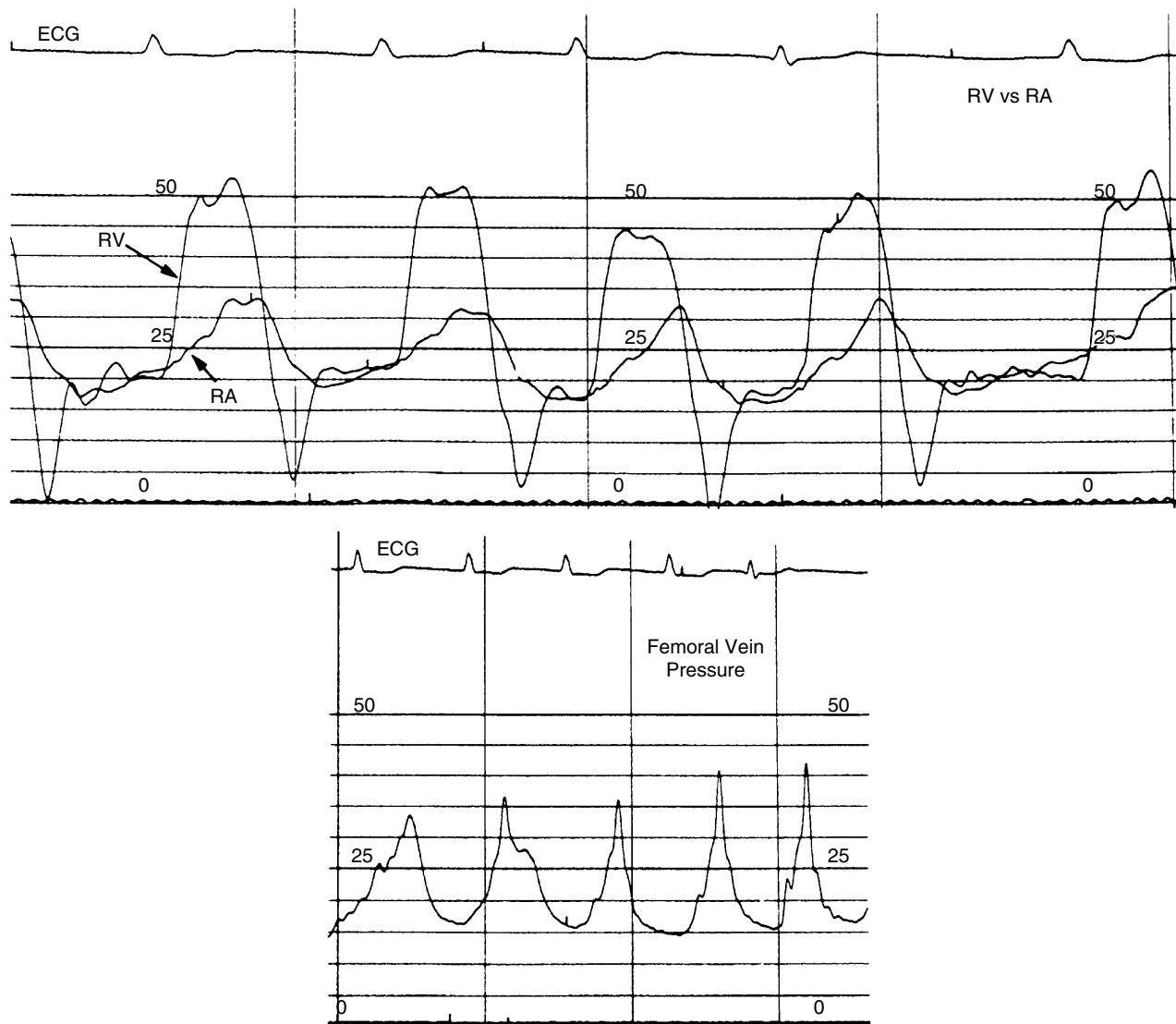
Right atrial and right ventricular pressures were measured in a 49-year-old woman with increasing abdominal girth, dyspnea at rest and exercise, and systolic and

diastolic murmurs that varied markedly with respiration (Figure 1.21). The right atrial pressure (upper panel) demonstrated a prominent regurgitant wave with fusion of A and V waves, an absent X trough, and a marked Y descent. Is there truly an A wave? No, the rhythm is atrial fibrillation. Also observe absent A waves on the right ventricular tracing (lower panel). The C wave of ventricular contraction can now be seen (arrow, lower panel). Because of the resonant qualities of some fluid-filled systems, the pressure waveform with a blunted A wave and X descent (Figure 1.22) may occasionally be confused with the “M” configuration of constrictive or restrictive physiology [10, 20]. The broad and wide upsloping right atrial pressure of tricuspid regurgitation is importantly associated with a persistent gradient of approximately 4 mm Hg across the tricuspid valve throughout diastole. Compare this pressure tracing to that seen on Figure 1.21, in which a diastolic right ventricular–right atrial pressure diastolic gradient is not present. These small pressure gradients are always significant in tricuspid valve disease [10].

A 66-year-old white female had severe tricuspid regurgitation in 1985 and underwent a procedure with a symptom-free period until five years later. A marked increase in abdominal girth and severe lower-extremity edema were the predominant complaints, along with mild paroxysmal nocturnal dyspnea and orthopnea. There was no chest pain. High-flow velocities across the tricuspid valve were demonstrated by echocardiography. Moderate left ventricular dysfunction was also present. The hemodynamic tracings of the right-heart pressures were measured with fluid-filled transducers through two catheters (Figure 1.23). The left-hand panel of Figure 1.23 demonstrates the elevated and matched right atrial pressures. The rhythm was atrial bigeminy. Note the loss of distinct right atrial A and V waves. When simultaneous right ventricular and right atrial pressures are measured (Figure 1.23, right-hand panel), a significant right atrial–right ventricular diastolic gradient can be seen. The tricuspid valve, five years after bioprosthetic valve implantation, had a mean gradient of 11 mm Hg with a cardiac output of 6.4 L/min, which yielded a valve area of 1.5 cm<sup>2</sup>. Importantly, matching of the two pressure transducers eliminated artifactual differences contributing to this gradient. As one can see, in significant tricuspid stenosis the gradient persists throughout diastole during both long and short cycles (compared to Figure 1.21). Repeat tricuspid valve replacement was subsequently performed.

### Pressure Wave Artifacts

If the measurement of blood pressure were automatically available without having to attend to transducer flushing, pressure line, and manifold connections, and erroneous settings on the recorders or kinks in catheters, the study of

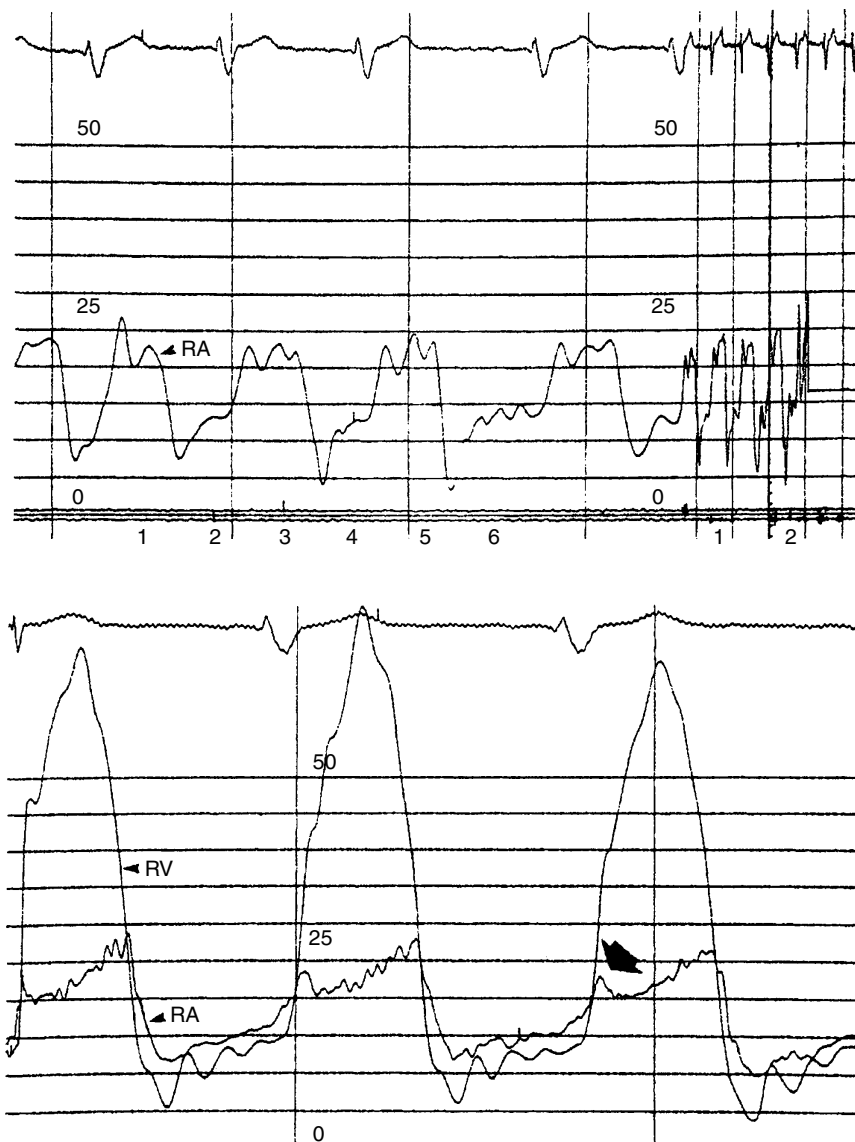


**Figure 1.20** Simultaneous right ventricular (RV) and right atrial (RA) pressures (0–50 mm Hg scale) through two fluid-filled channels in a patient with pulsatile neck veins. See text for details.

hemodynamics would be as routine and reliable as that of electrocardiography. However, as with any recording system that requires specially trained personnel and multiple combinations of different connector types, the mechanical and electrical artifacts of both fluid-filled tubes and high-fidelity recording instruments produce pressure wave artifacts that must be recognized as the major flaw in accurate hemodynamic data interpretation [21].

In the measurement of right atrial pressure, the most common artifacts include failure to match the zero positions or transducer gain sensitivity of the two fluid-filled systems. When tricuspid valve disease is suspected, precise calibration and equisensitivity of transducers are critical, because small gradients may have large clinical importance.

An increase in right atrial mean pressure during inspiration is a common indication (Kussmaul's sign) of physiologic abnormalities of atrial filling, especially prevalent in patients with constrictive or restrictive physiology. However, how does one explain the inspiratory increases in right atrial pressure in a 46-year-old woman with atypical chest pain without suspected pericardial disease? To the unknowing observer, this increase in right atrial mean pressure would be consistent with pathophysiology of constrictive pericarditis, but the mean right atrial pressure is only 4 mm Hg (Figure 1.24, top). It would be unusual for an asymptomatic, untreated person with significant constrictive physiology to have a low mean right atrial pressure. Whenever a suspected potentially erroneous physiologic event occurs during mean

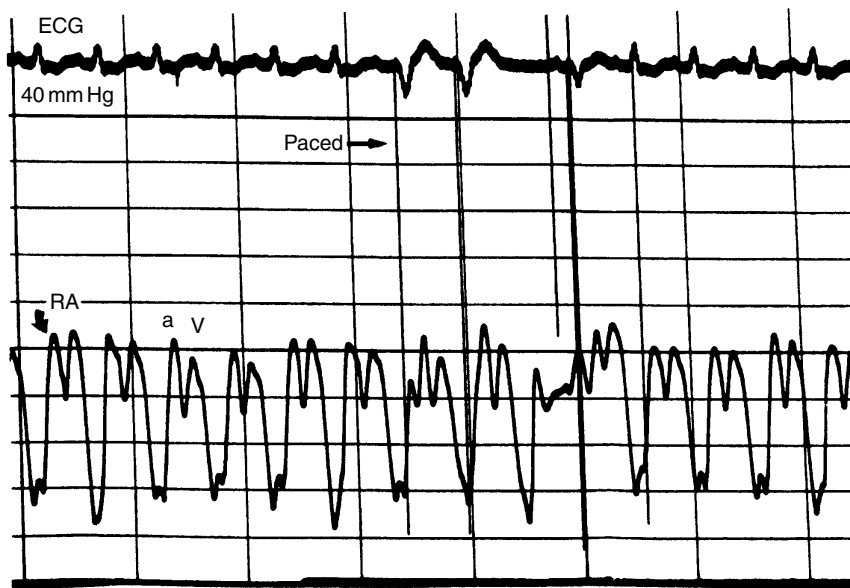


**Figure 1.21** Simultaneous right ventricular (RV) and right atrial (RA) pressures (0–50 mm Hg scale) through two fluid-filled channels in a patient with prominent waves in neck veins and varying murmurs. See text for details.

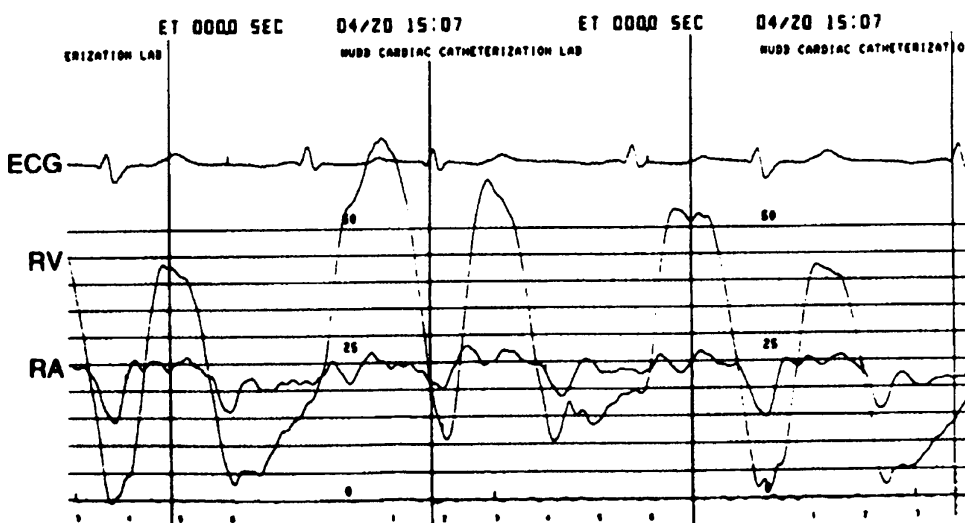
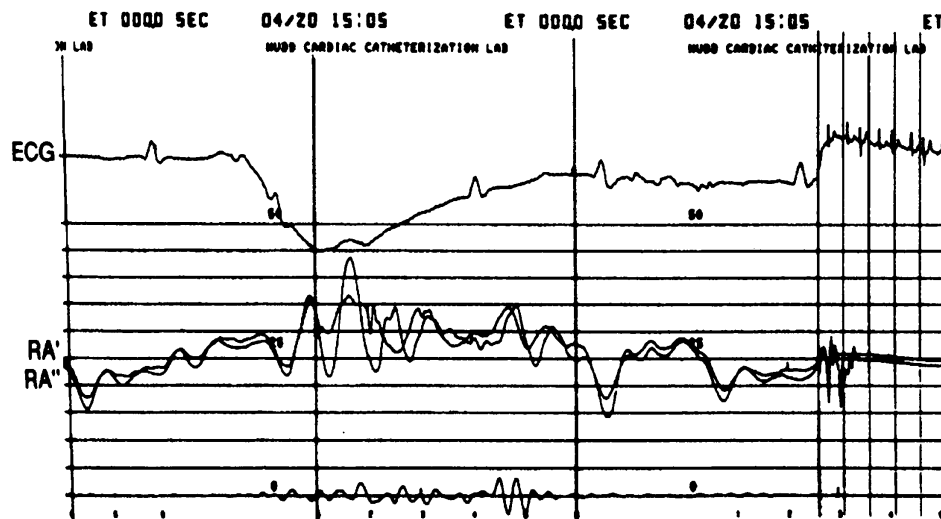
pressure recording, observe the phasic waveform generating this response. As can be seen on the lower panel of Figure 1.24, the phasic right atrial waveform is displayed during inspiration. The initial several beats demonstrate the normal fall in right atrial pressure with an increasing Y descent and then the catheter accidentally enters the right ventricle. The artifact of measuring right ventricular pressure during mean right atrial waveform recording is the explanation for an abnormal increase in right atrial mean pressure during inspiration in this patient, in whom pericardial disease was not present.

Another common and disturbing artifact of fluid-filled pressure systems, especially in measuring right atrial and other right-heart pressures, is that of excessive catheter

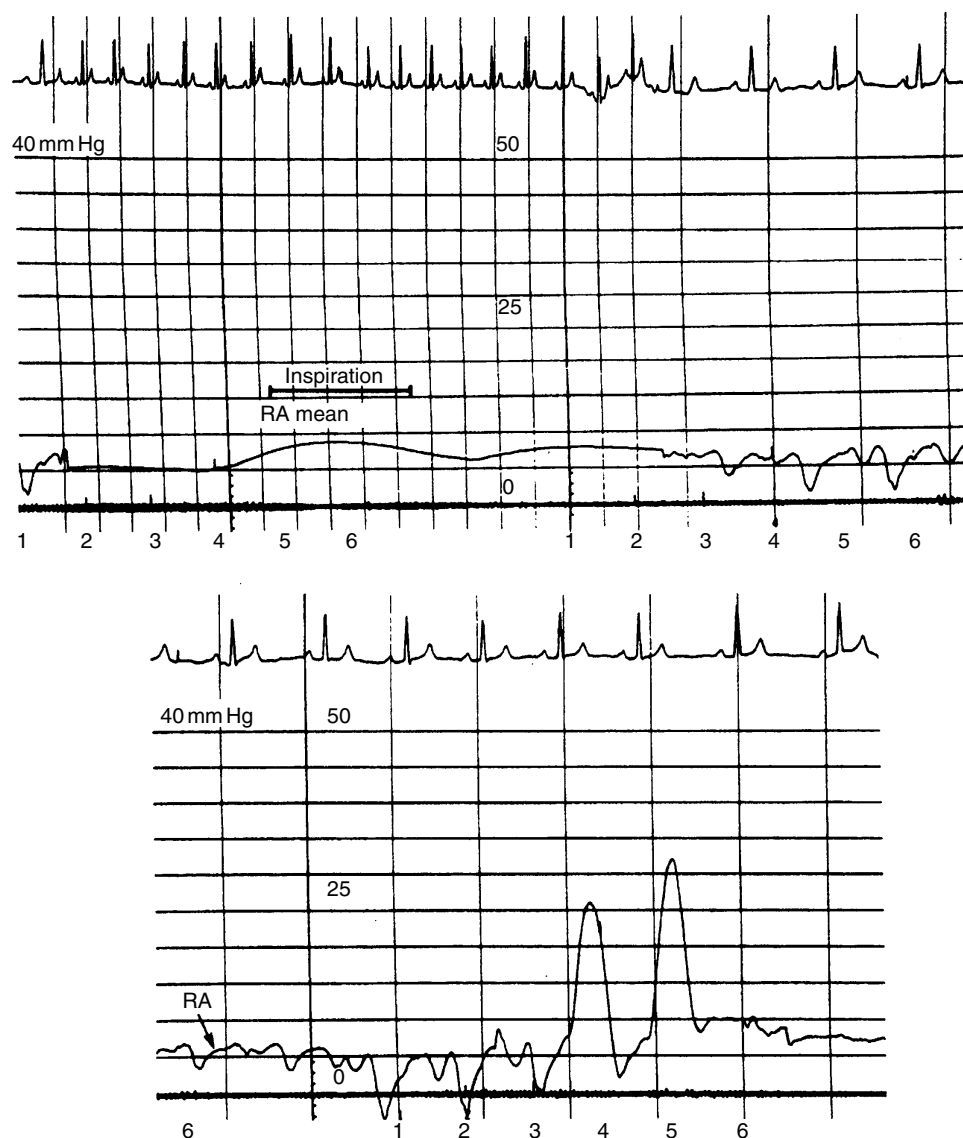
“fling” of an underdamped pressure (catheter, tubing, and transducer) system. This artifact is very common when using balloon-tipped pulmonary artery flotation catheters. The left side of Figures 1.25a and 1.25b shows right-heart pressures recorded prior to pressure system manipulation to reduce the underdamped signal. Interpretation of waveforms and other details of these pressure tracings cannot be discerned from the rapid high-frequency “ringing” artifact of the underdamped system. To improve hemodynamic recordings while continuously measuring pressure, a 50% saline and contrast solution was instilled through the catheter. The right-hand panel shows the distinct and strikingly elevated waveforms of a patient with congestive heart failure. The



**Figure 1.22** Right atrial (RA) pressure in a patient with dyspnea at rest (0–40 mm Hg scale). See text for details. *Source:* Grossman 1986 [10]. Reproduced with permission of Wolters Kluwer Health, Inc.



**Figure 1.23** Right ventricular (RV) and right atrial (RA) pressures (0–50 mm Hg scale) measured in a patient with severe ascites and peripheral edema. Left panel shows two matching fluid-filled transducers prior to crossing the tricuspid valve (0–50 mm Hg scale). See text for details.



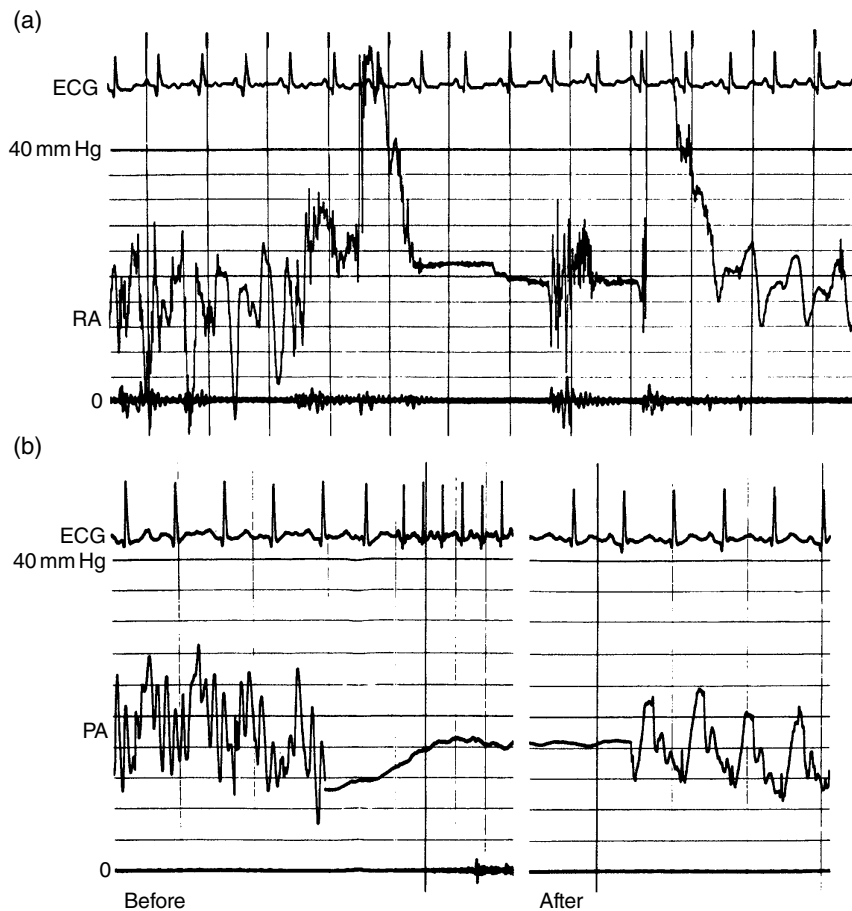
**Figure 1.24** Right atrial (RA) mean pressure during inspiration: Kussmaul's sign? See text for details.

small A and large V waves with the prominent Y descent are evident [22]. In the same patient, the catheter was flushed with saline and passed to the pulmonary artery, which also demonstrated a marked high-frequency ringing artifact. Contrast media was again instilled which correctly damped the pressure waveform, providing accurate identification of pulmonary artery pressures (Figure 1.25b, right-hand panel).

Whenever the damping characteristics of the fluid-filled systems are not satisfactory, maneuvers to improve the resonant characteristics should be conducted to achieve satisfactory pressure responses. Accurate determination of waveforms will aid in the determination of diseased conditions using pressure patterns.

## Pressure Wave Artifacts

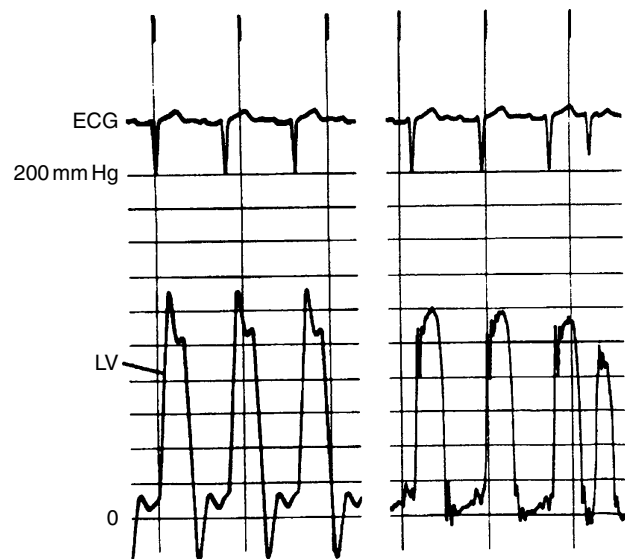
If the measurement of blood pressure were automatically available without having to attend to transducer flushing, pressure line, and manifold connections, as well as erroneous settings on the recorders or kinks in catheters, the study of hemodynamics would be as routine and reliable as that of electrocardiography. However, as with any recording system that requires specially trained personnel and multiple combinations of different connector types, the mechanical and electrical artifacts of both fluid-filled tubes and high-fidelity recording instruments produce pressure wave artifacts that must be recognized as the major flaw in accurate hemodynamic data interpretation.



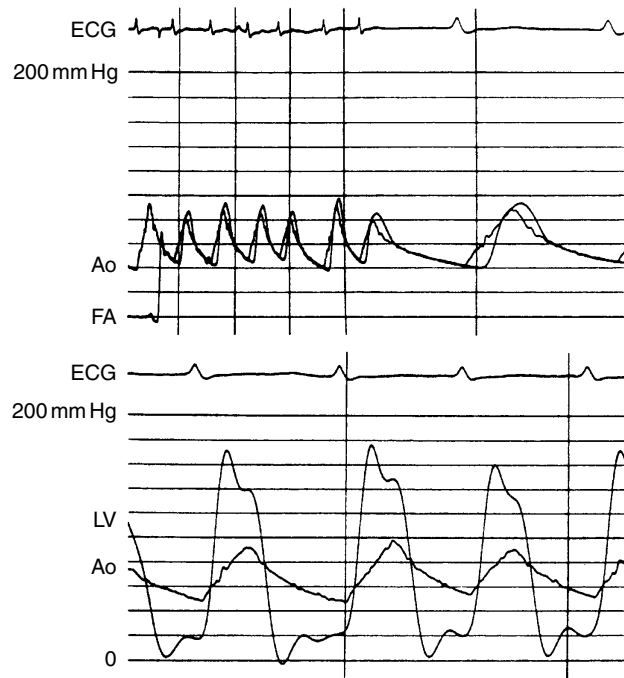
**Figure 1.25** (a) Right atrial (RA) pressure and (b) pulmonary artery pressure (G–40 mm Hg scale) with high-frequency artifact. Left panels are before and right panels after pressure system manipulation.

### Pressure System Resonance: The Under- and Overdamped Waveform

Probably the most common pressure wave artifacts of fluid-filled systems are the exaggerated ringing of underdamping and overly damped “rounded” waveforms. An underdamped pressure tracing is one in which the pressure wave is rapidly reflected within the system and produces an oscillating sinusoidal distortion of the pressure waveform [23]. This underdamped tracing is also called ringing, as in continued bounding of sound waves in a bell with a characteristic demonstration of the physics of reflected waves [24]. Commonly, an air bubble in the pressure line will be small enough to be rapidly accelerated and decelerated, moving the fluid column back and forth, resulting in ringing of the pressure tracing. The effect of a bubble in the pressure line connected to a 7 F pigtail catheter and fluid-filled transducer used to measure left ventricular pressure is illustrated in Figure 1.26, left panel. The bubble causes a high spike on the left ventricular upstroke and a large negative overshoot wave on the left ventricular pressure downstroke. When the pres-



**Figure 1.26** Left ventricular (LV) pressure with 7F pigtail catheter using fluid-filled transducers. Left panel demonstrates “ringing” artifact. See text for details.



**Figure 1.27** Overdamped left ventricle. (Top) Central and femoral (FA) pressures. (Bottom) Left ventricular (LV) and aortic (Ao) pressures in a patient with aortic stenosis. Note delay and unusual waveform in left ventricular pressure. See text for details.

sure line and transducer are properly flushed, the left ventricular pressure (Figure 1.26, right panel) shows an excellent and normal waveform, which should be expected with modern fluid-filled systems.

An overdamped waveform reduces the impact of the pressure wave, rounding the contours and delaying the upstroke and downstroke of the pressure wave. An example of pressure overdamping will be described later and is depicted in Figure 1.27.

## A Hemodynamic Data Collection Method

Hemodynamic studies require accurate data collection techniques. For complex cases, one must record simultaneous pressure waveforms, working with multiple transducers. Below are the steps commonly used to minimize artifactual pressure recordings.

### Step 1

Check the transducer connections to the recorder. This step is one of the greatest points of confusion and frustration in hemodynamic studies. It is not uncommon that the cables connecting the transducers to the table and the hemodynamic recorder are unplugged and

uncoiled, and scattered over the catheter table and floor. Numbering, labeling, and color-coding all cables and their inputs make reconnection easy and quick. In addition to clear labeling of the cables, an attachment device for the coiled cables to the tableside, such as Velcro strips or other tape, is also very helpful.

On the sterile field, it is also worth numbering and color-coding the transducers and tubing so that communications for recording the waveforms from the operators to the control room can proceed smoothly. For example, “zeroing transducer number 1; pressure is up on number 1; number 1 is a femoral artery pressure.” Clear communications are always helpful and this is the best method to reduce frustration, save time, and decrease confusion during hemodynamic measurements.

### Step 2

Set up the transducers on the sterile field or on the injector device. A small rack with transducer mounting brackets is placed opposite the operators and set at the patient’s mid-chest level. Transducers are connected electrically to the catheter table sockets, sending the signals to the recorder. The transducers are flushed with saline through plastic tubing to be connected to the catheters on the sterile field. The transducers are flushed to ensure that bubbles are eliminated. All connections to stopcocks and tubing should be flushed and tightened. If possible, the shortest and stiffest tubing should be used to produce the best pressure transmission pathway.

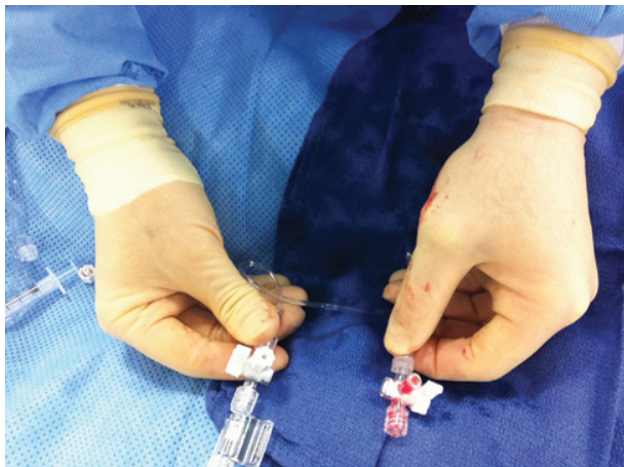
### Step 3

After flushing, the tableside transducers are zeroed at the mid-chest level of the patient. The transducers are opened to atmosphere; the recording technologist zeroes the signals on the hemodynamic recorder. The transducers are then closed. It may be necessary to reflush the transducers to be sure that they are free of air bubbles. Any remaining bubbles may produce underdamped pressure waveforms. This setup applies for multiple transducers and will provide accurate hemodynamic measurements for all cases.

### Technical Note

The ACIST power injection system (Eden Prairie, MN) has a pressure transducer mounted on a built-in bracket with rubber-padded backing through which the pressure is transmitted to an electrical sensor. Although the transducer on the ACIST device is accurate, because of the mechanical plate interface, the signal is delayed by about 50–100 msec relative to the ECG. The computer-measured left ventricular end-diastolic pressure may be inaccurate, since the timing mark of the R wave from

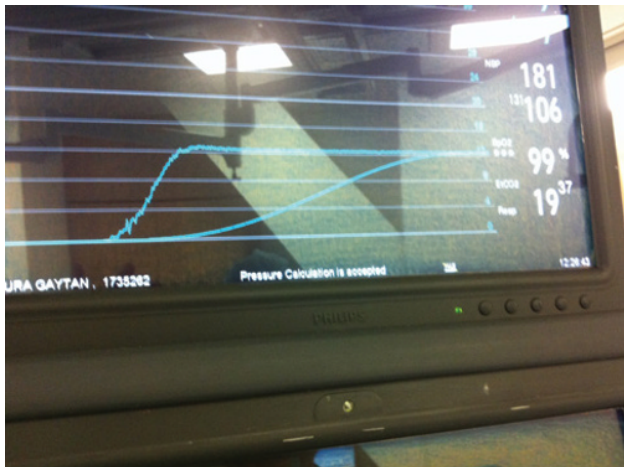
(a)



(b)



(c)



**Figure 1.28** (a) The transducer (left) is connected to a fluid-filled tube (right) and a stopcock, which is open to the air. (b) Lifting either the tubing as shown here or the transducer changes the pressure, but once zero is set with the position of the tubing, there is no need to adjust the transducer. Connect the tubing to the catheter and you are ready to measure. (c) A hemodynamic monitor showing the effect of first raising the tubing. The blue line goes up and then raising the transducer to the same level will produce the same effect.

the ECG is out of synchrony with the pressure wave (Figures 1.28, 1.29, and 1.30). For all simultaneous two-pressure hemodynamic studies, or any study in which high accuracy is desired, two tableside-mounted transducers may be preferred.

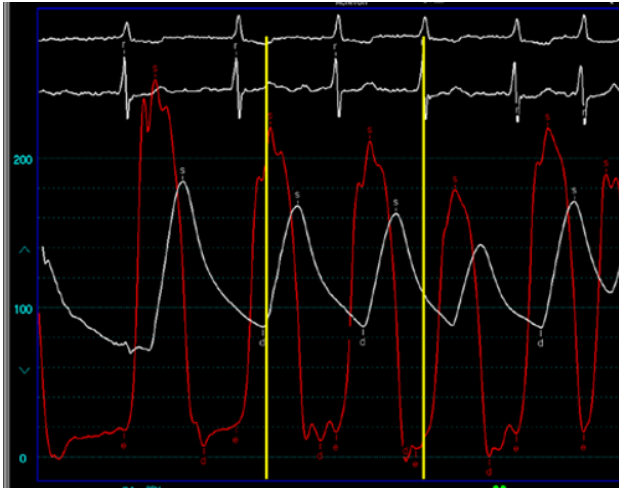
#### Step 4

Continuously review the pressure waveforms during acquisition. The waveforms should make sense for the catheter location, cardiac rhythm, and clinical situation. The pressure waveform should be timed correctly with the ECG and should be of an appropriate scale. For example, does an arterial pressure of 60/40 with a heart rate of 80bpm make sense in a perfectly comfortable, awake patient who is talking to you? First, check the patient. Then, check the scale factors on the recorder,

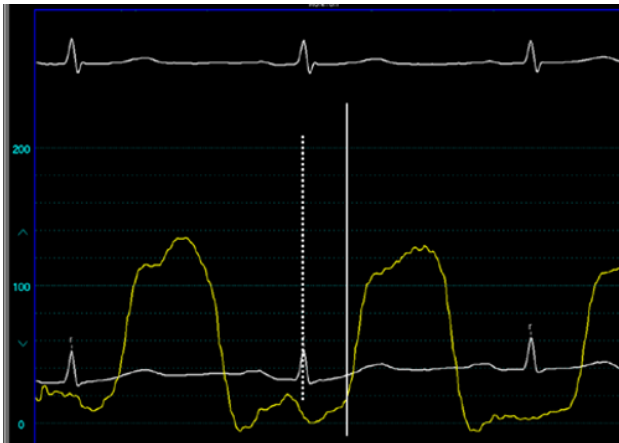
then the connections and tubing again, and whether or not the pressure transducer is connected to the left-sided or right-sided catheter. Errors like this are a common source of confusion among inexperienced personnel. Unusual waveforms should correlate to pathophysiology. If not, suspect some error in the recording technique, such as a loose connection, an air bubble, a clot in the line, a damped or kinked pressure tube or catheter, or a wrong recording scale. These checkpoints are necessary to record good-quality hemodynamics.

#### Technical Note on Using Simultaneous Right- and Left-Heart Hemodynamics

Usually, right-heart hemodynamics are performed in conjunction with left-heart hemodynamics to obtain a complete assessment of myocardial, pulmonary, and valvular



**Figure 1.29** Left ventricular and aortic pressures measured from table-side transducers. The vertical yellow lines indicate left ventricular end diastolic pressure (LVEDP) and timing to ECG. Note the delay in aortic pressure due to pressure transmission from the femoral arterial sheath-side arm. The computer has placed “d” on aortic diastolic pressures and “e” on LVEDP. The “e” position varies, but is mostly close to the true LVEDP. Scale 0–200 mm Hg. (See insert for color representation of the figure.)



**Figure 1.30** Left ventricular pressure recorded with an ACIST transducer, demonstrating delay in pressure relative to ECG. The R wave precedes the LVEDP by almost 100 msec. Manual selection of LVEDP is required. (See insert for color representation of the figure.)

function. Normally, when performing simultaneous right- and left-heart hemodynamics, a right-heart catheter is positioned in the pulmonary artery in the standard manner. After cardiac output is obtained, a pigtail catheter is positioned in the LV and LV hemodynamics and simultaneous wedge pressures are acquired to gauge mitral valve function. On pullback of the right-heart catheter, simultaneous RV and LV pressures are measured, especially useful in patients suspected of having constrictive or restrictive physiology. Finally, to evaluate aortic valve disease use a dual-lumen pigtail catheter or pressure wire and multi-purpose catheter. Right- and left-heart catheterizations

performed in this manner will provide a complete hemodynamic assessment in 95% of cases and provide an accurate understanding of aortic, mitral, tricuspid, and pulmonary valve disease with minimal extra maneuvers. These protocols have been described elsewhere [23]. Improving the precision, organization, clarity, and operational protocols will produce better hemodynamic data.

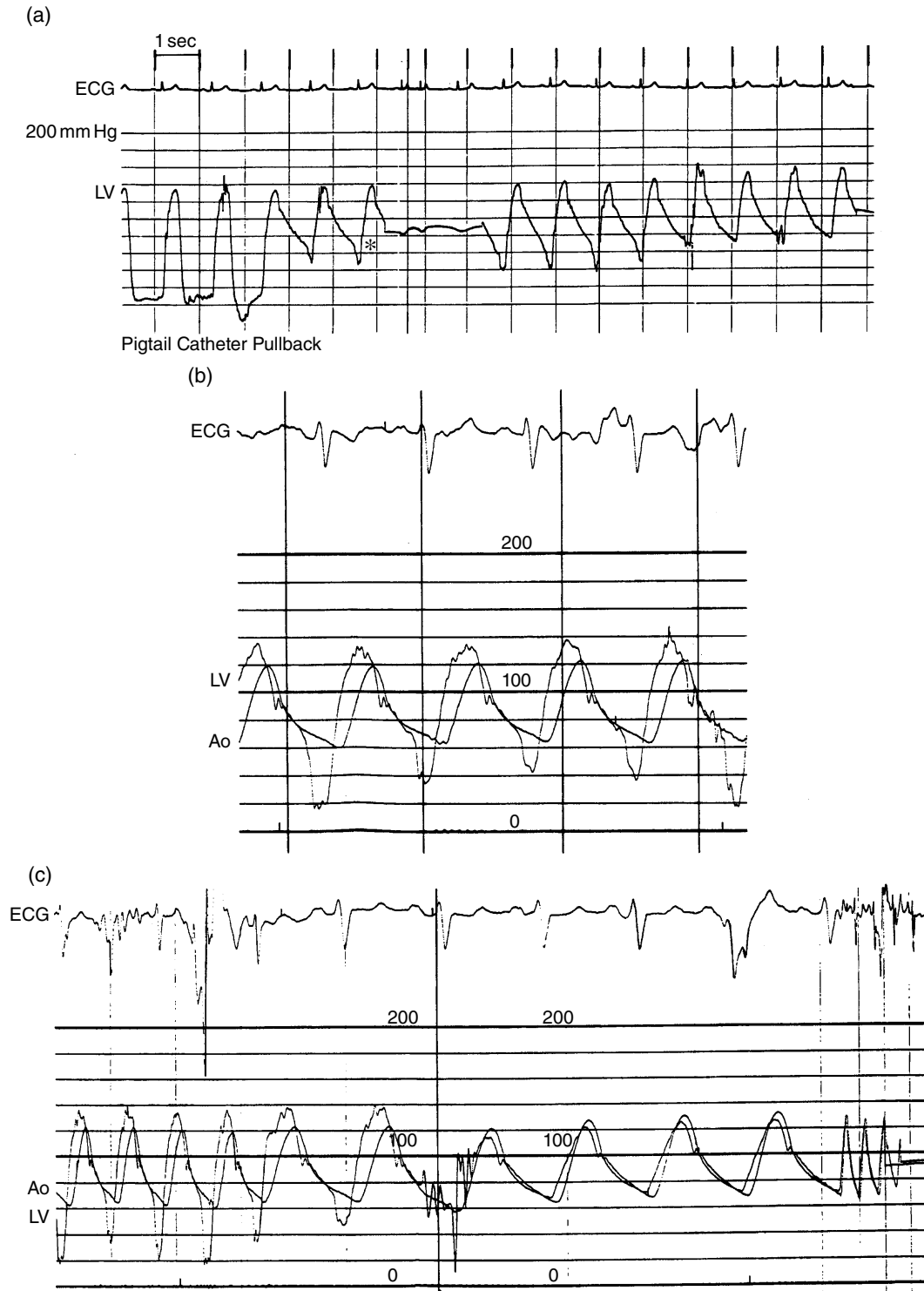
### Case of a Transiently Wide Pulse Pressure: Artifact or Episodic Aortic Insufficiency?

A continuous hemodynamic tracing was recorded during 7F left ventricular catheter pullback after ventriculography (Figure 1.31a). The pullback was easily and smoothly, but slowly, performed. The operator noted a wide pulse pressure which appeared to diminish over the next several beats. Should the operator reload for aortography to demonstrate transient aortic insufficiency? To a new student of hemodynamics, this mystifying physiology of waxing aortic insufficiency might require more study. However, a slow pigtail catheter pullback from the left ventricle might be incomplete, leaving a portion of the uncoiled pigtail in the left ventricle with several side holes still transmitting the lower left ventricular diastolic pressure falsely, reducing aortic diastolic pressure. This phenomenon is demonstrated again with simultaneous femoral arterial and pigtail catheter pressures (Figures 1.31b, c). The changing diastolic pressure is due to catheter movement, with a different number of pigtail catheter side holes moving across the aortic valve.

With complete catheter removal, two systemic pressures match without the diastolic pressure artifact (Figure 1.31c, right side). This artifact can be easily recognized by the unusual diastolic waveform, with a late diastolic shoulder and rapid dip differentiating it from a wide pulse pressure of valvular insufficiency.

### Case of a Late-Rising Central Aortic Pressure: Artifact or Pathology?

The most proximally measured aortic pressure wave rises before the pressure waves measured more distally. This constant physiologic requirement may be disturbed only by pressure waveform artifacts. Consider the hemodynamic data obtained in a 72-year-old man with aortic stenosis (Figure 1.27). Because of mild peripheral vascular disease, simultaneous pressures were initially obtained with a 6F femoral arterial sheath and a 5F pigtail catheter with fluid-filled transducers. The pressures measured before crossing the aortic valve demonstrated a good correspondence with two notable features: a slightly reduced femoral pressure overshoot consistent with mild peripheral vascular

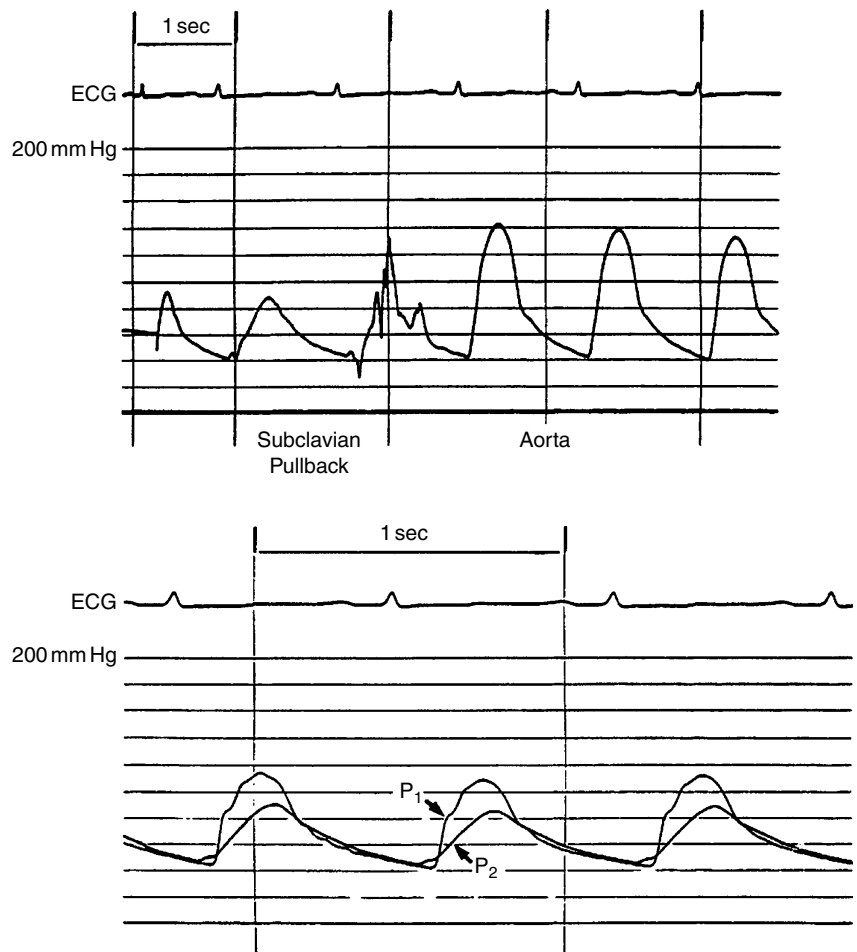


**Figure 1.31** (a) Left ventricular (LV) catheter pullback with transient aortic insufficiency. Note unusual diastolic waveform. See text for details. (b, c) LV and simultaneous aortic (Ao) pressures. See text for details.

disease and a slow central aortic pressure upstroke consistent with aortic stenosis. These pressure waveforms are acceptable for routine clinical use. Crossing the heavily calcified valve in the enlarged aortic root was

accomplished with the pigtail catheter and a 0.038" straight guidewire. Mild difficulty in advancing the pigtail catheter over the valve and into the left ventricle was encountered.

**Figure 1.32** (Upper panel) Catheter pullback from subclavian artery to aorta. Note gradient and waveform change. (Lower panel) Two central aortic pressures. P1 is in the aorta at the level of the left subclavian origin. Where is P2? See text for details.



Examine the simultaneous left ventricular and aortic pressures (Figure 1.27, lower panel). Why does left ventricular pressure rise after aortic pressure? The LV pressure is overly damped with a rounded contour. A delay in pressure transmission is caused by an LV catheter kink in crossing the valve that could not be eliminated by vigorous flushing. Pressure overdamping can be caused by inadequate flushing, leaving an air bubble or blood in the line, reducing the fidelity of pressure transmission. This problem may be exaggerated in small-diameter tubes and catheters. Increasing the fluid viscosity with contrast media would also produce the damped and delayed tracing. This artifact was eliminated by changing catheters, in this case to a 7 F or 8 F sheath with a 6 F or 7 F pigtail. A second arterial puncture or the transseptal approach as discussed elsewhere [25] would be an alternative solution. In Figure 1.27 (top panel), why is the central aortic (Ao) pressure different from the femoral pressure (FA)? The presence of peripheral vascular disease creates a gradient measurement between the central aortic pressure measured by a second small pigtail catheter placed in the ascending aorta and the original femoral artery catheter.

Another example of a late rising proximal aortic pressure is shown in Figure 1.32 (lower panel). P1 and P2 are pressure tracings from two fluid-filled catheters located in the thoracic aorta. P1 has a brisk upstroke with an anachrotic shoulder and dichrotic notch. P2 is an earlier rising tracing with a considerably slower upslope and attenuated resonant waveform characteristics. What conditions produced this pattern and from which locations are these pressures obtained? P1 is in the descending aorta below the left subclavian artery origin. P2 is inadvertently just beyond the ostial portion of the left subclavian artery, which had a significant narrowing, producing a 30–40 mm Hg systolic gradient, slow upstroke, and loss of the anachrotic shoulder and dichrotic notch. On pullback from the subclavian artery to the aorta, the systolic gradient and changing waveform are evident (Figure 1.32, upper panel). Subclavian stenosis and coarctation are the two conditions that can cause this pressure with catheters in the central aorta.

In the consideration of aortic coarctation (Figure 1.33A, top panel), one pressure waveform should be delayed with a slightly slower upstroke, but