HEMODYNAMIC ROUNDS
To Margaret and Anna Rose, the systole of my life. MJK
To Amy, Parker, and Taylor – the essential pieces to my life. MJL
To my wife Cindy, who keeps life fun while I am working. JAG
HEMODYNAMIC ROUNDS 2007

The following citations have been used in the chapters identified by chapter number. These chapters were originally published in *Catheterization and Cardiovascular Diagnosis* and comprise the basis for most of the chapters in *Hemodynamic Rounds*, Third Edition.


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PREFACE

As noted in the textbooks of cardiology, hemodynamics continue to be an integral part of the training experience and comprise validation for much of the pathophysiology obtained from clinical examination, echocardiographic study, and new imaging modalities. With the increased attention to visual medicine and angiography, the graphics of hemodynamics have been in decline. However, hemodynamics remain useful for diagnosis and treatment of the multitude of various and unusual cardiovascular conditions. It remains true that in today’s modern cardiology, hemodynamics are still critical to the diagnosis of valvular disorders and unusual cardiomyopathic conditions contributing to cardiac disability.

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 the journal of “Catheterization and Cardiovascular Diagnosis,” now renamed “Catheterization and Cardiovascular Intervention.” The case-based format limited itself to description of individual hemodynamic tracings, but was not presented in a formalized textbook fashion. The second edition of Hemodynamic Rounds extended this work and enlarged and reorganized it into new sections providing a more logical approach to the study of pressure waveforms and the associated pathology.

In the present edition of Hemodynamic Rounds, a further thematic approached to the understanding of pathophysiologic waveforms is provided. The text has been divided into 10 major parts (comprising Section I of this edition) incorporating the previously published works with new and dynamic tracings and incorporating the latest publications regarding hemodynamic topics as they have evolved into our modern practice.

Part 1 describes normal and pathophysiologic hemodynamic waveforms and is organized 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. LV end-diastolic pressure, simultaneous right- and left-heart pressures, and effects of nitroglycerin and pulsus alternans are also discussed.

Parts 2 and 3 cover valvular and valvuloplasty hemodynamics. In Part 4, constrictive and restrictive physiologic waveforms are described in detail. Cardiac arrhythmias are dealt with in Part 5. Hypertrophic obstructive cardiomyopathy is presented in Part 6. Coronary hemodynamics in Part 7 has also been expanded. The new concepts involving absolute and relative coronary reserve and pressure-derived fractional flow reserve are compared with the intent to help the practitioner understand practice in the laboratory on a daily basis. These findings can be used for decision-making during coronary angiography.

Parts 8 and 9 deal with particularly unusual hemodynamic problems involving adult congenital anomalies and hemodynamics, extra hearts and transplants, intra-aortic balloon pumps, and circulatory assist devices. Finally, in Part 10, right ventricular infarction is described by one of the world’s experts, Dr. Goldstein.

As a new and important aspect of hemodynamic rounds, Dr. Goldstein (in Section II of this edition) has undertaken the compilation of clinical and bedside applications of hemodynamics describing the correlation between 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. I continue to thank Dr. Frank Hildner, first editor and founder of Catheterization and Cardiovascular Interventions, formerly Catheterization and Cardiovascular Diagnosis for his involvement with this work, without whom this book would never have been published.

I would like to thank Margaret and Anna Rose, the continuing systole of my life as noted in our first edition, and I would like to extend my deepest appreciation to my co-editors and contributors to this work and to my fellows in training without them, there would be no point in these exercises.

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INTRODUCTION

MORTON J. KERN, MD, AND FRANK J. HILDNER, MD

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]. Mr. Hales took his place in medical history next to William Harvey with regard to studies of the human and animal circulation. De Motu Cordis [2] and Haemastaticks stimulated scientists interested 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 interest.

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 and correspond to currently accepted data obtained by computerized 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 and repeatedly confirmed computations. The numerous original achievements in hemodynamics provided to us by Hales are remarkable even by today’s standards and included the first direct and accurate measurement of blood pressure in different animals (see Figure) 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. Stephen 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. Hales 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 the observations of Hales was lacking, understanding the unique collection of data depends on interpreting descriptive material.

Our current appreciation of hemodynamics as provided in this book stems directly from a small group of modern physiologists active in the 1920s, among whom Dr. Carl Wiggers, from Western Reserve University in Ohio, emerges. Major advances in hemodynamic research arose from the development of recording instruments with improved fidelity able to capture and reproduce the waveforms of rapidly changing pressures during the various phases of cardiac contractions in the various heart chambers. 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 and relationships which are the benchmark against which the observations of the pressure tracings of classical diseased conditions, some of which are described herein, can be compared [3].

An interval of sixty years separates the originators of clinical cardiovascular anatomy and physiology from present day practitioners. What happened during that time should not be forgotten because it still affects us today. However, some of the lessons that have been learned, while still valid, tend to be ignored. From the time Claude Bernard coined the phrase “cardiac
catheterization” (1840) [4], laboratories of that type and name have been hemodynamic and physiology laboratories. Mter Forssman performed the first documented human cardiac catheterization on himself [5]—the nature of the work did not change, only the subjects. In the late 1930s, Cournand and Ranges [6] used the new right heart catheterization technique to investigate pulmonary physiology. With World War II, the scope and direction of their work changed to include hemorrhagic shock and drug effects on the circulation. But in those days, the most serious problems presented by patients related to congenital and rheumatic heart disease. Accordingly, laboratories around the world began publishing data on the hemodynamics and physiology of atrial septal defects [7], ventricular septal defects [8], stenotic and insufficient mitral and aortic valves and ventricular function. The beginning of invasive cardiology had come to an end.

Without doubt, the most important and crucial development needed for the advancement of the field of cardiovascular diseases was the cathode ray tube, a direct result of the war. Before the image intensifier [9, 10], cardiac fluoroscopy utilized high-dose radiation and required the physician to accommodate his 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 [11]. Without the
additional light provided by the image intensifier, “angiocardiography” was nothing more than a simple flat-plate radiograph, or perhaps a sequence of cut films obtained on the newly developed serial film changer [12]. Cineangiography was developed in the late 1950s through the persistent efforts of lanker (1954) [13] and Sones (1958) [14]. The addition of advanced imaging spurred the progress of catheter invasive techniques, which then permitted investigation of heretofore unapproachable anatomical sites, clinical conditions, and disease entities which in turn resulted in effective cardiac surgery. Once again, hemodynamic analysis was needed to explain what was being newly observed and to assist the development of medical and surgical interventions. After the basic mechanics of congenital anomalies and rheumatic abnormalities were confirmed, conditions related to occlusive coronary artery disease such as myocardial infarction, left ventricular aneurysms, mitral chordal, and septal rupture were investigated. Soon thereafter, newer concepts of systolic and diastolic myocardial mechanical function, hypertrophic obstructive and nonobstructive cardiomyopathy, electrophysiology relations, and other previously unappreciated conditions came under scrutiny. The final result was a body of knowledge that permitted development and use of the newly conceived noninvasive techniques including advanced physical examination (phonocardiographs, ballistocardiographs, etc.), exercise stress testing, radionuclide imaging, and echocardiography. In this age of imaging, even as it was during the previous fifty or sixty years, hemodynamic analysis remains absolutely necessary for a proper understanding and appreciation of all cardiovascular conditions and situations.

APPROACH TO HEMODYNAMIC WAVEFORM INTERPRETATION

With this background, we turn our attention from pressure waveforms to the interpretation of cardiac pathophysiology. Each chapter has been published or will soon appear in Catheterization and Cardiovascular Diagnosis and will serve to provide both novice and advanced cardiologists with classical and, at times, unique pressure tracings to emphasize the value of careful observation as the waveforms relate to different cardiac pathophysiology states.

It is clear that good-quality hemodynamic data are required for the quantitative determinations of physiologic conditions for most cardiovascular maladies. As in the days of Dr. Hales, some hemodynamic data are extraordinarily simple, such as using a sphygmomanometer for indirect assessment of systemic arterial pressure. Some hemodynamic data may also be complex, requiring catheterization with placement of multiple catheters within several chambers of the heart. Such data can then be used in the precise computations of pressure and flow to determine valvular gradients, myocardial contraction, relaxation, compliance, impedance and work [15–17]. Additional techniques, unknown to physiologists and cardiologists in decades earlier, have recently provided insight into the physiology of the coronary circulation. Intracoronary Doppler and vascular ultrasound imaging catheters can now provide information complementary to but previously unavailable through traditional angiographic methodologies.

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 has been 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 missing easily overlooked data collection steps. Time is also saved during procedure setup and data recording. The technical staff does not have to rethink what will happen for a unique and personal hemodynamic protocol of each different operator. Right-heart catheterization, sometimes performed sequentially with left-heart catheterization, may often be combined simultaneously with left-heart catheterization 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 [18], but the indications have become a subject for controversy [18, 19]. While some quarters feel routine right-heart catheterization is unjustified, others are equally adamant that patient care demands our maximum effort to provide optimum results. Unexpected congenital and hemodynamic abnormalities are found at right heart catheterization even with previous echocardiography. This has been pointed out by Shanes et al. [20] and Barron et al [21], even though they come to opposite opinions. However, there is no debate if right-heart catheterization is performed to evaluate patients with previous congenital heart disease, valvular heart disease, left- or right-heart failure, previous myocardial infarction,
cardiomyopathy, or any unexplained significant clinical historical or physical findings.

Left-heart hemodynamic protocols most often use a single pressure transducer, but simultaneous measurements of left ventricular and arterial pressure can easily be obtained through the side arm of an arterial sheath and the smaller catheter residing within using two transducers. Pressure obtained from an arterial sheath is satisfactory when at least a one French size larger sheath than the arterial catheter is used. After collecting the hemodynamic data, computations are made to clarify and enhance quantitative cardiac function. Measurements of cardiac work, calculation of flow resistance, valve areas, and shunt calculations are based on accurate hemodynamic data, arterial and venous blood oxygen saturations, and cardiac output determinations.

If the information is considered important enough to perform hemodynamic measurements, the operators should take the time to obtain pressure waveforms that are reliable and unequivocal, separating artifact from 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. The following section will highlight the important considerations for equipment used in daily hemodynamic measurements.

EQUIPMENT FOR HEMODYNAMIC STUDIES

A set of transducers, tubing and manifolds are employed for hemodynamic measurements which should be cost efficient, familiar, accurate, and simple to use for the laboratory. Although a variety of manifolds exist which are both disposable and reusable, the variety of transducers, tubing, and injection syringes should be cost efficient and easy to operate. Optimal hemodynamic pressure waves should be properly damped to reduce sinusoid “ringing” or overshoot artifact. Short, stiff tubing with a minimal distance from the end of the catheter to the transducer is desirable. Long tubing contributes to poor-quality tracings, introducing “fling” artifact due to the momentum of fluid through the tube. The zero position for hemodynamic measurements is also important. In some laboratories, the zero level is set at mid-chest, measured in the AP diameter of the patient (divided by 2) with the transducer connected by a fluid-filled tube to the zero level fixed at the table. When the transducer is raised above the zero level, pressure is lower. When the transducer is lower than the zero level, the pressure is higher. Setting an accurate zero before and at the conclusion of each measurement is minimally time-consuming and assures accuracy by eliminating recordings with erroneous zero baselines or transducer systems that have zero drift errors over time. The zero position at the mid-chest level can also be obtained by using two fluid-filled tubes connected to transducers. One tube is placed on top of the chest and the other at the back. The zero line manifold is then set at bedside height so that the two pressures are equidistant from this height. Artifacts related to under- and overdamping and suggestions to reduce these artifacts are described in Chapter 2.

Pressure Transducers

For most laboratories, table-mounted fluid-filled transducers produce acceptable clinical studies. Other devices are available which are suitable for special situations or requirements. Among these are miniaturized transducers mounted on the pressure manifold or placed in the pressure line. Some are disposable which obviates the need for sterilization but also adds to the cost. Other transducers are mounted on the end of the catheter and are inserted into the vessel or chamber being studied. These can be zeroed but require another pressure-sensing device for calibration. The specialized reusable micromanometer transducer-tipped catheters producing high-fidelity pressure recordings are required in the computation of the rate of rise of pressure with time ($dP/dt$) or relaxation ($-dP/dt$). Catheter reuse requires careful and meticulous cleaning, which is difficult and tedious at best. The cost of these devices usually prevents use in other than investigational pursuits.

Pressure Manifolds

Three- and four-port manifolds are available in disposable or reusable plastic configurations. In general, most laboratories set the first three ports for pressure, flush solution, and radiographic contrast media. A four-port manifold is also available and offers the advantage of an attached fourth port closed system for disposal of flush solutions. The waste fluid port (fourth port) minimizes contamination of personnel and laboratory equipment. The clear plastic manifolds are safe, practical, and disposable.

Physiologic Recorders

Every laboratory is equipped with a physiologic recorder with a multichannel photographic oscilloscope, electrocardiographic and pressure amplifiers and hard-copy printer capability. Most now use analog-to-digital signal converters to store and reproduce waveforms. A variety of specialized amplifiers (e.g., green dye curve calculators or signal differentiators) permit additional data collection. Multichannel (2–20 channels) units can
process, display, and record electrocardiographic, pressure signals and direct inputs from a variety of external sources. Although the number of recorded channels may be less than the number that can be displayed, for routine cardiac catheterization at least one electrocardiogram and one to three pressure signals are required. In complex cases such as electrophysiologic studies, congenital, valvular heart disease or hemodynamic research studies it is common to use between 6 and 18 channels. The physiologic recorder should be set up with amplifiers calibrated to reference pressure or voltage standards before each case. After the recorder is ready, pressure transducers are calibrated to a common pressure source. Differences in amplifiers or transducers can then be easily identified.

Recording artifacts may be responsible for confusing data. Examples of recording artifacts producing abnormal hemodynamic tracings are included in several chapters. The recording technician should demonstrate pressure scale changes and ensure correct time-line positioning to assist the physician in observing and collecting accurate and complete information.

CARDIAC OUTPUT METHODOLOGY

Critical to the calculations of nearly all hemodynamic data (systemic and pulmonary vascular resistances, as well as valve areas) is the accurate determination of cardiac output. The two methods most widely accepted for determining cardiac output have been reviewed [15, 16]. The Fick method assesses oxygen consumption with a polarographic cell or Douglas bag and blood oxygen saturations. The second method is indicator dilution technique, most commonly employing room temperature or iced saline using cold as the indicator. Green dye cardiac output curves are equally accurate but no longer used. Methods and limitations of these techniques have been described in detail [22]. The operators in the cardiac catheterization laboratory should familiarize themselves with the limitations and potential sources of error with both techniques.

REVIEWING WAVEFORMS

Pressure waveforms may be confusing for the cardiovascular fellow-in-training. After an intense training period in which the components of all pressure waves found in cardiovascular structures are incorporated, the young physician must be encouraged to continue practicing pattern recognition and deductive analysis. He should continue to strengthen his skills by performing systematic analysis of complete pressure data obtained on all indicated cases. This systematic examination includes a comparison of the pressure values across valves, an analysis of the pressure in all adjacent chambers, and the determination of whether the abnormalities are internally consistent with the clinical questions to be addressed. Finally, pressure calculation of resistance values and valve areas need to be confirmed (manual calculations to verify computer-managed data will, at times, be required). When reviewing physiologic tracings, every operator, whether expert or novice, should consider the following key points.

First, 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. Next, 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 right atrial A and V waves are commonly deformed by various arrhythmias, valvular disease, or pericardial and respiratory pathophysiologic states. Right and left ventricular waveforms are generally unaffected by most diseases, but the rate and position of the upslope and downslope of the pressure waves (relative to each other) should be brisk and characteristic. Electrocardiographic conduction abnormalities may alter the activation sequence of ventricular pressure. The presence of an exaggerated A wave in the ventricular tracings may identify chamber stiffness increased above normal limits. The early appearance of the A wave may also indicate first degree AV conduction block, a commonly observed phenomenon. Pressure artifacts should then be differentiated from true pathophysiologic waveforms. The type of artifacts due to catheter flogging, over or underdamping will be discussed in Chapter 2.

Finally, the interpretation of the waveforms should be made 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 and consideration of possible mechanisms of the various waveform phenomena forms the basis for the chapters in this book. The information will hopefully enhance the reader’s appreciation of seemingly trivial, but often important confirmatory data for
patients in discovery and confirmation of their cardiac pathophysiology.

REFERENCES

SECTION I

FUNDAMENTALS AND CLINICAL APPLICATIONS OF HEMODYNAMICS:
UNDERSTANDING THE PRESSURE WAVES IN THE HEART: THE WIGGER’S DIAGRAM

Everything you want to know about hemodynamics starts here. 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 ECG and corresponding pressure tracing.

The ECG “P” wave, the QRS, and the “T” wave are responsible for atrial contraction, ventricular activation, and ventricular relaxation, respectively. 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 are disturbed by arrhythmia, cardiac function is inefficient or ineffective as demonstrated on the various pressure waveforms associated with the arrhythmia.

The cardiac cycle is begun with the P wave. The P wave is the electrical signal for atrial contraction. The atrial pressure wave (“A” wave, point #1 in Figure I.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 LV pressure after the “A” wave is called the end-diastolic pressure. 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 both the LV and RV pressures increase rapidly. This period with 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, the heart begins relaxing and repolarization starts, there by generating a “T” wave. At 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 is concluded and diastole is underway. After aortic valve closure the ventricular pressure continues to fall. When the LV pressure falls below the 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.

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

To appreciate valve function and dysfunction, we study the pressure changes that normally open and close the valves. The aortic and pulmonary valves open in

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systole, when ventricular pressure exceeds aortic pressure (and RV exceeds PA pressure). Stenosis of these valves produces high-velocity systolic 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 failed 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 Wigger’s diagram to determine what the expected normal hemodynamic responses should be.

The Wigger’s diagram with periods of systole, diastole, and isovolumetric contraction and relaxation periods identified on Figure I.2.
A normal right atrial (RA) and pulmonary capillary wedge (PCW) pressure tracing (used as left atrial pressure) is shown in Figure I.3 demonstrating normal “A” and “V” waves with the associated “x”- and “y”-descents.