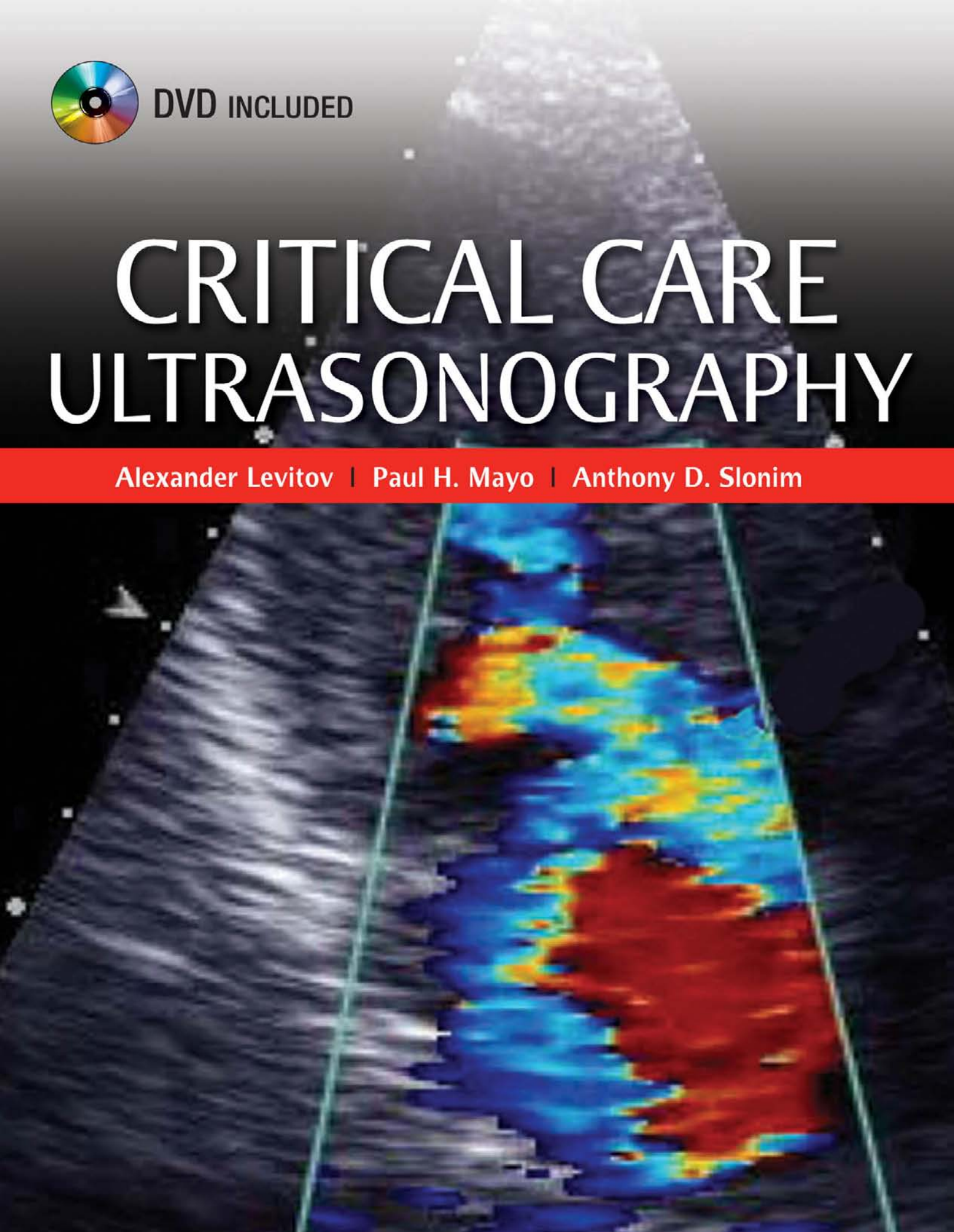

DVD INCLUDED
CRITICAL CARE
ULTRASONOGRAPHY
Alexander Levitov | Paul H. Mayo | Anthony D. Slonim

CRITICAL CARE ULTRASONOGRAPHY

Notice

Medicine is an ever-changing science. As new research and clinical experience broaden our knowledge, changes in treatment and drug therapy are required. The authors and the publisher of this work have checked with sources believed to be reliable in their efforts to provide information that is complete and generally in accord with the standards accepted at the time of publication. However, in view of the possibility of human error or changes in medical sciences, neither the authors nor the publisher nor any other party who has been involved in the preparation or publication of this work warrants that the information contained herein is in every respect accurate or complete, and they disclaim all responsibility for any errors or omissions or for the results obtained from use of the information contained in this work. Readers are encouraged to confirm the information contained herein with other sources. For example and in particular, readers are advised to check the product information sheet included in the package of each drug they plan to administer to be certain that the information contained in this work is accurate and that changes have not been made in the recommended dose or in the contraindications for administration. This recommendation is of particular importance in connection with new or infrequently used drugs.

CRITICAL CARE ULTRASONOGRAPHY

Alexander Levitov, MD, FCCM
Senior Staff, Department of Medicine
Carilion Clinic
Professor, Medicine
Virginia Tech-Carilion School of Medicine
Roanoke, Virginia

Paul H. Mayo, MD, FCCP
Director MICU
Long Island Jewish Medical Center
New Hyde Park, New York
Professor of Clinical Medicine
Albert Einstein College of Medicine
Bronx, New York

Anthony D. Slonim, MD, DrPH, FCCM
Vice President, Medical Affairs
Carilion Roanoke Memorial Hospital
Senior Staff, Departments of Internal Medicine and Pediatrics
Carilion Clinic
Roanoke, Virginia
Professor, Medicine and Pediatrics
Virginia Tech-Carilion School of Medicine
Roanoke, Virginia

New York Chicago San Francisco Lisbon London Madrid Mexico City
Milan New Delhi San Juan Seoul Singapore Sydney Toronto

ISBN: 978-0-07-171441-9

MHID: 0-07-171441-3

The material in this eBook also appears in the print version of this title: ISBN: 978-0-07-159297-0, MHID: 0-07-159297-0.

To Dr. Alexandra Arcus (1896–1981), Veda, Marguerite who made this book possible, and Irina, who makes everything possible.
A.L.

To my wife Charlotte Malasky, MD for all of her patience and support.
P.H.M.

To Terry, Michael, and Samantha . . . thanks for your love, support, and devotion.
A.D.S.

This page intentionally left blank

Contents

Contributors

Olivier Axler, MD, PhD, FCCP
Cardiovascular Department
Centre Hospitalier Territorial Gaston Bourret
Noumea, New Caledonia, France

Sameh Aziz, MD, FCCP
Senior Staff, Department of Medicine
Carilion Clinic, Roanoke, Virginia

Narinder P. Bhalla, MD
River Region Cardiology, Montgomery, Alabama

Michael Blaivas, MD
Professor of Emergency Medicine
Associate Professor of Internal Medicine
University of South Carolina, Columbia, South Carolina
Northside Hospital Forsyth
Department of Emergency Medicine, Atlanta, Georgia

William J. Brunelli, Jr., MPAS, RDMS, RDCS, PA-C
Radiology Associates of Roanoke, P.C.
Interventional Radiology
Lewis Gale Medical Center, Salem, Virginia

Christian Butcher, MD, FCCP
Assistant Clinical Professor of Medicine
Department of Medicine, University of Virginia
Charlottesville, Virginia
Assistant Professor of Clinical Medicine
Department of Medicine
Virginia College of Osteopathic Medicine
Blacksburg, Virginia
Pulmonary and Critical Care Faculty
Department of Medicine, Carilion Clinic
Roanoke, Virginia

James S. Cain, MD, FACP
Clinical Assistant Professor of Medicine
University of Virginia; Clinical
Assistant Professor of Medicine
Edward Via School of Medicine, Virginia Tech
Chief of Medicine Department
Medical Director for Dialysis Services
Carilion Roanoke Memorial Hospital, Roanoke, Virginia

Alan Cook, MD
Trauma Surgeon, East Texas Medical Center
Tyler, Texas

Peter Doelken, MD
Associate Professor, Division of Pulmonary
Critical Care, Allergy, and Sleep Medicine
Medical University of South Carolina
Medical University Hospital
Division of Pulmonary
Critical Care, Allergy, and Sleep Medicine
Charleston, South Carolina

Lewis Eisen, MD
Assistant Professor of Medicine
Division of Critical Care Medicine
Albert Einstein College of Medicine
Attending Physician, Division of Critical Care Medicine
Montefiore Medical Center, Bronx, New York

James E. Foster, II, MD, FACS, RVT
Asst. Professor of Clinical Surgery
University of Virginia
Medical Director, Noninvasive Vascular Laboratory
Carilion Clinic, Roanoke, Virginia

Heidi L. Frankel, MD
Professor of Surgery
University of Texas Southwestern, Dallas, Texas

Martin Goldman, MD
Professor, Medicine, Department of Cardiology
Mount Sinai School of Medicine
New York, New York

Adolfo Kaplan, MD
Pulmonary and Sleep Center of the Valley
Weslaco, Texas

Seth Koenig, MD
Attending Physician, Division of Pulmonary
Critical Care, and Sleep Medicine
Long Island Jewish Medical Center
New Hyde Park, New York

Pierre Kory, MD, MPA
Attending Physician
Division of Pulmonary and Critical Care Medicine
Beth Israel Medical Center, New York, New York

Alexander Levitov, MD, FCCM
Senior Staff, Department of Medicine, Carilion Clinic
Professor, Medicine
Virginia Tech-Carilion School of Medicine
Roanoke, Virginia

Julien Maizel, MD
Unite de Reanimation Medicale
Service de Nephrologie, CHU Sud, Amiens, France

Anthony R. Manasia, MD
Associate Professor of Surgery and Medicine
Department of Surgery, Mount Sinai School of Medicine
New York, New York

Paul H. Mayo, MD, FCCP
Division of Pulmonary, Critical Care, and
Sleep Medicine, Long Island Jewish Medical Center
New Hyde Park, New York

Dorothea McAreavey, MD, FACC
Critical Care Medicine Department
National Institutes of Health
Bethesda, Maryland

Marc Mikulski, MD
Anesthesiology and Critical Care Department
Centre Hospitalier Territorial Gaston Bourret
Noumea, New Caledonia, France

John M. Oropello, MD
Professor of Surgery and Medicine
Department of Surgery
Mount Sinai School of Medicine
New York, New York

Amitabh Parashar, MD
Assistant Professor
University of Virginia School of Medicine
Department of Internal Medicine
Charlottesville, Virginia
Assistnat Professor, Department of Internal Medicine
Virginia Tech Carilion School of Medicine
Carilion Roanoke Memorial Hospital, Roanoke, Virginia

Renée J. Roberts, MD
Assistant Professor of Anesthesiology and Pediatrics
Director of Anesthesia Support Services
Children's National Medical Center
The George Washington University Medical Center
Washington, DC

Rodney W. Savage, MD
Consultants in Cardiology, Roanoke, Virginia

Yefim R. Sheynkin, MD, FACS
Associate Professor of Clinical Urology
Department of Urology
State University of New York at Stony Brook
Department of Urology
Stony Brook University Medical Center
Stony Brook, New York

Michel Slama, MD
CHU Sud, Unité de Réanimation Médicale
Service de Néphrologie, Amiens, France

Anthony D. Slonim, MD, DrPH, FCCM
Vice President Medical Affairs
Carilion Roanoke Memorial Hospital
Senior Staff
Departments of Internal Medicine and Pediatrics
Carilion Clinic, Roanoke, Virginia
Professor, Medicine and Pediatrics
Virginia Tech-Carilion School of Medicine
Roanoke, Virginia

Balachundhar Subramaniam, MD
Assistant Professor, Department of Anesthesiology
Harvard Medical School
Director of Cardiac Anesthesia Research
Beth Israel Deaconess Medical Center
Boston, Massachusetts

Santhanam Suresh, MD, FAAP
Director of Research, Children's Memorial Hospital
Associate Professor of Anesthesiology and Pediatrics
Northwestern University
Feinberg School of Medicine, Chicago, Illinois

Daniel A. Sweeney, MD
Critical Care Department
National Institutes of Health, Bethesda, Maryland

Daniel Talmor, MD, MPH
Associate Professor of Anesthesia
Harvard Medical School
Director of Trauma Anesthesia and Critical Care
Department of Anesthesia and Critical Care
Beth Israel Deaconess Medical Center
Boston, Massachusetts

William Tsai, MD
Attending Physician
Critical Care Medicine and Emergency Medicine
Department of Pediatrics
Levine Children's Hospital at Carolinas Medical Center
Charlotte, North Carolina

Marguerite Underwood, RN, RDCS
Echocardiography Department
Carilion Clinic, Roanoke, Virginia

Antoine Vieillard-Baron, MD
Professor of Medicine
Université de Versailles Saint
Quentin en Yvelines, France
Hôpital Ambroise Paré
Intensive Care Unit, Boulogne, France

David A. Vitberg, MD
Emergency Department
Baltimore Washington Medical Center
Glen Burnie, Maryland

Kevin Wiseman, BS, RVT, RDMS
Non-invasive Vascular Laboratory
Carilion Clinic, Roanoke, Virginia

This page intentionally left blank

Foreword

As technology improves diagnosis and treatment, physicians are either "early adopters" or those who wait to see if a new approach is really better than the current standard. The "adopters" are challenged with becoming proficient in new skills, while those who resist need to be convinced that change is called for, and that it justifies the costs of time and money. Buying new equipment always provokes discussion of who pays for it, and what must be sacrificed. Finally, a new program may step on someone else's turf. There are disputes about who is best qualified to use the technology, along with calculations of the financial impact on existing programs and the potential legal liability of using new methods.

The introduction of ultrasonography into frontline critical care medicine is a case in point, and obstacles to its current wide use are similar to those that appeared with the introduction of other new technology that ultimately revolutionized the practice of pulmonary and critical care medicine. In the 1970s, flexible bronchoscopy gave pulmonary and critical care physicians a tool to expand their diagnostic abilities beyond the history, physical examination, and the chest radiograph. They had to learn how to use the bronchoscope, find the money to buy the equipment, and overcome the objections of many otolaryngologists and thoracic surgeons who strongly believed that people from internal medicine backgrounds had no business endoscoping the airways, no less performing biopsies of the airway and lung parenchyma. Of course, this resolved over several years, and flexible bronchoscopy is now a core procedure of pulmonary and critical care medicine physicians.

With ultrasonography, a similar conflict is now being played out between new practitioners, in this case critical care clinicians, and an "old guard" of radiologists and cardiologists. Ultrasonography has such strong utility in critical care medicine that all intensivists are strongly encouraged to become proficient in its bedside applications. Intensivists are capable of learning a variety ultrasound skills that greatly improve their effectiveness in bedside diagnosis and management, make their procedures safer, and liberate them from a dependence on other specialists who are not always immediately available to care for the critically ill patient. An intensivist is responsible for the whole patient; and now, armed with a good ultrasound machine and the right skills, is capable of acquiring images that answer urgent clinical questions, interpreting and acting on these images in the context of an overall management strategy.

The technology has existed for decades, and has been used daily by internists and intensivists in Europe and in parts of Asia. North American intensivists developed proficiency in ultrasonography only several years ago. Radiologists and cardiologists may fret about whether intensivists can acquire the competencies to use this technology properly, but with proper training and experience, ultrasound is now in the hands of ICU physicians who use it with excellent results for their patients.

A key element to training in critical ultrasonography is mastery of the knowledge base of the field. This textbook is designed to meet the needs of the critical care ultrasonographer who requires a comprehensive and coherent presentation of the core knowledge of this discipline; it achieves that goal admirably. This book is intended to be used by intensivists; its authors are expert practicing intensivists and ultrasonographers. Through years of dedicated study and direct experience, they use ultrasound every day in the diagnosis and management of patients with complex cardiac, pulmonary, renal, and digestive diseases. The chapters of this text review the basic technology and physics of

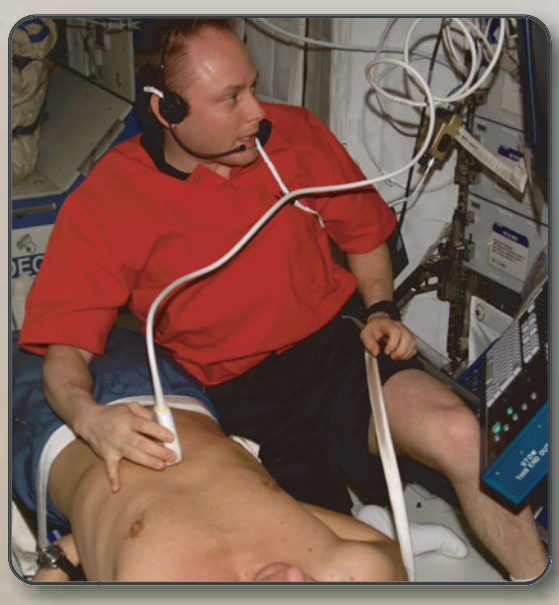
ultrasonography, and give detailed descriptions of its applications in diagnosis in each organ system, as well as its role in the performance of common ICU procedures.

This book defines the cognitive basis of the field; the intensivist who seeks to develop competence must combine this knowledge with hands-on bedside training in image acquisition and interpretation. For the experienced critical care ultrasonographer, the book provides a comprehensive reference for answering complex questions. Regardless of each clinician's current level of knowledge and skill, this text is an important resource for all practicing intenstivists, because like bronchoscopy in the 1970s, proficiency in ultrasonography will eventually be adopted by all intensivists, as the use of this technology will certainly serve our patients well.

Mark J. Rosen, MD, FCCP, FCCM, FACP
Chief
Division of Pulmonary, Critical Care and Sleep Medicine
North Shore University Hospital and
Long Island Jewish Medical Center
Professor of Medicine
Albert Einstein College of Medicine
Past President, American College of Chest Physicians

SECTION I

GENERAL PRINCIPLES AND IMPACT OF ULTRASOUND USE IN THE ICU



This page intentionally left blank

CHAPTER 1

The Use of Ultrasound in the ICU: Potential Impact on Care

Anthony D. Slonim

INTRODUCTION

Medical care for the critically ill usually advances in an incremental fashion. Physicians, for the most part, are a conservative group and critical care physicians are an important subgroup that tends to value a scientific approach and evidence-based decision-making. Experimental evidence requires time to generate, appropriate vetting through the peer review process, and then additional time prior to becoming engrained in clinical practice at the bedside for the benefit of patients. As a result, it is only through retrospective evaluation that the improvements in intensive care unit (ICU) care can be seen.

There are several important and relatively recent examples of this incremental approach in critical care, including low-tidal-volume ventilation, the management of hyperglycemia, and the use of hypertonic saline for acute elevations in intracranial pressure. Despite these well-defined examples, critically ill patients benefit from these approaches to a lesser degree than expected because their physicians fail to prescribe them in a large proportion of cases, thus compromising the quality of care for these critically ill patients. This is one example of how physicians can improve their own work by focusing on the elements of physician decision-making, particularly the process steps, and aligning them with the patients' needs.

Rarely, the quality of care for patient populations undergoes a major shift that can be thought of as revolutionary rather than evolutionary. These shifts, when viewed retrospectively, have usually involved major technological advances. For example, the use of fiberoptics in medicine has revolutionized the care of patients requiring diagnostic and therapeutic procedures. These patients now undergo relatively minor interventions as compared to what would have been experienced just a few decades ago. These shifts also involve practice settings. Surgeries formerly performed on inpatients are now performed on an ambulatory basis. Finally, these shifts involve physicians from different disciplines. Interventional radiologists are now performing procedures that previously required a surgeon. Cardiologists are now treating coronary syndromes in ways that previously required cardiac surgery.

Ultrasound use in the ICU is one such shift that decades from now will be viewed retrospectively as a revolutionary phenomenon that advanced the care of critically ill patients. However, the current challenge is to think prospectively, not retrospectively, about implementing this proven technology for diagnostic and therapeutic decision-making in a practice setting that is outside of the radiology suite and by providers who are neither radiologists nor cardiologists while the evidence base and applications are being further established. This book provides an opportunity to consider methods of applying this tool, in a thoughtful manner, at the bedside to advance the quality of care for this vulnerable subgroup of patients. Through an approach that evaluates the risks and benefits of using ultrasound in the ICU, physicians will be better able to understand how this technology can influence the care of their ICU patients.

HEALTH CARE QUALITY

Over the last 30 years, increased attention has been paid to the issues of quality health care. Donabedian provided a useful paradigm to consider the issue of quality by using structure, process, and outcome as three major components of the quality definition and applying it to health care. Since then, considerable effort has been put into further defining performance measures related to health care quality around six fundamental domains promulgated by the Institute of Medicine (IOM) in their seminal work titled *Crossing the Quality Chasm* and applied to a number of medical disciplines. These six domains include safety, effectiveness,

TABLE 1.1. The definitions of the IOM domains

IOM domain	*Definition*
Safety	To limit the unintentional harm associated with the delivery of health care
Effectiveness	To use evidence-based practices, the best scientific evidence, clinical expertise, and patient values to achieve the best outcomes for patients
Efficiency	To provide care that is done well and with limited waste
Equity	To provide care that is free from bias related to personal demographics like gender, race, ethnicity, insurance status, or income
Timeliness	To provide care without unnecessary wait and to assure that patients have access to the care they need
Patient-Centeredness	To provide care that reflects a focus on the patient's needs, including empathy, compassion, and respect

IOM indicates Institute of Medicine.

efficiency, equity, timeliness, and patient-centeredness (Table 1.1).

Safety

Safety is the domain concerned with medical errors that occur during the health care experience. These errors are classified as diagnostic errors, treatment errors, preventive errors, and "other" or unclassified errors. Diagnostic errors include delays or errors in diagnosis, the failure in applying appropriate diagnostic testing, and the failure to respond to the results of testing. Treatment errors result from errors in the performance of a procedure or test, delays in treatment, or providing care that is simply not indicated. Preventive errors occur from failing to provide prophylactic care or inadequately monitoring the patient. "Other" errors result from failures of the equipment or team, such as communication errors or performance issues.

Using this classification scheme, one can see how ultrasound use in the ICU may impact the safety of patients. Diagnostic errors may arise from operator inexperience in either acquiring or reading ultrasound images, leading to inaccurate or erroneous diagnoses. Artifacts that are misinterpreted represent another potential safety problem for patients. While ultrasound use in the ICU may compromise safety in important ways, there are also ways in which it improves care. Ultrasound as a diagnostic test can be applied when and where it is needed for the patients most likely to benefit. In addition, since the operator and interpreter are the same physician, the intensivist, the vulnerability of not having test results responded to is reduced.

Treatment errors are another category of safety errors. In this category, both test performance issues and using a test that is not indicated are important. If ultrasound is performed incorrectly, the results may be incorrect and be acted upon more quickly. Hence, ultrasound use in the ICU may compromise safety by its ready availability. The lead time from the performance of the procedure to a resulted report that can be acted upon in traditional diagnostic testing may be providing a safety net that disappears when the tool and operators are readily available. With ultrasound use in the ICU, a finding can be immediately acted upon for the benefit of the patient. The problem arises when the finding is a misinterpretation.

Ready availability of a test may also lead to excesses in use and treatment errors manifested in two specific ways. First, ICU physicians may use ultrasound because it is available and not because it is the best test to answer a particular clinical question. Second, the fact that a technology exists does not mean that it needs to be used on all patients. Care must be taken to assure that as a discipline, clinical questions are answered with the right tool and not the most technologically advanced or newest tool that happens to be available in the ICU. There is no need for ultrasound use when the physical examination will do just fine. However, there are opportunities to enhance the physical examination with a thoughtful and more in-depth assessment using ultrasound as an additional technique if one has been properly trained. If the test is not indicated, it is simply not indicated and its availability should not change the clinical indications. Ultrasound use can also improve safety with treatment errors because the findings are immediately available, communicated, documented in the record, and acted upon, all within a relatively short time span. In addition, by providing real-time guidance for invasive procedures, ultrasound allows direct visualization to assure accuracy of placement and avoidance of complications.

Preventive and unclassified errors can also compromise the safety of the ICU patient. Any time the intensivist is diverted from caring for the patient while

focusing on a procedure, safety events can occur from inadequate monitoring. Assuring that ultrasound is used in a broader context and that the "whole" patient remains under the watchful eye of the intensivist is important during the procedure. Safety is likewise improved by bedside ultrasound in this dimension because patients do not need to move from the ICU to receive their testing. Finally, the bedside ultrasound evaluation is only as good as the documentation and communication of the results. Failure to document in the medical record and report the care to colleagues creates risk to the patient that can be overcome by inserting a copy of the study and a report in the record and effectively communicating to staff and other physicians.

Effectiveness

Effectiveness is the domain concerned with using evidence-based principles, provider experience, and patient values in achieving the desired care. This includes the use of clinical guidelines that are evidence based, contemporary, and updated to keep pace with the evolving research. When evidence from randomized trials does not exist, other evidentiary methods can be used to help inform the care of patients.

When considering effectiveness, one needs to be careful not to fall into the trap of waiting for the literature to document all of the evidence with an available technology before it is used. That approach will take years and inadvertently prevent patients from benefiting from a useful bedside technology. Despite the fact that empiric evidence documenting the use of bedside ultrasound in the ICU is limited, there is face validity to recognizing that ultrasound is a simple diagnostic test that adds benefits to patients and has been performed in the disciplines of radiology, obstetrics, emergency medicine, and cardiology for a number of years. Further, the American Institute of Ultrasound in Medicine (AIUM), an organization that has existed since the 1950s, has a number of official statements, practice guidelines, and technical standards that can help to inform on the use of ultrasound more broadly, including in the ICU. In addition, the inexperienced operator and interpreter can also impair the effectiveness of the study.

Efficiency

Efficiency helps shape the use of health care resources by recognizing that there are limitations to the supply of resources that can be provided and by optimizing the value of the resources, by enhancing quality and limiting waste. The real opportunity for improving efficiency with ultrasound in the ICU is that it provides important answers to questions without the risk and danger of transporting a critically ill patient to another location. Further, having a test immediately available that can be performed by the providers caring for the patient helps to alleviate unnecessary steps in the process of obtaining and interpreting the test.

Equity

Equity is the domain responsible for assuring that health care is provided to patients without bias or discrimination based on personal demographics and for assuring equal access to health care services for patient populations. Ultrasound, when available as a tool in the ICU, provides ready and immediate availability on a 24/7 basis to patients regardless of their personal demographics. One potential problem is that currently the technology and expertise are not universally available. Hence, from an access perspective, the ICU to which a patient gets admitted may be unable to provide the service for the patients it serves. As a result, patients experience inequities in access to available technologies for diagnosis or treatment, particularly when ultrasound is unavailable 24/7 from the radiology department.

Timeliness

Timeliness is the domain responsible for assuring that patients receive the diagnostic and therapeutic services without delay when they need them. This improved access allows patients to receive the care they need when they need it. Similar challenges exist with timeliness as with equity. Namely, the ultrasound equipment and expertise may not be available 24/7 when the patient needs it, and the radiology department may also not have the service available.

Patient-Centeredness

Patient-centeredness is the domain where the patient and family are placed as the focal point of the health care experience. It also provides for the inclusion of patient wishes in the determination of what services are provided. Services that are patient-centered are those that provide care when and where it is needed by those who are most likely to benefit the patient. In addition, patient-centeredness ensures "service" aspects of health care quality including satisfaction with the

TABLE 1.2. Classification of ICU physician-specific quality components based upon Donabedian's structure, process, and outcome framework

Structure		
	"Bricks and mortar"	**ICU itself**
		Monitoring equipment
		Patient care equipment
		E.g.: Ventilators
		Ultrasound machines
		Medication pumps
	Personnel	**Physicians**
		ICU physicians
		Primary care physicians
		Consulting physicians
		Residents and fellows
		Other personnel
		ICU nurses
		Respiratory therapists
		Pharmacists
		Social workers
		ICU management
		Nursing director
		ICU medical director
		Hospital management
Process*	**Data gathering**	**Admission**
		History
		Thorough, timely, and accurate
		Physical examination
		Thorough, timely, and accurate
		Consultant input
		Thorough, timely, and accurate
		Diagnostic testing
		Appropriate test, performed
	Interpretation	**Pattern recognition from data**
		Clinical context from the patient
		Clinical knowledge based on training and experience
		Knowledge from EBM and current literature
	Decision-making	**Formulation of a plan consistent with patient choice**
		Medical treatment plan
		Surgical treatment plan
		Care management plan
	Action	**Gather further data**
		Revisit history
		Reexamine patient
		Perform further diagnostics
		Implement a care management plan
		Assure appropriate anticipatory measures
		E.g.: Gastrointestinal prophylaxis
		Deep venous thrombosis prophylaxis
		Assure appropriate therapeutic measures
		E.g.: Manage hyperglycemia
		Low tidal volume ventilation
		Elevate head of bed

(*continued*)

TABLE 1.2. (*Continued*)

	Implement the medical treatment plan Appropriate medication use based on EBM Appropriate diagnostic tests based on sensitivity and specificity Appropriate therapeutic plan based on EBM **Implement the surgical treatment plan** Right procedure Performed safely and correctly Intended outcome without complications **Perform a procedure** Right procedure Performed safely and correctly Intended outcome without complications
Outcome	**ICU mortality** **ICU morbidity** Physical disabilities Cognitive disabilities **ICU length of stay** Costs Duration of ICU therapies **Nosocomial infections** **Procedure complications**

**Physician-specific processes. ICU indicates Intensive care unit; EBM, Evidence-based medicine.*

technical aspects of the procedure. For ultrasound use in the ICU, the provision of the service at the bedside without unnecessary transportation, pain, and burden on the patient is representative of how ultrasound use can have a positive influence in this domain.

"DOCTOR QUALITY"

While the IOM report provides a useful framework for health care quality, physicians tend to think differently about health care quality. Specifically, when physicians consider quality they are often thinking about the care that they, rather than the health care team provides. Donabedian's constructs of structure, process, and outcome are particularly helpful here in assisting the physician in identifying his or her role in the provision of quality care to patients (Table 1.2).

Structure

From a structural perspective, the bricks and mortar of the ICU, including its walls, the monitors, the equipment, and maybe even the ultrasound machine are, by implication, what constitutes an ICU. However, the bricks and mortar alone do not make an ICU. It is the people, both providers and patients, and their expertise and interactions, that constitute the optimal delivery of ICU care. Structurally, physicians need to consider their role within the health care team. The organization of the ICU, how it is managed, and the management of other physicians, including primary care physicians, other consultants, residents and fellows determines the care the patients receive. The physician would be remiss not to consider the nurses, therapists, and ancillary departments who assure the appropriate delivery of care to the patients when the physician is not in attendance. When taken together, these elements are the structural components of ICU care to which Donabedian might refer (Table 1.2).

Process

Clinical processes, or the interactions between providers and their patients and providers with one another, are also important for physicians to consider. Nurses, in their discipline, can be very process focused, but physicians often lack this component in their training. Therefore, when asked to address specific process steps, like the implementation of the vascular access bundle, physicians often fail to recognize how such detailed specification of process actually makes a difference in outcome. However, some would argue that the

process steps are critical to patient care, particularly key physician processes (Table 1.2).

Doctor Processes and Medical Decision-Making

Taking a moment to consider doctor quality is helpful when considering how processes are operative in improving health care quality. The term "doctor quality" is used to describe the elements of the medical decision-making process that only physicians can influence. Doctor quality can be thought of through the key core processes of the ICU physician as he or she cares for the critically ill patient from ICU admission through discharge (Figure 1.1, Table 1.2). Traditional medical decision-making has four iterative steps that assist physicians with making decisions for their patients (Figure 1.1). The first step is data gathering. Physicians use their history, physical examination, diagnostic testing, consultants, and other members of the health care team to assist them in assuring that they have collected appropriate data upon which to base their clinical decisions (Table 1.2). The next step is for the physician to interpret the gathered data within the clinical context of the patient. This step involves assembling the collected data to see if it coalesces into a particular pattern and seeing if that pattern is consistent with the patient's presentation and findings. The next step is decision-making. Here, the physician may gather additional data by calling a consultant or ordering additional testing. If sufficient data has been gathered, the physician may formulate a medical treatment plan and reevaluate the plan's success as time progresses (Table 1.2). The physician may recommend or perform a procedure, the outcome of which may assist with diagnosis or treatment. Finally, as the last step of medical decision-making, the physician must take action. A plan that is not acted upon or a procedure or test that is thought about but not performed does not help the patient. These four steps allow the physician to think through and organize their work (Figure 1.1, Table 1.2).

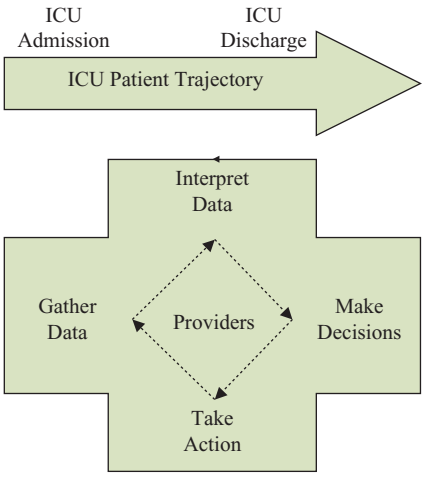


Figure 1.1. A model for medical decision-making that occurs throughout the ICU course.

Outcomes

Finally, outcomes represent the culmination of the health care experience. Physicians often focus on outcome measures as the result of their work. In the ICU environment, mortality is a traditional outcome measure that is important, quantifiable, and often discussed (Table 1.2). There are other outcome measures of relevance to ICU physicians, including the use of ICU specific therapies, length of stay, cognitive and physical outcomes, and morbidities arising from the episode of care (Table 1.2). However, since outcomes tend to be the end result of a series of process steps that are temporally distinct, it is often important for the physician to focus on both components of quality. Outcomes have been held in high regard for considerable time, almost to the exclusion of process measures. Physicians will only be able to improve the quality of care for their patients by focusing on both the process and outcome components of health care quality.

THE USE OF ULTRASOUND IN THE ICU: IMPACT ON CARE

The value of providing an overview to health care quality and the specific ways in which physicians are both affected and can affect it is that it provides a useful framework for further discussing the role of bedside ultrasound in the ICU and the potential to impact care for ICU patients. Table 1.3 uses the IOM domains and the medical decision-making process to help identify the opportunities to influence care in the ICU with the use of this important technology. By understanding the points in each of the IOM domains or the risk point in the decision-making process, the physician can approach the use of ultrasound in the ICU with improved recognition of the risks and benefits applied in this setting.

CONCLUSION

Overall, the use of bedside ultrasound is an important application of a well-described technology for diagnostic and therapeutic decision-making. Like most other new applications, it has risks and benefits associated

TABLE 1.3. The characteristics associated with ultrasound use in the ICU and their ability to impact care from the perspective of the IOM domains and physician-specific processes in medical decision-making

	Data gathering	*Interpretation*	*Decision making*	*Action*
Safety	Portable	Based on operator training/experience	Based on operator training/experience and confidence in findings	Improves procedure performance
	Available 24/7	Based on experience acquiring images		Avoids procedural complications by using direct visualization
	Noninvasive	Based on experience interpreting images		Avoids delays in interpretation
	No radiation exposure	Easy to learn		Misinterpretations may lead to errors in action
	Easy to learn	Expertise variable		Findings, interpretation, and actions need to be documented and communicated appropriately
	Expertise variable			
	Potential for excess use because of availability			
	May fail to use, more difficult to obtain, but better diagnostic tests			
	Availability causes overuse when traditional methods of physical examination would be fine			
	Attention to monitoring patient while performing ultrasound needs to be assured			
	Testing performed without moving patients and while maintaining ICU-level monitoring and therapy			
Effectiveness	Clear indications	Based on operator training/experience	Based on operator training/experience	Available EBM for ultrasound use in specific disciplines like radiology, emergency medicine, surgery, trauma, obstetrics/gynecology
	Limited EBM for ICU	Limited EBM for ICU	Limited EBM for ICU	Limited EBM for ICU
	Extensive EBM for ultrasound use generally	Extensive EBM for ultrasound use generally	Extensive EBM for ultrasound use generally	Extensive EBM for ultrasound use generally
	Requires acoustic window	Requires ability to distinguish artifacts		

(*continued*)

TABLE 1.3. (*Continued*)

	Data gathering	*Interpretation*	*Decision making*	*Action*
Efficiency	Operator/decision-maker are the same providing clinical context for focused evaluation	Operator/decision-maker are the same providing clinical context for focused evaluation	Lacks objectivity between operator and interpreter subjects to confirmatory bias	No lag time between decision and action; risky if decision is incorrect
	No need for patient transport	Immediate availability of information	May not be available in all ICUs 24/7	May not be available in all ICUs 24/7
	May not be available in all ICUs 24/7	May not be available in all ICUs 24/7		
Timeliness	May not be available in all ICUs 24/7	May not be available in all ICUs 24/7	May not be available in all ICUs 24/7	May not be available in all ICUs 24/7
				Operator/decision-maker are the same
	Not available in all ICUs	Not available in all ICUs	Not available in all ICUs	Not available in all ICUs
	Not available by all ICU providers	Not available by all ICU providers	Not available by all ICU providers	Not available by all ICU providers
Equity	Improves access for all ICU patients	Improves access for all ICU patients	Improves access for all ICU patients	Improves access for all ICU patients
	Not available in all ICUs	Not available in all ICUs	Not available in all ICUs	Not available in all ICUs
	Not available by all ICU providers	Not available by all ICU providers	Not available by all ICU providers	Not available by all ICU providers
Patient-centeredness	Alleviates the need for ICU patient transportation and its pain and risks	Operator/decision-maker are the same	Immediate information available for patient and family	Immediate access to specific interventions and next steps
	Expands the breadth of diagnostic and procedural capabilities	Lacks objectivity between operator and interpreter		

IOM indicates Institute of Medicine; EBM, Evidence-based medicine; ICU, intensive care unit.

with its use. For intensivists to optimize this technology for their patients, an understanding of their own decision-making process is important. I hope this chapter has provided a context upon which further use of this technology can be evaluated for the benefit of the critically ill patient.

Suggested Reading

Available at: http://www.aium.org/publications/guidelinesStatementsX.aspx#statements. Accessed October 25, 2008.

Available at: http://www.aium.org/publications/technical/techIntro.asp. Accessed October 25, 2008.

Morris AH. Developing and implementing computerized protocols for standardization of clinical decisions. *Ann Intern Med.* 2000;132:373–383.

Institute of Medicine Committee on Quality of Health Care in America. *Crossing the Quality Chasm: A New Health System for the 21st Century.* Washington, DC: National Academies Press; 2001.

Slonim AD, Pollack MM. Evaluating Pediatric Critical Care. In: Fink MP, Abraham E, Vincent JL, Kochanek PM, eds. *Textbook of Critical Care.* 5th ed. Philadelphia, Pa: Elsevier/Saunders; 2005:2207–2215.

CHAPTER 2

Physics of Sound, Ultrasound, and Doppler Effect and its Diagnostic Utility

Alexander Levitov

SOUND AND ULTRASOUND: ACOUSTIC PARAMETERS

All our lives we are surrounded by sounds. In fact, it is our ability to create and comprehend sounds in the form of speech that is integral to our human development. As physicians we assess heart sounds, breath sounds, and bowel sounds, but few will contemplate the nature of sound. Without understanding the physical properties of sound and their interactions with the surrounding medium, it is difficult to understand the images produced in clinical ultrasound. The critical care practitioner also often acts as a sonographer, whose responsibility is to operate the equipment, obtain images, distinguish between real structures and artifacts, and manipulate the transducer. Without a solid knowledge of basic sound principles these tasks are virtually impossible.

A sound is a wave created by a moving (vibrating) object and comprises areas of increased (compressions) and decreased (rarefactions) densities. This wave moves through a medium with a fixed speed (propagation speed), transmitting its energy, while the vibrating matter of the medium returns to its original position with each cycle (see Chapter 2 in enclosed DVD). When the sound wave reaches an object it is unable to penetrate, such as a wall, it may go around it (diffraction). This allows one to hear music around a corner. If the object is larger, such as a mountain, sound will bounce off (reflection) and return back to the source, creating a familiar phenomenon known as an echo. Echo was first described and named by the ancient Greeks.

Depending on the movement of the sound-generating object, the sound wave will acquire different characteristics known as acoustic parameters (Table 2.1). Some of those are related, while others are independent of each other. Though a sound wave is longitudinal with energy traveling in the same direction as the propagating wave, for the ease of representation it will be pictured as a transverse wave with energy distributed perpendicular to the direction of propagation like a wave on the surface of a pond (Figure 2.1).

Frequency and Period

The time necessary for the sound wave to complete one cycle is known as its period. The cycle is complete when the sound source has produced one vibration and the matter in the medium has returned to its original resting position. The period is measured in units of time (Table 2.1). One can probably use the fractions of the year, but that would be rather inconvenient, so most of the time it is measured in milliseconds ([msec] 1 thousandth), microseconds ([μsec] 1 millionth of a second) or nanoseconds (1 billionth). For example, a guitar D string takes 2.5 msec to completely travel across the reference object, such as a guitar fret, from left to right and left again to where it started; the sound it has generated will have a period of 2.5 msecs (Figure 2.2).

Related to the period is the frequency of the sound wave (Table 2.1). Frequency (f) is a number of cycles completed in 1 second. A standard measure of frequency is hertz (Hz), which was named for Heinrich Rudolf Hertz (1847–1894), the first person to transmit and receive radio waves. One Hz is 1 cycle per second. The same guitar string with the period lasting 2.5 msec will complete 400 periods in 1 second and therefore have a frequency of 400 Hz or 0.4 kilohertz (kHZ) (Figure 2.2). Frequency is the reciprocal of the period (Table 2.1). That is:

$$\text{Frequency} \times \text{period} = 1$$
$$\text{f (Hz)} = 1/\text{period sec}$$
$$\text{Period (sec)} = 1/\text{f (Hz)}$$

TABLE 2.1. Summary of acoustic parameters

Acoustic parameter	*Units*	*Determined by*	*Values in diagnostic ultrasound*
Period	μsec	Sound source	0.1–0.5 μsec
Frequency	MHz	Sound source	2–10 MHz
Amplitude	dB	Sound source	—
Power	Watts	Sound source	—
Intensity	Watts/cm^2	Sound source	0.001–100 W/cm^2
Wavelengths	mm	Source and medium	0.1–0.6 mm
Propagation speed	m/sec	Medium alone	1,500–1,600 m/sec

- kHz: kilohertz, thousands of cycles per second with a period of milliseconds
- MHz: megahertz, millions of cycles per second with a period of microseconds

Humans can hear sounds with frequencies ranging from 20 Hz to 20 kHz. Speech generates 100–220 Hz, and singing 50 Hz–1.5 kHz. The human ear is most sensitive to 3–4 kHz sounds. The sound in the range of human hearing is known as audible sound, or simply sound, with frequencies >20 kHz known as ultrasound and <20 Hz known as infrasound. Thus, a distinction between sound and ultrasound is really quite capricious if not entirely baseless. Many other animals have wider ranges of hearing. For example, the domestic cat can hear up to 50 kHz. Though an ultrasound wave is a sound wave in every respect, ultrasound waves (particularly in the MHz range), tend to travel in a straight line, and diffract less and reflect more off smaller objects than lower-frequency waves. Typical periods and frequencies for diagnostic ultrasound are 0.1–0.5 μsec and 2–10 MHz, respectively, and are determined by the ultrasound source known as a transducer, or a probe (Table 2.1). With frequencies, much greater than those of diagnostic ultrasound, ultrasound waves start to behave a lot like electromagnetic microwaves, but those frequencies are never reached in medical applications.

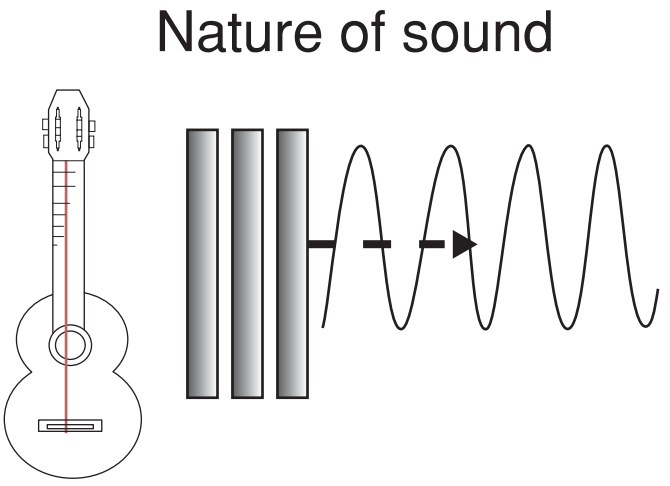


Figure 2.1. Guitar string vibration is creating a longitudinal wave. It is represented, however, as a transverse wave.

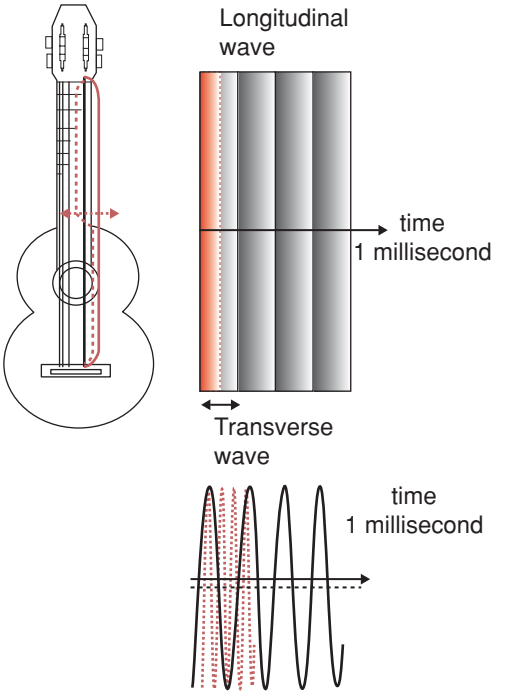


Figure 2.2. Guitar D string vibration is creating a longitudinal wave in fundamental (shades of gray) and first harmonic (shades of red) frequencies. The fundamental frequency longitudinal wave is represented below by a transverse wave of the same frequency. The red, dotted wave is a first harmonic wave with half the period and twice the frequency of the fundamental wave. Fundamental period is 2.5 of a millisecond; Fundamental frequency is 400 Hz (0.4 kilohertz [kHz]); First harmonic frequency is 0.8 kilohertz (kHz).

Most emitted and reflected sounds have a mixture of frequencies, with the lowest one being the so-called fundamental frequency, and its multiples are harmonic frequencies, or, simply, harmonics. These concepts are demonstrated by the movement of the guitar string (Figure 2.2).

AMPLITUDE, POWER, AND INTENSITY

As a sound wave propagates, the particles in the medium will move from the resting position. This creates areas of increased density and increased pressure on the surrounding area (compression). These changes in motion, pressure, and density will reach their peak and then subside with each vibration. The difference between a resting value of the parameter, such as density or pressure, and the peak one, during the period of the sound wave, is referred to as amplitude. You can pluck the guitar string softly (with less amplitude), creating less motion (pressure, density), or vigorously (with more amplitude), but because the string has the same period and frequency, the sound would not change. Simply stated, amplitude is the loudness or volume of the sound whether one can hear the sound or not (Table 2.1). In ultrasound applications it is often referred to as an output gain or acoustic power (Figure 2.3). Because amplitude is the difference or ratio between two values of the parameter with the same unit, it cannot be described in absolute units (Table 2.1). In other words, one can always produce a louder or softer sound. Therefore, a relative scale is used. The relative units of this scale are Bells. They are named for Graham Bell and are defined as 0.1 of the Bell or decibels (dB), which are the most commonly used units in acoustic measurements (Tables 2.1 and 2.2).

The scale is logarithmic, meaning that the number is described by how many multiples of 10 one needs to create it (Table 2.2). Thus, the logarithm of 100 is 2, of 1000 is 3, and of 1/100 is −2.

- $dB = 10 \times \log(A1/A2)$, where A1 and A2 are the amplitudes of sounds being compared.

More important, there are only two numbers to remember, i.e., 3 dB and 10 dB. 3 dB means twofold and 10 means tenfold increase in the measured parameter,

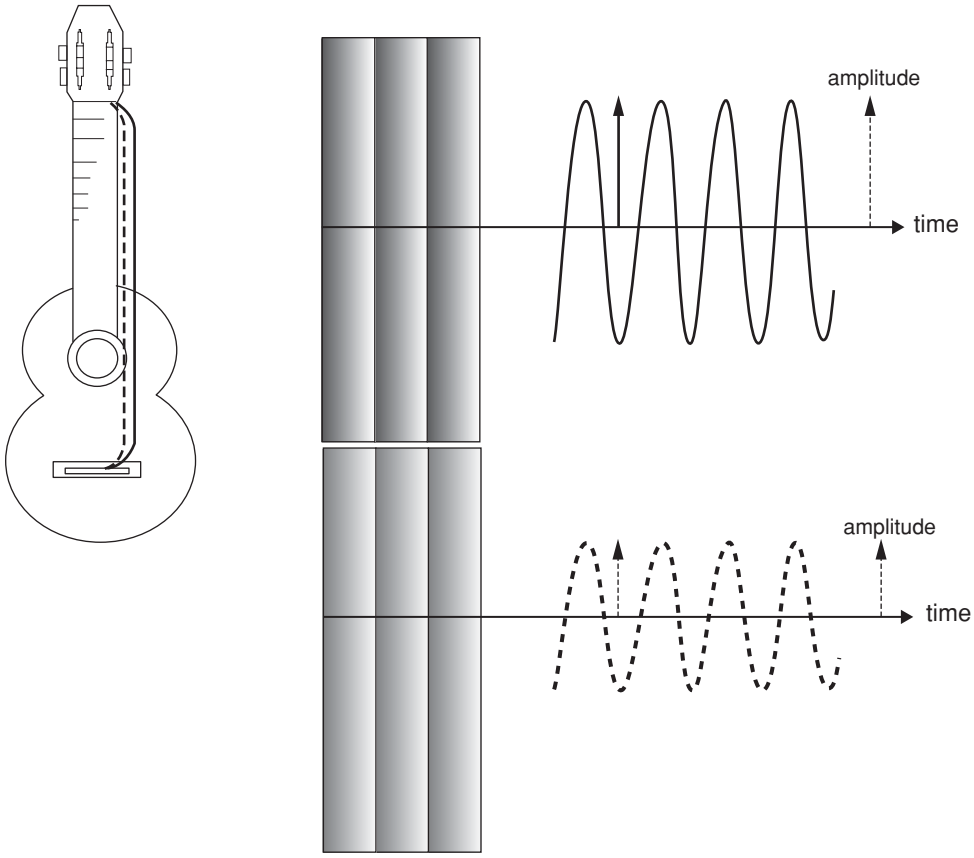


Figure 2.3. Different amplitude of the sound: —More medium displacement, higher amplitude; · · ·Less medium displacement, lower amplitude; Power = k × (amplitude)^2.

TABLE 2.2. Amplitude ratios and decibels

Ratio of sound 1/sound 2	*Decibels*
1000:1	30
100:1	20
10:1	10
4:1	6
2:1	3
1:2	−3
1:10	−10

therefore −3 dB is two times less (one half) and −10 is ten times less (0.1) than the original value. So if the parameter is pressure, then 3 dB sound will create twice the pressure on your eardrum (or be twice as loud) and 20 dB will be 100 times louder than the original one, while −3 dB will be only half that loud (Table 2.2).

It follows that power, or the amount of energy per second, delivered by the sound wave, is related to the amplitude and is in fact the amplitude squared (Table 2.1). When amplitude doubles, power increases fourfold.

$$\text{Power} = k \times (\text{amplitude})^2, \quad \text{where k is a coefficient}$$

The units of power are watts (Joules/sec), named for James Watt, the Scottish engineer. The power of the sound wave distributed over the unit of surface is known as intensity (Table 2.1). Intensity is defined as the amount of energy per second delivered to the surface area of the medium. Intensity (measured in watts/cm^2) therefore will be directly proportionate to power (amplitude) and inversely proportionate to the area over which that power is applied. The same sound beam with the same power when applied to a smaller surface area will deliver more energy to that area and is said to be more intense. The typical intensity of the diagnostic ultrasound equipment is 0.001 to 100 watts/cm^2 (Table 2.1). In the case of diagnostic ultrasound, the "medium" is human tissue and the intensity will predict the bioeffect of the ultrasound (Tables 2.1 and 2.3). Those effects can be rather dramatic. One important example is ultrasonic lithotripsy, where the intensity of the ultrasound is used to decimate renal stones.

The speed of sound (propagation speed) is a fixed number and is related solely to the medium (Tables 2.1 and 2.3). This is a far-reaching statement and a unique feature of the physics of wave propagation. The speed of moving objects is an algebraic sum of the speed of the object and the observer. A couple of examples can help to demonstrate this point:

TABLE 2.3. Speed of sound (propagation speed) in different tissues

Tissue	*Speed of sound (m/s)*
Lung	300–1,200
Fat	1,450
"Soft tissue"	1,540
Bone	2,000–4,000

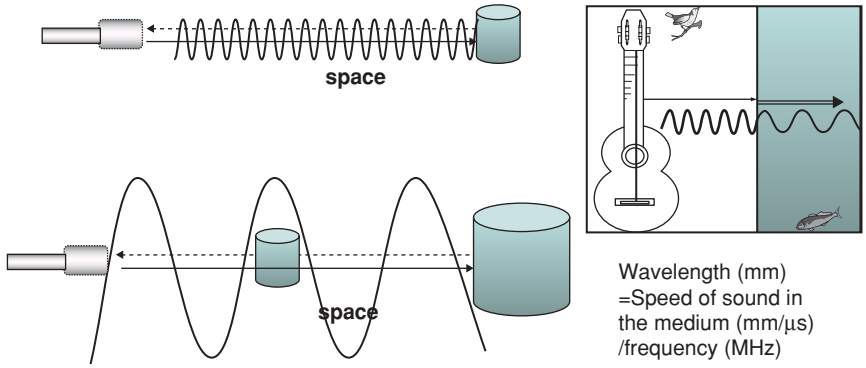
1. If a man is walking with the speed of 4 miles per hour (mph) down an escalator, which is moving at 3 mph relative to a stationary observer, he is moving at 7 mph.
2. If a police officer is chasing a getaway car and his car is moving at 110 mph, and the getaway car is moving at a 100 mph relative to that of the police officer, the speed of the chased car is −10 mph (the police officer is gaining on the getaway car).

It does not matter if the source of the sound or the observer is moving; the sound propagation speed remains the same (Table 2.1). A jet plane can fly faster than sound, but the roar of its engines will propagate at the same speed as if it were stationary on the ground; however, the frequency of the sound waves will change. If the sound is moving toward the observer, the period of the sound wave decreases and therefore the frequency increases (sound wave is compressed). If the sound is moving away, the opposite occurs (the wave is stretched). This is the so-called Doppler effect, which will be further discussed below, but for now remember: *nothing can change the speed of sound in a particular medium;* it is an established and fixed phenomenon.

Wavelength is the length of a single cycle and is measured in the units of length (Table 2.1, Figure 2.4A). A picture of the wavelength is easy to confuse with that of the frequency; in fact, they will look exactly the same, but remember that here the axis is space, not time. Wavelength is determined by the frequency of the sound and the speed of sound in the medium (Table 2.1).

$$\text{Wavelength (mm)} = \text{speed of sound in the medium (mm/}\mu\text{s)/frequency (MHz)}$$

Because the speed of sound in the medium is an intrinsic quality of the medium (Table 2.3), the wavelength in that medium will be inversely related to the

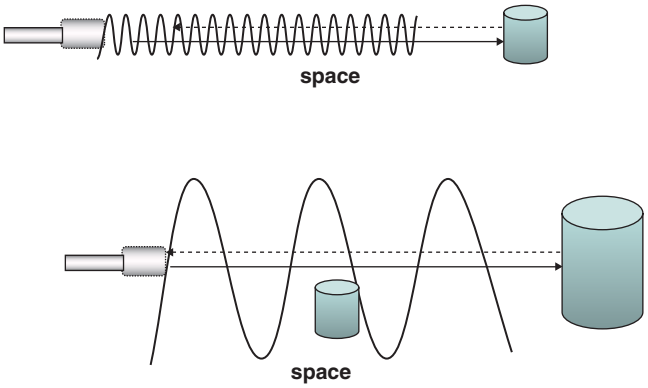


A

Figure 2-4A. Speed of sound in the medium is an intrinsic quality of the medium. As the speed of sound increases in the water, the wavelength becomes longer, while the frequency of the sound wave generated by the sound source does not change. In the same medium, higher frequency ultrasound will create shorter wavelength. Shorter wavelength ultrasound produces better images by reflecting off smaller objects. Wavelength (mm) = Speed of sound in the medium (mm/μs)/ frequency (MHz).

frequency of the sound wave generated by the sound source (Table 2.1). In the same medium, the higher frequency ultrasound therefore will generate a shorter wavelength. The longer the wavelength, the larger the size of the object the wave will need to encounter to be reflected. High-frequency ultrasound allows for reflection off smaller objects, which improves image quality (axial resolution) in the same medium (Figure 2.4B). In "soft tissue," sound with frequency of 1 MHz will have a wavelength of 1.54 mm, and 500 kHz a wavelength of 3.08 mm.

In "soft tissue": Wavelength (mm) = 1.54/f (MHz)



B

Figure 2-4B. Shorter wavelength and higher frequency ultrasound produce better images by reflecting off smaller objects. Because the speed of sound in the medium is an intrinsic quality of the medium, the wavelength in that medium will be inversely related to the frequency of the sound wave generated by the sound source.

So in diagnostic ultrasound the rule is: the higher the frequency, the shorter the wavelength, the better the image. Useful values of wavelengths in diagnostic ultrasound are 0.1–0.08 mm.

INTERACTIONS OF ULTRASOUND AND MEDIUM

As the ultrasound wave travels through the medium it releases some of its energy and reflects back to the source as an echo (Figure 2.6). The result is a dampening of the wave or reduction in its amplitude. This process is known as attenuation. Attenuation is dependent on the ultrasound frequency and the distance the ultrasound travels in the medium. Attenuation only affects the amplitude, the wave frequency, and frequency. The greater the distance the ultrasound has to travel, the more attenuation will occur. To illustrate the relationship between frequency and attenuation one just needs to rub one's hands together. The faster you do it (higher frequency), the hotter the hands will get, because more heat energy is released by friction. Attenuation is always measured in negative decibels, and in the soft tissues one MHz frequency signal will attenuate −0.5 decibels per every centimeter it travels (Figure 2.5).

$$\text{Attenuation} = (-0.5\,\text{dB/cm/MHz}) \times \text{travel distance (cm)}$$

Heat generation will be discussed in the biological effects of the ultrasound, but reflection is important to

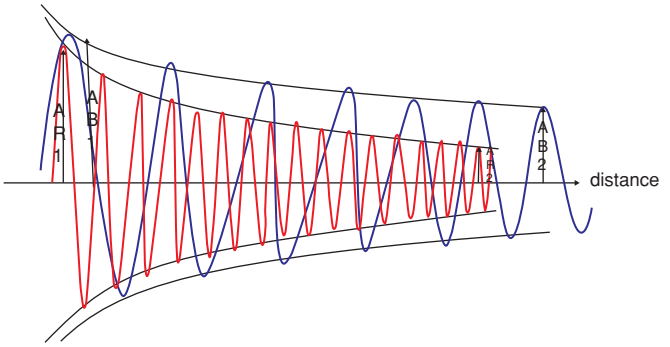


Figure 2-5. Both high-frequency red wave (R) and lower-frequency blue wave (B) attenuate (dampen) with distance. However, blue wave attenuates less. Ratio of initial amplitude A1 to final A2 illustrates the degree of attenuation and is measured in negative decibels (dB). AB1: AB2 > AR1: AR2.

TABLE 2.4. Relationship between elapsed time and distance to the reflective boundary in the "soft tissue"

Elapsed time (μsec)	*Depth of reflective boundary (cm)*	*Total distance traveled from the source and back (cm)*
13	1	2
26	2	4
52	4	8
130	10	20

In "soft tissue" if the elapsed time is 13 μs the reflector is 1 cm deep.

image formation. It will also be discussed later in more detail, but some basics need to be addressed now.

When the sound wave strikes a boundary between two different tissue layers, some of the energy will proceed further while the rest will return to the source of sound in the form of an echo. The timing and the energy of the echo signal will be related to the depth and the physical nature (acoustic impedance) of the boundary and will provide all the necessary information to create an image (Table 2.4, Figure 2.6). The sound wave that is not reflected will go on until the next tissue boundary is met. The same phenomenon will take place again until the signal is lost due to attenuation. Because energy is not created in the process, whatever is not reflected (or converted to heat and lost) will be transmitted. The amount of reflected ultrasound energy will depend on the physical properties of the boundary. Those properties can be summarized in a calculated number of acoustic impedance. Impedance is measured in units of Rayls (Z). The units are named for the physicist Robert John Strutt, 4th Baron Rayleigh.

$$\text{Acoustic impedance (Rayls)} = \text{density (kg/m}^3\text{)} \times \text{speed of sound (m/s)}$$

Typical impedance of "soft tissues" is 1.25 to 1.75 Mrayls (1,250,000–1,750,000 Rayls).

The best reflection can be achieved when sound strikes a boundary between two layers with large differences in acoustic impedance at a 90° angle of incidence (normal incidence). If there is no difference in

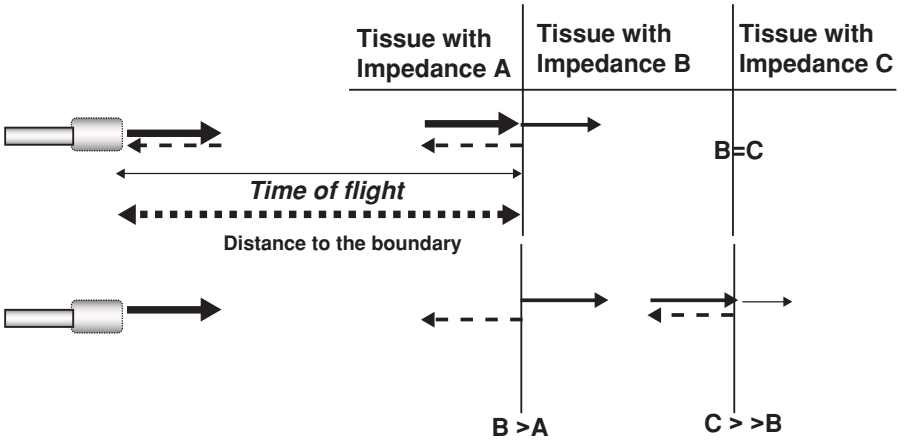


Figure 2-6. Normal incidence. If there is no difference in impedance between the two boundaries, no reflection will occur; the more the difference, the greater the amount of ultrasound is reflected back to the source. Distance to the boundary can be calculated from the time it takes for the ultrasound to reach the boundary and return to the source (time of flight, elapsed time). In the soft tissues, distance to the boundary (mm) = elapsed time (μs) × 0.77 mm/μs.

TABLE 2.5. Comparison of high and low frequency ultrasound waves

	Image depth	*Attenuation*	*Image quality*	*Axial resolution*	*Lateral resolution*
Low frequency 2–5 MHz	deep	low	lower	Lower (big number)	Lower (big number)
High frequency 5–10 MHz	shallow	high	higher	Higher (small number)	Higher (small number)

acoustic impedance between the two layers, no reflection will occur and no image can be formed (Figure 2.6). As a rule, at the boundary between two different "soft tissues" (i.e., fat/kidney) with similar impedance, only 1% of the sound is reflected in the form of echo and 99% is transmitted, while at soft tissues/bone interface roughly half of the sound is reflected back, and at tissue/air boundary, where the difference in impedance is the greatest, nearly all the energy is reflected (99%) and practically none is transmitted (Table 2.3). In this latter case, visualization of any structures below such boundary is impossible. The fine detail of the boundary (reflector) will only be reflected properly and visualized when the wavelength of the ultrasound is smaller than those details, thus the image will improve with an increase in ultrasound wave frequency (Tables 2.5 and 2.6). Regrettably, high-frequency waves also have high-attenuation rates and will not penetrate deeply into the tissues (Table 2.6). That can be partially remedied by analyzing only returning echoes with frequencies that are multiples of the fundamental (emitted) one, the so-called tissue harmonics. For example, if the emitted sound wave has a frequency of 5 MHz, only echoes with frequency of 10 MHz are analyzed. Selecting those higher frequency waves known as tissue harmonic imaging (THI) for image formation usually results in improved image quality.

When the two boundaries are separated by a distance much greater than the wavelength of the emitted ultrasound, they will appear separate on the image created by their respective echoes. However, as the boundaries get closer to each other, the timing interval between returning echoes becomes progressively shorter until finally they appear as a single object (Figure 2.7). The distance where two objects are perceived as two separate ones in the pass of the ultrasound wave (axial plane) is known as axial or longitudinal resolution. Resolution is measured in millimeters: the smaller the number, the better the image. The typical value of axial resolution for modern ultrasound equipment is about 0.1 mm (0.05–0.5 mm) (Figure 2.7).

For image formation the ultrasound machine (system) always assumes that the reflective boundary is struck by the ultrasound at a 90° angle. However, in reality it is seldom the case and the result is that images become difficult to interpret. If the sound wave strikes the boundary at a non-90° incidence angle (oblique incidence), reflection may never reach the source of sound and image formation will be impossible (Figure 2.8). Moreover, ultrasound transmission and reflection with oblique incidence is difficult to describe mathematically, and if reflection does reach the sound source, ultrasound equipment will interpret it as if there has been normal incidence and may place the image in the wrong place, creating artifact. With normal incidence, the transmitted fraction of the sound wave will always follow the original direction of the beam. However, with the oblique incidence, if the speed of sound is different between the two layers of the boundary transmission, a bend, or refraction, will occur. Refraction is governed by Snell's law: sine of transmission angle/sine of incidence angle = speed of sound medium1/speed of sound medium[2] (Figure 2.8).

Ultrasound equipment assumes the same speed of sound in all tissues (1540 msec) and accordingly will not account or compensate for refraction; therefore, refraction artifacts are common. To complicate things even further, once off the original pass, ultrasound might encounter unexpected reflective boundaries and take a "scenic" pass back to the transducer. None of these issues will be considered when the image is formed.

TABLE 2.6. Factors affecting quality of the ultrasound image

Image quality	*Depth*	*Wave frequency*	*Pulse*	*Focus*
Better image	Shallow sample	High frequency waves	Short SPL	Narrow focus
Worse image	Deep sample	Low frequency waves	Long SPL	Wide focus

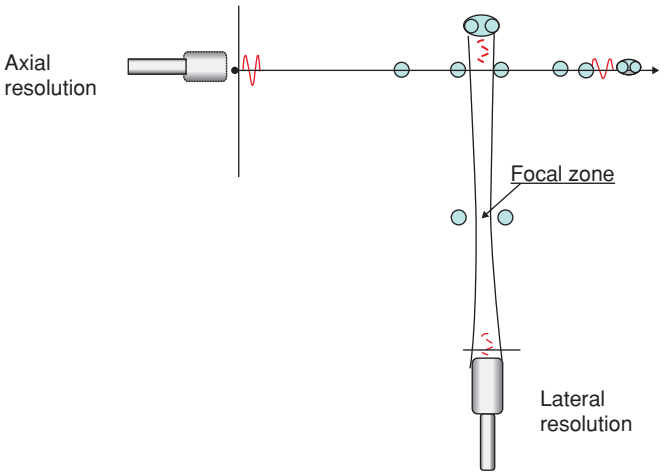


Figure 2-7. As two boundaries become closer in the pass of the ultrasound (axial plane), they will appear as one (limit of axial resolution). Two objects, appearing as one in the plane perpendicular to the pass of the sound, is a limit of lateral resolution. Axial resolution is always better (smaller number) than lateral resolution.

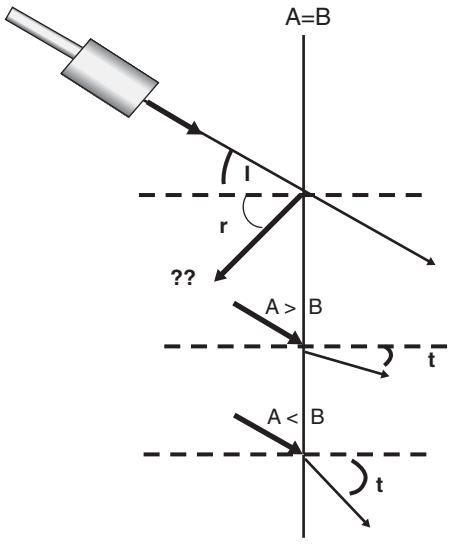


Figure 2-8. In oblique incidence, both transmission and reflection are unpredictable, except for the incidence angle l being equal to reflection angle r. If the speed of sound in both media is equal, transmission will follow the pass of the incident wave; otherwise refraction will occur. If the speed of sound in medium A is faster than in medium B, the transmission angle will be less than the incident angle. If the speed of sound in medium A is slower than in medium B, the transmission angle will be greater than the incidence angle (Snell's law). Sine t/ Sine i = propagation speed B/A.

But if the sound is traveling in a straight line and strikes the reflector at 90° incidence, it is very easy to determine the reflector's depth. For this, one only needs to know the time needed for the echo to return to the sound source (transducer) and the speed of sound (Figure 2.6). Position (depth) of the reflective boundary can be calculated using the following formula:

$$\text{Distance to the boundary (reflector) (mm)} = \text{elapsed time } (\mu\text{s}) \times 0.77\ \text{mm}/\mu\text{s}$$

In "soft tissue," if the elapsed time is 13 μs the reflector is 1 cm deep

WAVE INTERACTIONS

Besides interacting with the medium, the sound waves also interact with each other. As a longitudinal wave, the sound will transmit from the source in more or less concentric circles. An important analogy portrays this principle. One can sit behind a guitar player and still hear the music play. Due to wave interaction, however, when you sit in the front of the guitarist the music is louder because of the ways in which the sound waves interact with each other. Two in-phase waves will sum creating a wave with higher amplitude (constructive interference), while waves in the counterphase will subtract, resulting in one with a lower amplitude (destructive interference) (Figure 2.9).

A bullhorn is designed to produce even more constructive interference creating a sound beam traveling in the direction it points. The waves in the center of the beam have the highest amplitude. Ultrasound waves emitted by the transducer diffract less, making beam formation even more precise. Wave interactions are described by Huygens' principle, which states that all constructive and destructive interferences within the beam will produce an hourglasslike final shape (Figure 2.9). The narrowest area (waist) of the beam is known as the focal point or focus. The beam with the narrowest focus will be able to distinguish two side-by-side objects as separate, while the beam with the broader focal point will fuse them into one image (Figure 2.6). This concept is known as lateral resolution. High-frequency sound waves have a narrower focal point and higher lateral resolution. Therefore, high-frequency ultrasound waves will improve both axial and lateral resolutions and are preferred for their ability to produce superior image quality, but can only be used for visualization of superficial structures due to high attenuation.

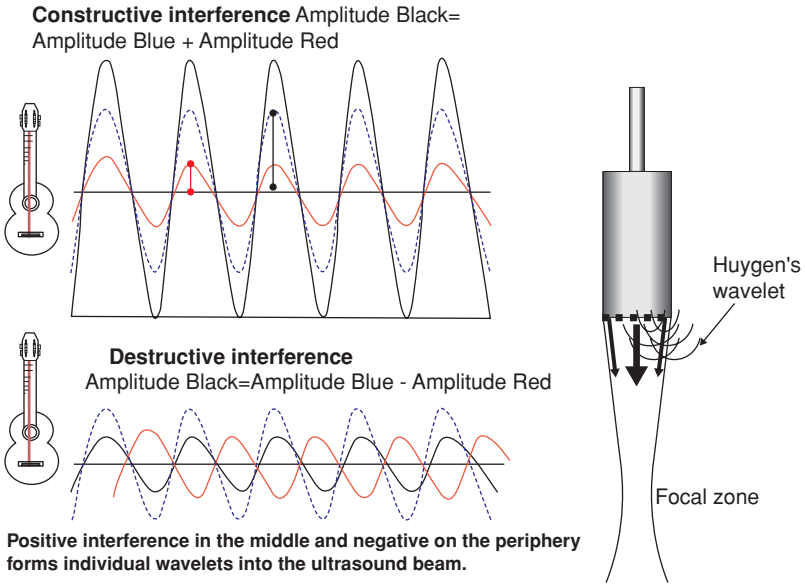


Figure 2-9. Interference and ultrasound beam formation. Positive interference in the middle and negative on the periphery form individual wavelets into the ultrasound beam.

CONTINUOUS-WAVE AND PULSE ULTRASOUND

An ultrasound wave can be emitted continuously as a light from the headlight of the car or in inpulses as a blinking turn signal. All imaging ultrasound waves are pulse waves, with an "on," or talking time, for producing a sound pulse and an "off" time for listening for the return echo signal. Imaging transducers serve as both transmitters and receivers.

The percentage of "on" time when the ultrasonic pulse is produced is known as the duty factor (Table 2.7). The usual duty factor in imaging ultrasound is 0.1–1%, so just as in a good human conversation, there is a lot of listening and very little talking. A duty factor of 100% means that continuous-wave, nonimaging (Doppler) ultrasound is used. The most familiar form of such a device is a "black-box Doppler" used for finding a pulsatile artery. A duty factor of 0% means that the ultrasound machine is off. Several other terms need to be introduced in regard to the pulsatile nature of imaging ultrasound.

Pulse duration (PD) is the time during which the sound is emitted in each on/off phase. It is usually 0.5–3 μsec, with each pulse being a short wave comprised of 2 to 4 cycles (Table 2.7, Figure 2.10). Pulse repetition period (PRP) is the time of the entire on/off cycle (Table 2.7). Because the pulse duration is fixed by the transducer, the only variable is an "off" or listening time. The deeper the imaging sample, the longer the time required for the echo pulse to return to the source, thus the longer the pulse repetition period necessary (Tables 2.4 and 2.7). Pulse repetition frequency (PRF) is a number of pulses emitted in one second (Table 2.7). Pulse repetition frequency is measured in hertz and usually is 1–10 kHz in imaging ultrasound and will vary depending on the depth of the imaging sample. Pulse repetition period and PRF are reciprocal numbers

TABLE 2.7. Summary of pulsed wave parameters

Parameter	*Determined by*	*Units*	*Typical value*
Pulse duration	Ultrasound source alone	μsec	0.5–3.0 μsec
PRP	Source/changes with depth of image	msec.	0.1–1.0 msec
SPL	Source and medium	mm	0.1–1.0 mm
PRF	Source/changes with depth of image	kHz	1–10 kHz
Duty factor	Source/changes with depth of image	%	0.1–1%

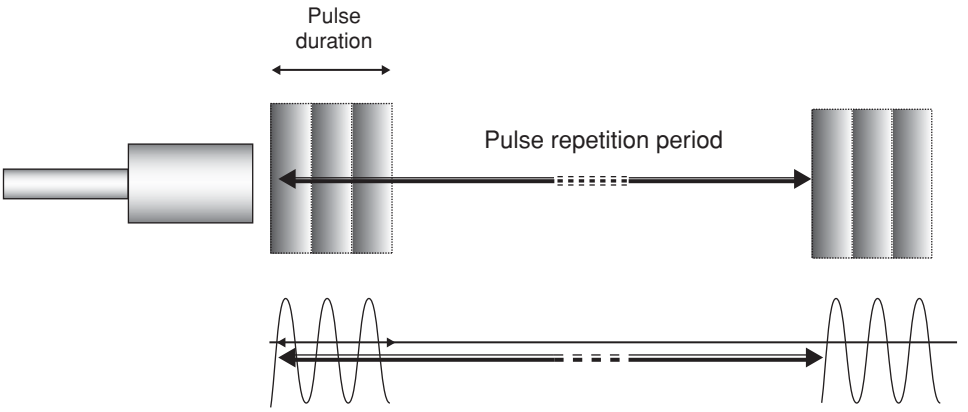


Figure 2-10. Pulse ultrasound. Pulse duration (PD) is the time during which the sound is emitted in each on/off cycle. It is usually 0.5–3μsec and comprises 2 to 4 cycles. Pulse repetition period (PRP) is the time of the entire on/off cycle. Though difficult to depict graphically, PRP is usually 100–1000 times longer than PD. Duty factor = PD/PRP. Pulse repetition frequency (PRF) is the number of pulses emitted in one second. PRP (sec) = 1/PRF (Hz), PRF (Hz) = 1/PRP (sec). The deeper the image, the longer the PRP, the lower the PRF.

related in the following ways:

$$\text{PRP (sec)} = 1/\text{PRF (Hz)}$$
$$\text{PRF (Hz)} = 1/\text{PRP (sec)}$$

Note that though it is measured in the same units, PRF has no relationship to the frequency of the ultrasound wave produced during pulse generation that is measured in MHz.

Spatial pulse length (SPL) is the length of the pulse in space with a typical value of 0.1–1.0 mm (Table 2.7). Shorter pulses, just as shorter wavelengths of the pulse wave, reflect off the smaller objects. Shorter pulses produce better images by improving axial resolution.

Axial resolution can be described by the following relationship:

$$\text{Axial resolution} = \text{SPL (mm)}/2$$

DOPPLER PHENOMENON AND ITS USE IN DIAGNOSTIC ULTRASOUND

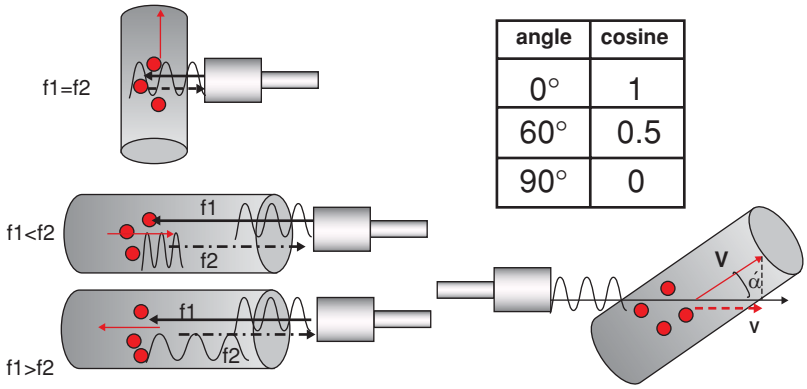
First described by Christian Doppler, the Doppler phenomenon simply states that if the source of sound (transducer) and the object reflecting the sound (reflector) are moving in relationship to each other, the frequency of the reflected sound wave will change. If the reflector is moving toward the sound source, the sound waves will be compressed to a higher frequency (positive Doppler shift). If it is moving away from the sound source, the sound waves will be stretched to a lower frequency (negative Doppler shift) (Figure 2.11).

Subtracting the incident frequency from the frequency of the returning echo Doppler shift can be recorded, and because it falls into the audible range of 20 Hz–20 kHz, it can be heard by the operator. It is important to remember that the shift itself is riding "piggyback" on the inaudible original ultrasound wave usually measured in MHz. So if the reflector (such as blood or cardiac structures) is moving, the velocity and the direction of the movement can be calculated and imaged from the value of Doppler shifts using the equation below:

$$\text{Reflector velocity} = (\text{Doppler shift} \times \text{propagation speed})/2 \times (\text{incident f} \times \text{cosine. incidence angle})$$

Of those, the cosine of the incident angle is most important. The cosine of 90° angle is "0" and no reflector velocity can be measured. In fact, the closer the incidence angle to 0° or 180°, the closer the measured velocity to an actual one (Figure 2.11). This presents a problem if the image of the organ, such as a blood vessel, is to be obtained simultaneously with the blood flow velocity. The images are best at 90° incidences, but then no Doppler information can be obtained. So a compromise has to be reached between the image quality and the Doppler data. This compromise is achieved with a 60° incidence angle, which is often utilized in vascular studies.

The oldest, simplest, and perhaps most used Doppler modality is a continuous-wave (CW) Doppler (Tables 2.8 and 2.9). In this case, one part of the transducer constantly emits the incident ultrasound wave



angle	cosine
0°	1
60°	0.5
90°	0

Figure 2-11. If the blood cell is moving toward the transducer, the frequency of the reflected signal will be higher than that of the emitted or incident one (positive Doppler shift). If the blood cell is moving away, the frequency of the returning signal will be lower than the incident frequency (negative Doppler shift). Doppler shift = f1 − f2. Doppler shift depends on the angle between the reflector (red blood cell) direction and the direction of the emitted sound wave. Doppler shift = 2× reflector speed × incident frequency × cos (angle $\acute{\alpha}$)/propagation speed. With 90° angle there is no Doppler shift and the reflector velocity calculations are impossible. Velocity calculations are most accurate with 0° incident angle. f1 = incident frequency, f2 = reflected frequency, $\acute{\alpha}$ = incident angle.

and the other constantly receives the returning wave. Processors subtract them and the resulting shift is heard by the operator. This is the construction of the "black box Doppler" found frequently in intensive care units (ICUs) and used by physicians and nurses when they are unable to palpate an arterial pulse (Figure 2.12). Continuous-wave Doppler is capable of detecting any flow velocity, but unable to tell where the sample is taken (Table 2.9). This "range ambiguity" results from large overlap between transmitted and received beams (Table 2.9).

When exact knowledge of the sample location is essential (i.e., stenotic aortic valve) pulsed-wave Doppler is used (Tables 2.8 and 2.9). Just as with the imaging pulsed ultrasound, the pulsed Doppler transducer serves both as a transmitter and a receiver. Echo signals from only one area, known as sample volume, are chosen for analysis, the rest are ignored (gating). This area is determined by the timing of the returned signal. This is known as range resolution (one knows exactly where the sample volume it taken) (Table 2.9). However, pulsed-wave Doppler has a fundamental problem. The transducer has to wait until the returned echo signal is received before the next incident pulse is transmitted (Table 2.8). Each pulse is a snapshot, and

TABLE 2.8. CW v. pulsed Doppler

Doppler modality	*Range resolution*	*Aliasing*	*High flow velocity detection*
Continuous wave	no	no	Unlimited
Pulsed Doppler	yes	yes	Limited by Nyquist frequency

TABLE 2.9. Comparison of Doppler modalities

Doppler modality	*Pros*	*Cons*
Continuous wave Doppler	Identifies high flow velocity jets, no aliasing	Range ambiguity
Pulsed wave Doppler	Range resolution (depicts location of the flow)	Aliasing with high flow velocities
Color flow Doppler	Direct 2-D flow information superimposed on anatomical images	Aliasing with high flow velocities

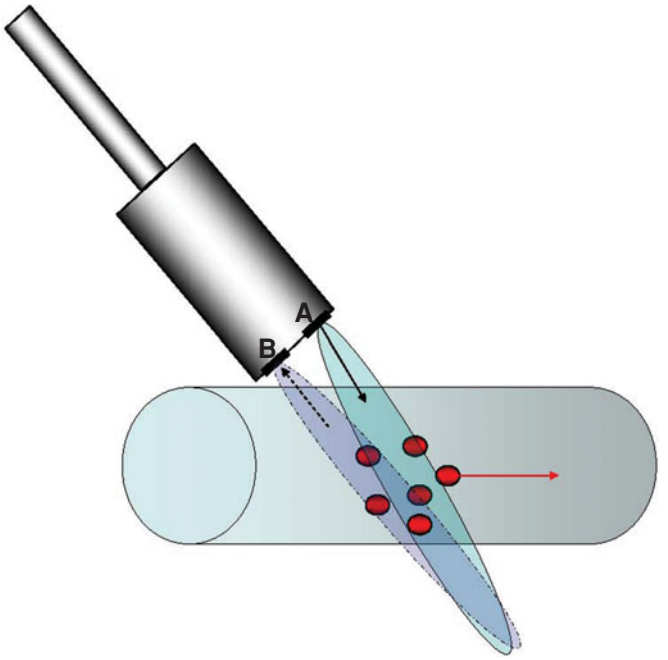


Figure 2-12. Continuous-wave Doppler. A is a transmitter element. B is a receiver element. The large area of overlap between the incident beam and the receiver beam results in an inability to assess where the sample is located. This is known as range ambiguity. Continuous wave can measure very high-flow velocities.

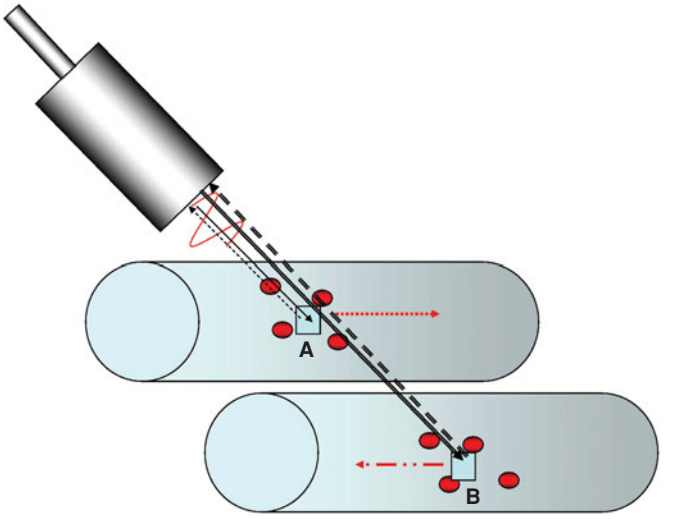


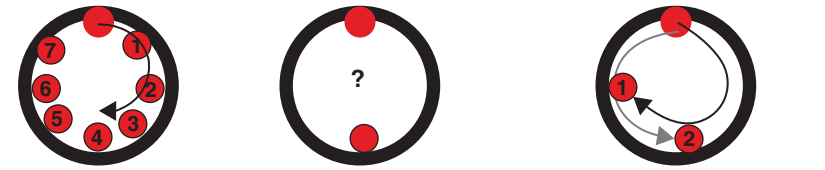
Figure 2-13. Pulsed-wave Doppler. The sample volume A is shallower and the position of the reflector (red blood cells) can be registered frequently. The sample volume B is deeper and the position of the reflector is sampled infrequently. Hence, the Nyquist limit = PRF/2 is exceeded and aliasing occurs.

the rate of those snapshots decreases with increasing depth of the sample due to a longer time of flight (Figure 2.13).

Because the depth of the sample and PRP are directly related (see pulsed ultrasound parameters above), one can also say that the longer the PRP (the shorter the PRF), the lower the rate of Doppler snapshot(s) that record the position of the reflector. That results in aliasing (Table 2.9, Figure 2.14).

Because of aliasing, with pulsed-wave Doppler measurements, high reflector velocities become inaccurate and the reflector may appear to be going in the direction opposite to the actual one (Table 2.8, Figure 2.14). Aliasing is a sampling error, occurring when the sampling rate is too slow in comparison with the reflector velocity. The following example will illustrate this further:

You are given snapshots of a unicyclist riding forward in a circus arena. You can see the position of the cyclist in the arena, but do not know if the cyclist's direction is going forward or backward. There is no timing mark on the snapshots and it takes him 5 minutes to complete the circle. If the snapshots are taken every minute, you will clearly see him moving forward. If sample snapshots are taken every 2.5 minutes, it is impossible to deduce if he is going backward or forward because he is only viewed on opposite sides of the arena. If the samples are taken every 4 minutes, you will have to conclude that the unicyclist is riding backward, because every sample snapshot will position him behind the previous one. He is still going forward, but low sampling frequency makes it appear otherwise. The same phenomenon explains why a plane propeller seems to



8:1 samples per rotaiton. Clockwise direction is obvious

2:1 samples per rotation. direction is obscure. Nyquist limit is reached

1:5 samples per rotation. Red dot seems to move counterclockwise (aliasing has occurred)

Figure 2-14. Nyquist limit and aliasing.

be moving in the direction opposite to its true course. The eye's sampling ability is exceeded by the rate of rotation, similar to the way in which in old cowboy movies the wagon wheels seem to rotate in the opposite direction of their true trajectory because the film rate was too slow.

Doppler frequency at which aliasing will occur is known as a Nyquist limit and is equal to half of the PRF of the pulsed-wave Doppler (Table 2.8).

$$\text{Nyquist limit (kHz)} = \text{PRF}/2$$

So the deeper the sample volume, the lower the PRF, and therefore the lower the Nyquist frequency limit and the more aliasing is observed.

Doppler shift is also directly related to the transducer frequency. The higher-frequency probe will produce more Doppler shift and more aliasing. With the blood flow velocity of 2 msec, the high-frequency (7 MHz) probe will produce a Doppler shift of 3 kHz and a low-frequency (3.5 MHz) probe will only produce 1.5 kHz. If the Nyquist frequency limit is 2 kHz, the first transducer will show aliasing and the second will not (Figure 2.15).

Methods of controlling aliasing:

1. Use shallower sample volume = increase PRF
2. Decrease carrier ultrasound frequency
3. Change Doppler angle to 0°
4. Use continuous-wave Doppler

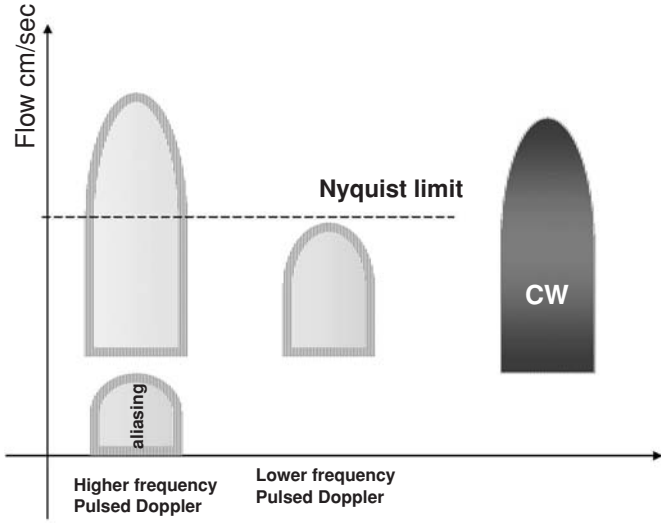


Figure 2-15. Eliminating aliasing. Aliasing can be eliminated by choosing the shallower sample (increased PRF), converting to continuous-wave Doppler, or choosing a lower-frequency pulsed-wave Doppler.

With pulsed Doppler, the Doppler frequency shift is very small (2300 times smaller) compared with the wave frequency of the pulse itself. This makes calculations from a single pulse difficult.

The problem can be partially solved by producing multiple ultrasound pulses (pulse packets or ensemble length) going in the same direction and interrogating the same sample volume. The Doppler shift of each pulse in the packet is measured separately, but then the packet is treated as a single pulse. This technique improves the accuracy of the velocity measurements and the sensitivity to low-flow states. Doppler shift is used to assess hemodynamic parameters such as stroke volume (SV) and to detect abnormal direction or velocity of blood flow in cardiac (echocardiography) and vascular ultrasound. Occasionally, it is also used in other modalities to detect movements of anatomical structures (e,g., pleura).

Doppler ultrasound has a number of important utilities. Normal blood flow is laminar, meaning that it is well organized, with the greatest flow velocity in the center and gradual decrease toward the vessel wall due to friction. At the level of the aorta, this bullet-like pattern represents blood ejection during systole. Doppler flow velocity will reflect it, describing different parts of the stream crossing the plane of Doppler interrogation over time. The integral of the flow velocity over time (velocity-time integral [VTI]) will enable the calculation of the vertical dimensions of the cross-section of the "bullet," while the aortic diameter (D) will allow the estimation at its base (cross-sectional area [CSA] = $(\pi \times \{D/2\}^2)$. The product will provide the volume of the "bullet" and thus stroke volume (Figure 2.16).

$$\text{SV} = \text{VTI} \times \text{CSA}$$

Those calculations are made by the equipment but are extremely dependent on the operator's ability to provide adequate Doppler data and proper measurement of the aortic diameter. As the blood flow reaches the area of the irregular lumen, predictable changes in flow velocity will take place at the site of the stenosis. The velocity will increase, reaching the highest velocity at the point of greatest narrowing (Figure 2.15). In the arterial bed there might be a loss of the pulsatile pattern, and in the venous bed a loss of phasic flow changes. At the exit point turbulent flow can develop. The pressure prior to stenosis (P_1) will increase and after the area of stenosis (P_2) will decrease, creating a pressure gradient. The predictable relationship between the flow velocity and pressure gradient was first described by Daniel Bernoulli. The Bernoulli principle

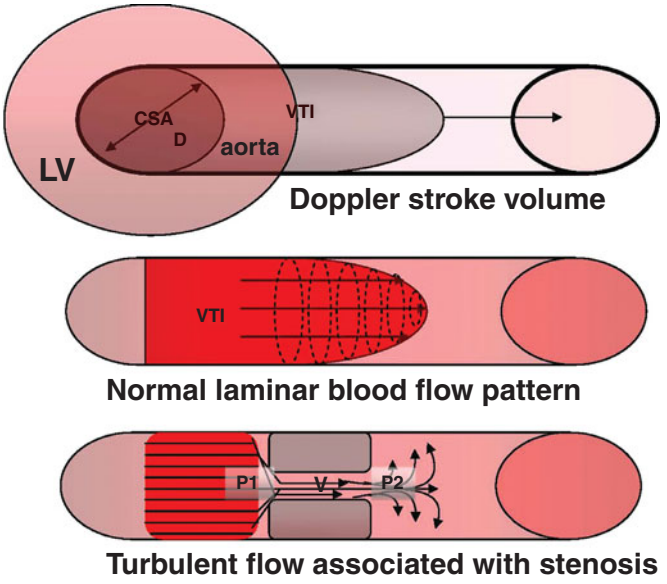


Figure 2-16. The normal laminar or parabolic blood flow pattern is bullet shaped with the highest flow velocity in the middle. Valvular or vascular stenosis is associated with an increase in pressure prior to the stenotic area, decreased pressure on exit, and increased flow velocity through the stenotic area. The pressure gradient is defined by the following relationship $P1 - P2 = 4 \times V^2$ (simplified Bernoulli equation). The cross-sectional area of the vessel (i.e., aorta) $CSA = \pi \times \{D/2\}^2$. VTI × CSA will describe the volume of blood passing through the area during systole. For the proximal aorta, this will be stroke volume.

states that as the speed of a moving fluid increases the pressure within the fluid decreases.

$$P_1 - P_2 = 4 \times (V_1^2 - V_2^2)$$

Where P_1 is prestenotic pressure, P_2 poststetsnotic pressure

V_1 is prestenotic flow velocity, V_2 poststetsnotic flow velocity

The simplified Bernoulli equation will allow calculating the pressure gradient from maximal (peak) flow velocity by using the following formula:

$$\Delta P = 4 \times (P\,\text{peak}^2)$$

Where ΔP is pressure gradient and V maximal flow velocity

The Bernoulli equation is used extensively in cardiac echocardiography and vascular ultrasound. Usually, the pressure calculations are made by the equipment. However, it is important for the physician to know how those numbers are generated.

COLOR FLOW DOPPLER

Color Flow Doppler is a multigated pulsed Doppler ultrasound technique, which means that multiple sample volumes are obtained and analyzed. Color flow Doppler is a pulsed modality and is therefore subject to aliasing and range resolution (Table 2.9). Multiple pulses in packets have different Doppler shifts that are averaged to provide mean or average flow velocities. Those flow velocities are measured not in one location, as with usual pulsed-wave Doppler or along the single line as with continuous wave, but on the two-dimensional grids and are usually combined with two-dimensional anatomical images (Table 2.9). The anatomical images are in black-and-white. Doppler information is provided in color, with negative Doppler shift (away from the transducer) usually depicted in the shades of blue and positive (toward the transducer) in the shades of red. Because it is not always presented in this way, a color map is provided to assist with identifying the direction and velocity of the flow. The upper part of the map shows flow toward the transducer and the lower part of the map shows flow away from it. The distance from the middle of the map bar is proportional to the flow velocity. In the upper part of the bar, the highest flow velocities are depicted on the top, and in the lower part, on the bottom (Figure 2.17). As has been alluded to, the returning echoes will have different Doppler shift frequencies. When knowledge of individual frequencies is essential, spectral analysis is used. Digital techniques such as autocorrelation and fast Fourier transform (FFT) are performed by the computer chips and are of little interest to this book's intended audience. Additional information on the matter of spectral analysis of Doppler signals can be obtained from the list of suggested readings.

QUALITY ASSURANCE

Knowing the basic principles of imaging and Doppler ultrasound will enable the physician to participate in and understand the quality assurance program that will guarantee optimal images and prevent unnecessary downtime. In the ICU, problems ranging from inconvenience to adverse patient outcomes may be avoided with these approaches. Though the equipment manufacturer might provide support for maintaining the equipment in the form of a service contract, the ultimate responsibility for quality assurance rests with the operator. In the case of ICU ultrasonography, the operator is nearly always the physician. Routine quality assurance will also alleviate medical–legal problems that

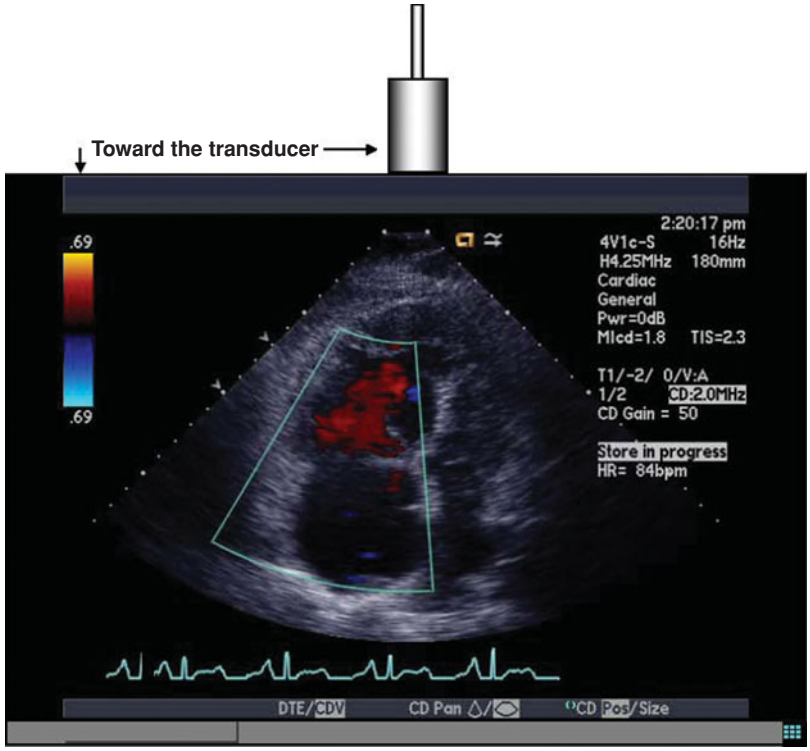


Figure 2-17. A colored map is provided for the colored Doppler of the right ventricle in this patient with pulmonary embolism.

may arise from the use of the ultrasound equipment, particularly for invasive procedures.

Usually, imaging ultrasound equipment is tested on phantoms with standard physical characteristics (Figure 2.18). The most commonly used ones are the American Institute of Ultrasound in Medicine (AIUM) 100 mm test-object and tissue-equivalent phantoms. Both are commercially available. In these phantoms, the speed of sound is 1540 msec. Objects with different acoustic impedance are placed in different positions to assess the equipment's ability to visualize (resolution) and properly estimate their position (calibration) (Figure 2.18). The sensitivity of the machine is evaluated by its ability to detect objects in the far field. Axial resolution is evaluated by objects placed at a certain depth in the pass of the ultrasound beam and so is vertical calibration. Lateral resolution and horizontal calibration are checked by the objects placed perpendicular to the direction of the beam (Figure 2.18).

Mock cysts and tumors with different acoustic impedance are also placed in the tissue phantom to check the equipment's ability to detect their diameter and characteristics (Figure 2.18). Doppler phantoms usually utilize belts or strings moving at a standard speed to evaluate the system's ability to detect direction and velocity of the moving objects. More sophisticated Doppler testing phantoms pump echogenic (visible with the ultrasound) fluid into plastic pipes at known velocities.

BIOEFFECTS

Ultrasound images are produced by inducing tissue vibrations at ultrasonic frequency. There is little evidence to suggest that those vibrations have any biologic consequences. The same does not hold true for the ultrasound energy converted into heat. For instance, at the soft tissue–bone interface, roughly half of the sound is reflected back, but the remainder of the energy is absorbed by the bone, creating temperature elevation at this tissue–bone interface. This tissue-heating is related to both the intensity and focus of the ultrasound beam. The more focused the ultrasound beam, the less the area of heat production, as the surrounding tissues dissipate thermal energy. This allowed the US Food and Drug Administration (FDA) and the AIUM to establish intensity limits: 100 mWatts/cm^2 for unfocused and 1000 mWatts/cm^2 (1 Watt/cm^2) for focused ultrasound.

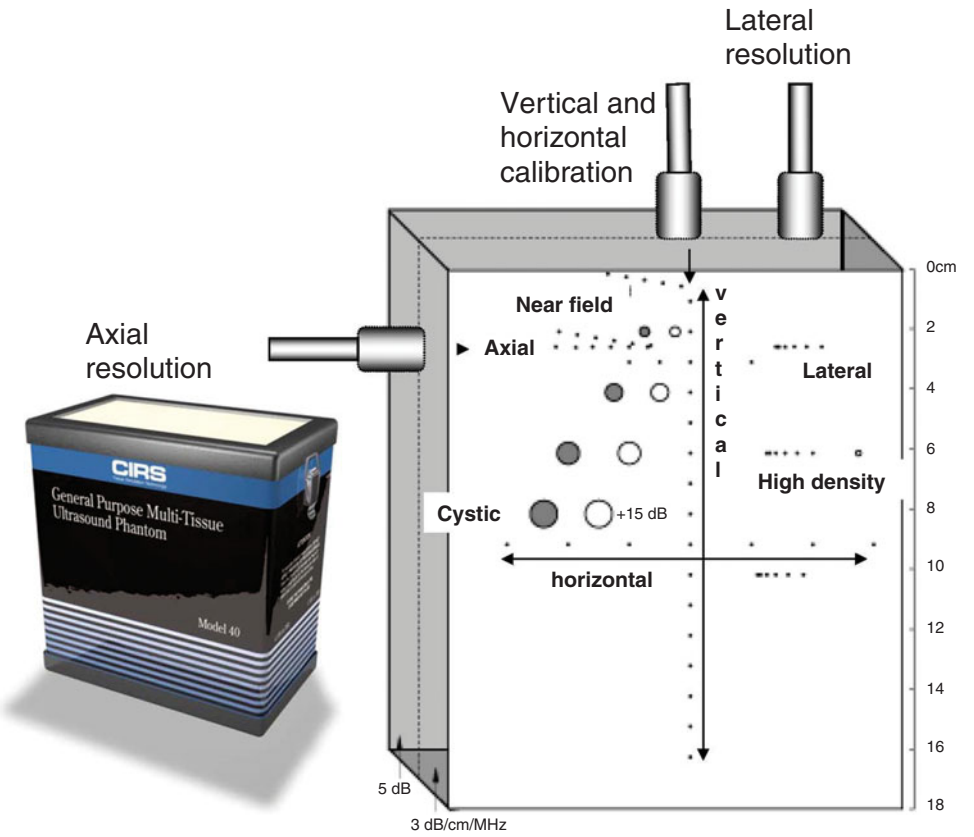


Figure 2-18. Tissue phantoms, with standard characteristics, can be used to assess axial and lateral resolution and calibration.

Aside from the thermal effects on the tissues, the other significant effect is called cavitation. Soft tissues contain microscopic areas of gas bubbles (gaseous nuclei) that can be heated by the ultrasound beam, resulting in their rapid expansion and ultimate bursting. This might create local mechanical stress and further tissue-heating. Though little evidence for cavitation has been shown with diagnostic ultrasound, the possibility of tissue injury by that mechanism definitely exists.

This possibility prompted the AIUM in 1988 to issue a safety statement that is applicable even now to the use of diagnostic ultrasound, including its application in critical care medicine. As for ultrasound, the AIUM suggested that:

1. No study should be *performed* without valid reason.
2. No study should be *prolonged* without valid reason.
3. The minimal output power should be used to produce optimal images if the ultrasound machine allows control of output power. (As low as reasonably achievable, or ALARA principle).

Suggested Reading

Edelman SK. *Understanding Ultrasound Physics.* 3rd ed. Huston, Tex: Esp Inc; 2004.

Hedrick WR, Hykes DL, Starchman DE. *Ultrasound Physics and Instrumentation.* 4th ed. St Louis, Mo: Elsevier Mosby; 2005.

Kremkau FW. *Diagnostic Ultrasound: Principles and Instruments.* 7th ed. St. Louis, Mo: Saunders Elsevier; 2006.

Miele FR. *Ultrasound Physics and Instrumentation.* 4th ed. Forney, Tex: Miele Enterpises; 2006.

Owen C, Zagzebski J. *Ultrasound Physics Review: A Q&A Review for the ARDMS Ultrasound Physics Exam.* Pasadena, Calif: Davies Publishing Inc; 2008.

CHAPTER 3

Transducers, Image Formation, and Artifacts

Alexander Levitov

TRANSDUCER STRUCTURE AND FUNCTION

Transducers are defined as devices converting one form of energy into another. In the case of ultrasound, electrical energy is converted into mechanical (acoustic) energy. The most familiar transducer is a telephone receiver, with an earpiece that converts electrical impulses into sound waves and a mouthpiece that converts sound energy into electricity. Imaging transducers combine both functions by emitting and receiving ultrasound pulses and converting them into electrical impulses for further processing. Non-imaging continuous-wave (CW) Doppler transducers, just like the telephone receiver, have two elements; one is constantly emitting sound and the other is receiving sound. Figure 3.1 shows the anatomy of the imaging transducer.

At the core of the ultrasound transducer (probe) is a sheet of piezoelectric material known as an active element, or simply the "crystal." It is usually made of lead zirconate titanate, or PZT. This material will create electricity when mechanically deformed (direct piezoelectric effect) and it itself deforms when electrical voltage is applied to its surface (reverse piezoelectric effect). The ability of some natural and man-made materials to create electricity when physically deformed was discovered by the brothers Pierre and Jacques Curie in 1880 and first used to produce ultrasound in sonar to track German U-boats during World War I in France in 1917. The piezoelectric effect of PZT irreversibly disappears as temperatures rise above 360° C (Curie point), making it impossible to sterilize ultrasound transducers with heat. The PZT crystal is one-half–wavelength thick (for the speed of sound in the active element itself). Connected to the PZT crystal is a wire that transmits electrical impulses from a pulse generator to the crystal during a pulse-generation phase, and away from it to the processor, during the "listening" phase, when an electrical impulse is generated in the PZT crystal by the returning echo. The listening phase is 10 times longer than the pulse duration, so the duty factor in imaging ultrasound transducers is 0.1–1% (see Chapter 2). The transducer can be also set to emit sounds of a so-called fundamental frequency, but receive echoes with frequencies that are multiples of the fundamental one. This tissue harmonic imaging is usually performed with returning frequencies that are twice (first harmonic) or even four times (second harmonic) higher than the fundamental one. Because the harmonic frequencies are generated in the tissues themselves, the image is resistant to certain artifacts and tends to be of a better quality. Behind the active element (PZT) is a backing or damping material. Just as a guitar string continues to produce sound after being struck once by the player, the electrical impulse, once it exits the PZT, will keep on ringing, producing longer pulse durations and spatial pulse lengths with deteriorating axial resolution. The backing material works like a guitarist's hand placed over the string, reducing the time that the PZT spends vibrating (ringing) after each electrical impulse and improving the image quality. The backing material is usually composed of tungsten-impregnated epoxy resin. Continuous-wave Doppler transducers emit sound waves constantly and therefore do not require or contain any backing material (Figure 3.2).

In front of the PZT crystal is a matching layer that is a one-quarter wavelength thick. The difference in impedance results in an increase in reflection. The impedance of the matching layer is between that of the PZT crystal and the skin, in order to increase the transmission of the ultrasound from the active element into the tissues. To further reduce the impedance difference between PZT and skin, ultrasound gel is used. The impedance of the gel is less than that of the matching layer but more than that of the skin, making ultrasound transmission relatively smooth (Figure 3.1).

Wire, backing material, PZT crystal, and the matching layer are all housed in a case to protect them from

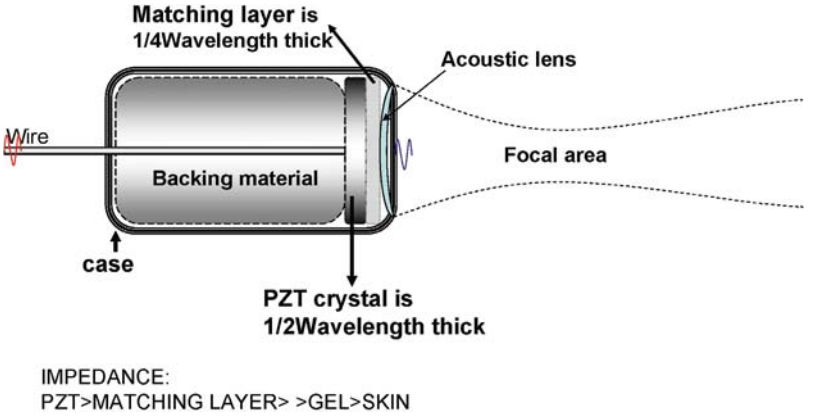


Figure 3.1. An imaging transducer both emits and receives signals. The PZT (piezoelectric) crystal converts electrical impulses from the wire into ultrasound and vice versa. A matching layer reduces internal reflections within the probe by gradually decreasing acoustic impedance. Backing material reduces the length of the pulse by preventing after-ringing (dampening effect) Acoustic lenses improve focus. The case prevents electrical shock exposure for the patient and the operator.

the elements and to protect the patient and the operator from an electrical shock (Figure 3.3). One should never attempt to use the transducer with a cracked housing or frayed wire.

Single-crystal transducers can produce several forms of imaging, of which only M-mode and two-dimensional (2D) (with mechanical scanning) are presently in use. A and B modes are only mentioned for their historical relevance (Figure 3.4). Both A-mode and B-mode will relate the strength of the signal to distance to the boundary where that signal is produced. This strength is either represented by the height (A-mode) or the brightness (B-mode) (Figure 3.4). B-mode of the single active element transducer will produce a series of dots arranged in a line. With multiple crystal or array transducers, each line produced by the single active element will coalesce with the one formed by its neighboring element to form a 2D image (see below). Because of this relationship, 2D images are sometimes called "B-mode," but this is technically incorrect. M-mode shows the position of the moving boundary over time, without reference to the signal's strength (Figures 3.4 and 3.5). M-mode is presently used in echocardiography and occasionally in noncardiac chest ultrasound for the diagnosis of pneumothorax, but 2D images are the primary mode used today. Two-dimensional imaging displays are used in all portable ultrasound machines available for intensive-care unit (ICU) use and in almost all diagnostic ultrasound equipment on the market.

Though 2D images can be produced by a single crystal transducer by mechanically moving the active element in a swinging motion across a scan plane (like moving a spotlight beam to see a deer in a night-time

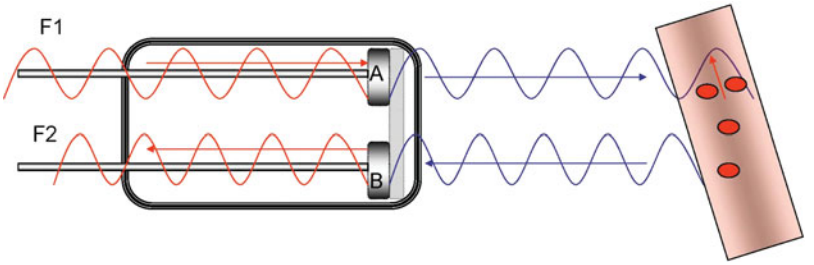


Figure 3.2. Continuous-wave Doppler transducer has two PZT crystals. One constantly emits and the other receives signals. Element A transmits continuous ultrasound waves with frequency f1. Element B receives frequency f2 (f1 − f2 = Doppler shift). Backing material is not necessary because continuous-wave signals require no dampening.

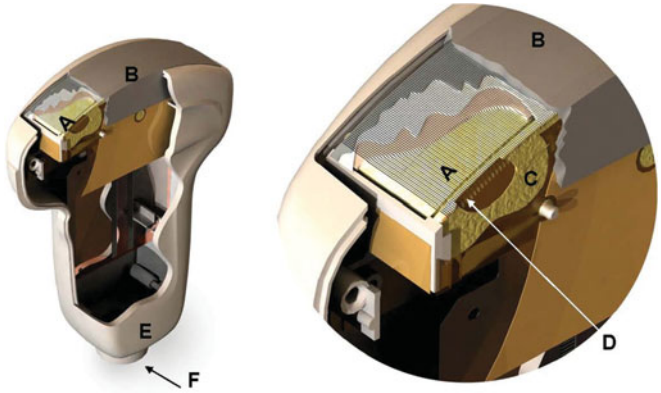


Figure 3.3. Components of the array transducer: (A) PZT crystals, multiple crystals (active elements), can be activated separately; (B) matching layer; (C) backing material; (D) wires to each PZT element; (E) case; (F) cable all wires are still separated within the cable.

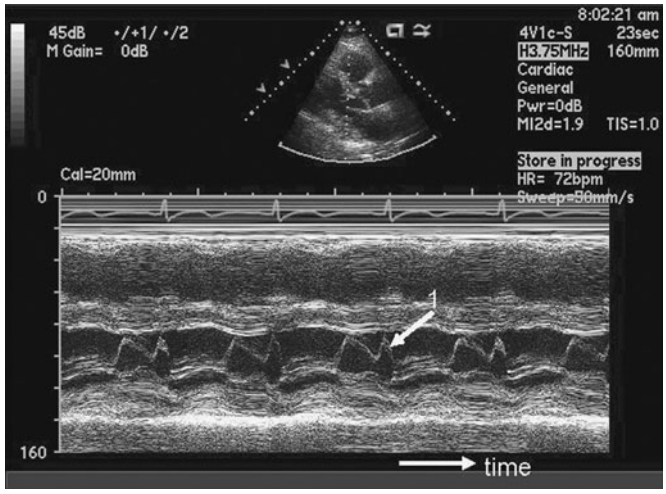


Figure 3.5. M-mode examination of the heart showing the position of different cardiac structures (intraventricular septum, mitral valve leaflets and inferior wall of left ventricle) plotted over time. Each 50 mm of the horizontal axis is 1 s. 1- Position of the anterior leaflet of the mitral valve at that time. Please note that brightness of the signal is of little relevance in an M-mode study.

meadow), most of the modern transducers are composed of multiple active elements (Figures 3.3, 3.6 and 3.7). These so-called transducer arrays contain multiple PZT crystals with a separate wire attached to each element (Figures 3.3, 3.6 and 3.7). The electronic circuitry allows for each element to be activated separately in a specifically designed order. Arrays of active elements can be placed in a straight line (linear array), in an arc (convex or curved arrays), in concentric circles (annular arrays), or even in a checkerboard pattern (three-dimensional arrays) (Figures 3.3, 3.6 and 3.7). According to the sequence of element activation, transducers can also be divided into sequential arrays or phased arrays.

With sequential-array probes, groups of PZT crystals, usually arranged in linear or curved arrays, are fired in a sequence starting from one end to the other, five to 10 elements at a time, with each group firing

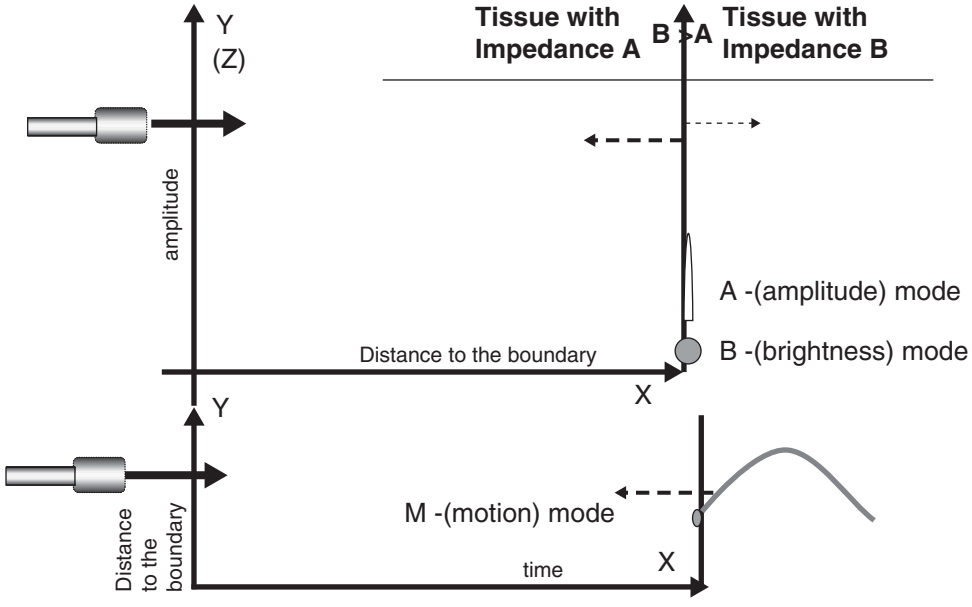


Figure 3.4. Display modes: A-mode displays amplitude of the signal and reflector depth; B-mode displays the same parameters, but the amplitude of the signal is represented by its brightness and not the height; M-mode displays reflector depth over time.

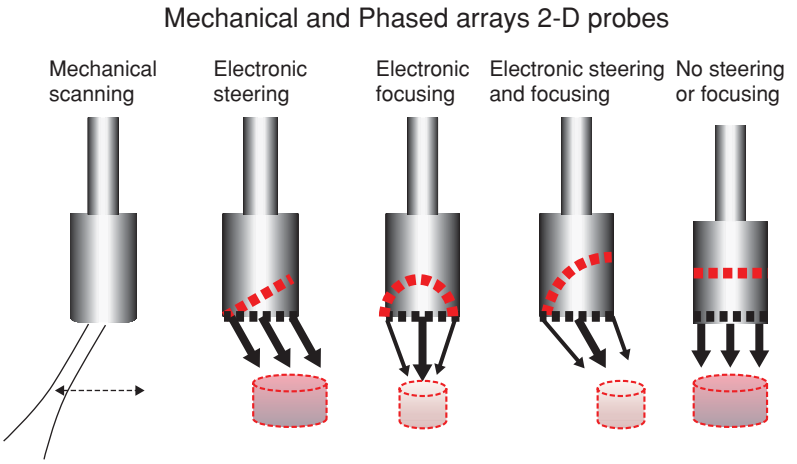


Figure 3.6. Mechanical scanning and phased-array probes offer a large acoustic footprint in the far field through a small window. They are common in cardiac ultrasound where the window is limited by intercostal spaces. In addition, phased-array probes offer electronic steering (sweeping) and focusing of the ultrasound beam. The operator is capable of selecting single or multiple focal points and the width of the sweep. A lack of moving parts also makes phased-array probes more reliable and durable.

immediately after its neighboring group. This is similar to a "wave" in a baseball stadium. When the activation sequence reaches the opposite end of the transducer, the process starts again. Linear sequential-array probes produce images only of the size of the transducer with a fixed focus, since each crystal in the array has its own focal zone and there is an inability to steer the beam. The image produced has a uniquely characteristic square shape (Figures 3.7 and 3.8). The linear-array probes are more common in vascular ultrasound. Convex sequential-array probes tend to be larger, with a fixed focus (for the same reason), but the image has a sector shape with a blunted top. Convex arrays have the advantage of a large near field and even larger far field and are used extensively in abdominal ultrasound, where large images are necessary (Figures 3.7 and 3.9).

However, most transducers that critical care physicians will encounter will be either linear or convex

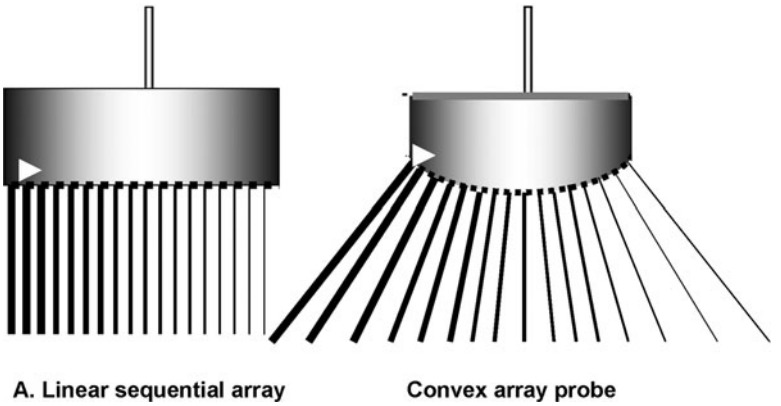


Figure 3.7. Two-dimensional imaging. Linear sequential arrays consist of multiple PZT crystals (elements) arranged in a line. Each one is connected to a separate wire. Elements are activated in groups from one end of the transducer to the other. A similar arrangement is present in a convex array, but the elements are arranged in a curve, giving this type of the transducer a wider view (larger footprint) in the far field. Elements in curved-array probe can be activated individually or in small groups.

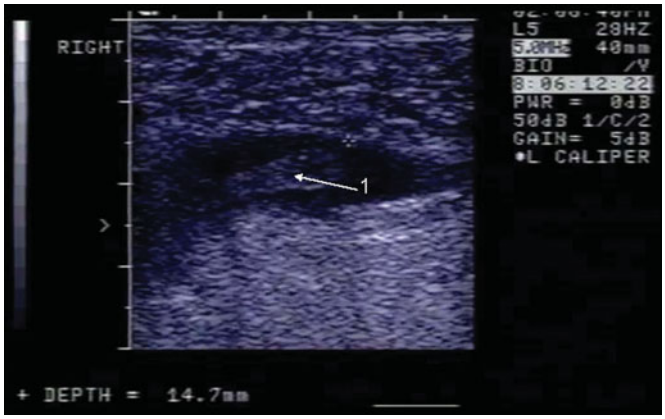


Figure 3.8. Vascular image (right common femoral vein) produced by linear sequential array transducer. Notice the image is square and is of the same size as a vessel. The solid-looking structure 1 in the middle of the vessel is a thrombus.

phased arrays. In phased-array probes, both beam steering and focusing is achieved electronically by sequenceing the PZT crystal activation. Each active element is activated with an approximately 10-nanosecond delay in a pattern created by a beam former in the ultrasound machine. Each PZT crystal in the array receives a signal in that predetermined pattern. There is also a similarly spaced delay in signal reception by the ultrasound machine. If the delay pattern is from the left to the right of the array, the beam is steered to the left. If the delay pattern is from right to left, the beam will be steered to the right. This will create the sweeping necessary to form 2D images without moving the active element (Figures 3.6 and 3.10).

The entire sweep from one side of the probe to the other will produce one imaging sector or frame. If the

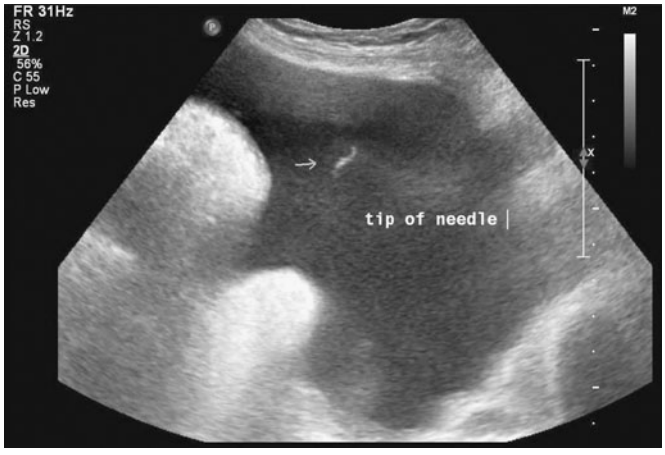


Figure 3.9. Image of the abdomen produced by the convex-array transducer. Notice a large acoustic footprint in both near and far field.

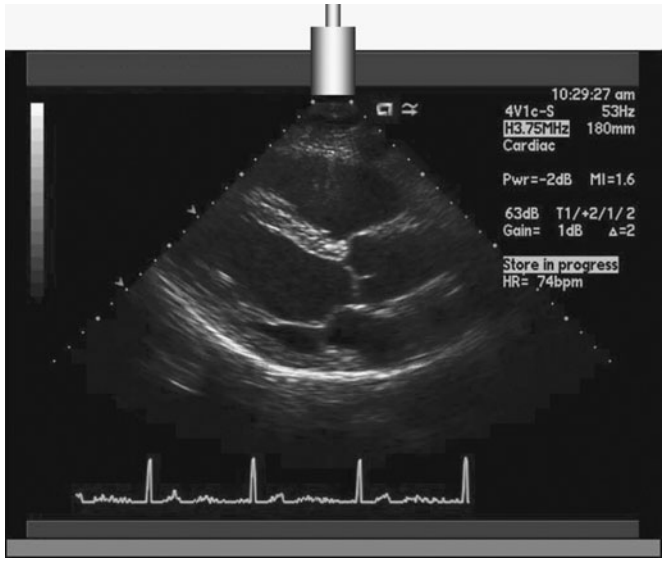


Figure 3.10. Echocardiographic image produced by the phased-array probe. Electronic steering enables the production of an image of the heart from a small acoustic window (intercostal space).

activation pattern is parabolic, the beam will be focused on a particular depth and the combination of patterns will produce both focusing and steering. Phased-array probes, regardless of how the active element patterns are arranged, are now the predominant probes used in echocardiography and are being used more frequently in vascular and general ultrasound.

The quality of the detail of the 2D image (spatial resolution) produced by an array probe depends on the number of separate ultrasound beams (lines) generated by the probe and the width of the sector necessary to produce the image. The line density, therefore, will depend on the number of the PZT crystals in the array probe and the sector width. To visualize the entire heart, for example, one will need a wider sector than the one necessary to visualize just the mitral valve. A cross-section of the aorta will need a wider sector than a cross-section of the carotid artery. The more lines per sector, the higher the line density, and the better the image quality and spatial resolution will be. Shallow images allow higher ultrasound frequency with better axial resolution per line and improved overall image quality (spatial resolution) (Figure 3.11, Table 3.1). One complete sweep of either a mechanically or electronically steered beam will produce one frame of the 2D image. The sequence of the frames will create a movie clip that can be observed or recorded by the operator. The number of frames per unit time is the temporal resolution of the exam. For continuous motion to be perceived as continuous, a minimal frame rate of 15

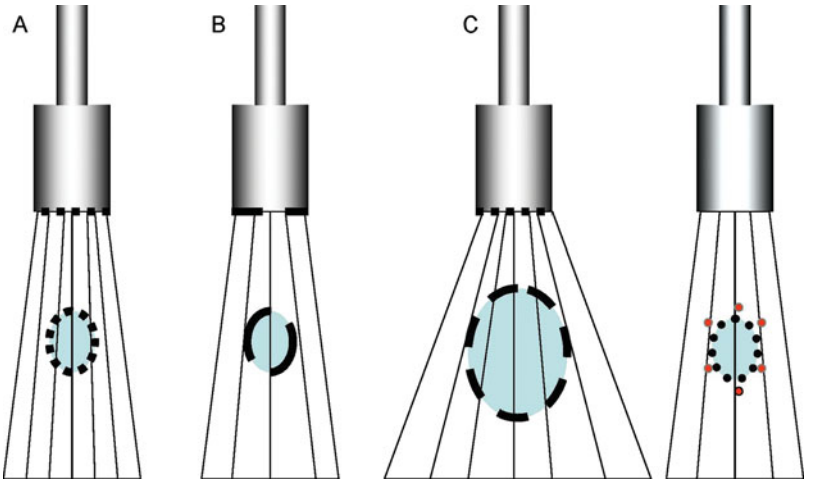


Figure 3.11. Probe A produces more ultrasound beams (lines) than probe B, with a resulting improvement in image quality (better spatial resolution). Probe C produces as many lines as probe A, but a wider sector decreases line density and degrades spatial resolution. Multifocusing requires more pulses per line and improves spatial resolution.

frames per second is necessary to provide a minimally acceptable temporal resolution. The more frames per second, the higher the temporal resolution. For a moving structure, like the heart, the higher the temporal resolution, the more real time is the image. The number of frames is limited by the time necessary to create a single frame. In any imaging, the previous pulse has to be received by the transducer and processed before the next one can be generated (Figure 3.11).

Temporal resolution is determined by the speed of sound in the medium and not controlled by the operator. The greater the distance the sound has to travel to deeper images, the longer the listening time of the transducer, the line density, and the amount of pulses necessary to create a frame (sector width and number of focal points) (Table 3.1).

In addition to commonly used imaging transducers, several specialty transducer types should be mentioned. Multidimensional transducers include 2D arrays used to create 3D ultrasound images (Figures 3.12 and 3.13) and one and one-half–dimensional arrays used to improve vertical components of the image by reducing beam width. Three-dimensional–image capabilities are presently unavailable in portable ICU ultrasound equipment, but will undoubtedly become standard in the relatively near future. Annular phased arrays are automatically focused in multiple planes and are steered mechanically. These are used mostly in OB-GYN imaging. Vector arrays combine linear-, sequential-, and phased-array technologies in one transducer (Figure 3.12).

The construction of a pulsed Doppler transducer is very similar to that of a single-crystal imaging transducer, mentioned above. Continuous-wave Doppler transducers emit ultrasound waves continuously, just as their name implies, and therefore do not require any backing material (Figures 3.2 and 3.3). Different 2D transducer types are summarized in Table 3.2. Most modern transducers combine imaging and Doppler capabilities in a single probe.

IMAGE FORMATION

The formation of the ultrasound image requires proper flow of electrical impulses into the transducer and interpretation of direction, strength, frequency, and

TABLE 3.1. Factors determining spatial and temporal resolution of the 2D image

Improved 2D resolution	*Depth*	*Sector width*	*Line density*	*Focusing*
Spatial	Shallow	Narrow if the line density increases (zooming in)	High	Multiple
Temporal	Shallow	Narrow if the line density is unchanged	Low	Single

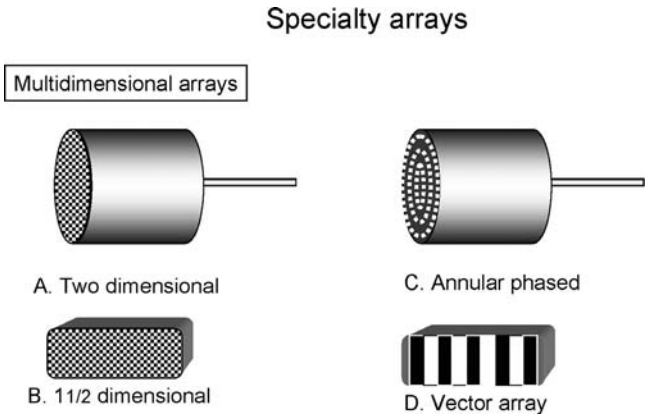


Figure 3.12. (A) Two-dimensional array is used to produce three-dimensional images. It has as many active elements in the vertical as in the horizontal plane. (B) One and one-half–dimensional array has more elements in the horizontal than vertical plane, makes a thin slice, and improves resolution in the vertical dimension. (C) In annular phased probe, PZT crystals are arranged in a concentric circular pattern and are activated from the innermost circle out. Each circle is focused on the particular depth (central shallower than outer ones), making it focused in all planes. Annular phased transducers are steered mechanically. (D) Vector arrays combine phased-array and sequential-array technology.

timing of the returning signal. The ultrasound machine or system makes all of that possible. Once an image is formed, it is displayed on a screen, usually in a digital format. In modern ultrasound systems, particularly portable ones that the ICU physician is likely to encounter, computer chips are increasingly used to simplify operation and optimize images (Figure 3.14). However, six common components are used in all ultrasound machines, irrespective of size or function, namely:

1. The master synchronizer organizes and times the flow of electrical signals within the system.

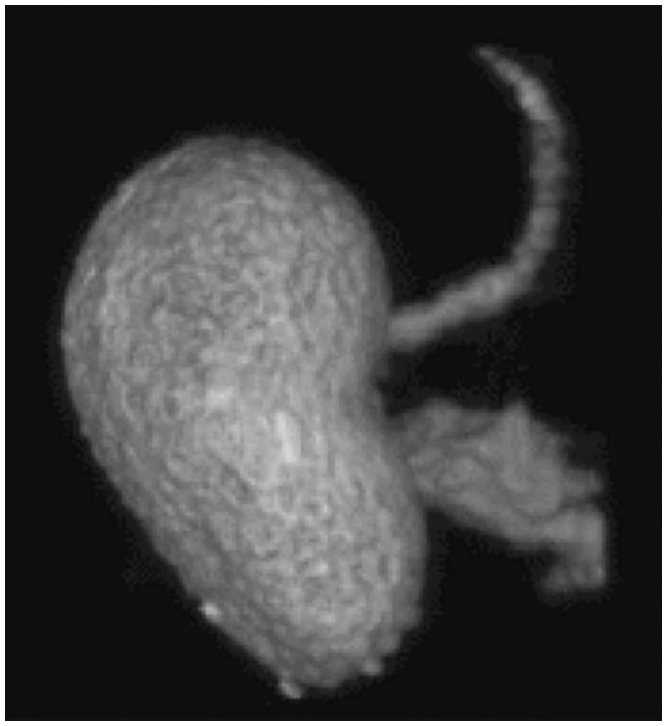


Figure 3.13. Three-dimensional ultrasound image of the kidney, produced using two-dimensional array.

2. The pulser or beam former controls the firing pattern in the transducer and pulse amplitude, pulse-repetition frequency (PRF), and pulse-repetition period (PRP).
3. The transducer converts electrical signals received from the beam former into a sequence of ultrasound pulses and converts returning acoustic pulses into electrical impulses.
4. The receiver/processor contains the necessary elements for conversion of the returning electrical impulses into images (Table 3.3).
5. The display (screen, audio speakers, recording devices) presents data for interpretation and storage (usually in digital format).

TABLE 3.2. Comparison of different 2D transducer types

Transducer type	*Image shape*	*Steering*	*Focusing*
Mechanical	Sector	Mechanical	Fixed
Linear sequential array	Rectangle	None	Fixed
Linear phased array	Sector	Electronic	Electronic
Annular phased	Sector	Mechanical	Electronic
Convex sequential	Blunted sector	None	Fixed
Convex phased	Blunted sector	Electronic	Electronic
Vector	Flat top sector	Electronic	Electronic

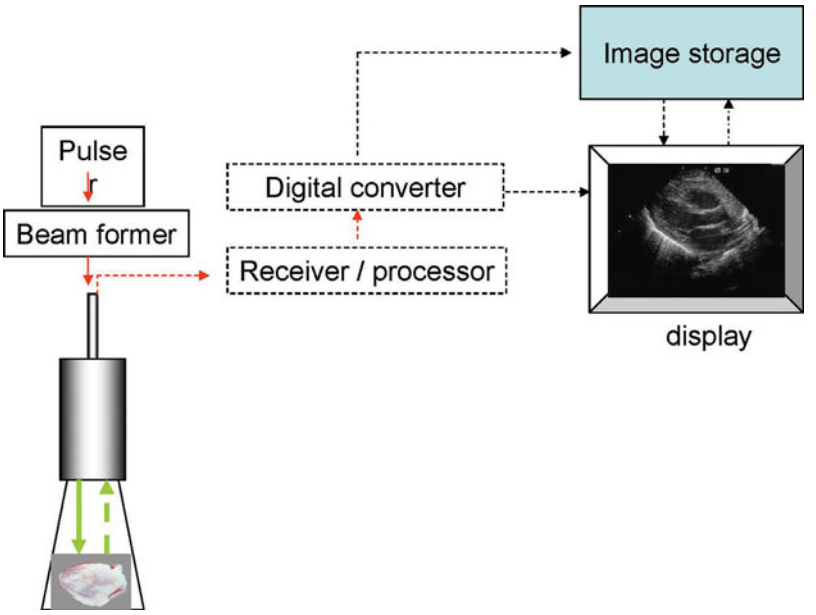


Figure 3.14. Basic components of the modern ultrasound system. The pulser generates electrical impulses organized by the beam former to electronically focus and steer (sweep) the ultrasound beam generated by the transducer. Active elements in the transducer convert electrical impulses into mechanical (acoustic) energy sent into the tissues. Incoming ultrasound echoes are converted into electrical impulses by the transducer and sent into the receiver/processor. The processor deciphers electrical information and an image is created. In modern systems, the image is displayed and stored in a digital format. Outgoing information is represented by solid arrows: red for electrical, green for ultrasound signals. Incoming information is represented by broken arrows.

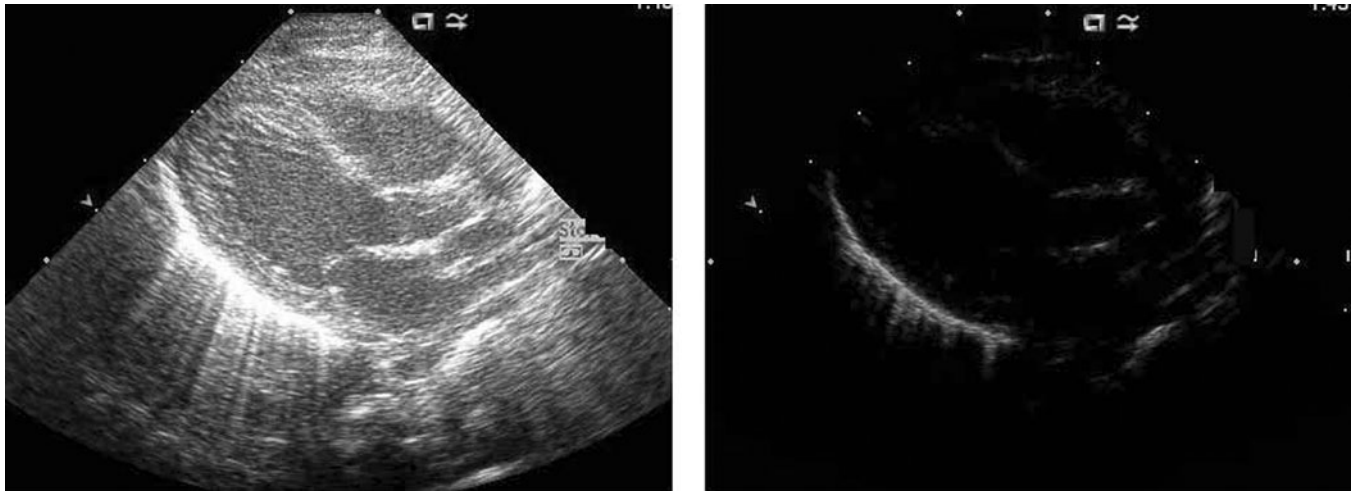
6. Storage devices, also known as an archive, keep information more or less permanently for further review and to meet legal requirements.

Since the physician–sonographer will never come in contact with the master synchronizer, it is mentioned here, but will not be discussed further. Pulses or phased-array beam formers produce the sequence of electrical pulses that excite PZT crystals in the transducer. For the array transducer, separate electrical impulses are produced and timed separately for each PZT crystal. The voltage of those pulses may reach 500 volts, thus they present a real electrical hazard to the patient and the operator. *One should never use a transducer with a defective housing or frayed wire, no*

TABLE 3.3. Functions of the receiver/processor

Receiver functions	*Adjustable*	*Processing*
Amplification	Yes	(Receiver gain) all signals are amplified and image becomes brighter when increased
Compensation	Yes	(TGC) deeper signals are amplified more
Compression	Yes	Gray-scale map is changed, dynamic range is decreased
Demodulation	No	Form and the direction of the signals are changed
Rejection	Yes	Only weak signals are rejected, strong are not affected

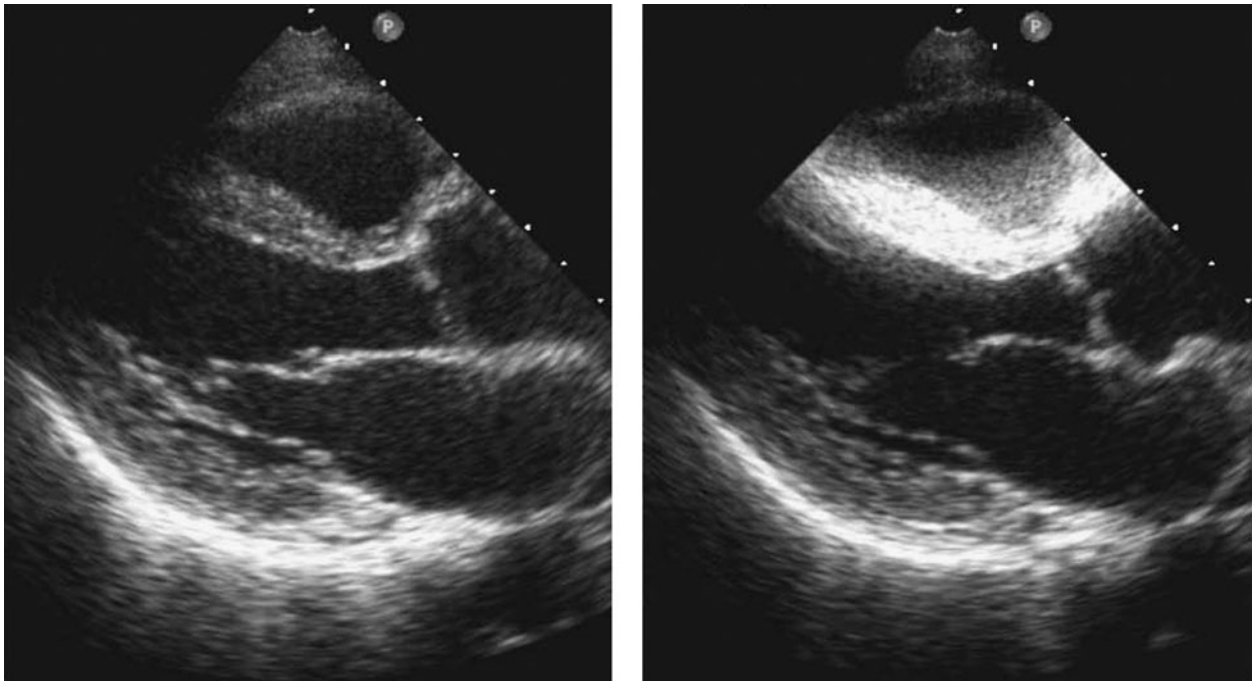
TGC indicates time-gain compensation.



A B

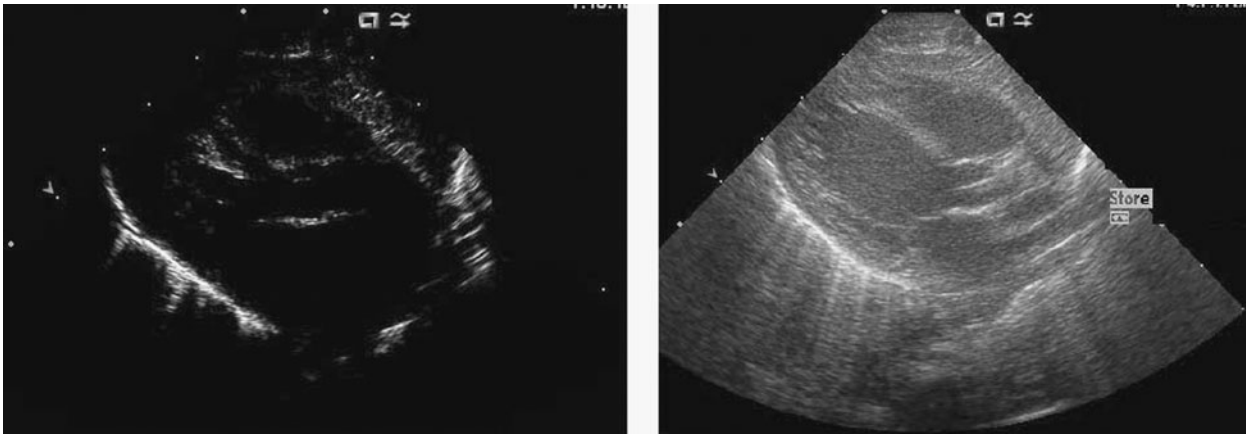
Figure 3.15. Receiver gain controls the brightness of the entire screen. (A) Amplification settings that are too high; (B) settings that are too low. In either case, image quality is degraded.

matter what the circumstances. Traditionally, the amplitude of the output pulses (transducer output) was controlled by the operator, who could increase the amplitude of the electrical pulses in the pulser. In some larger echocardiography machines, this is still possible. High transducer output improves the signal-to-noise ratio and image quality. Presently, in almost all portable ultrasound systems, transducer output is set by the manufacturer and cannot be adjusted by the sonographer. The higher the amplitude of the electrical impulses and therefore the transducer output, the higher the potential to experience the adverse biological effects of ultrasound. Because manufacturers tend to improve image quality by setting the transducer



A B

Figure 3.16. (A) Normal time gain compensation settings. (B) Overcompensation (too high of a gain) in the near field. Image quality is deteriorated (no fine details can be seen in the near field). Notice that both images are nearly identical in the far field. (Image courtesy of D. Adams, RDCS.)



A B

Figure 3.17. In most portable ultrasound systems compression is set automatically. In some, it is controlled by the operator, but in either case it sets the dynamic range of the image (the shades of gray representing the differences between the brightest and the darkest areas). In many ways it is analogous to the contrast. (A) Image is overcompressed; it has narrow dynamic range with bistable (black and white) appearance, but also a high contrast. (B) Image is undercompressed; it has a wide dynamic range with multiple shades of gray, but the contrast is low. In either case, the image quality is degraded.

output to the highest safe amplitude, it is incumbent upon the physician–sonographer not to prolong the examination unnecessarily.

A receiver/processor processes impulses received by the transducer and makes them suitable for the display (Table 3.3). The returning ultrasound and therefore electrical impulses are very weak and need to be amplified. The amplification, also known as receiver gain, is controlled by the operator and increases amplitude of all signals received by the transducer. In almost all imaging modalities, the amplitude (strength, volume) of the signal is presented on the screen as brightness. Increasing the receiver gain will increase the brightness of the entire image (Figure 3.15). After the signals have been amplified, signal compensation takes place. Compensation treats returning signals discriminately, depending on the depth of the image. Because the depth is derived from the time of flight, the control is known as time-gain (TGC) or depth-gain (DGC) compensation. Attenuation makes signals from greater depths (arriving later) disproportionately weaker compared with shallower (earlier arriving) echoes. Time-gain compensation amplifies returning signals to a greater degree if they have been received from the deeper parts of the image. Higher frequency traducers will produce quicker attenuating signals and require more TGC. Larger ultrasound systems usually have multiple controls increasing amplification of each depth separately, but in portable ICU equipment there may be only two TGC controls: shallow gain and deep gain, and the smoothness of the transitions is determined by the computer processing chips, which are often proprietary (Figure 3.16).

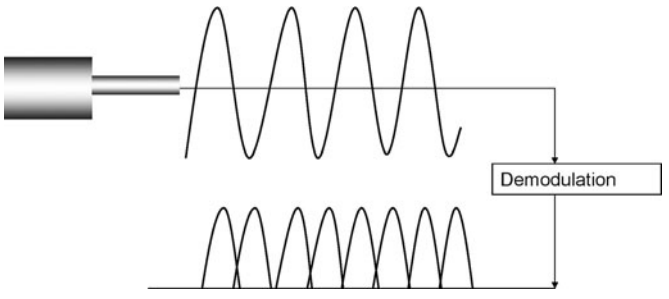


Figure 3.18. Demodulation converts all negative electrical impulses into positive ones with the same amplitude, making image formation possible, as negative electrical impulses cannot be processed further.

After compensation, signal compression takes place (Figure 3.17). Compression brings all signals within the brightness range visible to the human eye. Relative relationships between the signal amplitude continue to be the same (i.e., the highest are still the highest and the lowest are still the lowest), but the highest amplitude signals are reduced by an established number of decibels (dB) while the lowest amplitude signals are increased, so the difference between the highest and the lowest amplitude signals (dynamic range) is diminished. For example, if the original signal has a dynamic range of 100 dB and a compression of 30 dB takes place, the resulting dynamic range will be 70 dB. Visually, the

dynamic range is represented by the gray scale of the image and is controlled either by the operator or set automatically by the processor, as in most portable systems. The wider the dynamic range (the less the compression), the more shades of gray represent the differences between the darkest and the brightest parts of the image and the lower is the contrast. The narrow dynamic range increases contrast, but makes images bistable (back and white), with lesser detail.

The vibration of the PZT crystal creates an alternating electrical current with positive and negative phases. because ultrasound cannot identify negative electrical impulses, all negative voltages are converted to positive ones with the same amplitude (rectified). Then the signals are enveloped where all the changes in amplitude are evened out. Rectification and enveloping are collectively known as demodulation. Demodulation is performed by the processor in all systems and cannot be controlled by the operator (Figure 3.18).

After completion of demodulation, some low-level signals are rejected. Rejection does not affect high amplitude (bright) signals because they are usually meaningful to image formation. Low-amplitude signals can be rejected by the sonographer through the entire image if they do not appear meaningful and reduce image quality. Rejection can be fully or partially relegated to

TABLE 3.4. List of assumptions for ultrasound imaging

Assumptions	***Validity***
1. Ultrasound travels in a straight line from the transducer to the reflector and back.	Regrettably, this assumption is rarely valid. Although ultrasound pulses might approach the reflector in a relatively straight line, they probably will not strike the reflector at a 90° angle. Therefore, reflected echoes might never reach the transducer or return to it after being reflected from multiple other reflective boundaries.
2. The transmitted portion of the ultrasound continues to travel in a straight line, until it encounters another reflector, at which time the reflected echo will again return to the transducer in a straight line.	The transmitted portion of the pulse is the subject of refraction and is likely to continue its pass in a slightly different direction. It may also return to the transducer after encountering one or several secondary reflective boundaries.
3. Imaging ultrasound always strikes the reflector at a 90° angle.	This may be a valid assumption, but usually is not. It is also in stark contradiction to the next assertion regarding Doppler measurements if the single transducer is used for both.
4. Doppler ultrasound always strikes the moving reflector at a 0° angle.	This one is almost never correct and obviously contradicts the prior assumption if the same transducer is used for both imaging and Doppler.
5. All reflections arise only from the structures positioned along the axis of propagation of the ultrasound beam (pulse).	The axis of beam propagation itself is distorted by refraction, nonorthogonal reflections, and unexpected reflective boundaries.
6. The plane of 2D ultrasound sweep is very thin (has essentially no thickness).	This is incorrect. The ultrasound beam, just like a light beam, has a diameter and may simultaneously encounter and reflect off multiple structures.
7. The speed of sound in soft tissues is 1540 m/s.	This is incorrect. In fact, the human body does not contain any generic soft tissues. As the speed of sound is different in different tissues, and the distance to the reflector is calculated based on this assumption, this distance is never correct. Therefore, the position of the reflector is an estimated, and not a real, anatomical one.
8. The intensity of the reflection is related to the nature of the tissue.	The intensity of the reflection depends upon the interaction between multiple reflective boundaries and the ultrasound. Structures below the boundary with a higher difference in impedance may not be visualized (acoustic shadowing).
9. Two-dimensional ultrasound provides information in real time.	Depending on the temporal resolution of the ultrasound system and the depth of the reflector, the image formation is delayed and the motion of the reflective boundary is again an approximation of its real motion.

TABLE 3.5. Common compromises necessary to obtain the "best possible image"

Compromise	*Reason*	*Operator control*
High vs. low frequency transducers	High frequency improves image quality, but limits the penetration.	Choose the appropriate transducer for the image depth and the view with the least depth.
High vs. low PRF	High PRF improves image quality, but limits the penetration.	Choose the view with the least depth that will adequately visualize the structure.
High vs. low transducer output	High transducer output improves signal-to-noise ratio, but may have more bioeffects.	Transducer output may be controlled by the operator in some systems.
Line frequency in the 2D image	More lines improve spatial resolution, but worsen temporal resolution.	Choose views with the narrowest sector that will adequately visualize the structure.
Multifocusing	Improves spatial resolution, but worsens temporal resolution.	Operator preference in system and transducer choice.
Image quality vs. Doppler	Best image at 90°, best Doppler at 0° incident angle.	Choose the view that will give an adequate information.
CW vs. pulsed or colored Doppler	CW measures high-flow velocity; pulsed is subject to aliasing, but provides sample location.	Choose both, when necessary to obtain complementary information.

CW indicates continuous wave; PRF, pulse-repetition frequency.

TABLE 3.6. Commonly used ultrasound terminology

Term	*Definition*
Static Characteristics	
Anechoic	Also called "echo-free," and refers to the parts of the image that produce no returning signal. These occur below the boundary with high acoustic impedance (shadowing) or in liquid-filled structures, i.e., cysts.
Hypoechoic	A portion of the image may produce fewer returning echoes than the surrounding tissues, and appears less bright than the other parts of the image. An area of necrosis is a prime example.
Isoechoic	Refers to tissues with the same brightness and presumably produces similar echo return.
Hyperechoic	Portions of the image that appears either brighter than the surrounding tissues or brighter than expected. For example, the mitral valve as compared to the rest of the heart or carotid artery calcifications as compared to the normal artery.
Echohomogeneous	Any structure can be homo- or heterogeneous, depending on whether it has similar or different echo characteristics throughout.
Dynamic (Movement) Characteristics	
Akinetic	A structure or a part of an organ that should be moving, but does not (e.g., inferior wall of left ventricle in inferior myocardial infarction).
Hypokinetic	A structure that is moving less than expected (same example as for akinetic).
Diskinetic	A structure that is moving in the direction opposite to what is expected. Also known as paradoxical motion (e.g., the intraventricular septum in massive pulmonary embolism or the acute phase of left ventricular aneurysm).
Hyperkinetic	A structure that is moving too much compared with what is expected (i.e., left ventricle in patient with early hypovolemic shock).
Doppler Characteristics	
Laminar phasic	Characteristic of flow of reflectors and their position in the structure of travel. Normal venous flow.
Laminar pulsatile	Characteristic of flow of reflectors and their position in the structure of travel. Normal arterial flow.
Turbulent flow	Flow velocity depends on the cardiac structure or the caliber of the vessel, but normally will seldom exceed 2 m/s. Always abnormal, and sometimes described as mosaic in colored Doppler.

TABLE 3.7. Common artifacts and their clinical significance

Artifact	*Clinical significance*
Acoustic shadowing	When ultrasound reaches an object with very high acoustic impedance (attenuation), it will not be able to penetrate it any further. This will create acoustic shadowing, which is a linear anechoic or hypoechoic area covering deeper structures so that they cannot be visualized or displayed. Acoustic shadowing is used to diagnose high-attenuation objects such as gallstones or heavy calcified vessel walls. Another cause for acoustic shadowing is the refraction at the edge of a circular structure. An analogy is the circular appearance of the sun on a sunny day shining in your eyes. You can see very little the closer you get to it. This phenomenon is called shadowing by refraction (edge shadowing), and on the ultrasound image it will produce a hypoechoic line parallel to the sound beam. No anatomical structures will be visible in that "shadow." Anatomical edges of round organs such as the heart, kidneys, testicles, or a baby's head will be prone to produce such artifacts (Figure 3.20).
Reverberations and ring-down (comet-tail) artifacts	If the ultrasound reaches two reflectors, it might reflect multiple times like a candle standing between two mirrors. The resulting image will have a "Venetian blind" appearance, with equally spaced multiple lines perpendicular to the direction of the ultrasound beam's propagation. If the distance between the parallel lines diminishes, they may become confluent. Those merged reverberation artifacts are known as the "comet-tail" sign (Figure 3.21). These are solid hyperechoic lines that appear to visualize the ultrasound beam itself. Reverberations are common in echocardiography in the apical 4-chamber view where the ultrasound beam is "bouncing" between layers of pericardium or between pericardium and epicardium, which are both high-impedance boundaries. In chest ultrasound, reverberation artifacts become clinically important. They are produced by the signal trapped between parietal and visceral pleura, which are both strong reflectors. The presence of reverberation artifacts implies that both pleural layers are in close proximity and virtually rules out pneumothorax. Normally, lung tissue is not visualized by contemporary ultrasound systems because the sound propagation speed in the lungs is way below the expected 1540 m/s (propagation speed error). But as the lung tissue becomes more dense from fluid accumulation (pulmonary edema) or inflammatory changes (pneumonia, ARDS) the speed of sound increases. This improves transmission of the ultrasound back to the transducer and converts distinct reverberation artifact lines into comet-tail artifacts. The number of comet-tail lines is thought by some to correlate with the degree of pulmonary edema or inflammatory changes and may become a useful diagnostic and prognostic tool in critically ill patients.
Enhancement	If the sound passes through an area of lower attenuation, structures located beneath appear hyperechoic. This hyperechoic band parallel to the ultrasound beam is known as an acoustic enhancement and will often be used to differentiate cysts (lower-attenuation structures) from cystic tumors and abscesses that have higher attenuation (Figure 3.22). The other type of enhancement, known as banding, occurs in the focal area of the single-focus transducers. It is a hyperechoic stripe perpendicular to the direction of the ultrasound beam. Banding became increasingly rare with modern transducers used in the portable ICU ultrasound machines.
Mirror image	The area of very high acoustic impedance may serve as an acoustic mirror deflecting the ultrasound beam to the side. The ultrasound system assumes that sound travels in a straight line, and thus is unable to recognize redirected beams as such. It will always place the image created by the deflected beam (mirror artifact) deeper than the correct anatomical position of the true reflector. This is because the redirected beam will take longer to reach the transducer (Figure 3.23). A high reflective boundary will be located between the anatomical reflector and the artifact.
Propagation speed errors	The ultrasound system assumes that the speed of sound in soft tissues is 1540 m/s. So if the actual propagation speed is higher, the reflector will be placed shallower, and if it is slower, deeper than the actual anatomical position. If the propagation speed differs significantly from the one assumed (i.e., silicone gel prosthesis or lung tissue), the position will vary significantly as well.

(*continued*)

TABLE 3.7. (*Continued*)

Artifact	*Clinical significance*
Refraction artifacts	If the sound strikes the boundary obliquely or if the propagation speed in two adjacent mediums differs, propagation with the bend or refraction will take place. The difference in propagation speed will compensate for the increased distance, and the reflection of the refracted beam will reach the transducer nearly simultaneously with the echo of the pulse that stuck the reflector with normal incidence. Therefore, the refraction artifact will be placed by the ultrasound system side by side with the true anatomical reflector (Figure 3.24). Because of this, one cannot say which one of the images is the reflector and which is an artifact.
Lobes	Lobes are caused by the parts of the ultrasound beam propagating in the direction other than the beam's main axis (violation of the Assumption 5 above) Because very few modern ICU ultrasound systems use mechanical or single PZT crystal transducers, side lobes specific to this kind of transducers will not be discussed further here. Commonly used array transducers produce the so-called grating lobes. Grating lobes are second copies of the reflector placed side by side with the reflector itself. They occur if the ultrasound beam is wider than the reflector itself. Use of tissue harmonic imaging greatly reduces the occurrence of this artifact.
Doppler artifacts	Two common Doppler artifacts are ghosting and cross talk. Ghosting is a Doppler shift produced by moving anatomical structures (i.e., pulsatile vessel wall), rather than the blood flow. Ghosting can be eliminated by rejecting low-level Doppler shifts with wall filters. Alternatively, ghosting can be helpful in identifying if the reflective boundary is moving, which in the case of pleural layers will rule out pneumothorax. Cross talk is a mirror image artifact as applied to the Doppler phenomenon. It can be caused by a high receiver gain or the Doppler incident angle of near 90°. It is seen as the identical flow pattern appearing above and below baseline and can be remedied by decreasing receiver gain or changing the incident angle. Strictly speaking, aliasing is another Doppler artifact but it was fully discussed previously and will not be discussed here.

ARDS indicates acute respiratory distress syndrome; ICU, intensive care unit; PZT, lead zirconate titanate.

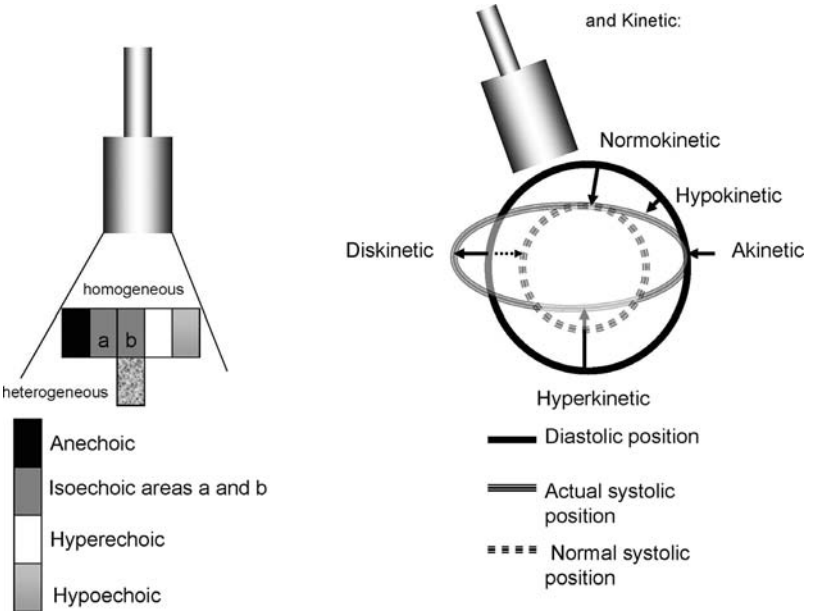
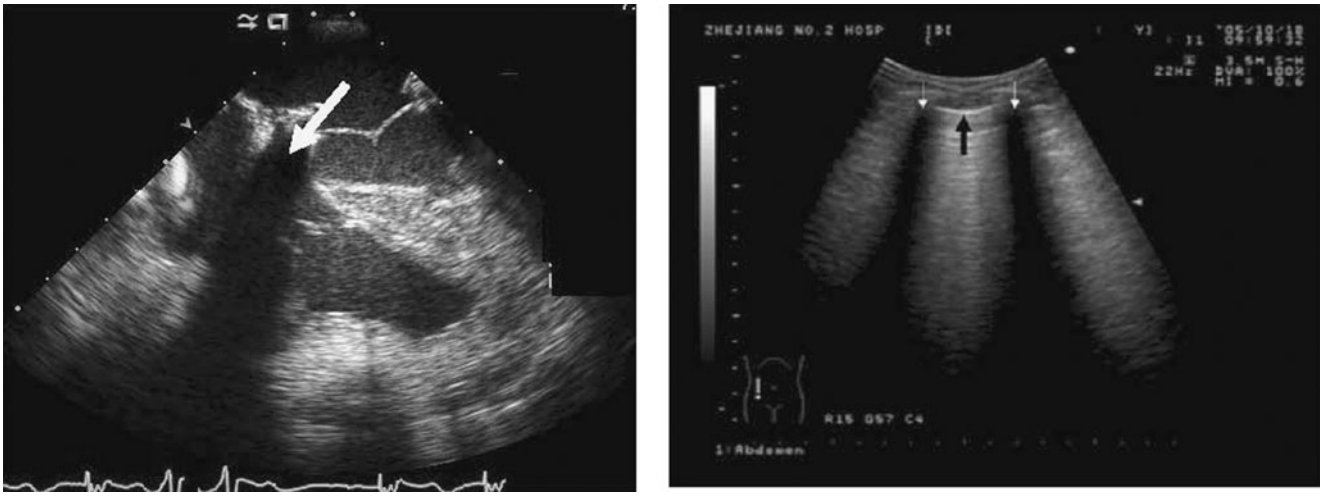


Figure 3.19. Image characteristics static (left) and kinetic (right).

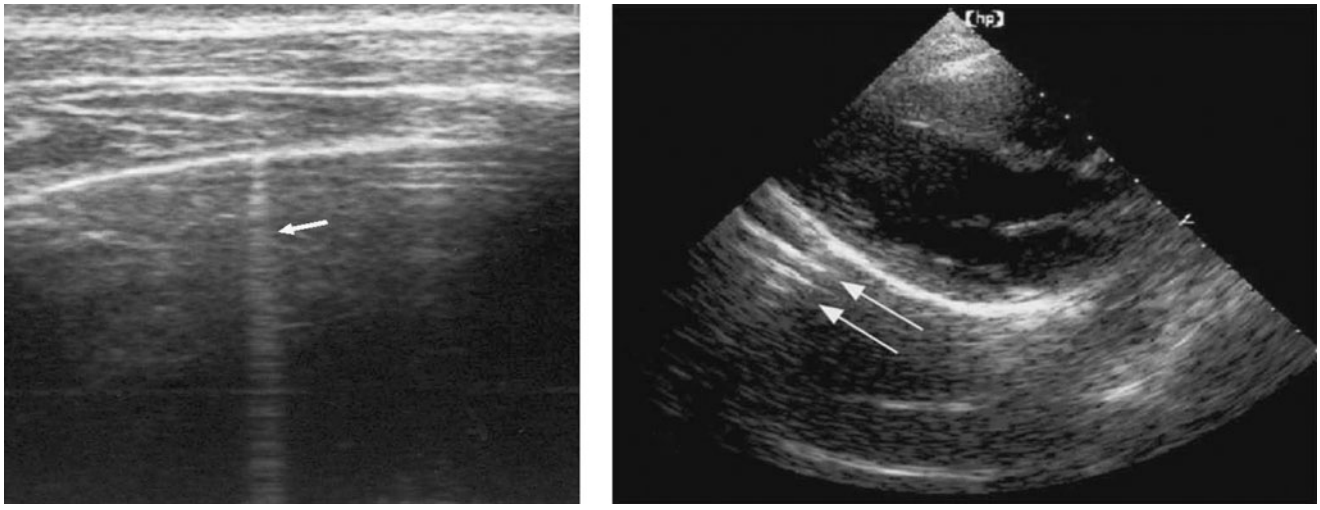


A B

Figure 3.20. Two examples of acoustic shadowing. Part A is a transesophageal echocardiogram where shadowing of the severe calcification is preventing visualization of deeper structures (white arrow). In Part B rib calcifications are utilized to identify the pleural line (black arrow) formed by parietal and visceral pleura. Presence of motion (shimmering) in that location helps to rule out pneumothorax during ultrasound evaluation of the lung.

the computer chips in some portable ultrasound systems. Just as compression rejection increases the contrast, it narrows the dynamic range and results in some loss of the detail (Figure 3.17).

In modern ultrasound systems, processed signals are usually sent to a digital converter to be displayed in a digital format on the screen. Here again, computer chips are employed to improve the image. Digital images have the advantage of being virtually permanent, easy to disseminate, and allow, to some degree, the return to the original image format to do more analysis should new questions be raised. Digital archiving (a picture archiving and communication system, or PACS) can be entered easily at any time, after the images have been stored. The quality of the digital image is proportionate to the number of pixels available



A B

Figure 3.21. (A) Reverberation artifact on the lung ultrasound. "Comet-tail" sign helps to rule out pneumothorax (white arrow). Multiple similar artifacts may indicate an increase in lung stiffness due to congestion or inflammatory changes. (B) Classic reverberation artifacts (echocardiogram) are represented by the "Venetian blind" pattern produced by the pericardium and just causes the deterioration of image quality.

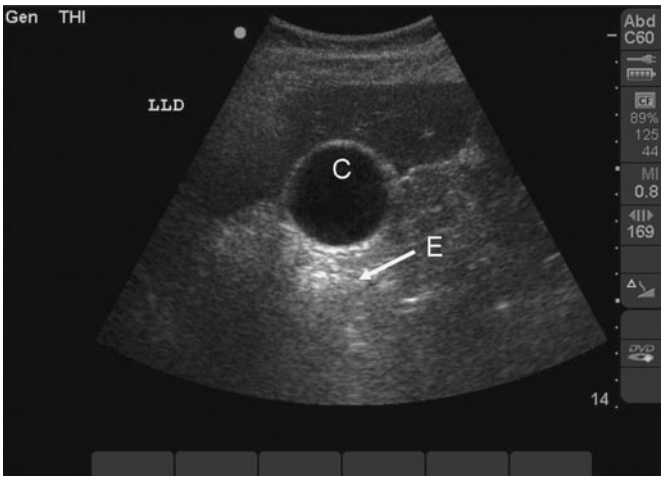


Figure 3.22. This image of a low-attenuation lesion (anechoic cyst C) shows a hyperechoic band parallel to the ultrasound beam, known as an acoustic enhancement (E), and will often be used to differentiate cysts (lower-attenuation structures) from tumors (high-attenuation structures) that will not produce this artifact.

for the recording. At the present time, digital formats do not limit image quality. Throughout the ultrasound system the dynamic range is reduced from >100 dB in the transducer to 10–20 dB in the recorded images.

As one can see, the image generated by the ultrasound system is the result of complex processing. This makes this imaging modality the least intuitive of all, with the potential exception of nuclear medicine imaging. What complicates it even further is the fact that ultrasound imaging is based on a series of assumptions and the formation of the image requires multiple compromises (Tables 3.4 and 3.5). When the assumptions are faulty and compromises need to be made, image quality suffers and artifact production results. This by itself is neither bad nor good; in fact, some artifacts are an important part of the ultrasound diagnosis. However, it is important for the operator to be able to recognize which part of the image is real and which is not. The goal is not to achieve an "ideal image," reflecting anatomical reality, but a "best possible image" that differs from the reality to a greater or lesser degree.

ARTIFACTS

Terminology

Prior to a discussion of different ultrasound artifacts, some important and common terms to describe ultrasound images, both static and moving, should be introduced. This terminology will enable the operator to describe the image, provide the information for medical records and to other health professionals, and better understand radiology and cardiology reports related to the ultrasound images (Table 3.6, Figure 3.19).

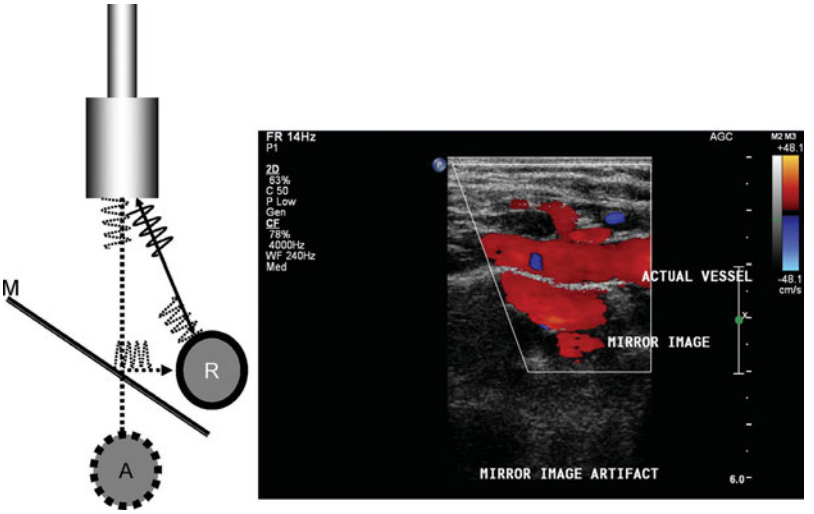


Figure 3.23. An ultrasound beam reaches anatomical reflector R by two routes, directly and after being reflected from a high-impedance boundary (mirror M). Because the reflected pulse takes longer to reach the transducer, the mirror artifact image A is placed deeper than the real reflector. Colored Doppler is also a subject to mirror image artifact as in this case of the carotid artery image.

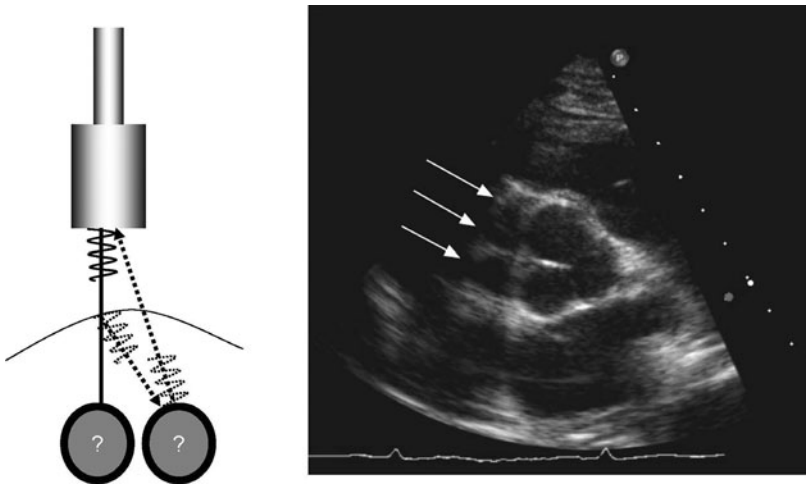


Figure 3.24. Aortic duplication due to the refraction artifact (white arrows). Due to incomplete nature of duplication, the position of the artifact vs. anatomical position of the true reflector can be identified. This is not always the case. (Image courtesy of D. Adams, RDCS.)

Artifacts and Image Alterations

The combination of complex processing, physical limitations, and partially valid assumptions is capable of producing artifacts. Artifacts result from a discrepancy between the image interpretation and reality and include imaging errors, operator errors, and interpreter errors. Interpreter errors can be avoided by a solid knowledge of the ultrasound physics, image formation, ultrasound system, and human anatomy. With the exception of operator error, most common artifacts are caused by the discrepancy between the true physics of ultrasound and assumptions about image formation. Violations to the assumptions of image formation are known as acoustic artifacts. Most acoustic artifacts are seen on a single view and cannot be confirmed on subsequent views of the same anatomical structures. Others will disappear when corrective measures are taken by the operator. Persistent artifacts in multiple views might also signify system malfunctions and require a call to the manufacturer or service engineer. The most common ultrasound artifacts and their clinical significance are presented in Table 3.7.

Suggested Reading

Edelman SK. *Understanding Ultrasound Physics.* 3rd ed. Spring Texas: Esp Inc; 2004.

Hedrick WR, Hykes DL, Starchman DE. *Ultrasound Physics and Instrumentation.* 4th ed. St Louis, Mo. Elsevier Mosby; 2005.

Kremkau FW. *Diagnostic Ultrasound: Principles and Instruments.* 7th ed. St Louis, Mo. Saunders Elsevier; 2006.

Miele FR. *Ultrasound Physics and Instrumentation*. 4th ed. Forney, Tex. Miele Enterpises; 2006.

Owen C, Zagzebski J. *Ultrasound Physics Review: A Q&A Review for the ARDMS Ultrasound Physics Exam.* Pasadena, Calif: Davies Publishing Inc; 2008.

This page intentionally left blank

CHAPTER 4

Training of the Critical Care Physician as Sonographer

Alexander Levitov, Paul H. Mayo, and Anthony D. Slonim

INTRODUCTION

This chapter presents some important issues related to training and competence in critical care ultrasonography. The field is likely to undergo rapid growth in the next few years. Intensivists should be aware of some of the challenges they will face in achieving competence in bedside ultrasonography and the potential solutions to those challenges. This chapter will present a discussion of some of the issues related to training in critical care ultrasonography and will describe a more concrete and prescriptive approach to the issue of training, developed and implemented for our own trainees, which serves as a model for ensuring that the knowledge and skills can be applied to the care of the patient. Table 4.1 provides a list of common terms that are important to the discussion of training.

TRAINING: A VIEW FROM AROUND THE WORLD

United States

Critical care ultrasonography is a relatively new application of a well-established technology. In the United States, there is not yet a means for the intensivist to become formally certified in the field, and the relevant professional societies have not yet developed a clear definition of what constitutes competence. It is instructive to examine some examples of what other specialties have done to resolve these issues both in the United States and abroad.

In the United States, ultrasonography is a well-established part of emergency medicine. It is a required part of emergency medicine residency training, and the emergency medicine professional societies have established the minimum requirements for ultrasound during residency in terms of the number and types of scans required for the completion of training. Completion of residency training in emergency medicine, by definition, indicates that the physician is competent in emergency medicine ultrasonography. No further special indication of competence is required. For the attending-level clinician who completed training before ultrasonography was incorporated into emergency medicine training, the professional societies have developed specific training objectives to allow the clinician to become competent.

While there are no specific requirements for training in ultrasound for the general internal medicine training programs, in internal medicine subspecialty training programs, proficiency in the use of ultrasound to guide the placement for central venous catheters and thoracentesis is highly recommended, but not required, for critical care and pulmonary medicine subspecialty training. No threshold numbers of performance are required. For cardiology training, echocardiography is a core procedural skill that is recommended by the Board.

In pediatrics, the American Board of Pediatrics has included knowledge of the indications for diagnostic ultrasound in the content requirements for the subspecialty examinations in critical care medicine, but there is no formal requirement for the procedural aspects of ultrasound use in critical care. The subboard of pediatric emergency medicine provides specific recommendations for knowledge and performance of ultrasound in ectopic pregnancy, but does not address other areas specifically. For pediatric cardiology, there are specific knowledge and performance requirements for transthoracic and transesophageal echocardiography.

Residency training in obstetrics and gynecology in the United States has included significant experience in the use and application of ultrasound at the bedside. All residents graduating since the early 1980s are deemed competent by virtue of their training in residency. Prior training requires a demonstration of applicable

TABLE 4.1. Common terms important in discussing training

Competence	• *Competence* refers to the implicit, internalized knowledge of critical care ultrasonography that enables intensivists to use the technique for the clinical benefit of their patient; this requires the skill, knowledge, and capacity to be proficient in the field. • At the most basic level, any definition of competence is based on a standard developed by an authoritative group. • In the United States, major national professional societies have traditionally been involved with the definition of competence. In addition, the American Board of Medical Specialties and the Accreditation Commission for Graduate Medical Education have been responsible for defining competence that is relevant to postgraduate medical education including critical care fellowship training.
Certification	• *Certification* is defined as the process by which competence is formally recognized by an external agency. • In the United States, there is presently no accepted process for certification in critical care ultrasonography. A formal definition of competence is clearly a necessary prerequisite to developing criteria, standards, and a training process leading to certification.
Credentialing	• *Credentialing* is defined as the method whereby an individual hospital or other specific entity grants a clinician permission to practice based on an assessment of legitimacy of training, knowledge, and skills. • Credentialing is a local, hospital-based issue that is related to competence and certification insofar as the hospital may require competence and certification (if available) before granting credentials (or privileges) to perform critical care ultrasonography.
Training	• *Training* refers to the process by which the intensivist develops the knowledge, skills, and competence in critical care ultrasonography. • Training of the intensivist encompasses hands-on image acquisition, immediate bedside image interpretation, and the cognitive ability to apply the results at the bedside of the critically ill patient.

knowledge and skills to be proficient in the use of the equipment, the acquisition of images, the interpretation of images, and application within the clinical context at the bedside.

The American College of Surgeons has provided a voluntary and tiered mechanism for surgeons that provides criteria to ensure appropriate knowledge, skills, and competence in the performance of specific ultrasound examinations. The College also provides recommendations for methods to ensure competence on an ongoing basis.

International

In Japan, ultrasonography is fully incorporated into all levels of residency training. For example, internal medicine residents perform hundreds of scans during their training, and Japanese intensivists become routinely skilled in many aspects of ultrasonography. The Japanese approach to ultrasonography is very different from that provided in the United States, given that it is a ubiquitous part of training and is regarded as a routine part of medical practice.

In Europe, Germany has been a leader in incorporating ultrasonography into internal medicine training at all levels. Medical residency training has specific requirements that mandate training in ultrasonography. For example, the typical medical residency training program requires that 400 abdominal ultrasound examinations be performed by a trainee before graduation. This approach to ultrasonography as a routine part of internal medicine training applies in the intensive care unit (ICU), where bedside ultrasonography is considered to be a part of the everyday practice of critical care medicine.

In France, the intensivist community has developed specific training requirements for advanced-level critical care echocardiography. Critical care fellows train for one year in a parallel track with cardiology fellows and then spend a second year training in a centrally accredited critical care echocardiography fellowship program. They are required to perform a defined minimum

number of transthoracic and transesophageal echocardiograms, and must pass an examination.

HOW TO TRAIN INTENSIVISTS IN CRITICAL CARE ULTRASOUND

Define Competence

The first step in developing the process of training and certification in critical care ultrasonography is to clearly define what constitutes competence in the field. The definition of competence should be objectively measurable so that the trainee, the trainer, and independent evaluators can recognize the knowledge, skills, and aptitudes that define it (Table 4.1). Defining competence also allows a "training framework" to be established so that competence can be achieved. An important principle for establishing a definition of competence is to specify that it is a minimum standard. The field of ultrasonography is extant. Hence, it becomes important for the critical care physician to have the relevant bodies of knowledge specific to their practice. Unfortunately, the rule book that defines these standards has not yet been written. However, professional societies in the United States and Europe are in the process of cooperatively developing such a document. While competence is usually used to specify a minimal standard, intensivists who wish to develop expertise in ultrasonography should not be dissuaded from advancing their knowledge and skills.

Advanced Competence

While some authorities believe that competence is a yes or no proposition, where the bedside clinician is either competent to perform critical care ultrasonography or is not, others believe that there may be different levels of competence in the field. In this regard, a tiered program, akin to the American College of Surgeons' verification program, is useful particularly for helping physicians who desire achieving competence at higher levels. Neri and colleagues described a similar system of graded competence for critical care and the cardiology community uses a three-level approach to rank competence in echocardiography. A three-level system highlighting this approach is described in detail later in this chapter. The different levels of designated competence may have implications in terms of teaching, administrative, or academic advancement, but may also unnecessarily complicate the determination of competence.

The Relationship between Competence and Training

Competence requires training. One approach to defining training requirements is to develop guidelines that establish the duration of training and the specific course work requirements, and are specific in establishing threshold numbers and types of ultrasound studies that must be performed by the trainee. The problem with this approach is that the training effects cannot be determined. A trainee who performs a perfunctory five-minute long, poorly supervised cardiac examination receives training credit but with little training effect. When compared with the training effect of a one-hour comprehensive cardiac examination carefully supervised by an experienced echocardiographer, the outcomes are quite different. Quantification of the numbers of contact hours and completed examinations may not equate with competence. An alternative approach to training is to deemphasize the numerical assessment of training and to focus on determination of actual competence through formal assessment of the knowledge and skills needed to show competence. This outcome assessment can be done with a knowledge-based written component and a proctored hands-on examination to demonstrate the skills of image acquisition and interpretation. The goal of this process would be certification, but this is presently not available in the United States.

Training during Fellowship

Training in critical care ultrasonography is best accomplished during subspecialty fellowship training. Based on our experience, training can be easily incorporated into the fellowship curriculum. For those trainees who seek training in advanced critical care echocardiography, the French model where the critical care fellow may rotate with cardiology fellows for comprehensive training in general echocardiography followed by focused training in advanced critical care applications.

In the United States, the major barrier to fellowship-level training remains the lack of qualified attending faculty and dedicated ultrasound machines can be suggested. To be successful, a basic requirement for incorporating ultrasound training into critical care fellowship would be 24-hour availability of a fully capable ultrasound machine that can be used as needed by the clinical faculty. As the American Board of Internal Medicine (ABIM) places more stringent requirements on critical care and pulmonary fellowship training,

including specific training in ultrasound guidance of vascular and pleural access among other procedures, it is likely that the need for additional equipment and faculty will increase.

Training after Fellowship

For the attending-level clinician who did not receive training in critical care ultrasonography during fellowship, training can be a challenging proposition. Time constraints and economic pressures may make it difficult to access training. The absence of an organized process for certification results in credentialing processes that are defined on a local or regional rather than national level. Fortunately, for the intensivist with a casual interest in ultrasound use in the ICU, the knowledge base of the field is well defined. It may be found in textbooks and review articles and through widely available courses. Competence, however, requires mastery of the cognitive and practical components of the field. Therefore, several general suggestions may be helpful for attending physicians who have more than a casual interest and are seeking training.

Critical care ultrasonography lends itself to division into several distinct and stand-alone modules, including, for example, basic and advanced echocardiography, and vascular access, diagnostic, pleural, lung, and abdominal ultrasound. While it may desirable for the bedside intensivist to be competent in all, it may not be necessary, because all modules may not be equally relevant to the requirements of their practice. One approach is for the interested intensivist to undertake training in those aspects of critical care ultrasonography that are most relevant to their practice. For example, mastery of ultrasound-guided vascular access and pleural ultrasonography are very straightforward and have immediate application for the intensivist. Once proficient in these modules, the clinician may decide to proceed with more comprehensive training that fits their practice needs and time allotment.

As a next step, proficiency in lung, abdominal, vascular diagnostic ultrasonography, and basic critical care echocardiography can be addressed. Because advanced critical care ultrasonography requires a considerable time commitment, the decision to pursue training in this module should be carefully considered. If so interested, the intensivist should be prepared to fulfill the requirements for Level 2 echocardiography, as defined by the cardiology community, and may wish to demonstrate their mastery of the content by taking the echocardiography board examination.

Training in Image Acquisition

An important element of proficiency in ICU ultrasonography is image acquisition. Much of this experience is obtained as the critical care ultrasonographer personally performs studies at the bedside of the patient and uses the results to guide the management of the critically ill patient. The only way to become proficient at image acquisition is through repetitive practice. While a course may offer hands-on training, it is often insufficient to achieve competence. Having a skilled physician–sonographer providing supervision during hands-on training for image acquisition is helpful but rarely available. Ultrasound technicians are an excellent resource for assisting the physician interested in training in image acquisition.

Training in Image Interpretation

Competence in image interpretation requires the review of a large number of normal and abnormal images. To some extent, this requirement is met during training in image acquisition. The best means of gaining experience with image interpretation is to read images with an experienced ultrasonographer. This may be easy for the fellow in training who has access to faculty, but may present problems for the attending-level clinician practicing in the community.

Because static images are often insufficient for developing competence, dynamic real-time studies are now being incorporated into textbooks, onto DVD-based training materials, and in courses.

Documentation of Training

It is important to document all ultrasound training activity. This includes a record of all hands-on scanning activity, including a procedure logbook or image-documentation log, image-interpretation time, and course work. This information may be useful when a formal certification process is developed. Further, it may be necessary for documentation of competence and expanded privileging at the local level.

AN EXAMPLE OF A PROGRAM FOR TRAINING

Included here is one example of an approach for training that we have used successfully to train physicians interested in developing the necessary competence to perform critical care ultrasonography at the bedside. Training and achievement of higher competency levels are delineated from 1 to 3, with increasing competence

represented by a higher number. It provides a tiered template, which represents a starting point for other institutions and perhaps even for the establishment of standards and competency validation. Although developed for adult medical ICU patients, it should be adaptable for use in other populations such as critically ill children.

Basic Knowledge

Because ultrasound image generation follows the basic laws of acoustics, knowledge of the laws of physics is an essential requirement for competence in all modalities of ultrasonography. Moreover, the choice of transducer for a particular examination is important and must be informed by an understanding of the relationships between the physical properties of the ultrasound wave and image formation. Similarly, the understanding of Doppler phenomenon is required for cardiac and vascular sonography. The trainee must understand the inherent limitations of portable devices including the reductions in image quality and have a working knowledge of the anatomy and physiology of the heart, vessels, chest, abdomen, and extremities to guide image interpretation so that a plan of action can be developed at the point of care.

Technical Aspects of Image Acquisition

In point-of-care ultrasonography, the device operator and image interpreter are often the intensivist, which puts a set of unique technical demands on the physician performing the study. These demands include the knowledge of transducer selection, transducer manipulation, and adjusting the equipment settings. The operator must appreciate the influence of the gain, filters, and depth and the use of tissue harmonics on image optimization. When a physician–sonographer obtains images, the physician must be also competent enough to provide oversight, quality control, and education.

Knowledge, Training, and Skills for Physician–Sonographers

With this approach, training and competence in different ultrasound techniques can be advanced simultaneously. This is possible because the core body of knowledge related to one technique is often represented as a foundational element in the other techniques. Each of the techniques will be described briefly here. The specific knowledge, training, and skill requirements for each technique and level of training within the technique are presented in Table 4.2.

Focused Assessment of Transthoracic Echocardiogram (TTE)

Focused assessment of TTE allows the clinician to rapidly and more safely acquire clinical information than with invasive hemodynamic monitoring. The general indications for this focused assessment include:

- Hemodynamic instability due to ventricular failure
- Hypovolemia
- Pulmonary embolism
- Acute valvular disease
- Acute ventricular septal defect (VSD)
- Ventricular wall perforation
- Cardiac tamponade

In addition, ultrasonography may inform the clinician about the reasons for oliguria, persistent hypoxemia, sepsis, or failure to wean from mechanical ventilation.

Although the training requirements for echocardiography have been suggested by the American Society of Echocardiography (ASE), to include three months of training, the performance of 75 examinations, and the interpretation of 150 examinations, we believe that these recommendations are conservative and that a balanced training program can place skilled physician–ultrasonographers at the bedside making clinical decisions that benefit patients without undue risk (Table 4.2).

Transesophageal Echocardiography (TEE)

The guidelines jointly established by ASE and the Society of Cardiovascular Anesthesiology are well established and applicable to Critical Care TEE. Critical care practitioners should be allowed to participate in necessary TEE training and be held to the same standards as anesthesiologists for these procedures. In fact, the TEE guidelines may provide an effective roadmap for the development of critical care TTE certification.

Vascular Ultrasound and Ultrasound-Guided Central Venous and Arterial Catheterizations

Nearly all larger vessels can be imaged with ultrasound. Ultrasound guidance reduces complications and is increasingly being considered the standard of care for jugular vein and subclavian vein cannulation. The verification of catheter position permits rapid and safe use of the catheter prior to radiologic confirmation.

Other uses of ultrasound include the assessment of vein patency and a diagnostic evaluation for pulmonary

TABLE 4.2. Knowledge, training, and skill requirements for different categories of ultrasound

Category	*Level*	*Knowledge*	*Training*	*Skills*
Focused assessment of TTE				
	1	Basic knowledge of ultrasound physics	For physicians already in practice, 32 hours of formal ultrasound education	Ability to independently choose proper transducer and ultrasound system settings to perform an adequate bedside echocardiographic examination
		Indications for vascular sonography	For physicians in training, one month rotation in point-of-care ultrasound	
		Basic knowledge of appropriate transducer choices and manipulations	Perform and interpret 25 supervised and 25 partially supervised bedside echocardiograms. All echocardiograms are subject to review by supervising physician. (Supervising physician should meet level 3 definition for point-of-care sonography or echocardiography as described in ACC/AHA clinical competence statement) (3)	Ability to independently obtain adequate 2-dimentional echocardiographic images in subcostal, apical, and parasternal views
		Basic spatial orientation and blood flow patterns (velocity) in major vessels	Progression to level 2 within one year's time	Competency to distinguish between normal and abnormal left and right ventricular dimensions and function
		Basic ability to distinguish adequate and inadequate images		Ability to recognize presence of significant pericardial effusion and distinguishing it from pleural effusion
		Ability to distinguish between the artery and the vein utilizing knowledge of anatomical position		Recognize gross abnormality in valvular function
		Ability to assess catheter position in the vessel by the ultrasound		Ability to incorporate knowledge obtained from bedside echocardiography into the care of critically ill patients
		Ability to communicate results to others and provide documentation for medical records		Letter from supervising physician verifying level 1 competency
	2	All requirements for level 1	For practicing physician. Additional 32 hours of formal ultrasound education	Ability to independently choose proper transducer and ultrasound system settings to perform an adequate bedside echocardiographic examination, including two-dimensional and M-mode study as well as colored flow, pulsed Doppler, and CW for all valvular structures, left ventricular outflow tract, and aorta

		Detailed knowledge of ultrasound physics	For physician in training, one additional month rotation in the point-of-care ultrasound	Competency to distinguish between normal and abnormal left and right ventricular dimensions and function
		Detailed knowledge of appropriate transducer manipulations	Perform and interpret an additional 25 supervised echocardiograms and an additional 25 unsupervised studies (subject to independent random review by supervising physician). (Supervising physician should meet level 3 definition for point-of-care sonography or echocardiography as described in ACC/AHA clinical competence statement)	Numerically assess left ventricular ejection fraction and right ventricular systolic pressure
		Advanced ability to distinguish adequate and inadequate images	Progress to level 3 within two years' time	Ability to recognize presence of significant pericardial effusion and signs of pericardial tamponade
		Knowledge of Doppler echocardiographic principles (colored, pulsed, and continuous-wave)		Recognize abnormality in valvular function and assess severity of valvular dysfunction utilizing Doppler modalities
		Ability to assess adequacy of CW, pulse Doppler, and colored Doppler images		Ability to incorporate knowledge obtained from bedside echocardiography into the care of critically ill patients
		Ability to relate Doppler studies to the direction and velocity of blood flow		Letter from supervising physician verifying level 2 competency
		Ability to perform ultrasound guided Pericardiocentesis in the simulated environment		
	3	Detailed knowledge of concepts described in levels 1 and 2	16 hours of ultrasound-related CME a year	Serve as a mentor to levels 1 and 2 operators
		Additional 16 hours of ultrasound education a year	Yearly performance and interpretation of at least 100 FATE echocardiograms	Assume responsibility for quality control and participate in ultrasound education
			Active participation in echocardiographic case reviews with other level 3 operators (including AHA/ACC level 3)	Reaccreditation every 3 years for all level 3 operators is highly advisable
Vascular Ultrasound				
	1	Basic knowledge of ultrasound physics	For physicians already in practice, 32 hours of formal ultrasound education	Ability to independently choose proper transducer and ultrasound system settings to perform an adequate bedside two-dimensional vascular examination

(*continued*)

TABLE 4-2. (*Continued*)

Category	*Level*	*Knowledge*	*Training*	*Skills*
		Indications for vascular sonography	For physicians in training, one month rotation in the point of care ultrasound	
		Basic knowledge of appropriate transducer choices and manipulations	Perform and interpret 25 supervised vascular ultrasounds and perform 20 large vessel cannulations, with first 5 in simulated environment and at least 15 venous	Ability to independently obtain adequate 2-dimentional venous images of lower (above the knee) and upper extremities
		Basic spatial orientation and blood flow patterns (velocity) in major vessels	Progress to level 2 within one year's time	Competency to distinguish between normal and abnormal venous images and identify common vascular abnormality (clot)
		Basic ability to distinguish adequate and inadequate images		Ability to incorporate knowledge obtained from bedside vascular study into the care of critically ill patients
		Ability to distinguish between the artery and the vein utilizing knowledge of anatomical position		Letter from supervising physician verifying level 1 competency
		Ability to assess catheter position in the vessel by the ultrasound		
		Ability to communicate results to others and provide documentation for medical records		
	2	All requirements for level 1	For physicians already in practice, an additional 32 hours of formal ultrasound education	Ability to independently choose proper transducer and ultrasound system settings to perform an adequate bedside two-dimentional and Doppler vascular examination
		Detailed knowledge of ultrasound physics, including Doppler (CW, pulsed, and colored)	For physician in training, one additional month rotation in the point-of-care ultrasound	
		Detailed knowledge of appropriate transducer choice and manipulations	Perform and interpret an additional 25 supervised and 25 unsupervised vascular ultrasounds including CW, pulsed Doppler, and colored-flow Doppler studies (10 arterial)	Ability to independently obtain adequate 2-dimentional venous images and Doppler flow velocity measurements of lower (above the knee) and upper extremities
		Advanced ability to distinguish adequate and inadequate images	Perform an additional 20 supervised and 10 unsupervised large vessel cannulations (at least 10 of them arterial)	Competency to distinguish between normal and abnormal venous images and identify common structural venous abnormality and abnormal blood flow patterns

	Ability to assess normal vs. abnormal arterial and venous flow pattern	All unsupervised procedures are subject to random review by level 3 mentor/ supervisor	Competency to distinguish between normal and abnormal arterial images and identify common structural arterial abnormality and abnormal flow patterns
	Ability to utilize flow augmentation in diagnosis of venous thrombosis	Procedures resulting in inability to cannulate or any complications are subject to mandatory review by level 3 supervisor	Ability to diagnose arteriovenous fistula arterial aneurism and pseudoaneurism
	Ability to assess catheter position in the vessel by the ultrasound		Ability to incorporate knowledge obtained from bedside vascular study into the care of critically ill patients
	Ability to assess and grade the severity of atherosclerotic occlusive disease in major peripheral arteries		Letter from supervising physician verifying level 1 competency
	Ability to recognize the presence of arteriovenous fistula, and pseudoaneurism		
3	Detailed knowledge of concepts described in level 2	Yearly performance and interpretation of at least 50 vascular ultrasounds, excluding ultrasound-guided arterial and venous cannulations	Serve as a mentor/supervisor to level 1 and 2 operators
	Additional 16 hours of ultrasound education a year	Active participation in vascular laboratory case reviews with other level 3 operators, including physicians certified as vascular ultrasound interpreter by ARDMS	Assume responsibility for quality control and participate in ultrasound education
			Reaccreditation every 3 years for all level 3 operators is highly advisable
Upper airway, chest, abdomen, retroperitoneal space, and small parts ultrasound			
1	Basic knowledge of ultrasound physics	For physician already in practice, 32 hours of formal ultrasound education	
	Indications for general sonography	For physician in training, one month rotation in the point-of-care ultrasound	

(*continued*)

TABLE 4-2. (*Continued*)

Category	*Level*	*Knowledge*	*Training*	*Skills*
		Basic knowledge of appropriate transducer choices and manipulations	Perform and interpret 25 supervised an 25 unsupervised general body ultrasounds and perform 20 supervised ultrasound-guided taps of fluid collections or cavities, no less than 10 of which should be pleural fluid. All studies are subject to independent random review by supervising physician. Supervising physician should meet level 3 definition for point-of-care ultrasonography. The first five invasive procedures will be done in the simulated environment	
		Basic spatial and anatomical orientation	Progression to level 2 within one year	
		Basic ability to distinguish adequate and inadequate images		
		Ability to diagnose presence of pleural effusion and pneumothorax utilizing ultrasound of the chest		
		Ability to diagnose intraabdominal free fluid (ascitis, hemoperitoneum) and abnormal collections		
		Ability to identify common pathology of great vessels (Aorta, IVC, SVC) i.e., aneurisms, dissection IVC obstruction and thrombosis		
		Ability to suspect the presence of hydronephrosis		
		Ability to communicate results to others and provide documentation for medical records		
		Ability to assess catheter position in pleural or peritoneal space or collection by ultrasound guidance		

2	All requirements for level 1	For physician already in practice, an additional 32 hours of formal ultrasound education	All unsupervised procedures are subject to random review by level 3 mentor/supervisor
	Detailed knowledge of ultrasound physics	For physician in training, one additional month rotation in point-of-care ultrasound	Procedures resulting in inability to perform or any complications are subject to mandatory review by level 3 supervisor
	Detailed knowledge of appropriate transducer choice and manipulations	Perform and interpret an additional 25 supervised and 25 unsupervised general body ultrasounds	
	Knowledge of Doppler principles and their application in general ultrasonography	Perform an additional at least 10 supervised and 10 unsupervised ultrasound-guided general procedures, one half of which should be thoracentesis	
	Advance knowledge of spatial and anatomical orientation		
	Advanced ability to distinguish adequate and inadequate images		
3	Detailed knowledge of concepts described in level 2	Yearly performance and interpretation of at least 25 general body ultrasounds	Serve as a mentor/supervisor to levels 1 and 2 operators
	Additional 16 hours of ultrasound education a year	Active participation in the ultrasound department case reviews with other level 3 operators (including radiologists)	Assume responsibility for quality control and ultrasound education

ACC indicates American College of Cardiology; AHA, American Heart Association; ARDMS, American Registry of Diagnostic Medical Sonographers; CME, continuing medical education; CW, continuous wave; FATE, Focused Assessment of the Transthoracic Echocardiogram; IVC, Inferior Vena Cava; SVC, Superior Vena Cava; TTE, transthoracic echocardiogram.

emboli. Major arterial occlusions or other abnormalities that contribute to patient compromise may be discovered and improve the understanding of the patient's condition, thereby helping to inform an effective plan of care. Further, ultrasound can facilitate the placement of arterial catheters for hemodynamic monitoring, particularly in the hypotensive patient. Doppler studies will help to delineate flow pattern, direction, and velocity.

General Ultrasonography of the Upper Airway, Chest, Abdomen, Retroperitoneal Space, and Small Parts Ultrasound

Ultrasound of the neck (larynx, trachea) may discover unsuspected lymphadenopathy or abscess and provide an additional safety margin for bedside tracheotomy. It may also provide information on the success of endotracheal intubation and perhaps yield information to predict the success of extubation. Ultrasound examination of the paranasal sinuses allows physicians to diagnose sinusitis at the bedside and provides guidance for a therapeutic bedside puncture.

In the chest, the ability to assess the pleural space with ultrasonography enables the operator to instantaneously diagnose a pneumothorax or pleural effusion and provides effective guidance for the safe removal of pleural fluid or air for diagnostic and therapeutic reasons. Abnormal conditions of the thoracic aorta may also be diagnosed with ultrasound.

Abdominal ultrasound can detect ascites and provide guidance for safe paracentesis. Intraabdominal collections can be identified and drained, if necessary, thus improving the diagnosis and therapy of sepsis with an intraabdominal source. Liver and gallbladder conditions may yield a diagnosis of the site of infection, providing an opportunity for further investigation and therapy. The retroperitoneal space can also be visualized through the abdomen, allowing the operator to interrogate the kidneys and abdominal aorta, potentially providing an explanation for anuria or confirming the presence, absence, size, and possible dissection of abdominal aortic aneuryms. Ultrasonographic assessment of the urinary bladder may be helpful in anuric patients. Soft-tissue ultrasound can assist with the diagnosis of necrotizing fasciitis by visualizing soft-tissue air or abscess.

Ultrasound of lumbar spine can assist with a spinal tap where anatomical markers are difficult to obtain clinically. Nerve visualization can assist with the infiltration of local anesthesia. Training and credentialing for focused assessment with sonography for trauma (FAST), though related, are defined elsewhere and are outside of the discussion.

The Future of Medical Ultrasound

Given the breadth of the discipline of ultrasonography and its emerging clinical applications, new subdivisions for training are likely to be created, and critical care practitioners will have to tailor their training program to meet the demands of their practice.

Ultrasound-friendly simulation platforms and specially adapted cadavers provide an ideal opportunity for sharpening invasive procedural skills and sparing patients the "see one, do one, teach one" approach.

Important, the critical care professional societies including the American College of Chest Physicians and the Society of Critical Care Medicine along with the American Institute for Ultrasound in Medicine will have to respond to the pressing needs of the critical care community for developing a set of guidelines for the training and credentialing of the physician–sonographer in the ICU, and the American Board of Internal Medicine and other specialty boards will have to evaluate the critieria for certification in this emerging technology.

In the early 1980s, ultrasound spread from the radiology department into the obstetrics and cardiology practices. A decade later, it started to find its way into emergency departments and, more recently, into the ICU.

The relatively low cost of equipment, ready availability, and ease of training and use allows medical ultrasound to become an ideal candidate as a primary diagnostic tool in even the most remote or medically underserved areas, and may replace the stethoscope as a primary assessment tool in the next century, as medical students become trained on this device.

Suggested Reading

Goldstein SR. Accreditation, certification: why all the confusion? *Obstet Gynecol*. 2007;110:1396–1398.

Manasia AR, Nagaraj HM, Kodali RB, Croft LB, Oropello JM et al. Feasibility and potential clinical utility of goal-directed transthoracic echocardiography performed by noncardiologist intensivists using a small hand-carried device (SonoHeart) in critically ill patients. *J Cardiothorac Vasc Anesth*. 2005;19(2):155–159.

Neri L, Storti E, Lichtenstein D. Toward an ultrasound curriculum for critical care medicine. *Crit Care Med*. 2007; 35(suppl):S290–S304.

Pearlman AS, Gardin JM, Martin RP, et al. Guidelines for optimal physician training in echocardiography. Recommendations of the American Society of Echocardiography Committee for Physician Training in Echocardiography. *Am J Cardiol.* 1987;60:158–163.

Pearlman AS, Gardin JM, Martin RP, et al. Guidelines for optimal physician training in transesophageal echocardiography: Recommendations of the American Society of Echocardiography Committee for Physician Training in Echocardiography. *J AM Soc Echo* 1992; 5(2):158–194.

Quinones MA, Douglas PS, Foster E, at al. ACC/AHA clinical competency statement in echocardiography. *J Am Coll Cardiol*. 2003;41:687–708.

Sargsyan AE. *Principles of Clinical Medicine for Space Flight* M. Barratt and S. Pool (Eds.) Springer 2008.

Available at: https://www.abp.org/ABPWebSite/certinfo/subspec/suboutlines/crit.pdf. Accessed October 29, 2008.

Available at: https://www.abp.org/ABPWebSite/certinfo/subspec/suboutlines/card.pdf. Accessed October 29, 2008.

Available at: https://www.abp.org/ABPWebSite/certinfo/subspec/suboutlines/emer.pdf. Accessed October 29, 2008.

Available at: http://www.facs.org/fellows_info/statements/st-31.html. Accessed October 29, 2008.

Available at: http://www.abim.org/certification/policies/imss/pulm.aspx. Accessed October 29, 2008.

Available at: http://www.abim.org/certification/policies/imss/card.aspx. Accessed October 29, 2008.

Available at: http://www.abim.org/certification/policies/imss/ccm.aspx. Accessed October 29, 2008.

This page intentionally left blank

CHAPTER 5

Pediatric Critical Care: Use of Bedside Ultrasonography

William Tsai and Anthony D. Slonim

INTRODUCTION

The pediatric intensive care unit (PICU), like other intensive care units (ICUs), is a dynamic place that provides multidisciplinary care with the integration of numerous medical and surgical subspecialists who come together with a common goal, the care of a critically ill child. Predictably, the diseases span the spectrum of adult ICU care and range from acute illnesses like septic shock and sepsis-related cardiomyopathy to hemorrhagic shock with traumatic visceral rupture. While the problems are similar to those encountered in adult ICUs, three additional layers of complexity are important to understand when caring for a critically ill child, namely age, size, and developmental status, and all of these have relevance for critical care ultrasonography. First, many differential diagnoses encountered in the PICU are age dependent, which is important for the ultrasonographer to remember when performing ultrasound for diagnostic purposes on a child. Second, a child's size may range from <2 to >200 kg, which has important implications for the technical aspects of ultrasound procedures. Finally, children may not be able to cooperate with an examination or procedure as adults are, making the use of ultrasonography, a pain-free and noninvasive tool, an ideal method for extending one's physical examination. Bedside ultrasound is an important and evolving tool for pediatric intensivists and can be used to evaluate many disease processes, assist in procedural interventions, and assess for complications related to those procedures. This chapter aims to provide a practical discussion on the use of bedside ultrasonography in the PICU.

DEVICES

Ultrasound use in the PICU ranges from being an aid for vascular access to being a versatile instrument that is able to perform an acute, comprehensive assessment of the critically ill child at the bedside and monitor response to critical treatment. Common indications for bedside ultrasound in the PICU are listed in Table 5.1.

Similarly, equipment also ranges from simple ultrasound with the use of a linear probe for vascular access to an instrument with multiple probes that can be manipulated and enhanced to provide the best visualization of cardiac, abdominal, vascular, and thoracic structures (see Chapter 3). Pediatric-sized probes are also available. Small, hockey stick–style linear probes have a small footprint and are able to provide excellent images in less accessible areas of the body such as the neck or axillae. Small phased-array probes are also available for focused echocardiography but are infrequently necessary.

In some PICUs, a portable notebook-type ultrasound system is placed on a mobile cart with a curvilinear abdominal probe, a linear high-frequency probe, and a low-frequency cardiac probe. The probes can be manipulated in terms of frequency, depth of ultrasound beam, and use of Doppler technology. Many systems have a very short power-on-to-scan time and require very little manipulation to provide good images. They are lightweight, easily maneuvered, and have a very small footprint. These systems receive regular use in vascular access, thoracic and abdominal ultrasonography, and focused echocardiography.

VASCULAR ACCESS

The use of procedural ultrasound in vascular access is more efficient and safer than techniques using palpation and landmarks. Of importance, the tool is not a substitute for knowing the appropriate landmark-based approaches to central venous catheterization, but it can facilitate the procedure. Verghese and Alderson demonstrated that the routine use of internal jugular central venous line (CVL) placement in children under ultrasound guidance resulted in fewer attempts and fewer complications. Maecken demonstrated that the inconsistent location and relationship between the

TABLE 5.1. Indications for the use of ultrasound in the pediatric intensive care unit

- Procedural
  - Vascular access
  - Thoracentesis
  - Paracentesis
  - Pericardiocentesis
- Focused echocardiography
  - Pericardial tamponade
  - Ventricular function
  - Volume status
- Thoracic ultrasound
  - Pneumothorax
  - Pleural effusion
- Abdominal ultrasound
  - Ascites/hemoperitoneum

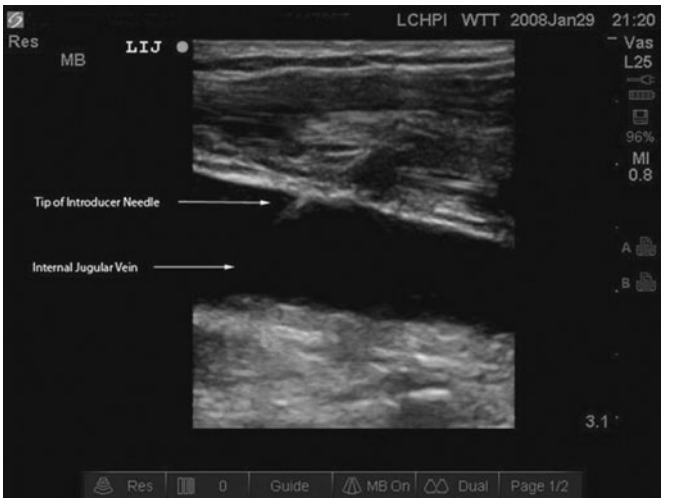


Figure 5.2. Internal jugular vein being accessed with an introducer needle.

internal jugular vein and carotid artery makes ultrasound guidance a useful tool.

Ultrasound can benefit in several ways. First, a survey of the vessels prior to deciding on an access site is useful in children with vascular and anatomic abnormalities or who have disease processes that predispose to venous clotting or have undergone multiple cardiac catheterization procedures through access of the femoral vessels. Second, ultrasound confirms the position and relative position of the vein and its relationship to the artery and other anatomic structures (Figures 5.1 and 5.2, and Video 5.1 in enclosed DVD). Third, placement of the catheter using the landmark method or by palpating the artery can be imprecise and ultrasound contributes additional specificity. Fourth, children, because of smaller structures and more subcutaneous fat, frequently do not have reliable landmarks. Fifth, it can be used to confirm proper placement of the catheter (Figure 5.3). Finally, after difficult and multiple attempts it can confirm whether a perivascular hematoma is going to prohibit the proper cannulation of the vessel. Please refer to Chapter 30, for a step-by-step guide to the use of ultrasound in venous cannulation.

Video 5.2 in the DVD shows a longitudinal image of an internal jugular vein with a guide wire in the lumen. Confirmation of vessel cannulation has been made prior to dilation of the vessel.

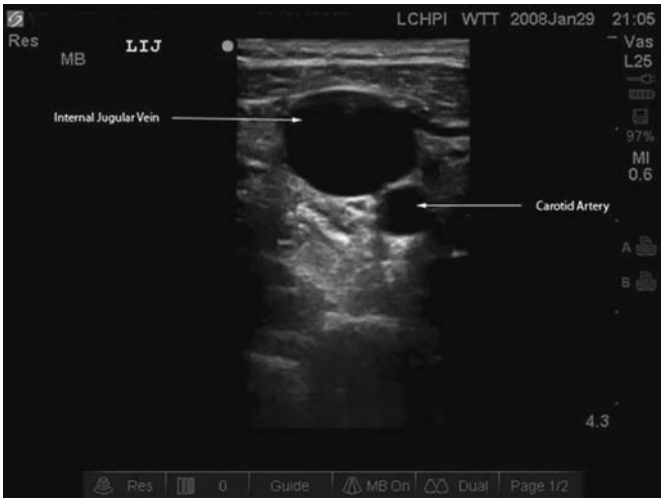


Figure 5.1. Left internal jugular vein with juxtaposed carotid artery.

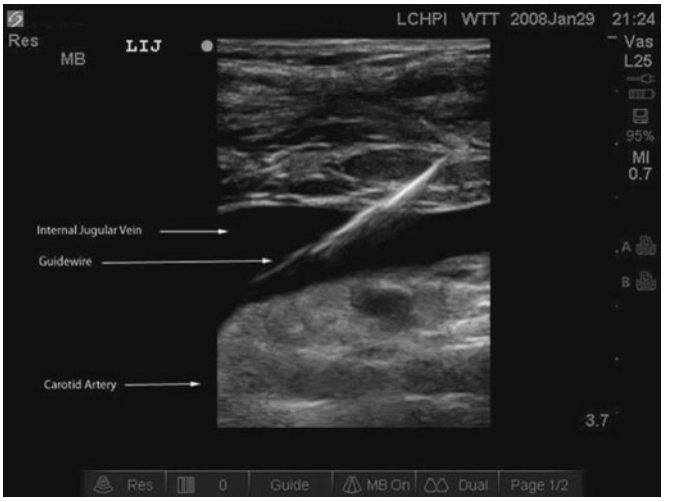


Figure 5.3. Internal jugular vein with an intraluminal guidewire.

FOCUSED ECHOCARDIOGRAPHY

In the pediatric cardiac ICU, focused echocardiography aids in the management of postoperative cardiac patients who are predisposed to pericardial effusion or tamponade, depressed cardiac function, controversies regarding the assessment of volume status, and the presence of hemodynamically significant pleural effusions. In addition, with more advanced techniques, right-sided heart failure can be assessed quickly with an assessment of right ventricular (RV) volume, tricuspid regurgitation, and paradoxical septal wall motion. These findings may be used to augment data from other hemodynamic measurements to confirm RV failure whether because of a right ventriculotomy or the presence of pulmonary hypertension. Similar findings are seen in adult patients and older pediatric patients who have hemodynamically significant pulmonary embolism.

The need to assess cardiac function may be underestimated in the general PICU. The use of focused echocardiography, a limited echocardiogram that has as its goal the hemodynamic assessment of gross ventricular function, pericardial tamponade, ventricular dilation, and assessment of volume status (Table 5.2), may be useful in guiding patient management in undifferentiated, fluid-resistant hypotension.

While septic shock in children has classically been considered a hyperdynamic state with either high or preserved cardiac output, Ceneviva et al. described that 60% of pediatric patients with septic shock have a decreased cardiac output. The sepsis-induced cardiomyopathy has been classically studied in children with meningococcemia but has also been studied quite extensively in adult patients.

In the pediatric patient with undifferentiated, fluid-resistant hypotension, bedside-focused echocardiography may be valuable. It allows the rapid assessment of global cardiac function and left ventricular chamber dimensions, and identifies hemodynamically significant pericardial effusions, findings that may influence management. Spurney and colleagues demonstrated that with limited training, and limited echocardiographic views (Figures 5.4 and 5.5), PICU physicians are capable of diagnosing significant pericardial effusions, decreased left ventricular (LV) systolic function, and LV enlargement. What is perhaps more important is that focused-bedside echocardiography allows the ability to perform serial bedside examinations and allows the important assessment and reassessment of the adequacy and efficacy of therapy (Videos 5.3 and 5.4 in enclosed DVD).

The assessment of volume status in the PICU is extremely important and assessments using physical examination may be inaccurate, particularly in the edematous child. Echocardiography has been validated for LV volume measurements, and assessment of LV end-diastolic volume (LVEDV) on parasternal short and

TABLE 5.2. Focused echocardiography

Cardiac function assessment
Left ventricular enlargement
Pericardial effusion
Inferior vena cava dynamics

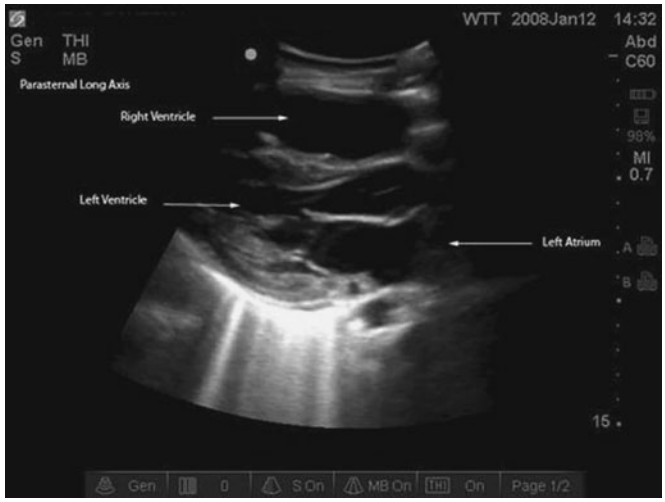


Figure 5.4. Parasternal long axis view of the heart during focused echocardiography.

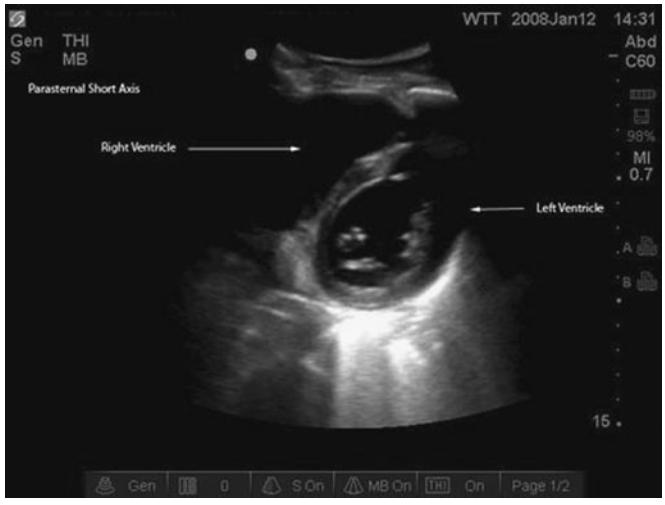


Figure 5.5. Parasternal short axis view of the heart during focused echocardiography.

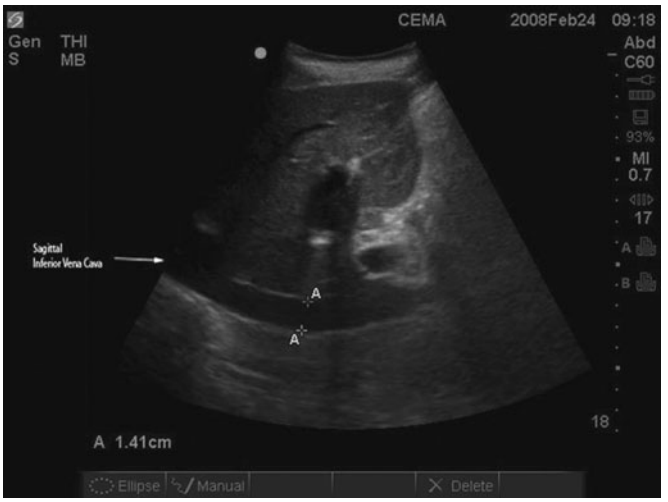


Figure 5.6. Assessment of IVC diameter.

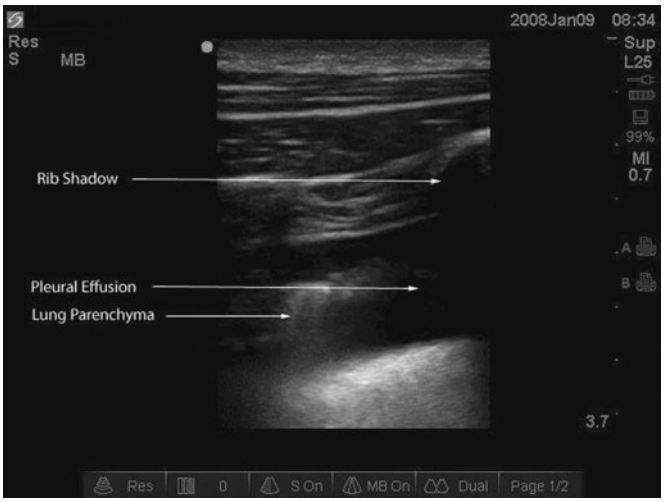


Figure 5.7. Simple pleural effusion.

parasternal long axis may guide volume management. For example, an LV chamber with complete collapse or obliteration of the LV cavity guides management in a way that is markedly different than in a patient with an LV cavity that is clearly dilated and poorly functioning.

The assessment of inferior vena cava (IVC) diameter and its collapse during inspiration may also be used to assess volume status. This method has been validated in adults to differentiate right atrial (RA) pressures <10 mm Hg or >10 mm Hg. Inferior vena cava dilation without a normal reduction in caliber during inspiration usually indicates elevated RA pressures. Inferior vena cava measurements during mechanical ventilation are less reliable because of the IVC dilation that is frequently seen normally while on the ventilator. However, a small diameter, or collapsed IVC will reliably exclude elevated RA pressures (Figure 5.6).

Much of the research done on IVC dynamics has been performed on adult patients. More investigation into pediatric patients must be performed before widespread use of this technique can be validated with empirical evidence.

THORACIC ULTRASOUND

While initially it seems that ultrasound of the chest would be limited because air is a poor medium for ultrasound wave conduction, lung ultrasound is quite useful in the PICU for evaluating pneumothorax, pleural effusions, and pulmonary edema. The assessment of pleural effusion is useful in the ICU setting because it provides a rapid assessment of the size, quality, and location of the effusions. In patients such as those with Fontan physiology who are predisposed to hemodynamically significant pleural effusions, the rapid assessment and ultrasound-assisted drainage may be emergent and life saving (Figure 5.6). On dynamic video, the lung can be seen swinging into view with each ventilator breath (Video 5.5 in enclosed DVD). Formal diagnostic ultrasound frequently fails to provide the complete picture a bedside clinician needs in properly assessing the size, quality (Figures 5.7 and 5.8, and Video 5.6 in enclosed DVD), and location of pleural effusions.

Pneumothorax

The assessment of pneumothorax can be extremely useful in those patients with a thoracic air leak. Pleural sliding, or shimmering (Figure 5.9), occurs on thoracic ultrasound when the visceral and parietal

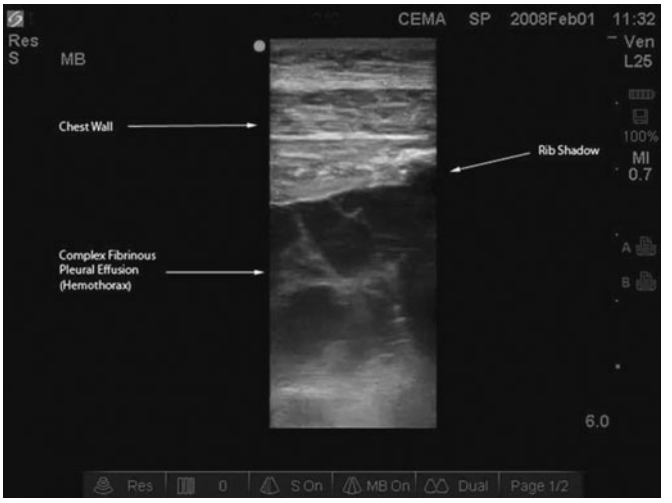


Figure 5.8. Complex pleural effusion.

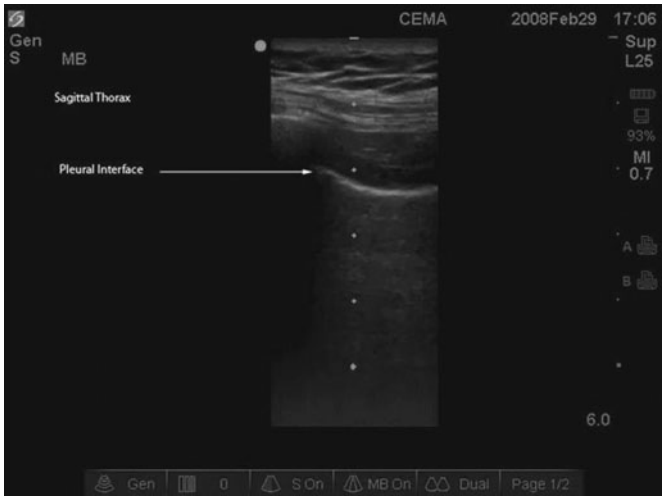


Figure 5.9. Pleural sliding.

pleura are apposed to one another and are sliding past each other because of movements of the diaphragm, or with mechanical ventilation. Dynamic images of pleural sliding are quite striking and its presence denotes the mobile apposition of the visceral pleura with the parietal pleura (Video 5.7 in enclosed DVD). The lack of sliding (Video 5.8 in enclosed DVD), however, does not prove that a pneumothorax is present and the clinician must proceed through the differential diagnosis for the lack of lung sliding (Table 5.3). Once it has been determined that the lack of lung sliding is due to pneumothorax, the transducer can be moved over the hemithorax to determine if loculation is present and the extent of the pneumothorax (Video 5.8). Thoracic ultrasound can help determine the best and safest place to insert a chest tube or pigtail drain.

Airway Ultrasound

The use of ultrasound in confirming endotracheal intubation is still early in its evolution. It remains difficult to determine if this technique is useful in real time. Galicinao et al. examined this issue in the pediatric emergency department and the PICU settings and were

TABLE 5.3. Differential diagnosis for absence of pleural sliding

Pneumothorax
Pleural effusion
Pleural scarring
Poor respiratory effort
Mainstem intubation
Mainstem occlusion

TABLE 5.4. Procedural ultrasound

Vascular access
Central line placement
PICC line placement
Arterial line placement
Pericardiocentesis
Paracentesis
Thoracentesis
Airway intubation

PICC indicates peripherally inserted central catheter.

able to report a high success rate in confirming endotracheal intubation in children. In addition, they were able to show specific instances where ultrasound was superior to CO_2 detection in determining tube placement. Currently, more research in this use is necessary before it gains widespread use.

PROCEDURAL ULTRASOUND

Table 5.4 shows the critical care procedures where ultrasound guidance is useful. While critical care procedures have traditionally been performed without adjunctive measures, the use of ultrasound now greatly influences efficiency and safety. Not only does ultrasound identify the optimal access points, but it also shows the anatomical relationships of internal structures and organs. While perhaps not as useful in patients with normal anatomy, it can be extremely useful in patients with abnormal anatomy or abnormal structural relationships. For example, in patients with hepatomegaly secondary to increased abdominal

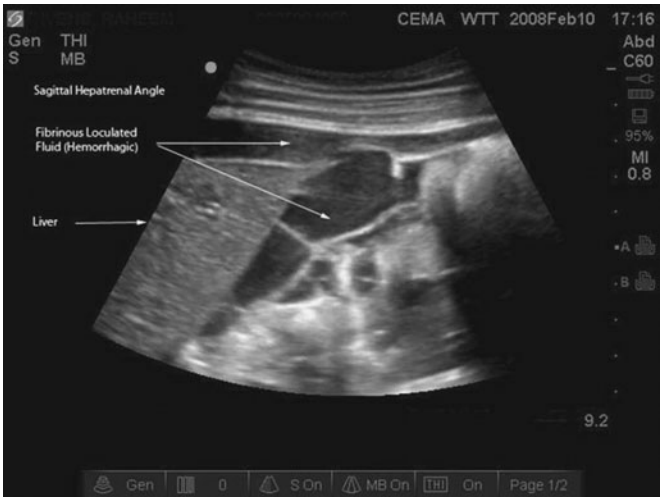


Figure 5.10. Hemorrhagic ascites in ECMO patient.

pressures, the insertion of a right-sided chest tube for an effusion or pneumothorax may come perilously close to the liver if the liver is encroaching into the chest. Abdominal ultrasound can help to identify the quantity and quality of ascitic fluid before attempting drainage. Figure 5.10 and Video 5.9 (in enclosed DVD) provide images of hemorrhagic ascites in a patient on extracorporeal membrane oxygenation (ECMO) who spontaneously bled into the peritoneum.

CONCLUSION

There are many artificial barriers to using bedside ultrasound in critically ill children. From the resistance of radiologic services, to cardiologists who disagree with limited focused emergency echocardiography, to the perception on the part of intensivists that past practice never required the use of ultrasound, to the difficulties associated with billing and liability; nonetheless, the use of ultrasound can enhance practice in the PICU and care for critically ill children in other settings as an extension of the physical examination.

Without proper training and expertise, however, the use of bedside ultrasonography may be misleading and may result in diagnostic and procedural mistakes. Additional empiric evidence and experience are necessary to show the benefits of bedside critical care ultrasonography in pediatrics.

Suggested Reading

Alderson PJ, Burrows FA, Stemp LI, Holtby HM. Use of ultrasound to evaluate internal jugular vein anatomy and to facilitate central venous cannulation in paediatric patients. *Brit J Anaesth.* 1993;70:145–148.

Barbier C, Loubieres Y, Schmit C, et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responsiveness in ventilated septic patients. *Intensive Care Med*. 2004;30:1740–1746.

Ceneviva G, Paschall JA, Maffei F, Carcillo JA. Hemodynamic support in fluid-refractory pediatric septic shock. *Pediatrics*. 1998;102:e19.

Feissel M, Michard F, Faller JP, Teboul, JL. The respiratory variation in inferior vena cava diameter as a guide to fluid therapy. *Intensive Care Med*. 2004;30:1834–1837.

Galicinao J, Bush AJ, Godambe SA. Use of bedside ultrasonography for endotracheal tube placement in pediatric patients: a feasibility study. *Pediatrics*. 2007;120:1297–1303.

Hind D, Calvert N, McWilliams R, et al. Ultrasonic locating devices for central venous cannulation: meta-analysis. *BMJ.* 2003;327:361.

Kircher BJ, Himelman RB, Schiller NB. Noninvasive estimation of right atrial pressure form the inspiratory collapse of the inferior vena cava. *Am J Cardiol*. 1990;66:493–496.

Maecken T, Grau T. Ultrasound imaging in vascular access. *Crit Care Med.* 2007;35:S178–S185.

Merx MW, Weber C. Sepsis and the heart. *Circulation.* 2007; 116:793–802.

Rudiger A, Singer M. Mechanisms of sepsis-induced cardiac dyspfunction. *Crit Care Med*. 2007;35:1599–1608.

Schiller NB, Shah PM, Crawford M, et al. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. *J Am Soc Echocardiogr*. 1989;2:358–367.

Spurney CF, Sable CA, Berger JT, Martin GR. Use of a hand-carried ultrasound device by critical care physicians for the diagnosis of pericardial effusions, decreased cardiac function, and left ventricular enlargement in pediatric patients. *J Am Soc Echocardiogr.* 2005;18:313–319.

Thiru Y, Pathan N, Bignall S, et al. A myocardial cytotoxic process is involved in the cardiac dysfunction of meningococcal septic shock. *Crit Care Med*. 2000;28:2979–2983.

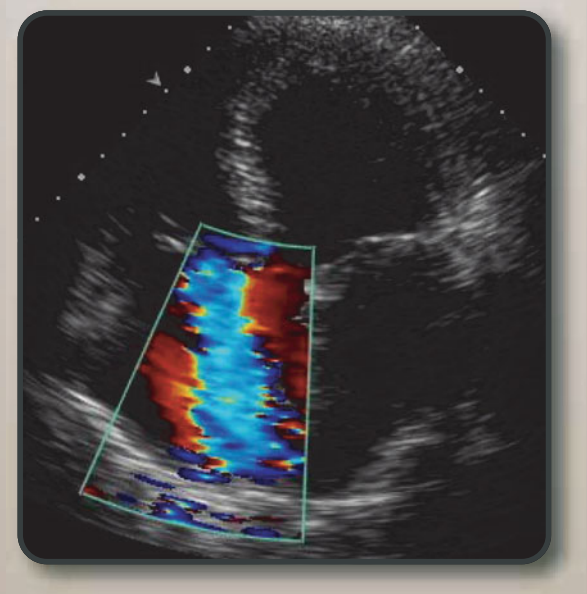
Verghese ST, McGill WA, Patel RI, Sell JE, Midgley FM, Ruttimann UE. Ultrasound-guided internal jugular venous cannulation in infants: a prospective comparison with the traditional palpation method. *Anesthesiology.* 1999;91:71–77.

Verghese ST, McGill WA, Patel RI, Sell JE, Midgley FM, Ruttimann UE. Comparison of three techniques for internal jugular vein cannulation in infants. *Paediatr Anaesth.* 2000;10:505–511.

Vieillard-Baron A, Chergui K, Rabiller A, et al. Superior vena caval collapsibility as a gauge of volume status in ventilated septic patients. *Intensive Care Med*. 2004;30: 1734–1739.

SECTION II

CARDIAC SONOGRAPHY IN THE ICU



This page intentionally left blank

CHAPTER 6

Goal-Directed Echocardiography in the ICU

John M. Oropello, Anthony R. Manasia, and Martin Goldman

INTRODUCTION

Goal-directed echocardiography (GDE) in the intensive care unit (ICU) setting is defined as a basic echocardiogram done with specific, focused objectives. It comprises a rapid, real-time, visual, two-dimensional echocardiographic assessment of cardiac preload, global and regional wall motion, and the pericardium at the bedside using either transesophageal or transthoracic methods. This chapter will discuss the rationale for intensivist-performed GDE, scope of GDE skills and knowledge, indications for GDE, choosing between transesophageal and transthoracic examinations, performance and interpretation of the GDE examination, equipment considerations, and training for acquiring the necessary skills for GDE.

RATIONALE FOR INTENSIVIST-PERFORMED GDE

Echocardiography has been predominantly performed by cardiologists who undergo extensive training for the performance of comprehensive echocardiographic examinations.[1] Over the past 1–20 years physicians, particularly in the fields of anesthesiology[2] and emergency medicine (EM),[3–6] have adopted components of the echocardiographic exam that are particularly suited to the unique needs of their particular patient population. Training guidelines have been published in basic and advanced perioperative comprehensive echocardiography for anesthesiologists and EM physicians.[5–7] Ultrasound is now an accepted part of the EM curriculum, having been adopted by the American College of Graduate Medical Education (ACGME) and now incorporated into residency training programs.[8] However, because significant differences exist between the use of echocardiography by these disciplines and that of critical care, guidelines specific for intensive care physicians need to be circumscribed.

Echocardiography provides information that is essential in clinical decision-making in critically ill patients to assess cardiac function and relative volume status. When compared with the indirect hemodynamic data from the relatively more invasive process of pulmonary artery catheterization,[9–12] echocardiography provides direct visualization of cardiac anatomy, and information on abnormal biventricular function and volume status as well as potentially compromising pericardial effusions. Goal-directed echocardiography in hemodynamically unstable patients, performed at the bedside by the physician caring for the patient, can provide immediate critical information about the cardiovascular system that is not available by other means and that can impact therapy in 30–40% of patients.[13,14] The aim of intensivist-performed GDE is to rapidly assess the hemodynamically unstable patient at the bedside to provide an immediate, personalized treatment in the ICU setting. While the standard comprehensive echocardiographic examination, which encompasses M-mode, 2D-echo, pulsed, continuous-wave, and color Doppler with calculations, requires approximately one hour,[15] the ICU-focused echo examination pertinent only to the immediate clinical scenario greatly reduces image acquisition and interpretation time while still maintaining diagnostic integrity[16,17]; it favors specificity of diagnosis over sensitivity.

SCOPE OF GDE KNOWLEDGE AND SKILLS

Performance of an ICU GDE requires an understanding of the indications for GDE in critically ill patients, the principles of ultrasound, and cognitive and technical skills to perform GDE in the ICU in critically unstable patients. The cognitive elements of performance of basic echocardiography include understanding of basic ultrasound principles, "knobology" of the machine, and a basic understanding of cardiac anatomy and function.

TABLE 6.1. Clinical indications for goal-directed echocardiography

Clinical indications	*Possible echocardiographic findings*
Acute hemodynamic instability including unexplained tachycardia, hypotension, shock, and changes in perfusion, e.g., decreased urine output, or other signs of decreased organ perfusion	Decreased preload, LV dysfunction, LV outflow tract obstruction, pericardial tamponade, RV dysfunction, proximal pulmonary embolism, severe valvular stenosis, intracardiac mass
Acute respiratory failure	LV or RV dysfunction, proximal pulmonary embolism
Acute pulmonary edema	LV dysfunction, LV outflow tract obstruction, severe valvular stenosis
Evaluation of cardiac arrest	Presence or absence of ventricular contraction, decreased preload, LV dysfunction, LV outflow tract obstruction, pericardial tamponade, RV dysfunction, proximal pulmonary embolism
Failure to respond appropriately to initial resuscitation	Decreased preload, LV dysfunction, LV outflow tract obstruction, pericardial tamponade, RV dysfunction
Serial monitoring of the response to resuscitation	Follow changes in LV preload and contractility
Unexplained increased trend in serum lactate, V-aCO_2 difference, or decreased trend in SvO_2	Decreased preload, LV dysfunction, LV outflow tract obstruction, pericardial tamponade, RV dysfunction
Assessment of pacemaker capture	Presence or absence of ventricular contraction

LV indicates left ventricle; RV, right ventricle; SvO_2, mixed venous oxygen saturation; V-aCO_2, venous-arterial CO_2 difference.

The method of probe placement may be either transesophageal or transthoracic and the trainee may train in either or both skills. All aspects of the echocardiographic examination—image acquisition, image interpretation, and clinical application—should be the responsibility of the critical care physician in charge of the bedside management of the patient. In this situation, clinicians can integrate the immediate information gained by ultrasonic examination with their knowledge of the real-time clinical situation to establish a more accurate diagnosis and guide ongoing therapy.

GDE encompasses the ability to assess ventricular preload, detect a volume-depleted ventricle, assess left ventricular contractility and wall motion, recognize dilation of the right ventricle, and detect significant pericardial effusion. Clinicians who achieve basic level competence in GDE must recognize the limitations of their skills. Basic training does not prepare the practitioner to perform a comprehensive echocardiographic evaluation that encompasses assessment of valvular pathology, endocarditis, cardiac thrombus, aortic dissection, or other complex conditions outside the basic examination. The trainee should confirm any unexpected findings with either a formal echo exam or a rapid review by a more skilled echo performer. Therefore, ideally, images should be stored digitally for documentation, reviews, and future comparison.

INDICATIONS FOR GDE

Goal-directed echocardiography rapidly provides information about cardiac filling, wall motion abnormality, pericardial effusion, and approximate circulatory volume status.[13,14] The indications for GDE (Table 6.1) include to evaluate and manage acute hemodynamic decompensation or shock, to guide resuscitation, to determine a cardiogenic cause of acute respiratory failure or pulmonary edema, and to provide critical information when monitors indicate a worsening state of organ perfusion.

CHOOSING TRANSESOPHAGEAL VS. TRANSTHORACIC EXAMINATION

Although transesophageal echocardiography (TEE) is considered an advanced imaging modality performed by cardiologists and cardiac anesthesiologists only after mastery of transthoracic echocardiography (TTE), TEE can be performed by intensivists, especially

those with experience in invasive interventions such as intubation and bronchoscopy. The technique of esophageal intubation in the setting of a tracheally intubated patient who is sedated represents more familiar territory to the intensivist than to most cardiologists. Transesophageal probe placement is not as difficult for an intensivist who routinely inserts nasal and orogastric tubes, performs endotracheal intubation, and often administers sedation in the face of hemodynamic instability. As a result, it takes the average intensivist a much shorter time to become proficient at safely and consistently passing a transesophageal probe. Intensivists can become proficient at TEE probe placement and GDE image acquisition after an average of eight procedures.[13] Especially in the context of GDE, the advantages of TEE are higher resolution images than TTE, and virtually guaranteed appropriate transducer location within the distal esophagus and proximal stomach, providing excellent echocardiographic windows compared with window searching by TTE. Critically ill patients are more likely to have difficult TTE imaging windows due to intubation, patient positioning, body habitus, pathologic thoracic air collections around the chest, and chest wall bandages. Because TEE probes do not need as much penetration, they can image at higher frequencies (5–7 MHz) and provide greater resolution than TTE.

Transesophageal echocardiography provides an accurate and reliable assessment of intracavitary dimensions, global and regional right ventricular (RV) and left ventricular (LV) wall motion, and valve function, and it images the great vessels including the proximal pulmonary arteries. An added advantage of TEE is that the proximal pulmonary arteries can be evaluated for evidence of pulmonary thromboembolism. Although the use of TEE is not totally without risk, it is much less invasive than the pulmonary artery catheter (PAC), and is probably associated with fewer complications.[18,19]

The disadvantages of TEE include that it is relatively invasive compared with TTE and cannot be performed on patients with significant oropharyngeal, esophagogastric pathology, or acute upper gastrointestinal (GI) bleeding. Despite its relatively more invasive nature, TEE is a very safe procedure with a less than 1/10,000 incidence of esophageal perforation. Complications related to sedation, aspiration and the dislodgement of airway tubes can still occur, but are relatively uncommon.

Transthoracic echocardiography has the advantage of being totally noninvasive and more readily available than TEE, but it can be challenging to obtain adequate images in the critically ill patient. Despite the limitations of TTE, intensivists can obtain an adequate GDE examination in 94% of patients in the ICU.[14]

All things considered, TTE probably represents an easier starting point for most intensive care physicians in GDE. However, for critical care physicians, we advocate learning both methods, as there will be occasions when image acquisition is not possible with TTE. If a patient is not critically ill to require intubation, the patient should have a TTE examination or have the TEE exam when a cardiologist is available.

PERFORMANCE AND INTERPRETATION OF THE GDE EXAMINATION

The components of performing an echocardiographic examination are application or placement of the probe, obtaining the necessary views specific to the method (TEE vs. TTE), image acquisition, and data interpretation.

Goal-Directed Transesophageal Echocardiography

The performance of TEE is a multistep process involving (1) esophagogastric intubation, (2) TEE probe manipulation, (3) image acquisition and processing, and (4) data interpretation. A key assumption of the TEE training is skill with esophageal intubation and emphasis on the performance and interpretation of the echocardiogram examination. Intensivists become comfortable with TEE after performing an average of 8–10 TEE examinations and complete a GD TEE examination in an average of 12+/−7 minutes.[13]

The two views necessary to perform GD TEE are the transgastric short-axis window and the esophageal four-chamber window.

Transgastric Short-Axis Window

This window is obtained by passing the TEE probe to approximately 40 cm into the proximal stomach and anteflexing the tip. By moving the probe more distally or proximally circumferential, images of the LV apex, midpapillary muscle level, and base (mitral valve level) are obtained. This view is useful for assessing LV (volume and function) and the presence of a pericardial effusion. The midpapillary muscle level is used to assess end-systolic and end-diastolic area that correlates with LV volume (preload) (Figure 6.1, and Video 6.1 in enclosed DVD). Previous studies have confirmed the

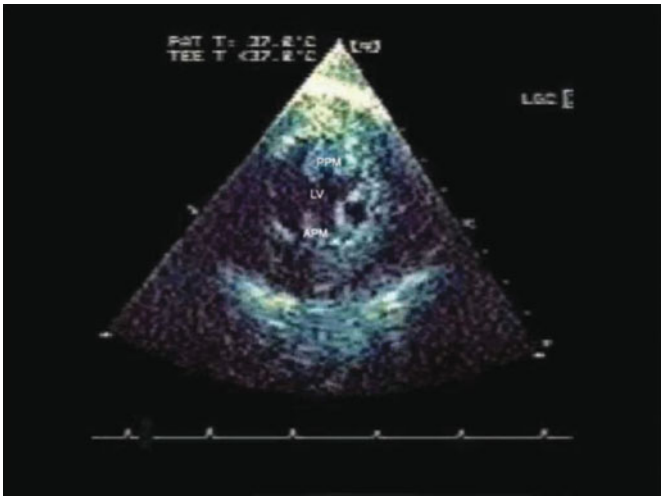


Figure 6.1. Transgastric short-axis window; midpapillary muscle level. LV indicates left ventricle; PPM, posterior papillary muscle; APM, anterior papillary muscle (see Video 6.1 in enclosed DVD).

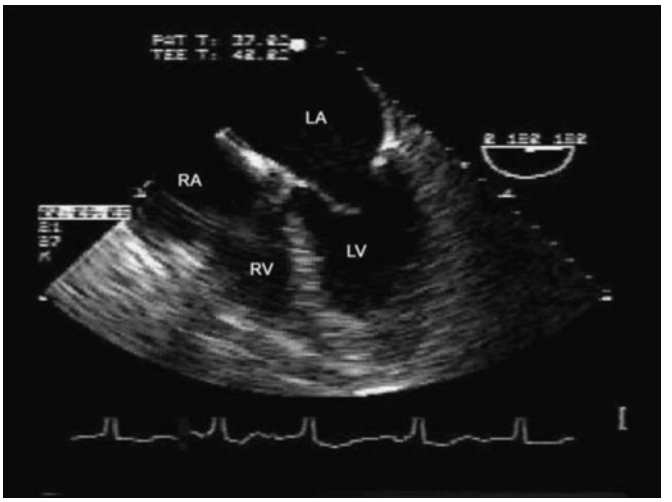


Figure 6.2. Transesophageal four-chamber window. LA indicates left atrium; RA, right atrium; LV, left ventricle; RV, right ventricle (see Video 6.2 in enclosed DVD).

efficacy of a short-axis plane for evaluation of the LV ejection fraction.[21]

Esophageal Four-Chamber Window

This window is obtained by withdrawing the probe into the midesophageal level and relaxing or retroflexing the tip, opening up a four-chamber view of the heart. In this view the right atrium, right ventricle, left atrium, left ventricle, interatrial septum, interventricular septum, tricuspid valves, mitral valves (MV), and pericardium can be visualized. By slight positioning of the probe, the left ventricular outflow tract (LVOT), the aortic valve, and the relationship between the LVOT and the anterior leaflet of the MV can be seen (Figure 6.2, and Video 6.2 in enclosed DVD).

Basal Short-Axis Window

The basal short-axis window is obtained by advancing the TEE probe to a depth of 25–30 cm, which corresponds to a position posterior to the left atrium. Slight anteroflexion and withdrawal of the transducer will allow visualization of the aortic valve, proximal ascending, proximal coronary arteries, atrial appendages, superior vena cava, atrial septum, pulmonary veins and the proximal pulmonary arteries. The left atrial appendage appears as a triangular extension of the left atrium. It is important to note that the pectinate muscles appear as muscular ridges within the appendage and must not be mistaken for thrombi. When the transducer is withdrawn 1–2 cm while anteflexing the tip, the pulmonary artery trunk with the right and left pulmonary arteries can be visualized (Figure 6.3).

Goal-Directed Transthoracic Echocardiography

The performance of TTE involves identifying an appropriate "window" to penetrate the chest wall and soft tissue for ultrasound penetration to the heart and manipulation of the TTE probe on the chest wall, image acquisition and processing, and data interpretation. The left lateral decubitus position, when possible, brings the heart closer to the chest wall, facilitating better imaging windows. The average time for image acquisition and interpretation by noncardiologist intensivists is 10.5+/−4.2 minutes.[14]

The four basic views necessary to perform GD TTE are the parasternal longitudinal-axis window,

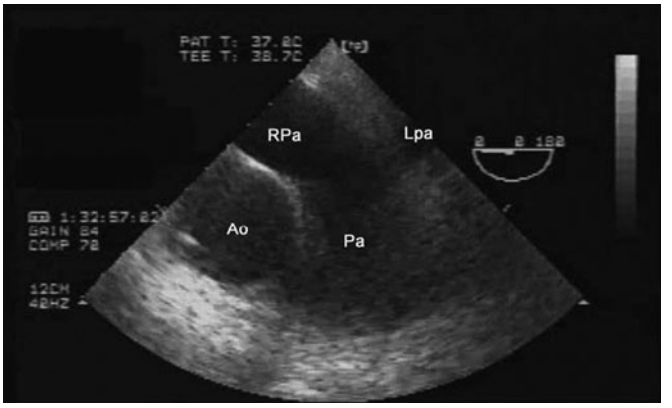


Figure 6.3. Basal short-axis window. Ao indicates aorta; Pa, main pulmonary artery; RPa, right pulmonary artery; Lpa, left pulmonary artery.

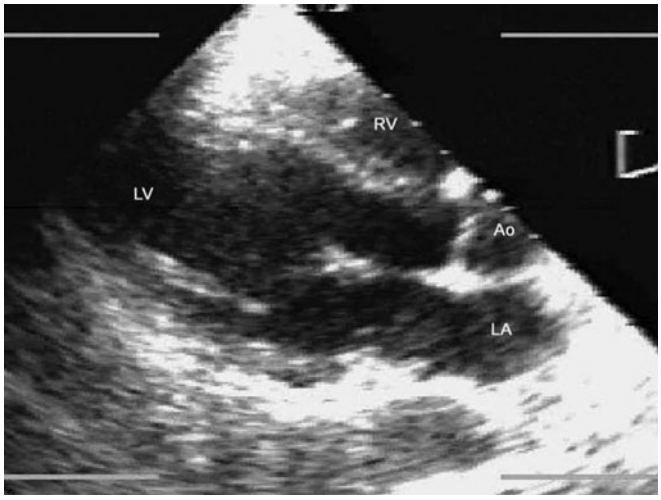


Figure 6.4. Parasternal longitudinal-axis window. LA indicates left atrium; LV, left ventricle; Ao, aorta; RV, right ventricle (see Video 6.3 in enclosed DVD).

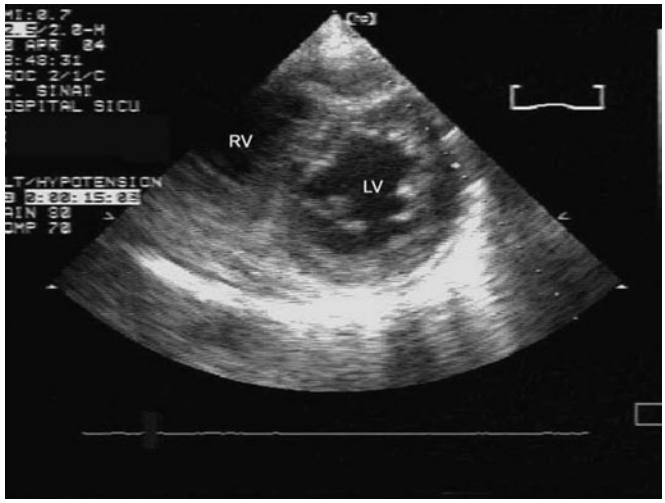


Figure 6.5. Parasternal short-axis window. RV indicates right ventricle; LV, left ventricle (see Video 6.4 in enclosed DVD).

the parasternal short-axis window, the apical four-chamber window, and the sub-xiphoid window.

Parasternal Longitudinal-Axis Window

This window is obtained by placing the TTE probe near the left of the sternum at the 4th intercostal space and rotating the probe into a diagonal plane from the right shoulder to the left flank. In this view the aortic valve, left ventricle, interventricular septum, right ventricle, LVOT, mitral valve, and pericardium can be seen. (Figure 6.4, and Video 6.3 in enclosed DVD).

Parasternal Short-Axis Window

This window is obtained by placing the TTE probe near the left of the sternum at the 4th intercostal space and rotating the probe in a diagonal plane from the left shoulder to the right flank. By angling the probe, circumferential images of the LV apex, midpapillary muscle level, and base (mitral valve level) are obtained. This view is useful for assessing LV preload, end-diastolic area and end-systolic area, septal and LV wall motion, and the presence of pericardial effusion. (Figure 6.5, and Video 6.4 in enclosed DVD).

Apical Four-Chamber Window

This window is obtained by placing the probe over the apical beat if palpable, or anticipated location of the apex and obtaining a four-chamber view of the heart. In this view the right atrium, right ventricle, left atrium, left ventricle, interatrial septum, interventricular septum, tricuspid valves, mitral valves, and pericardium can be visualized (Figure 6.6, and Video 6.5 in enclosed DVD).

Subxiphoid Window

The subxiphoid view is obtained by positioning the imaging probe just below or to the right of the xiphoid process with the patient in the supine position. When possible, the patient's knees should be bent to allow relaxation of the abdominal muscles and with the patient taking a full inspiration. This allows the heart to move closer to the probe. The right ventricular apex as well as the mid- and basal portions of the right ventricle are visualized. The inferior interventricular septum and the anterolateral left ventricular wall are also seen. Since the interatrial septum runs somewhat perpendicular to the ultrasound beam, atrial septal defects

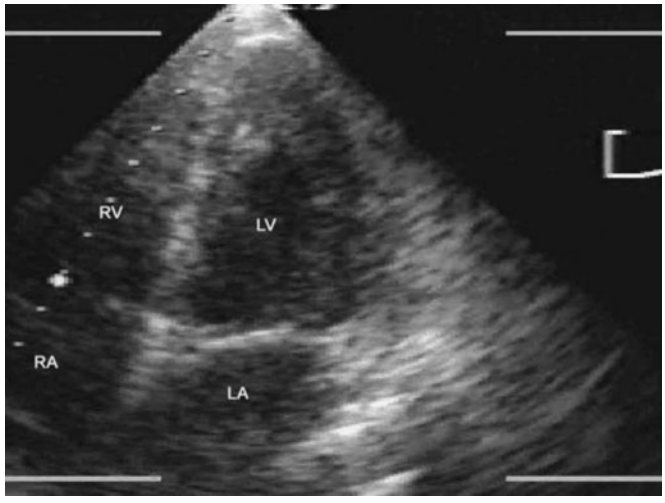


Figure 6.6. Apical four-chamber window. LA indicates left atrium; RA, right atrium; LV, left ventricle; RV, right ventricle (see Video 6.5 in enclosed DVD).

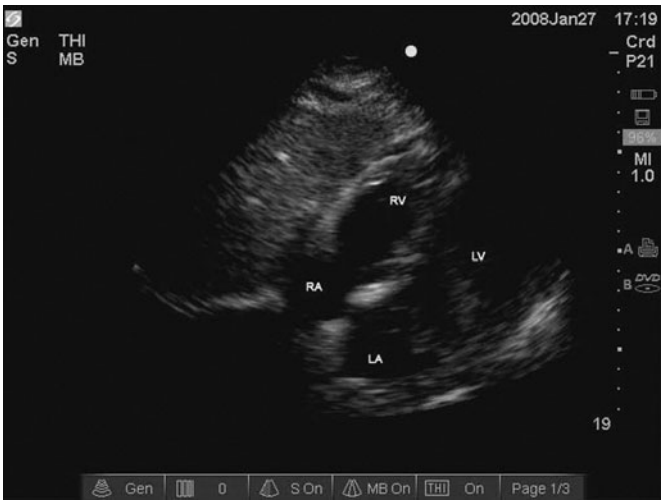


Figure 6.7. Subxiphoid window. LA indicates left atrium; RA, right atrium; LV, left ventricle; RV, right ventricle.

and atrial septal aneurysms are best detected in the subxiphoid window. Right ventricular dimensions and wall thickness are best evaluated using this window. When the transducer is angulated more medially, the hepatic veins and the inferior vena cava entering the right atrium are visualized. The abdominal aorta is visualized when the transducer is moved slightly to the left (Figure 6.7).

Assessment of Preload

The diagnostic criterion for normal preload is a normal overall end-diastolic LV cavity size at the midpapillary level on transgastric short-axis view (TEE) or parasternal shortaxis view (TTE). In this midpapillary short-axis view, decreased preload is defined as a decreased LV end-diastolic area (EDA) [normal: 22 ± 4 cm^2] and an even greater decreased end-systolic area (ESA) [normal: 8.5 ± 2 cm^2], or near end-systolic obliteration of the LV cavity. This combination of changes leads to an increased fractional area change (FAC = (EDA − ESA)/EDA) on the midpapillary short-axis view. The criteria for increased preload are an increased LV EDA and ESA and a normal-to-decreased FAC on the midpapillary short-axis view of the LV. Although normal ranges are given, it must be noted that there is significant variability in the reference values for the normal echocardiographic examination.[22] Determining the limits of discrimination between "normal" and a certain condition, e.g., decreased preload, also requires visual comparisons of relative sizes during the 2D-echo examination (Video 6.6 and Video 6.7 in enclosed DVD).

Assessment of Contractility

Left ventricular function is visually assessed on the longitudinal four-chamber view and at three levels (basal, midpapillary, and apical) of the transgastric short-axis view. Normal LV function is defined as a normal FAC and the absence of regional wall motion abnormalities. The criteria for hypocontractility, or more accurately, reduced wall thickening, are global or segmental wall motion abnormalities with overall decreased LV function (Video 6.8 in enclosed DVD). Hypercontractility is defined as a global LV hyperkinesis, or marked increased wall thickening, not just tachycardia (Video 6.9 in enclosed DVD). The assessment of LV contractility may be accompanied by colored Doppler (to screen for gross aortic and mitral regurgitation) and examination of the aortic outflow tract to recognize systolic anterior motion of the mitral valve (to assess for left ventricular outflow tract obstruction).

Assessment of the Left Ventricular Outflow Tract

The left ventricular outflow tract can be assessed in the four-chamber view, and with slight manipulation the aortic valve relationship with the anterior leaflet of the mitral valve can be seen to detect the phenomenon of dynamic left ventricular outflow tract obstruction, a condition associated with a reduced preload and inotropic medications that cause the anterior leaflet of the mitral valve to obstruct the aortic outflow tract (Figure 6.8), or significant narrowing of left ventricular

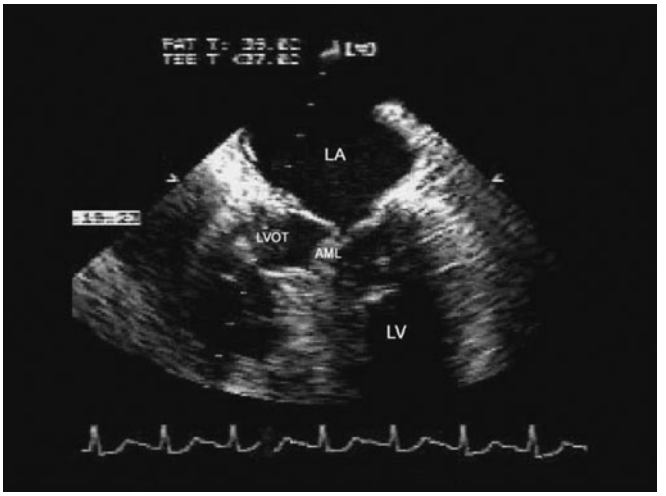


Figure 6.8. Left ventricular outflow tract obstruction. LA indicates left atrium; LV, left ventricle; AML, anterior mitral valve leaflet; LVOT, left ventricular outflow tract. Note the AML moving into and obstructing the LVOT on systole (SAM indicates systolic anterior motion).

outflow tract due to small volume and hypercontractile LV muscles. Typically, the pulmonary artery catheter has demonstrated low cardiac output (CO) and high pulmonary artery occlusion pressure, but the heart is not hypocontractile nor dilated: echocardiography reveals a hyperdynamic heart with small, almost empty end-systolic volume. This condition cannot be diagnosed with a PAC and the treatment is "counterintuitive" to the high wedge and low CO: the treatment is fluid and withdrawal of inopressors. More commonly, even in the absence of frank outflow tract obstruction, ventricular underfilling, and normal or elevated wedge pressure (or central venous pressure [CVP]) are encountered.

Assessment of Pericardium

The four chambers and surrounding pericardium of the heart are visualized for global function and abnormal fluid collections, differentiating pericardial from pleural and ascitic fluid. Two-dimensional echocardiographic signs of tamponade physiology in the presence of pericardial effusion include right atrial collapse, right ventricular diastolic collapse, noncollapsible inferior vena cava and hepatic veins, respiratory shifting of the interventricular septum, and Doppler variation in LV or RV outflow of >25 %. Signs and symptoms for significant pericardial effusion, or tamponade, such as Beck's triad, are often nonspecific or lacking in critically ill patients on positive pressure ventilation or positive end-expiratory pressure (PEEP) (Figure 6.9 and Video 6.10 in enclosed DVD).

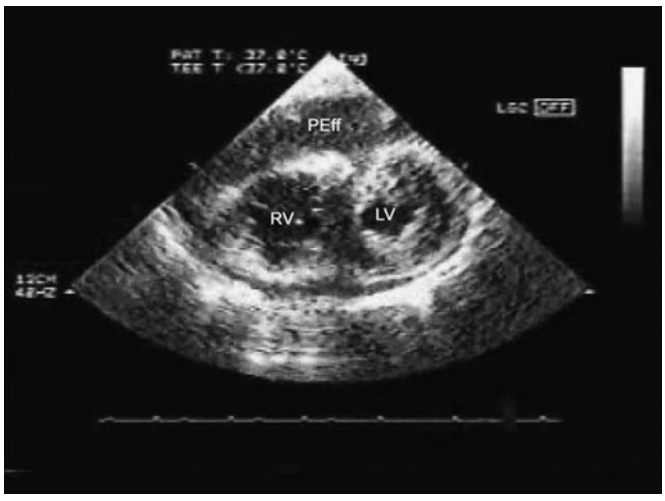


Figure 6.9. Pericardial effusion; transgastric short-axis window. RV indicates right ventricle; LV, left ventricle; PEff, pericardial effusion (see Video 6.10 in enclosed DVD).

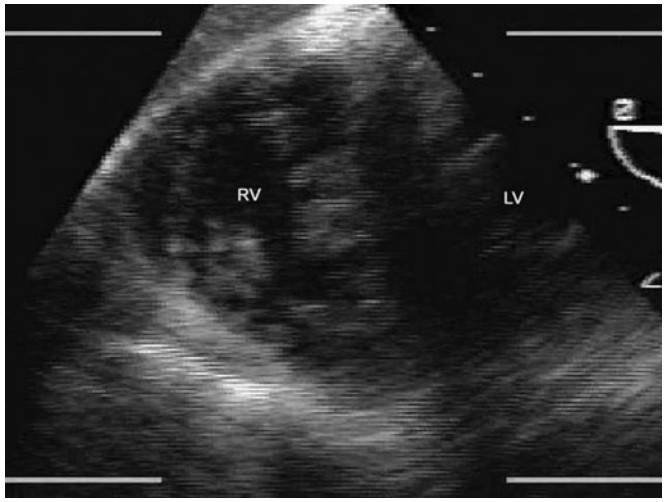


Figure 6.10. Right ventricular hypertrophy. RV indicates right ventricle; LV, left ventricle. Note the thickened RV wall and small end-diastolic area.

Assessment of the Right Ventricle

Both the preload and contractility of the right ventricle (RV) can be determined using the analogous views used to determine the same variables for the LV, namely the midpapillary level on the transgastric short-axis view (TEE) or parasternal short-axis view (TTE) and the longitudinal four-chamber view. Although the stroke volumes of the right and left ventricles will be equivalent overall; in acute conditions they may not be equal. In addition, the function (e.g., contractility, preload, and ejection fraction) may be markedly different. A rapid visual assessment of RV function can be helpful in determining the etiology of acute hemodynamic instability. The RV may be markedly hypertrophic in patients with cor pulmonale or long-standing pulmonary hypertension, (Figure 6.10) or dilated and hypocontractile in acute pulmonary hypertension.

Assessment of Valves

The assessment for complex valvular disease is beyond the GDE exam; if the sole indication for an echocardiogram is to rule out endocarditis or assess for particular valvular pathology, the exam should be performed by a level 2 examiner (see Chapter 4).

However, there are two aspects concerning valvular pathology that do concern the basic-scope echocardiographer. When performing a GDE, e.g., for acute hypotension, the valves are visualized and the examiner should know normal valvular anatomy so that gross abnormalities, e.g., large vegetations or valvular deformities (stenosis, severe regurgitation, or abnormal

masses), may be detected and an appropriate referral for a comprehensive exam can be made. Finally, the assessment of LV preload and contractility should be accompanied by a basic colored Doppler evaluation of the mitral and aortic valves to rule out significant mitral or aortic regurgitation that impacts net forward systemic cardiac output.

EQUIPMENT CONSIDERATIONS

It is highly recommended to have an ultrasound machine exclusively available for use in the ICU to gain the hands-on experience necessary to learn.

Ultrasound Devices

There is a wide selection of ultrasound devices available from a number of manufacturers.[22,23] They range from refrigerator-sized, full-function devices that can cost over US$250,000 dollars, to smaller units (e.g., laptop computer–sized or even pocket-sized, i.e., Siemens ACUSON P10, Siemens Healthcare, Malvern, PA) with limited capability (Table 6.2) that currently cost from US$10,000 to over $100,000, depending on the number and types of probes (TEE or TTE) ordered. For a GD examination, a device capable of performing 2D echocardiography with basic color Doppler is sufficient. All of the smaller devices have surface probes and several manufacturers now offer an optional TEE probe.

TEE Probe Selection

To expedite insertion in patients who commonly have indwelling endotracheal or tracheostomy tubes, the smallest-size transesophageal probe available should be chosen. This will depend on the manufacturer of the ultrasound machine, although the general trend is that over time transducer probes become smaller, with more capability.[25] Monoplane probes image in only the transverse plane, biplane probes allow the user to switch from a transverse to a longitudinal plane, and multiplane probes allow imaging in any selected angle between the transverse and longitudinal. When using a multiplane probe, selection of 0° will provide imaging in the transverse plane. In the GD TEE study,[13] a pediatric monoplane 5-MHz probe with a shaft diameter of 7.1 mm and distal tip 10 mm wide and 8 mm thick, was chosen. More recently, a pediatric probe of similar dimensions comes with multiplane capability. Even adult multiplane probes can be inserted; however, they are relatively larger, thus more difficult to pass.

For GD echocardiography, regardless of the transesophageal transducer probe capability, it is recommended that images be obtained in only the transverse plane earlier in training. The transverse plane facilitates visualization not only of the transgastric short-axis view, but also of the transesophageal four-chamber view, in which even the apex and the anteroseptal and lateral walls can be clearly seen. This simplifies the

TABLE 6.2. Features comparison of cardiac ultrasound machines

	Full-sized unit	*Mobile cart mounted unit**	*Handheld device***
Relative price with probes	++++	++−++	+−++
Weight	~300 lbs	~20 lbs	~5–10 lbs
Power supply	AC	AC or DC	AC or DC; DC only
TTE probe capability	All	All	All
TEE probe capability	All	Most	Some
M-mode capability	All	All	Some
2D echo capability	All	All	All
Doppler: color	All	All	Most
Doppler: pulse wave	All	All	Some
Doppler: continuous-wave	All	All	Some
Image storage	All; full	Most; full	Some; limited

** These units are in general larger and heavier than laptop computers and need mobile carts for transport to the bedside.*
*** Includes pocket-sized units as well as laptop computer–sized units that are often mounted on mobile carts to protect the device, avoid theft, and aid in positioning the unit at the bedside. AC: needs to be plugged into outlet; DC: battery powered; All = all models; Most = most models; Some = some models. Note: the capabilities feature sets of the smaller devices continues to grow, hence the purchaser should consider the budget and compare the features offered at the time of purchase.*

image interpretation and reduces the chances for misinterpretation of cavity size or wall motion.

TTE Probe Selection

A versatile probe that can image at frequencies between 2.5 MHz and 5 MHz may also be used for general ultrasound imaging applications (e.g., pleura, lungs, abdomen, etc.) in the critically ill patient.

Recording

In terms of documentation, it is important to record echocardiographic studies for later review for medical, educational, quality control, and legal purposes. Most of the large, full function devices have digital image capability but some of the handheld devices may not. When using a handheld device, it is important to make sure that it can record digital video to a memory card for later downloading and archiving or that it can be connected to an external digital video recording system. In the event that an acute or emergent diagnostic exam is performed but not recorded, the findings must be documented in the medical record.

TRAINING: ACQUIRING SKILLS IN GDE

Consensus competency guidelines for intensivists seeking to perform basic echocardiography have been published.[26] Subsequent guidelines will deal with education, training, and documentation of competency. In the meantime, the authors of this chapter offer the following suggestions.

Trainees in GD critical care echocardiography must be licensed physicians enrolled in or with completed accredited residency training. The cognitive and technical skills necessary to perform GD echocardiography are outlined in Tables 6.3 and 6.4. The key training recommendations are outlined in Table 6.5. The achievement of these skills depends on structured independent study consisting of reading, audiovisual aids, web-based education, computer-assisted instruction,

TABLE 6.3. Training objectives for goal-directed basic echocardiography: cognitive skills

Understanding of:

1. Basic ultrasound principles
2. The operation of ultrasound machines including the controls
3. Equipment handling, infection control, electrical safety operation
4. The indications and contraindications and complications of TEE
5. The indications for TTE
6. Normal topographic cardiac anatomy
7. The transgastric short-axis and esophageal longitudinal-axis views (TEE)
8. The parasternal longitudinal and short-axis, and apical four-chamber views (TTE)
9. The echocardiographic evaluation of preload
10. The echocardiographic evaluation of global and regional wall motion
11. The echocardiographic presentation of pericardial effusion and tamponade and to differentiate pericardial from pleural effusion
12. The presentation of dynamic left ventricular outflow tract obstruction
13. The echocardiographic presentations of severe sepsis
14. The echocardiographic presentation of myocardial ischemia and infarction
15. The echocardiographic presentation of acute pulmonary embolism and basal view to detect proximal pulmonary emboli (TEE)
16. Basic normal valvular anatomy and detection of stenosis
17. Basic colored Doppler to detect significant mitral or aortic regurgitation

TEE indicates transesophageal echocardiography; TTE, transthoracic echocardiography.

TABLE 6.4. Training objectives for goal-directed basic echocardiography: technical skills*

1. Facility with the operation of ultrasound machine "knobology" and how to produce quality images
2. Facility with insertion of TEE probe safely in a tracheally intubated patient
3. Facility to acquire transgastric short-axis and longitudinal four-chamber views (TEE)
4. Facility with parasternal longitudinal and short-axis views and apical four-chamber views (TTE)
5. Ability to scan the ventricle in short axis from base to apex and identify the midpapillary muscle level for assessment of left ventricular end diastolic and end systolic area
6. Ability to recognize normal vs. markedly abnormal cardiac structures
7. Ability to detect significant abnormalities in cardiac preload
8. Ability to detect significant abnormalities in left ventricular contraction and wall motion (global and regional)
9. Ability to perform rapid visual online assessment of dynamic ventricular function ("eyeballing"), e.g., global ventricular filling and function
10. Ability to detect significant pericardial effusions
11. Ability to recognize echocardiographic artifacts
12. Ability to communicate echocardiographic results to health care professionals, the medical record, and patients
13. Ability to recognize limitations and when to call for advanced echocardiographic backup both acutely and electively as indicated

*See also Chapter 4.
TEE indicates transesophageal echocardiography; TTE, transthoracic echocardiography.

attendance of seminars, supervised performance of echocardiographic examinations under the direct supervision of an experienced, advanced echocardiographer (cardiologist or level 2–trained intensivist), independently performed examinations recorded and reported to the supervisor, and interpretation of studies performed by others but presented to the supervisor. The director of the echocardiographic training program should be a physician with advanced training (level 3) and demonstrated expertise in intensive care GDE. Training should begin with an intensive seminar—at least 10 hours of instruction, focused on the elementary principles of cardiac ultrasound examination, echocardiographic data interpretation, the basic operation of echocardiographic equipment, and 2D echocardiographic examination of the LV as seen from the midpapillary short-axis and longitudinal four-chamber views. The trainee should also observe echocardiography examinations conducted in the ICU and, where possible, in the echocardiography laboratory so that basic trainees can gain regular and frequent exposure to teaching and clinical resources within that laboratory. After this basic orientation, the trainee begins performing GDE under the direct guidance of a physician (intensivist or cardiologist) experienced in GDE. Under appropriate supervision, the trainee learns to operate the ultrasonograph, place the transducer probe, and perform the GDE examination. The level of supervision is subsequently modified depending on the competence acquired by individual intensivists. Trainees graduate to the unsupervised level after they demonstrate competence in intubation (for TEE), image optimization, and interpretation of LV function, as deemed by the supervisor. This may occur after approximately 10 to 20 procedures, depending on the prior experiences of the trainee. However, a practitioner with advanced training must review every examination performed by the trainee. All echocardiographic examinations performed by trainees should be digitally recorded and reviewed weekly with the supervisor with respect to the accuracy of data interpretation in those patients not examined under their direct supervision. Trainees should keep a log of examinations performed and reviewed to document the extent of their training. Minimum numbers of cases for competence can be delineated (Table 6.5) but these numbers

TABLE 6.5. Key training recommendations for goal-directed basic echocardiography

Transesophageal goal-directed echocardiography

	Minimum suggested number*	***Achievement of competency***
Esophageal intubation**	5	Ability to consistently and safely place probe in sedated and intubated patient and position probe to acquire transgastric short-axis and transesophageal longitudinal four-chamber views.
Personally performed exams+	10	Ability to acquire transgastric short-axis and transesophageal longitudinal four-chamber views of sufficient quality to evaluate ventricular filling and function and the pericardium. Ability to distinguish between normal and abnormal anatomy.
Total number of cases reviewed++	20	Ability to interpret ventricular preload and left ventricular wall motion both global and gross regional (septum, anterior wall inferior wall). Ability to diagnose pericardial effusion and distinguish from pleural effusion. Ability to assess for signs of dynamic left ventricular outflow tract obstruction including LV preload and systolic anterior motion of the anterior leaflet of the MV.

Transthoracic goal-directed echocardiography

	Minimum suggested number*	***Achievement of competency***
Personally performed exams+	10	Ability to acquire parasternal longitudinal and short-axis views and apical four-chamber views of sufficient quality to evaluate ventricular filling and function and the pericardium. Ability to distinguish between normal and abnormal cardiac anatomy.
Total number of cases reviewed++	20	Ability to interpret ventricular preload and left ventricular wall motion both global and gross regional (septum, anterior wall inferior wall). Ability to diagnose pericardial effusion and distinguish from pleural effusion. Ability to assess for signs of dynamic left ventricular outflow tract obstruction including LV preload and systolic anterior motion of the anterior leaflet of the MV.

**Achievement of competency under the direct supervision of an experienced advanced echocardiographer is more important than the exact number of examinations and it is possible that more or fewer exams may be required.*
***Performed in the presence of an experienced advanced echocardiographer. +Performed by the trainee, then interpreted and reported by the trainee to an experienced advanced echocardiographer (TTE should be performed in the presence of the supervisor until images are deemed satisfactory), ++this includes personally performed exams as well as cases archived and presented by other examiners but interpreted by the trainee; may include both TEE and TTE exam. LV indicates left ventricle; RV, right ventricle; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography.*

are less important than the depth of the clinical experience, quality of training, and assessment by the supervising advanced echocardiographer. These guidelines also do not specify the duration of training. Experience and the depth of clinical experience determine the time needed to achieve the goals. The trainee must also be taught how to effectively convey, document, and integrate clinically the results of examinations. Finally, all noncardiologist echocardiographers must recognize the limitations of the scope of their exam and obtain a formal complete study to confirm any unusual unexpected finding. Consultation on an emergent or elective basis should occur as appropriate in complex situations, such as suspected endocarditis, aortic dissection, or valvular disease, or in the case of confounding or questionable findings.

References

1. Clinical competence in adult echocardiography. A statement for physicians from the ACP/ACC/AHA Task Force on Clinical Privileges in Cardiology. *Circulation.* 1990; 81: 2032–2035.
2. Shanewise JS, Cheung AT, Aronson S, et al. ASE/SCA guidelines for performing a comprehensive intraoperative multiplane transesophageal echocardiography examination: recommendations of the American Society of Echocardiography Council for Intraoperative Echocardiography and the Society of Cardiovascular Anesthesiologists Task Force for Certification in Perioperative Transesophageal Echocardiography. *Anesth Analg.* 1999;89:870–884.
3. Mayron R, Gaudio FE, Plummer D, Asinger R, Elsperger J. Echocardiography performed by emergency physicians: impact on diagnosis and therapy. *Ann Emerg Med.* 1988; 17:150–154.
4. Mateer J, Plummer D, Heller M, et al. Model curriculum for physician training in emergency ultrasonography. *Ann Emerg Med.* 1994;23:95–102.
5. American College of Emergency Physicians. ACEP emergency ultrasound guidelines-2001. *Ann Emerg Med.* 2001; 38:470–481.
6. American College of Emergency Physicians. Use of ultrasound imaging by emergency physicians. *Ann Emerg Med.* 2001;38:469–470.
7. Cahalan MK, Stewart W, Pearlman A, et al. American Society of Echocardiography and Society of Cardiovascular Anesthesiologists task force guidelines for training in perioperative echocardiography. *J Am Soc Echocardiogr.* 2002;15:647–652.
8. Heller MB, Mandavia D, Tayal VS, Cardenas EE, Lambert MJ, et al. Residency training in emergency ultrasound: fulfilling the mandate. *Acad Emerg Med.* 2002;9:835–839.
9. Hansen RM, Viquerat CE, Matthay MA, et al. Poor correlation between pulmonary arterial wedge pressure and left ventricular end-diastolic volume after coronary artery bypass graft surgery. *Anesthesiology.* 1986;64:764–770.
10. Raper R, Sibbald WJ. Misled by the wedge? The Swan-Ganz catheter and left ventricular preload. *Chest.* 1986;89:427–434.
11. Fontes ML, Bellows W, Ngo L, Mangano DT. Assessment of ventricular function in critically ill patients: limitations of pulmonary artery catheterization. Institutions of the McSPI Research Group. *J Cardiothorac Vasc Anesth.* 1999;13:521–527.
12. Kumar A, Anel R, Bunnell E, et al. Pulmonary artery occlusion pressure and central venous pressure fail to predict ventricular filling volume, cardiac performance, or the response to volume infusion in normal subjects. *Crit Care Med.* 2004;32:691–699.
13. Benjamin E, Griffin K, Leibowitz AB, et al. Goal-directed transesophageal echocardiography performed by intensivists to assess left ventricular function: comparison with pulmonary artery catheterization. *J Cardiothorac Vasc Anesth.* 1998;12:10–15.
14. Manasia AR, Nagaraj HM, Kodali RB, et al. Feasibility and potential clinical utility of goal-directed transthoracic echocardiography performed by noncardiologist intensivists using a small hand-carried device (SonoHeart) in critically ill patients. *J Cardiothorac Vasc Anesth.* 2005;19:155–159.
15. Oh JK, Seward JB, Tajik AJ. *The Echo Manual.* 2nd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 1999.
16. Hu BS, Saltiel F, Popp RL. Effectiveness of a limited training in echocardiography for cardiovascular diagnosis. *Circulation.* 1996;94:I–253 (suppl)
17. Kimura BJ, Pezeshki B, Frack SA, DeMaria AN. Feasibility of "limited" echo imaging: characterization of incidental findings. *J Am Soc Echocardiogr.* 1998;11:746–750.
18. Manasia A, Griffin K, Oropello J, Leibowitz A, DelGuidice R, et al. A comparison of transesophageal echocardiography and the pulmonary artery catheter in critically ill patients. *Chest.* 1994;106:100A.
19. Sohn DW, Shin GJ, Oh JK, Tajik AJ, Click RL, Miller FA Jr, Seward JB. Role of transesophageal echocardiography in hemodynamically unstable patients. *Mayo Clin Proc.* 1995;70:925–931.
20. Daniel WG, Erbel R, Kasper W, et al. Safety of transesophageal echocardiography. A multicenter survey of 10,419 examinations. *Circulation.* 1991;83:817–821.
21. Gorcsan J 3rd, Snow FR, Paulsen W, Nixon JV. Noninvasive estimation of left atrial pressure in patients with congestive heart failure and mitral regurgitation by Doppler echocardiography. *Am Heart J.* 1991;121:858–863.
22. Vasan RS, Levy D, Larson MG, Benjamin EJ. Interpretation of echocardiographic measurements: a call for standardization. *Am Heart J.* 2000;139:412–422.
23. DeCara JM, Lang RM, Spencer KT. The hand-carried echocardiographic device as an aid to the physical examination. *Echocardiography.* 2003;20:477–485.
24. Liang D, Schnittger I. Accuracy of hand-carried ultrasound. *Echocardiography.* 2003;20:487–490.
25. Spencer KT, Goldman M, Cholley B, et al. Multicenter experience using a new prototype transnasal transesophageal echocardiography probe. *Echocardiography.* 1999;16:811–817.
26. Mayo PH, Vieillard-Baron A, et al. ACCP/SRLF Statement on competence in critical care ultrasonography. *Chest.* In press.

CHAPTER 7

Transthoracic Echocardiography: Image Acquisition and Transducer Manipulation

Seth Koenig and Paul H. Mayo

INTRODUCTION

Transthoracic echocardiography (TTE) has major application in the intensive care unit (ICU). Proficiency in TTE allows the intensivist to determine the diagnosis of cardiopulmonary failure, develop management strategies, and follow the results of therapeutic interventions with serial examinations. By definition, critical care echocardiography (CCE) is performed by the intensivist in the ICU. The clinician acquires and interprets the image at the bedside, and uses the information to guide management. It follows that the intensivist must have a high level of skill in image acquisition, which requires a working knowledge of ultrasound physics, machine controls, and transducer manipulation. This chapter will review important elements of image acquisition with emphasis on transducer manipulation. The reader is referred to Chapters 2 and 3 for a comprehensive discussion of physics and machine controls.

BASIC AND ADVANCED CRITICAL CARE ECHOCARDIOGRAPHY

Proficiency in CCE can be separated into basic and advanced levels. Basic CCE is performed as a goal-directed examination using a limited number of views. It is designed to answer very specific clinical questions at the bedside. Proficiency in advanced CCE requires a high level of skill in all aspects of image interpretation and acquisition. Advanced CCE allows a comprehensive evaluation of cardiac anatomy and function using TTE and Doppler echocardiography. Both basic and advanced CCE require skill in image acquisition.

Technical Issues

The performance of TTE has challenges that relate to the fact that the heart is surrounded by lung and ribs, both of which block ultrasound transmission. Since ribs block ultrasound waves, cardiac transducers are designed with a small footprint to scan through the small rib interspace. During scanning, left arm abduction may increase the size of the interspace. Aerated lung also block ultrasound, so that positioning the patient in the left lateral decubitus position may be helpful because in this position the heart is moved from behind the sternum, and the left lung moves laterally, thus exposing more of the heart for examination. While the left lateral decubitus view improves visualization from the parasternal and apical views, the supine position is best for the subcostal examination.

The critically ill patient may be difficult to place in a favorable scanning position. Patients on ventilatory support, particularly when hyperinflated, may have very poor parasternal and apical windows. Very often, the subcostal view yields the only acceptable image. Transthoracic echocardiography image quality may be poor in the edematous or muscular patient. Obesity presents a special challenge for two reasons. It attenuates the penetration of ultrasound. In addition, abdominal obesity elevates the diaphragm, particularly when the patient is supine and when passive on ventilatory support. The heart is then rotated into a more vertical position. This makes it difficult to obtain properly oriented parasternal views. The presence of chest dressings, wounds, or subcutaneous air also degrade TTE image quality. Transesophageal echocardiography (TEE) is always an alternative in the patient who fails TTE. Artifacts in echocardiography relate, in part, to the fact that the heart is a highly mobile organ in constant motion within the thorax. Translational, torsional, and rotational movement of the heart may be misinterpreted as reflecting actual cardiac contractile function.

In addition to these challenges, CCE is performed in a difficult operating environment. The patient is often

surrounded with multiple ICU devices, so that positioning of the machine and operator may be difficult. The light level in the patient room is often too bright for optimal screen displays. The echocardiographer is under pressure to complete the examination rapidly because the patient is critically ill and other patients demand immediate attention. The results of the study frequently require immediate response, so that image acquisition and interpretation must be accurate. The intensivist should always attempt to obtain the best image quality. However, image quality may be limited in the critically ill and below the standards mandated by standard cardiology echocardiography practice. The intensivist must come to terms with this clinical reality while always attempting to obtain the best image quality possible.

Nomenclature

Transthoracic echocardiography examines the heart in tomographic planes obtained by positioning the transducer and "slicing" the heart through different planes. By obtaining multiple views of the heart, the examiner integrates the information to yield a comprehensive evaluation of cardiac anatomy and function. The addition of Doppler analysis yields important information related to cardiac pressures and flows. The American Society of Echocardiography (ASE) has defined the standard tomographic views of the heart.[1] The three standard image planes are as follows:

1. The long-axis plane is parallel to the long axis of the left ventricle (LV). This is defined by a line that goes through the LV apex and the center of the base of the LV intersecting with the center of the aortic valve (AV).
2. The short-axis plane is perpendicular to the long-axis plane.
3. The four-chamber plane is perpendicular to both the short- and long-axis views. This is defined by a plane that goes through the LV apex and intersects the LV and right ventricle (RV) and atria.

The various tomographic planes of TTE are characterized by the position of the transducer required to obtain the image (the window) and the resulting image plane (the view). Transducer manipulation occurs as follows:

1. *Move:* The transducer is shifted to a different position on the thorax.
2. *Tilt:* The transducer is tilted or rocked along the same tomographic plane without moving it.
3. *Angle:* Without moving the transducer, angulation is changed to obtain adjacent tomographic planes.
4. *Rotate:* The transducer is rotated without moving, tilting, or angling it in order to obtain orthogonal tomographic planes.

Image orientation is standardized for adult TTE. The transducer position is projected at the top of the screen. The image orientation marker is set to the upper right of the screen. In the long-axis view, superior or cephalad cardiac structures project to the right of the screen. In the short-axis view, left-sided cardiac structures project to the right side of the screen. This is reverse to the orientation used for abdominal, thoracic, and vascular ultrasonography.

The basic or goal-directed CCE examination generally includes five views without major Doppler analysis, while the advanced CCE examination includes a minimum of 11 views of the heart with comprehensive Doppler measurements. At each view, the examiner may choose to obtain one or more tomographic planes by tilting and angling the transducer. There is no officially sanctioned sequence for image acquisition. However, regardless of the sequence used, the intensivist should use a methodical approach to image acquisition for the initial examination. The examination should always be performed in standard sequence. This reduces the likelihood that views will be omitted. Typically, CCE uses sequential follow-up examinations of the heart to check for response to therapy, progression or regression of disease, and new problems. Follow-up examinations may be very limited. Certain situations do not permit any but the briefest examination. For example, echocardiography performed during cardiopulmonary arrest may include only several seconds of a subcostal view during a pulse check.

THE TTE EXAMINATION

What follows is a description of transducer use required to obtain the standard views of TTE. The discussion does not include a detailed review of Doppler measurements. For each view, the specific clinical utility for the intensivist with proficiency in advanced CCE is mentioned. For the four views that are key elements of the basic CCE examination, some common pitfalls are highlighted, which may be helpful to the inexperienced echocardiographer.

Parasternal Long-Axis View

The transducer is placed in the 3rd or 4th intercostal space adjacent to the sternum, with the transducer

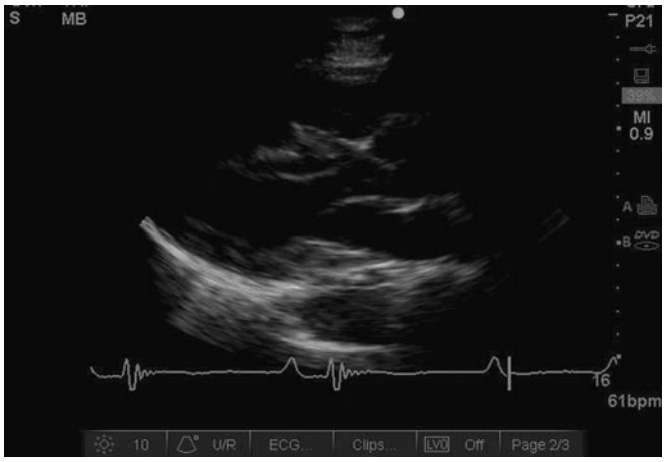


Figure 7.1. Parasternal long-axis view (basic CCE view).

mark pointing to the patient's right shoulder. Movement of the transducer either caudad or cephalad brings the parasternal long axis into view. If adequate image quality is not obtained, positioning the patient toward the left lateral decubitus position may improve image quality. Left-arm abduction may open the intercostal spaces enough to reveal a better acoustic window. The examiner should not accept an off-axis view simply because an image of the heart appears. As with orientating a camera lens, the transducer should be moved around to obtain the best tomographic view. With minor movement and angulation, the examiner seeks a view that bisects the mitral valve (MV) and aortic valve (AV) and includes the LV cavity in longest axis (Figures 7.1 and 7.2, and Videos 7.1 and 7.2 in enclosed DVD). The image should be orientated so that the aorta is displayed on the right, with the LV cavity on the left of the screen. The RV outflow track and chest wall appear at the top of the screen. Posterior structures, such as the left atrium and pericardium, appear at the bottom of the screen. While the perfect long-axis view displays the heart horizontally, technical limitations such as patient positioning, body habitus, mechanical ventilation, and examiner inexperience may yield a more vertical view of the heart. These limitations must be accepted and interpreted as appropriate to the clinical situation.

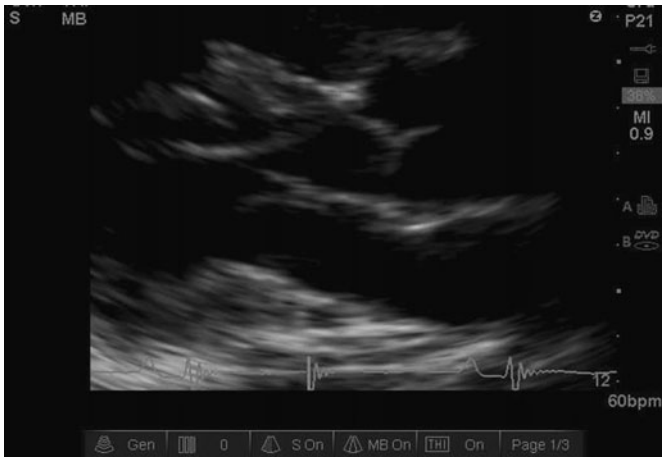


Figure 7.2. Parasternal long-axis view of the aortic valve and mitral valve.

The parasternal long-axis view visualizes both the aortic root and the right and noncoronary leaflets of the AV. The anterior and posterior mitral valve leaflets are also viewed well. Once an acceptable image is obtained, the transducer may be tilted to view the ascending aorta or more of the LV cavity. Minimal angulation allows assessment of the medial and lateral parts of the mitral apparatus.

Clinical utility: Assessment of ejection fraction (EF), RV/LV wall thickness, LV segmental wall function, RV/LV/left atrial (LA) chamber size and function, evaluation of AV/MV anatomy and function, descending aorta, pericardial space, coronary sinus size, M-mode measurements, and colored Doppler interrogation of the AV and MV.

The parasternal long-axis view is a standard view for basic CCE. It allows assessment for pericardial effusion, LV/RV size and function, and septal kinetics. For the basic CCE echocardiographer, the pitfalls of this view include the following:

1. Inaccurate assessment of RV size. The RV size may be underestimated; the apical four-chamber and subcostal views are favored for assessment of RV size. This is because the parasternal long axis affords a view of the right ventricular outflow track predominately.
2. Inaccurate assessment of LV size and function. Off-axis views of the LV due to rotation or angulation may lead to erroneous assessment of LV size and function. If the initial view places the AV in the center of the screen, the LV cavity may be better visualized by moving or tilting the transducer to include the LV cavity.
3. Inaccurate assessment of MV and AV function. The MV and AV may appear to be anatomically normal on 2D view, but can have substantial degrees of regurgitation discernable only with colored or spectral Doppler analysis. Proficiency in basic CCE does not allow the examiner to reliably exclude severe valvular regurgitation. Colored Doppler has limitations not intuitively obvious to the inexperienced examiner. These include gain settings ("dial a jet"), wall jet effect (Coanda effect), angle effect (both of

transducer and by Doppler interrogation angle relative to the jet), and shadowing by surrounding structures such as a prosthetic valve apparatus or a calcified annulus.

Right Ventricular Inflow and Outflow Long-Axis Views

From the parasternal long-axis view, the transducer is angled medially. For the novice sonographer, this is accomplished by angling the top of the transducer toward the patient's left shoulder without lifting it off the patient's chest. This shifts the tomographic view to a position that will display the RV and atrium. One may need to move the transducer slightly superiorly or inferiorly, keeping the transducer mark toward the left shoulder. This results in a view of the right atrium (RA), tricuspid valve (TV), and RV. The anterior and septal leaflets of the TV are visible (Figure 7.3, and Video 7.3 in enclosed DVD). Care must be taken to start with a good or "on axis" parasternal long-axis view in order to visualize the right ventricular inflow and outflow views. To obtain the latter, the sonographer can move the transducer slightly medially toward the sternum, while tilting toward the base of the heart and angling the top of the transducer toward the patient's right hip. This results in a long-axis view of the RV outflow tract, the pulmonic valve (PV), and the pulmonary artery (PA). These views are not part of the basic CCE examination.

Clinical utility: Evaluation of TV/PV anatomy and function and RA/RV anatomy; and colored/spectral Doppler analysis (TV/PV regurgitation, cardiac pressures, assessment for intracardiac shunt, and measurement of shunt fraction).

Parasternal Short-Axis Views

From the parasternal long-axis view, the transducer is rotated 90° clockwise without angulation or tilting. This results in cross-sectional views of the heart. While this seems to be an easy assignment, the learner may be frustrated by unintended migration of the transducer. A good short-axis view follows from a good long-axis view. It is important to concentrate on acquisition of the "best" long-axis view before the transducer is rotated. Rotation of the transducer may be achieved using a two-handed approach: Keeping the transducer hand steady while rotating with the other hand will give the best results. The transducer is rotated until the short axis of the heart is obtained. The transducer marker will be positioned at the 1–2 o'clock position, facing the patients left shoulder. The cross-sectional view obtained by rotation will depend upon the pre-rotation parasternal long view and any unintentional angulation. By angling the transducer along a right-shoulder-to-left-hip axis, multiple tomographic views of the heart may be obtained. By angling toward the base of the heart, the aortic level (Figure 7.4, and Video 7.4 in enclosed DVD) comes into view. This short-axis view results in a cross-section of the AV. Medial tilting shows the TV, while lateral tilting and superior angling of the transducer permits visualization of the PV and proximal PA (Figure 7.5, and Video 7.5 in enclosed DVD). Angling the transducer inferiorly results in a cross-sectional view of the anterior and posterior leaflets of the MV (Figure 7.6, and Video 7.6 in enclosed DVD). Further inferior angulation of the transducer results in a cross-section of the LV at the level of the anterolateral and posteromedial papillary muscles (Figure 7.7, and Video 7.7 in enclosed DVD). One should attempt to

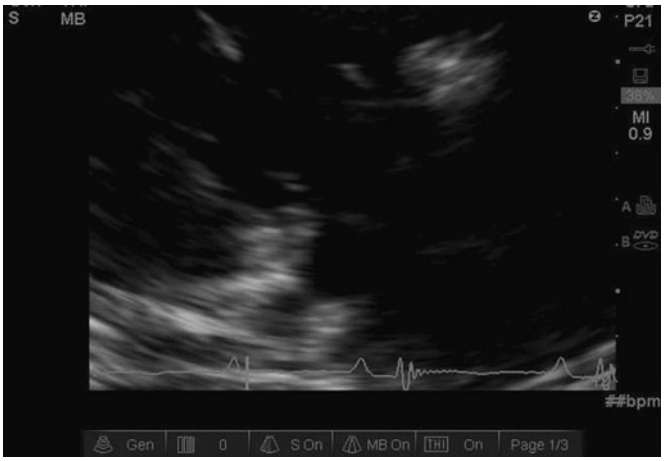


Figure 7.3. Parasternal long-axis view of the tricuspid valve inflow.

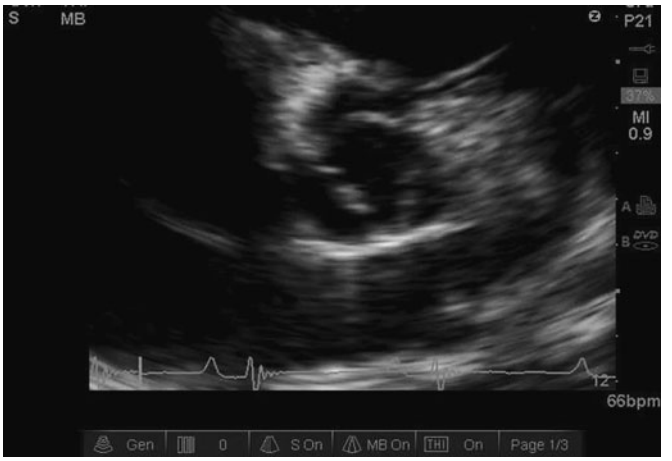


Figure 7.4. Parasternal short-axis view of the aortic valve.

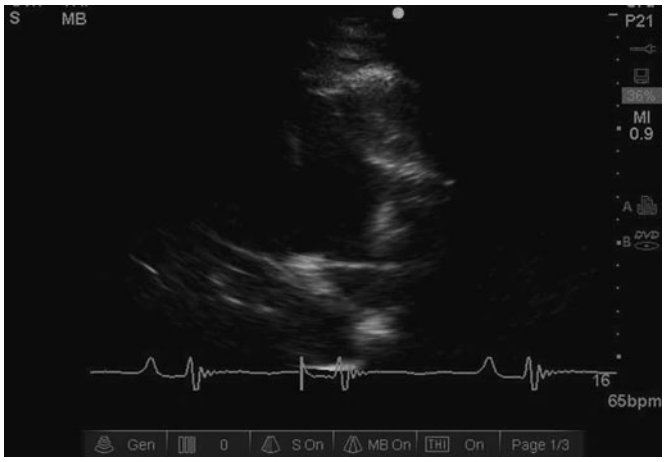


Figure 7.5. Parasternal short-axis view of the pulmonary artery.

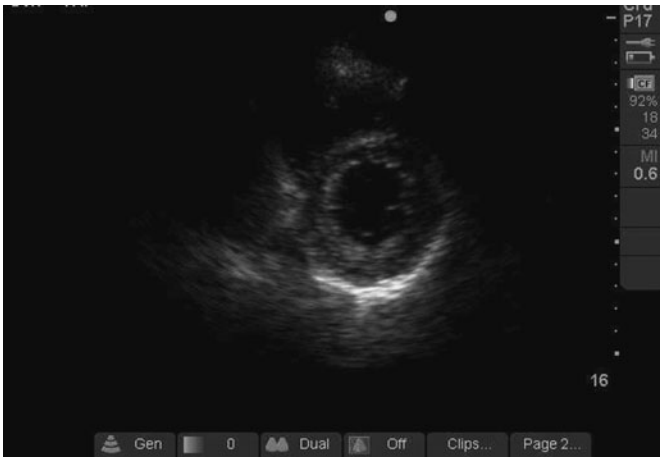


Figure 7.7. Parasternal short-axis view of the left ventricle (basic CCE view).

position the LV cavity in the center of the screen, which may require moving or tilting the transducer.

Clinical utility: Assessment of EF, RV/LV wall thickness, LV segmental wall function, and RV/LV chamber size and function; evaluation of AV/TV/PV/MV anatomy and function; and colored/spectral Doppler analysis (AV/TV/PV/MV regurgitation, cardiac pressures, intratrial shunt).

The parasternal short-axis view at the midventricular level is a standard view for basic CCE. It allows assessment for pericardial effusion, LV/RV size and function, and septal kinetics. For the basic CCE echocardiographer, the pitfalls of this view include the following:

1. Inaccurate assessment of LV configuration. The normal LV should be circular in short axis. An elliptical appearance results from an off-axis view related to a non-perpendicular tomographic plane. An off-axis view may result in inaccurate diagnosis of segmental wall contraction abnormality or septal flattening ("D"-shaped heart). The supine position, ventilatory support with lack of diaphragmatic movement, obesity, and elevation of intraabdominal pressures may all cause the heart to be rotated such that the long-axis view tends to assume a more vertical position on the screen in the critically ill. This results in an off-axis view of the LV in the transverse scanning plane. This cannot be corrected by transducer manipulation. An alternative method of obtaining a short-axis view of the LV is to use the subcostal approach.
2. Inability to visualize the RV free wall. Estimates of RV size require visualization of the RV wall, which may be difficult in the parasternal short-axis view of the LV. The apical four-chamber and subcostal views are superior for assessment of RV size and function.

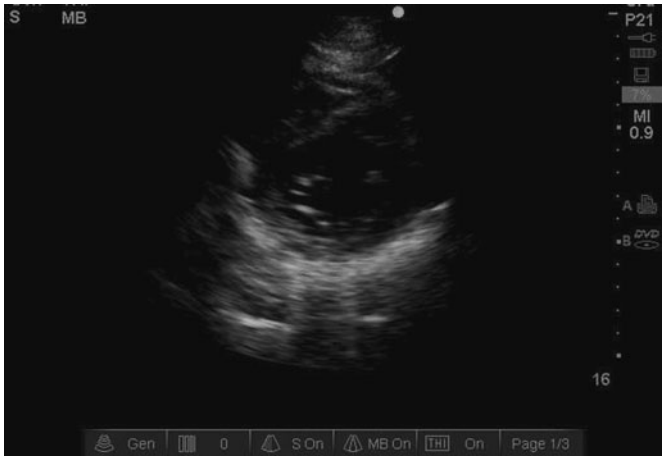


Figure 7.6. Parasternal short-axis view of the mitral valve.

Four-Chamber View and Variants

Apical Four-Chamber View

The transducer is placed at the anatomic apex of the LV with the tomographic plane bisecting the ventricles and atria (Figure 7.8, and Video 7.8 in enclosed DVD). Patient position may have to be optimized. This may be difficult in the critically ill patient, so an assessment of the view before patient repositioning may be prudent. The left lateral decubitus position is best. It may be helpful to move the patient's left arm away from the chest wall. The transducer marker should be facing the patient's left shoulder at the 3–4 o'clock position. A good starting position to place the transducer is just lateral and inferior to the nipple. One may need to move the transducer in different directions, i.e., up or down or side to side, until an adequate window is found.

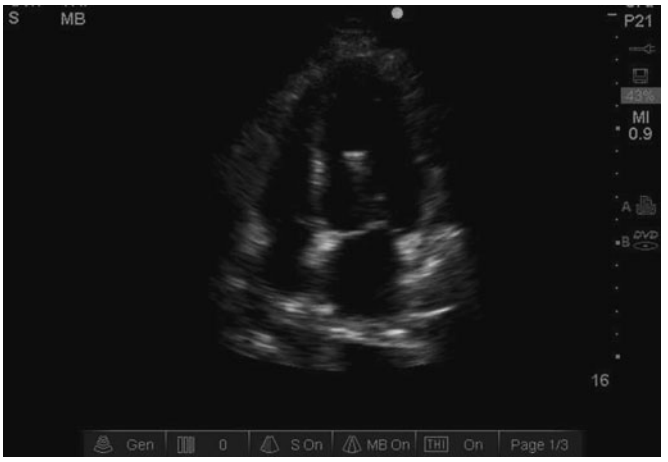


Figure 7.8. Apical four-chamber view (basic CCE view).

Clinical utility: Assessment of EF; measurement of EF/stroke volume (SV) by Simpson's method, LV/RV wall thickness, LV segmental wall function, LV/RV chamber size and function, and LA /RA size; evaluation of MV/TV anatomy and function; and colored/spectral Doppler analysis (MV/TV regurgitation/stenosis, cardiac pressures, and diastolic function).

The parasternal apical four-chamber view is a standard view for basic CCE. It allows assessment of LV/RV size and function, septal kinetics, and pericardial effusion. It is particularly important for identifying RV enlargement by the RV/LV ratio method. For the basic CCE echocardiographer, the pitfalls of this view include the following:

1. Off-axis image. The apical four-chamber view is the most difficult for the basic-level echocardiographer to obtain. It is best achieved in a steep lateral decubitus position, which is often not possible in the critically ill patient. Lung may block the view, particularly if the patient is on ventilatory support, and cycling of the ventilator may yield intermittent imaging as well as translational artifact. The window may be small and the transducer manipulation challenging in terms of angulation and tilting. The inexperienced scanner may easily accept a view that shows all four chambers but is off axis. This can result in the inaccurate assessment of LV/RV size and function.
2. Inaccurate RV/LV size ratio. This ratio is a critical parameter of basic CCE. Off-axis view, inability to visualize the RV free wall, and counterclockwise transducer rotation may result in underestimation of RV size. In the latter case, counterclockwise rotation will cause the RV to disappear completely. The subcostal view is the best alternative approach in the case of suboptimal image quality in the apical four-chamber view position.

Apical Five-Chamber View

From the apical four-chamber view, the transducer is angled anteriorly to obtain an image of the left ventricular outflow tract and AV. This view is not usually part of the basic CCE examination.

Clinical utility: Measurement of SV, determination of preload sensitivity by dynamic indices (see Chapter 10), and colored/spectral Doppler analysis of the AV and left ventricular outflow tract (LVOT).

Apical Two-Chamber View

From the apical four-chamber view position, the transducer is rotated counterclockwise 60° without movement, angulation, or tilting to obtain a view of the LV and LA (Figure 7.9, and Video 7.9 in enclosed DVD). This view is not usually part of the basic CCE examination.

Clinical utility: Assessment of EF; measurement of EF/SV by Simpson's method, LV wall thickness, LV segmental wall function, LV chamber size and function, and LA size; evaluation of MV anatomy and function; and colored/spectral Doppler analysis (MV regurgitation/stenosis, cardiac pressures, and diastolic function).

Apical Three-Chamber View

From the apical two-chamber view, the transducer is rotated counterclockwise 60° without movement, angulation, or tilting to obtain a view of the LV, LA, and

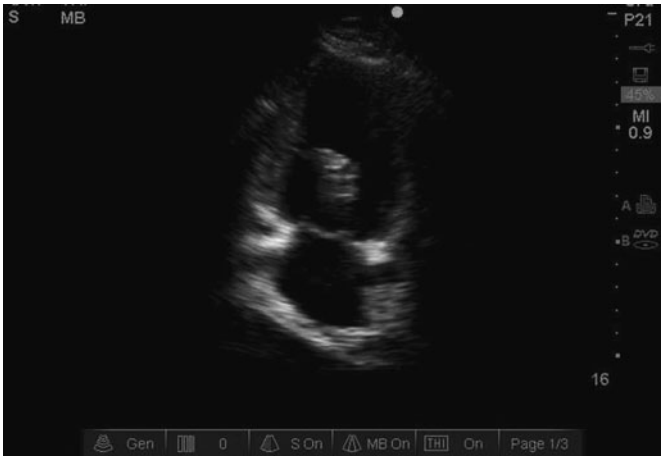


Figure 7.9. Apical two-chamber view.

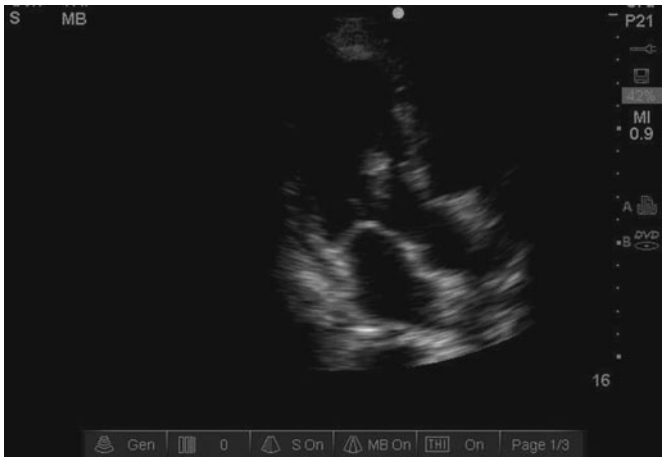


Figure 7.10. Apical three-chamber view.

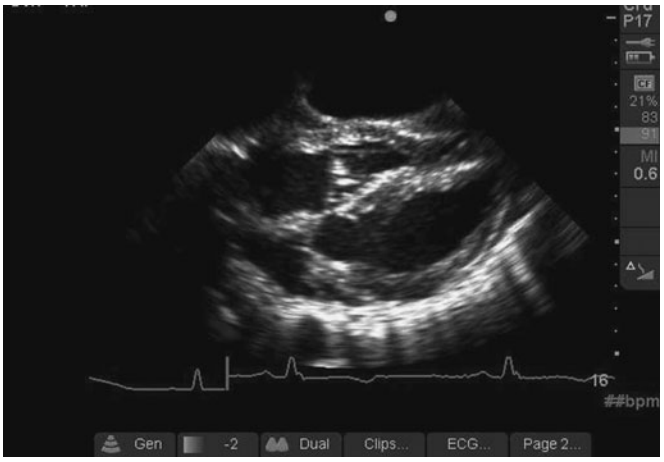


Figure 7.11. Subcostal long-axis view (basic CCE view).

RV (Figure 7.10, and Video 7.10 in enclosed DVD). The view is similar to the parasternal long-axis view. This view is not usually part of the basic CCE examination.

Clinical utility: Assessment of EF; measurement of SV; determination of preload sensitivity by dynamic indices (see Chapter 10), LV wall thickness, LV segmental wall function, LV chamber size and function, and LA size; evaluation of MV anatomy and function; and colored/spectral Doppler analysis (MV regurgitation/stenosis, cardiac pressures, and diastolic function).

Subcostal Four-Chamber View

This view is best obtained with the patient lying supine. The transducer is placed just below the xiphoid process, pointing toward the left shoulder with the index mark pointing to the left. This view requires the transducer to be held on its top surface, as some or most of its bottom surface will be contacting the patient. This yields a four-chamber view, with the tomographic plane sectioning the heart from the right side through to the left (Figure 7.11, and Video 7.11 in enclosed DVD).

Clinical utility: Assessment of EF; measurement of LV/RV wall thickness, LV segmental wall function, LV/RV chamber size and function, LA /RA size, and intraatrial septal anatomy and function; evaluation of MV/TV anatomy and function; and colored/spectral Doppler analysis (TV regurgitation).

The subcostal view is a standard part of basic CCE. It allows assessment of LV/RV size and function, septal kinetics, and pericardial effusion. It is particularly important for identifying RV enlargement by RV/LV ratio method. For the basic CCE echocardiographer, the pitfalls of this view include the following:

Off-axis view: The subcostal view is often the best quality view in the critically ill, particularly in patients on mechanical ventilatory support who are hyperinflated. It is also the preferred view in cardiac arrest. The examiner should scan perpendicular to the RV free wall and adjust the view so as to obtain the largest RV size.

Subcostal Short-Axis View

From the subcostal long axis, the transducer is rotated counterclockwise 90° without movement, angulation, or tilting to obtain a cross-section of the LV similar to that obtained with the parasternal short-axis midventricular view (Figure 7.12, and Video 7.12 in enclosed DVD). The transducer is then angled medially to examine the AV. Slight counterclockwise rotation then yields a long-axis view of the RV outflow tract, PV, and main

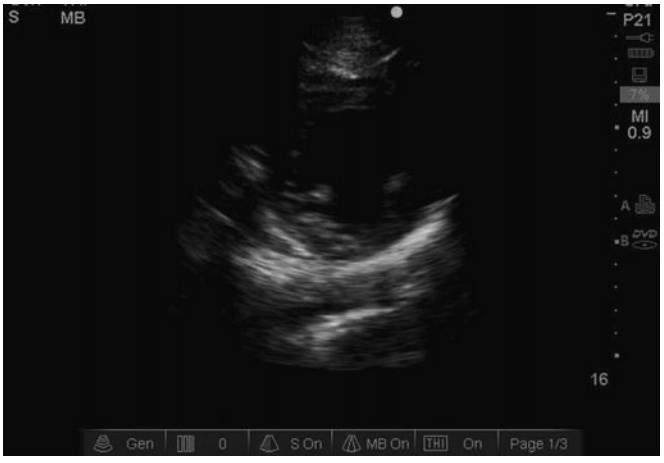


Figure 7.12. Subcostal short-axis view (basic CCE view).

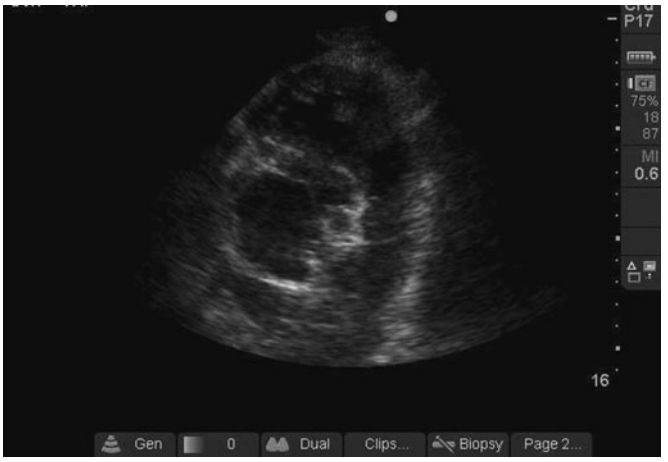


Figure 7.13. Subcostal pulmonary artery view.

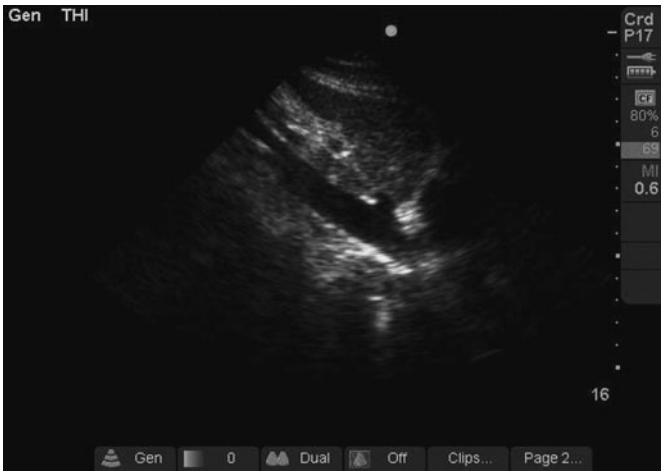


Figure 7.14. Inferior vena cava view (basic CCE view).

pulmonary artery to its bifurcation (Figure 7.13, and Video 7.13 in enclosed DVD).

Clinical utility: Assessment of EF; measurement of RV/LV wall thickness, LV segmental wall function, and RV/LV chamber size and function; evaluation of AV/TV/PV/MV anatomy and function and pulmonary artery anatomy; and colored/spectral Doppler analysis of AV/TV/PV/MV.

The subcostal view of the LV in short axis is a standard part of basic CCE. It allows assessment of LV/RV size and function, septal kinetics, and pericardial effusion. The pitfalls of this view include the following:

Off-axis view: The subcostal view is often the best quality view in the critically ill, particularly in patients on mechanical ventilatory support who are hyperinflated. It is also the preferred view in cardiac arrest. The transducer should be orientated such that the LV cavity is circular. An oval LV suggests an off-axis view.

Inferior Vena Cava (IVC) View

From the subcostal short-axis view, the transducer is tilted inferiorly and angled medially to obtain a long-axis view of the IVC (Figure 7.14, and Video 7.14 in enclosed DVD).

Clinical utility: Determination of preload sensitivity, assessment of right atrial pressure, and spectral/Doppler analysis of the hepatic veins.

The IVC view is a standard part of basic CCE. It allows determination of preload sensitivity. The pitfalls of this view include the following:

1. Misidentification of the aorta for the IVC. This can be avoided by careful attention to angulation. The aorta is to the left of the midline, while the IVC is to the right of the midline.
2. Off-axis view: Determination of preload sensitivity requires an accurate measurement of IVC diameter. The scanning plane must be orientated along the midline of the IVC and along its longitudinal axis in order to assure accurate diameter measurement.
3. Translational artifact: Preload sensitivity is determined by IVC diameter change when the patient is on ventilatory support without spontaneous respiratory effort. When the ventilator cycles, the liver is displaced by diaphragmatic movement. This may move the IVC out of the initial scanning plane, giving the impression of diameter change that actually is a translational artifact.

Suprasternal and Supraclavicular Views

The transducer is placed in the suprasternal or supraclavicular area with the scanning plane directed toward the heart and great vessels. The transducer may be manipulated in order to obtain the axis appropriate to the study question. These views are not part of the basic CCE examination.

Clinical utility: Evaluation of aorta anatomy and colored/spectral Doppler analysis of aortic and superior vena cava flow.

CONCLUSION

Competence in basic and advanced CCE requires skill in image acquisition, as the frontline intensivist personally performs and interprets the echocardiogram at the bedside of the critically ill patient. Proficiency at

transducer manipulation is therefore a mandatory part of proficiency in CCE. This Chapter reviews transducer manipulation and serves as a guide for the intensivist interested in developing skill at CCE.

Reference

1. Henry WL, DeMaria A, Gramiak R, et al. Report of the American Society of Echocardiography Committee on Nomenclature and Standards in Two-dimensional Echocardiography. *Circulation.* 1980;62:212–217.

This page intentionally left blank

CHAPTER 8

Transesophageal Echocardiography: Image Acquisition and Transducer Manipulation

Pierre Kory and Paul H. Mayo

INTRODUCTION

The value of performing transesophageal echocardiography (TEE) in intensive care unit (ICU) patients is well established. Although transthoracic echocardiography (TTE) is an excellent diagnostic tool in the ICU, TEE has superior diagnostic accuracy and therapeutic impact in several clinical situations, particularly for patients in shock states.[1–3] Several authors have demonstrated that TEE findings lead to major therapeutic decisions between 43% and 68% of the time.[1,4–6] Transesophageal echocardiography produces superior image quality due to the position of the probe proximate to the heart, allowing for the use of higher frequency ultrasound with superior resolution of cardiac structures. Although improvements in imaging, software, and portable systems have reduced the rates of inadequate image quality seen with TTE, there remain a significant percentage of patients in the ICU whose image quality with TTE is inadequate. Many factors can account for this including inadequate patient positioning, lung hyperinflation, obesity, edema, and the presence of chest devices, wounds, and dressings. Transthoracic echocardiography results in adequate image quality in approximately 55% of mechanically ventilated ICU patients, with the remaining 23% and 22% of studies being of suboptimal and poor quality, respectively.[3] In addition to overcoming poor image quality of TTE, TEE is often necessary for the evaluation of specific diagnoses in the ICU such as endocarditis, embolic sources, intracardiac shunt, aortic dissection, and loculated pericardial effusion. For hemodynamic assessment, TEE is the only method to assess superior vena cava (SVC) variation, a predictor of volume responsiveness.[7] When compared with helical computed tomography (CT), TEE has similar sensitivity and specificity for suspected central pulmonary embolism (PE) associated with right ventricular dilatation.[8,9]

Many factors have combined to increase the use of TEE in the ICU.[10]

1. The increasing use of ultrasound and echocardiography in the ICU has made intensivist skills in these modalities more prevalent.
2. The presence of ultrasound units dedicated to the ICU has reduced the acquisition cost of TEE.
3. The perspective of TEE as a risky and invasive procedure requiring sedation with a risk of airway compromise is removed in mechanically ventilated and sedated ICU patients. Transesophageal echocardiography in ICU patients has had consistently low complication rates: 0% among three studies totaling 304 patients[1,4,6] with two additional trials reporting rates of 1.6% and 2.6%.[2,5] The most common complication is sedative-induced hypotension requiring additional vasopressor use.
4. Limited or goal-directed TEE examination is of significant value, yielding important information in a short period of time, with several authors reporting exam times of $<$15 minutes.[4,11]
5. Skill in performance and interpretation of limited TEE examination can be acquired by intensivists after as few as eight to 10 supervised TEE exams and one didactic session.[4]

In this chapter, an overview of the technical and performance aspects of TEE including discussion of the goal-directed TEE exam will be discussed.

PREPARATION AND PATIENT SELECTION

Transesophageal echocardiography is a minimally invasive and safe procedure with few but definite risks.[12] Complications are rare, especially in the ICU (see

above) and include esophageal abrasion, perforation, and bleeding. These can be avoided by proper patient selection and by minimizing rotational movement of the endoscope tip while under flexion (see below Transducer Manipulation). Transesophageal echocardiography is contraindicated in the setting of esophageal varices, strictures, bleeding, recent surgery, tumor, or diverticuli. It is mandatory that a complete history be obtained that addresses the risk of esophageal injury during TEE. If such conditions are present, a barium swallow or endoscopic evaluation of the esophagus is recommended prior to the procedure.[13,14] Risks associated with sedation such as airway compromise and transient hypotension are similar to those of other endoscopic procedures.

The TEE approach in the ICU is dependent on whether the patient is already mechanically ventilated and sedated. For such patients, the majority of preparatory tasks have already been completed and the procedure avoids most of the risk, time, and materials needed. In the following section, we describe our approach to two categories of patients in the ICU: the patient who is not on ventilatory support and the patient who is endotracheally intubated and receiving mechanical ventilation.

Nonintubated Patients

Patients are kept fasting for a period of six hours, intravenous access is established, and cardiac and oxygen saturation monitoring is begun. An appropriate history defining risks of esophageal injury and medication allergies is obtained. Risks of the procedure are explained and consent is obtained. The mouth is examined for dentures, loose teeth, and lesions, and all hardware is removed. Local anesthesia of the oropharynx is applied by sprays, nebulizers, or direct "painting" of the posterior tongue and throat with viscous lidocaine. A rare complication of local anesthesia is methemoglobinemia.

Before esophageal intubation, the TEE probe is inspected for defects in the waterproof covering. Patients are placed in a left lateral decubitus position, with the head of the bed elevated 30° to prevent aspiration during the procedure. Supplemental oxygen is provided. The neck is gently flexed while the endoscope is inserted orally in the midline, keeping the face of the transducer positioned anteriorly. With the probe at the esophageal inlet, the patient is asked to swallow while the operator simultaneously advances the probe. Advancement should continue unless resistance is met. The probe is kept in neutral position during any advancement or withdrawal. Gagging often occurs until the probe is advanced beyond the carina or >25 cm beyond the teeth.[13] The procedure is performed with conscious sedation using appropriate doses of intravenous agents. Narcotic and/or sedative agents often work well for this procedure.

Endotracheally Intubated Patients

Since mechanically ventilated patients have a secure airway that facilitates intubation, they require much less airway preparation. Our approach is to augment intravenous sedation so that the patient is unconscious during the TEE exam. Topical anesthesia is not required. In other respects, the assessment of patient risk and monitoring is similar to that described for the unintubated patient.

In mechanically ventilated patients, we recommend using a standard intubating laryngoscope to insert the TEE probe into the esophagus under direct vision. If resistance is met at any point during intubation or the performance of the study, force should never be applied to advance the endoscope. Alternatively, the TEE probe may be inserted "blindly." This may be facilitated by neck flexion, jaw thrust, and ensuring a midline insertion while avoiding rotation of the probe.

Equipment

Modern TEE equipment utilizes a multicrystal, phased-array transducer placed at the tip of a flexible endoscope. The probe can be advanced within the esophagus, and positioned directly posterior to the heart, with excellent resolution of cardiac structures.

Historically, TEE probes were equipped with a monoplane transducer that could provide only a single-plane view of the heart. Typically, this was a transverse view from behind the left atrium, similar to the TTE apical four-chamber view but with the apex in the far field of the TEE image. The development of multiplane probes allows the transducer to be rotated or "spun" to any position between 0° and 180°. This provides multiple views of the heart when combined with movements of the endoscope such as advancement, turning, or flexion.

An advantage of the proximity of the TEE probe to the heart is that it enables the use of frequencies between 5 MHz and 7.5 MHz. This results in excellent resolution of posterior cardiac structures when compared with TTE. However, anterior cardiac structures may require reduction of the ultrasound frequency for better penetration, at the expense of reduced resolution.

We typically start with 7.5 MHz frequency, and adjust when needed to view structures that are further from the transducer.

TRANSDUCER MANIPULATION

The American Society of Echocardiography (ASE) has described 20 standard TEE views of the heart[12] that are obtained by operator manipulation of the probe. Each view requires a specific position and orientation of the transducer with respect to the heart, achieved through the following maneuvers (see Figures 8.1–8.3):

1. Advancement/withdrawal of probe: accomplished by changing the distance of the transducer from the mouth (distances noted on shaft of endoscope).
2. Flexion of probe from the neutral position in four directions: accomplished by rotation of the control knobs on the shaft of the endoscope. Movement of the large knob results in anteflexion of the probe face (pointing or facing of the probe in an anterior/superior-directed view) or retroflexion of the probe face (pointing or facing of the probe in an inferior/posterior-directed view). Rotation of the small knob on the shaft of the endoscope results in right and left flexion of the probe face.
3. Turning of the probe to the right or left side: accomplished by twisting the shaft in a counterclockwise (left side of patient) or clockwise motion (right side of patient).
4. Rotation: accomplished by changing the plane of orientation of the crystal *within* the probe. This is a confusing aspect of TEE for the beginner. If the operator is standing to the left of the patient and looking down at the patient, the face of the transducer will be facing anteriorly, i.e., toward the operator. The

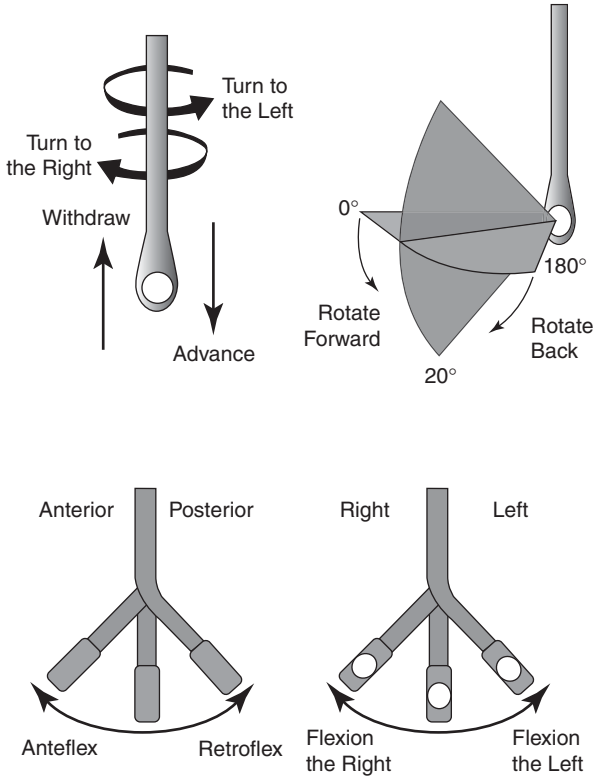


Figure 8.1. Probe manipulation and view orientation. Terminology used to describe manipulation of the probe and transducer during image acquisition. (From Ref. 12.)

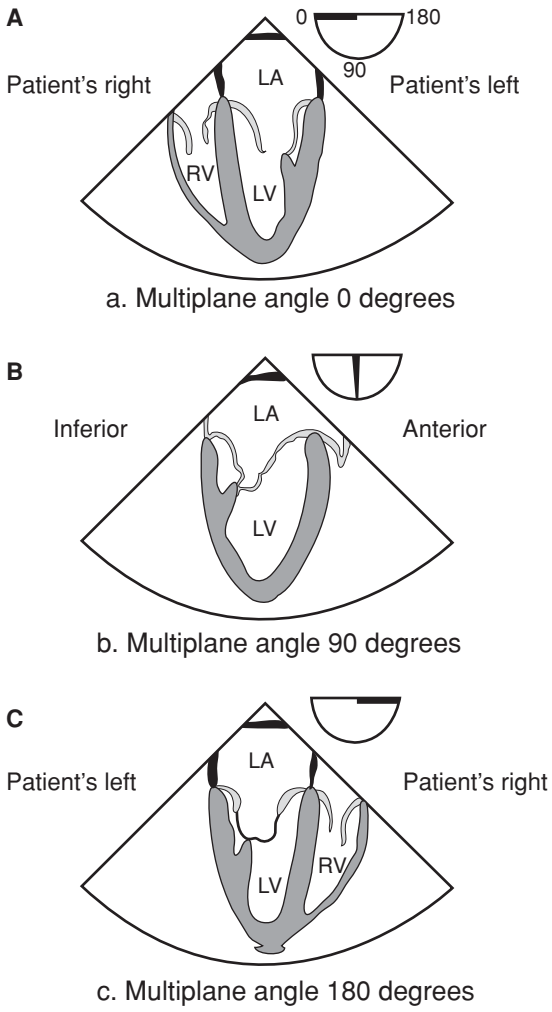


Figure 8.2. Probe manipulation and view orientation. Conventions of image display followed in the guidelines. Transducer location and the near field (vertex) of the image sector are at the top of the display screen and far field at the bottom.
(A) Image orientation at multiplane angle 0°.
(B) Image orientation at multiplane angle 90°.
(C) Image orientation at multiplane angle of 180°. LA indicates left atrium; LV, left ventricle; RV, right ventricle. (From Ref. 12.)

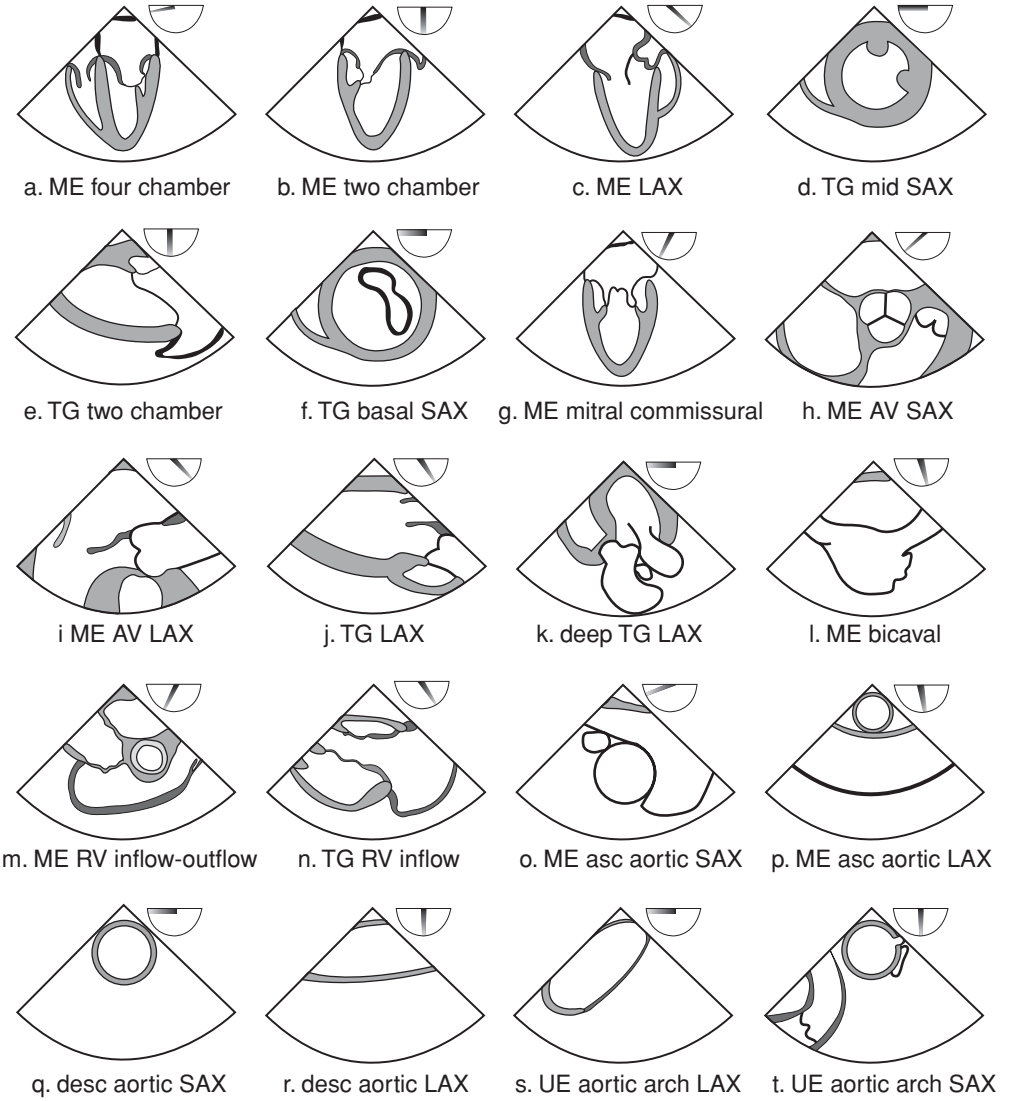


Figure 8.3. Standard TEE views. 20 cross-sectional views composing the recommended comprehensive transesophageal echocardiographic examination. Approximate multiplane angle is indicated by the icon adjacent to each view. AV indicates aortic valve; asc, ascending; desc, descending; LAX, long axis; ME, midesophageal; RV, right ventricle; SAX, short axis; TG, transgastric; UE, upper esophageal. (From Ref. 12.)

0° position represents the patient's right side. Rotation of the transducer occurs in a counterclockwise fashion via an electronic switch that allows for 1° incremental changes. The exact orientation of the transducer is represented by the angle indicator on the screen with values between 0° and 180°.

ORIENTATION OF TEE VIEWS

Knowledge of the standard TEE view orientation relies on two main principles:

1. The ultrasound beam always originates from behind or underneath the heart. The top or "apex" of the screen always displays structures closest to the esophagus, i.e., the atria and great vessels when the endoscope is in a neutral position within the esophagus, and the inferior wall of the heart when anteflexed from within the stomach. Structures in the far field of the screen typically represent anterior structures. The one exception to this convention is the deep gastric apical four-chamber view, which results in the apex of the heart being projected at the top of the screen.
2. The orientation is described by the degree rotation of the ultrasound beam plane. For example, 0° pertains to the transverse plane, with the leftmost part

of the screen pertaining to the rightmost part of the patient (similar to the orientation of a chest x-ray). Increasing the degree rotation corresponds to a counterclockwise rotation of the scan plane; thus, a 90° view results in a longitudinal view, with superior structures to the right of the screen and inferior structures to the left of the screen.

Additionally, operators should be familiar with the degree orientation of the four *primary* TEE views: (1) 0°: transverse plane; (2) 45°: short-axis view of aortic valve; (3) 90°: oblique, long-axis view; and (4) 135°: "true" long-axis view. These four primary views, when combined with turning and flexion, can produce all 20 of the standard ASE views.

THE COMPLETE TEE EXAMINATION

The complete TEE exam as defined by the ASE consists of 20 standard views (Figure 8.1). Attainment of all views requires a significant time commitment. An alternative approach is to perform a goal-directed or limited TEE that focuses on views that answer a specific clinical question. Cardiology-trained echocardiographers generally perform a complete TEE examination with full assessment of all cardiac structures for two reasons: (1) to avoid missing a diagnosis and (2) to reduce the necessity for a repeat examination. Time constraints of the intensivist may make this approach difficult to accomplish. In addition, the ease of performance in an intubated patient makes repeated examination straightforward. The intensivist may also identify very specific indications for TEE that do not require a comprehensive examination (e.g., determination of preload sensitivity, identification of acute cor pulmonale pattern).

We support the value of goal-directed TEE in the critically ill. However, we often perform a complete TEE examination for two reasons: (1) to thoroughly assess for relevant cardiac pathology and (2) to train ICU fellows in the performance of a comprehensive TEE examination. This is an important goal for a teaching service. Once an intensivist is fully trained in TEE examination, goal-directed or limited examination can be performed by this individual. In the following sections, we first describe the sequence and planes of view that make up a complete TEE examination. This is followed by description of a goal-directed or limited examination.

TEE VIEWS

The sequence of TEE image acquisition varies between institution and practitioner. There is no "correct" sequence. However, common to the comprehensive TEE examination is a need for a systematic approach to image acquisition. We describe a specific sequence to perform a comprehensive TEE examination. It results in a stepwise anatomic evaluation of all cardiac structures. There are other sequences that provide identical information. The important principle remains to perform the examination in the same way every time. For documentation and review purposes, three cardiac cycles are captured in a digital format for each view.

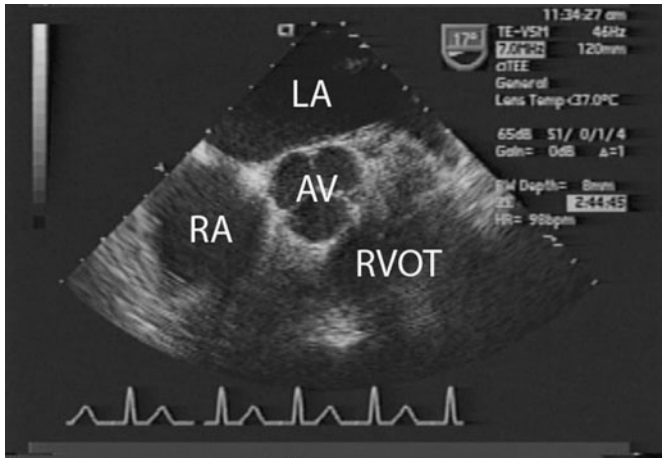


Figure 8.4. Aortic valve short axis view. With the AV in the center of the image, the coaptation point of the AV cusps can be clearly seen. Note the posteriorly positioned LA, the right-sided RA with the RVOT coursing anteriorly to the AV. AV indicates aortic valve; LA, left atrium; RA, right atrium; RVOT, right ventricular outflow tract.

Aortic Valve (AV)

Aortic Valve Short-Axis View (Figure 8.4)

1. Transducer positioning: In neutral position, the probe tip is advanced approximately 30–35 cm until the AV appears in short axis, positioned in the center of the screen. At the 0° position, the beam plane is not a true short axis due to the tangential course of the aorta as it leaves the heart. To achieve a "true" short axis of the AV, the beam plane is rotated to approximately 30–45° so that a clear view of all three aortic valve leaflets and commisures with a coaptation point is seen (Video 8.1).
2. Diagnostic utility: Aortic valve morphology, aortic stenosis, aortic regurgitation.

Aortic Valve Long-Axis View (Figure 8.5)

1. Transducer positioning: From the short axis, the probe is rotated an additional 90° until the entire

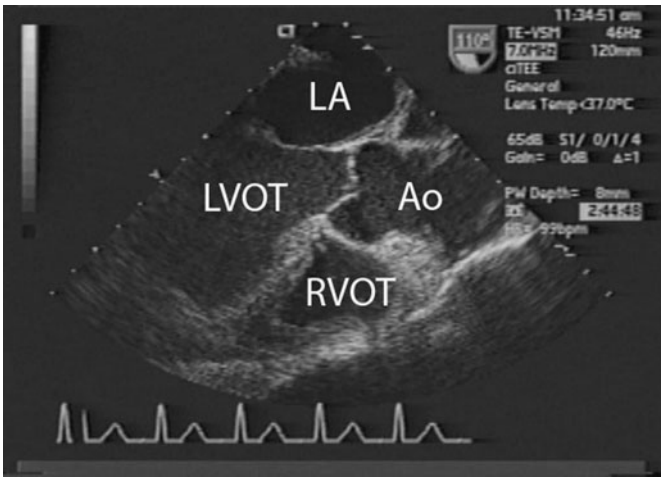


Figure 8.5. Aortic valve long axis view. The LVOT, AV, and proximal aorta can be seen in continuity exiting from the LV. Note the short-axis view of the RVOT anterior to the aorta in this view. AV indicates aortic valve; LV, left ventricle; LVOT, left ventricular outflow tract; RVOT, right ventricular outflow tract.

course of the left ventricular outflow tract (LVOT), AV, and the proximal ascending aorta are seen. A slight turn to the right (clockwise turn of the transducer handle) is sometimes necessary to obtain the image (Video 8.2).

2. Diagnostic utility: LVOT, AV, aortic root, ascending aorta.

Left Atrium (LA), Left Atrial Appendage (LAA), and Pulmonary Veins (PV)

LAA and Left PV Views (Figure 8.6)

1. Transducer positioning: The transducer is returned to the 0° position at the AV level, followed by a leftward turning of the probe (endoscope handle is turned counterclockwise), bringing into view the triangular shape of the LAA with pectinate muscles and the left superior pulmonary vein (anteflexion is sometimes necessary for an optimal view). The inferior pulmonary vein can then be seen by slight advancement of the probe with retroflexion. Depth is reduced to allow for clear examination of LAA contents. A 90° rotation is then performed to further examine the LAA and left PV (Video 8.3).
2. Diagnostic utility: LAA thrombi, LAA flow velocity, PV inflow analysis.

Left Atrial View (Figure 8.7)

1. Transducer positioning: From the left PV view, a slow clockwise turn of the endoscope handle allows for complete scanning of the LA as the probe is swept across the left atrium. Slight advancement and withdrawal is performed to fully inspect the inferior-superior extent of the LA (Video 8.4).
2. Diagnostic utility: LA thrombus/mass.

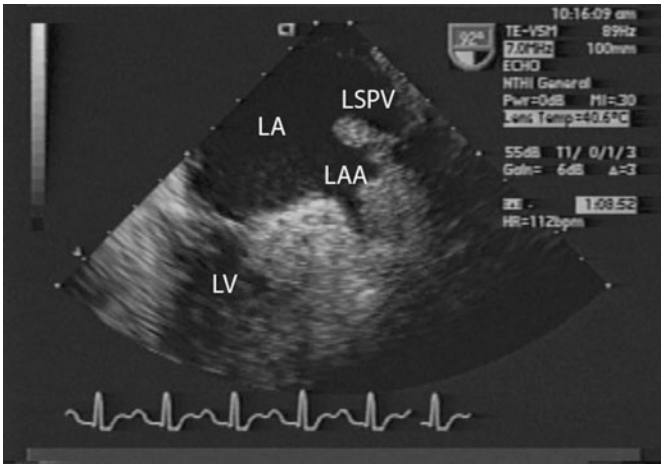


Figure 8.6. Left atrial appendage view. The LAA can be seen as a triangular structure upon leftward turning of transducer from the LA. Note the LSPV entering superior to the LAA. LA indicates left atrium; LAA, left atrial appendage; LSPV, left superior pulmonary vein.

Right PV View

1. Transducer positioning: Following LA examination, slight withdrawal and continued clockwise turning of the endoscope handle allows for visualization of the right superior PV as it enters the LA. Slight retroflexion will then bring the right inferior PV into view.
2. Diagnostic utility: PV inflow analysis.

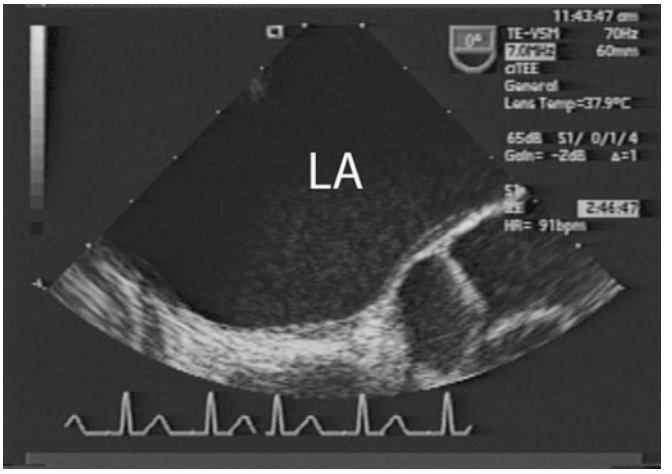


Figure 8.7. Left atrial view. At minimal depth, excellent resolution of the entire contents of the LA can be achieved. LA indicates left atrium.

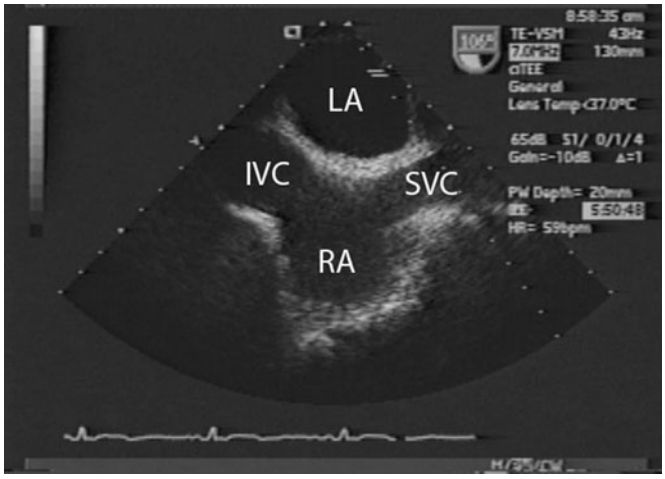


Figure 8.8. Bicaval view. The RA is positioned centrally, the probe is rotated to 90°, allowing for visualization of the IVC and SVC entering the RA in the same plane. The LA and atrial septum are also well seen in this view. IVC indicates inferior vena cava; LA, left atrium; RA, right atrium; SVC, superior vena cava.

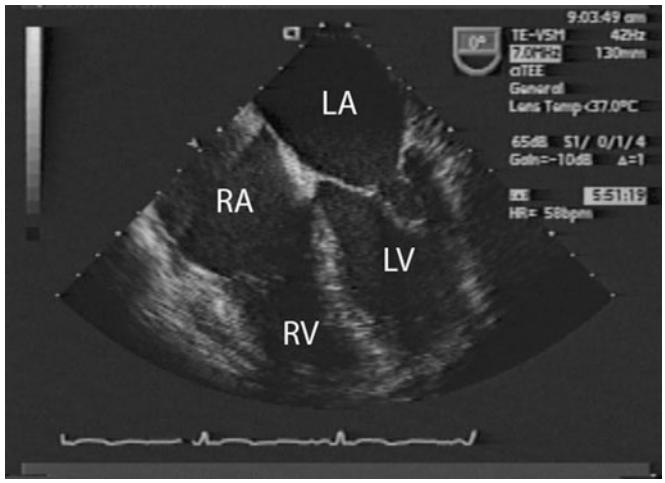


Figure 8.9. Four-chamber view. With the apex in the far field, all four cardiac chambers can be seen, including both atrioventricular valves.

Superior Vena Cava (SVC), Inferior Vena Cava (IVC), Right Atrium (RA), Atrial Septum

SVC Views (Figure 8.8)

1. Transducer positioning: In the 0° plane starting from the right PV view, counterclockwise turning of the handle will bring the RA into view. Slight withdrawal of the probe allows for a short-axis view of the SVC, followed by a 90° rotation for the long-axis view of the SVC (Video 8.5).
2. Diagnostic utility: SVC thrombus, SVC catheter position, assessment of volume responsiveness.

Right Atrium Bicaval View (Figure 8.8)

1. Transducer positioning: Returning the transducer to the 0° plane and advancing the probe slightly brings the RA into view. 90° rotation produces the bicaval view, showing the inferior vena cava and superior vena cava entering the RA in one longitudinal plane. Clockwise and counterclockwise turning of the handle allows for full evaluation of atrial contents as well as the atrial septum. Particular attention is paid to the atrial septum in this view, in order to assess for shunts, atrial septal defect (ASD), masses, or thrombi.
2. Diagnostic utility : RA contents, patent foramen ovale, atrial septal aneurysm, ASD, shunt assessment using agitated saline injection.

Left and Right Ventricles (LV, RV), Mitral and Tricuspid Valves (MV, TV)

Four-Chamber View (Figure 8.9)

1. Transducer positioning: In the 0° plane, the probe is advanced to the level of the mitral valve. Increasing the depth allows for assessment of the four cardiac chambers in a single plane, analogous to the transthoracic apical four-chamber view. Minor rotation adjustments and retroflexion may be required to optimize the view of all chambers. In addition, MV and TV anatomy are assessed (Video 8.6).
2. Diagnostic utility: RV and LV chamber size and function, anatomy and function of the TV and MV, inferoseptal and anterolateral LV wall contractility, MV inflow, tissue Doppler of lateral annulus.

Two-Chamber View (Figure 8.10)

1. Transducer positioning: Rotating 90° brings the LA and LV into a long-axis view (Video 8.7).
2. Diagnostic utility: LA and LV chamber size and function, anatomy and function of the MV, inferior and anterior LV wall contractility, MV inflow analysis.

Three-Chamber View

1. Transducer position: Further rotation to approximately 120° results in a long-axis view of the LVOT, along with a view of anteroseptal and inferolateral walls.
2. Diagnostic utility: LVOT anatomy, AV anatomy and function, anteroseptal/inferolateral LV wall contractility.

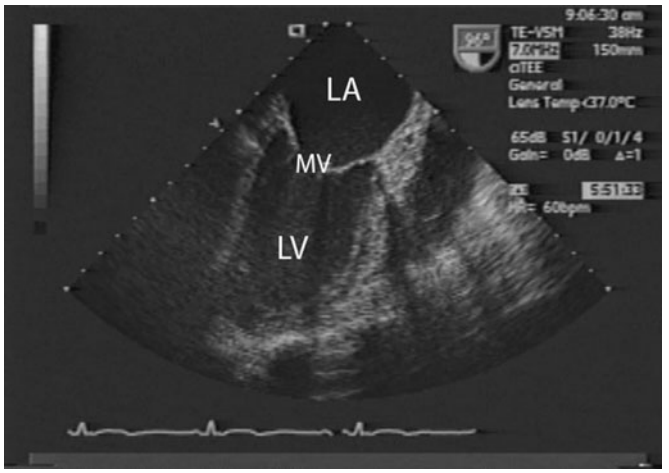


Figure 8.10. Two-chamber view. At 90° rotation from the four-chamber view, the LA and LV can be seen in long axis, allowing for assessment of the LV anterior and inferior walls. LA indicates left atrium; LV, left ventricle.

Gastric Short-Axis Biventricular View (Figure 8.11)

1. Transducer positioning: The probe is advanced in the neutral position at 0° to approximately 40–45 cm and anteflexed. This results in a short-axis view of both ventricles that is similar to the TTE parasternal short-axis midventricular view. By withdrawing the probe slightly, a "fish mouth" view of the mitral valve may be obtained. By advancing slightly, a view of the papillary muscle level is obtained (Video 8.8).
2. Diagnostic utility: Circumferential analysis of LV segmental wall contractility, RV size and function, septal kinetics.

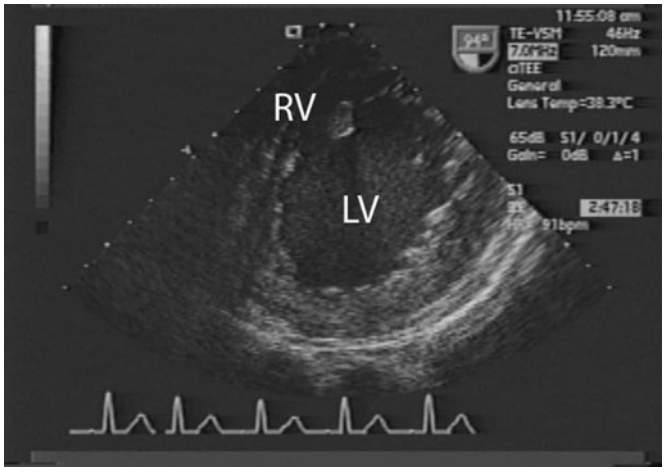


Figure 8.11. Gastric short axis view. Circumferential view of the LV and RV is achieved. LV indicates left ventricle; RV, right ventricle.

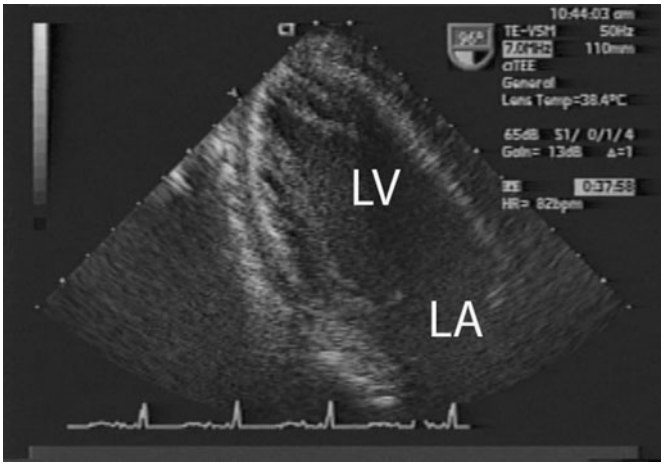


Figure 8.12. Deep gastric two-chamber view. The LA and LV are seen with the apex in the near field of the image. LA indicates left atrium; LV, left ventricle.

Gastric Long-Axis View

1. Transducer position: Rotation to 90° yields a long-axis view of the LV.
2. Diagnostic Strengths: Inferior and anterior LV wall contractility, MV anatomy and function.

Deep Gastric View (Figure 8.12)

1. Transducer positioning: Advancing the probe in neutral position to approximately 45–50 cm with anteflexion positions the transducer at the LV apex. Counterclockwise turning is often required to optimize the view. Further anteflexion allows for a five-chamber view, which includes the LVOT and AV (Video 8.9).
2. Diagnostic utility: MV, TV, AV anatomy and function, LV, RV size and function. LVOT outflow measurements for quantitation of stroke volume, dynamic indices of preload sensitivity.

Aorta and Pulmonary Artery (PA)

Aortic Views (Figure 8.13)

1. Transducer position: In neutral position, the probe is turned counterclockwise with slight withdrawal until the descending aorta is visualized in short axis. Depth is decreased to an appropriate extent. Slow withdrawal of the probe allows for complete visualization of the descending aorta. Clockwise turning of the probe beginning at the left subclavian allows for examination of the aortic arch, followed by slight advancement for evaluation of the ascending aorta. Orthogonal views may be obtained during the "pull-back" as indicated (Video 8.10).
2. Diagnostic utility: Aortic pathology.

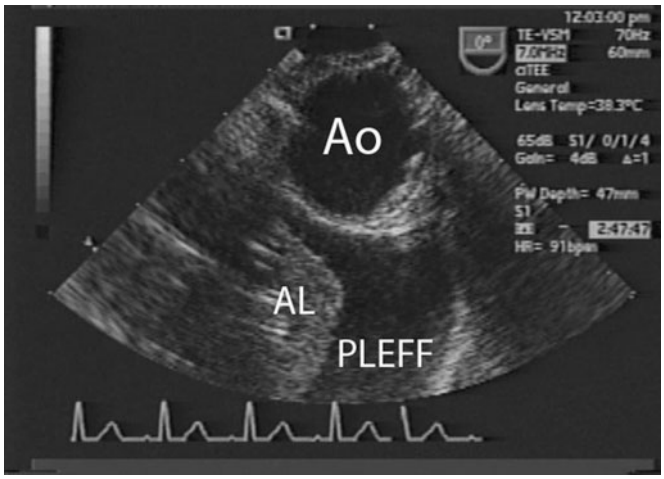


Figure 8.13. Descending aorta. Circumferential view of the aortic wall can be seen in the near field. Note the pleural effusion (PLEFF) and atelectatic lung (AL) anterior to the aorta.

RV Outflow Tract (RVOT), Pulmonary Artery View (Figure 8.14)

1. Transducer position: Starting at the AV level at 0°, the transducer orientation is increased to approximately 120–135° in order to visualize the RVOT, pulmonic valve, and proximal PA. This is followed by withdrawal of the probe while simultaneously decreasing the rotation angle down to 0°. This maneuver takes practice, as it requires simultaneous withdrawal and rotation. This results in a view of the main pulmonary artery and the proximal right PA and left PA. Once the proximal right (RPA) and left (LPA) pulmonary arteries are visualized, rotation to 90° combined with clockwise and counterclockwise turning allows for further visualization of these structures (Video 8.11).
2. Diagnostic utility: Anatomy and function of the RVOT, main PA, and proximal RPA/LPA.

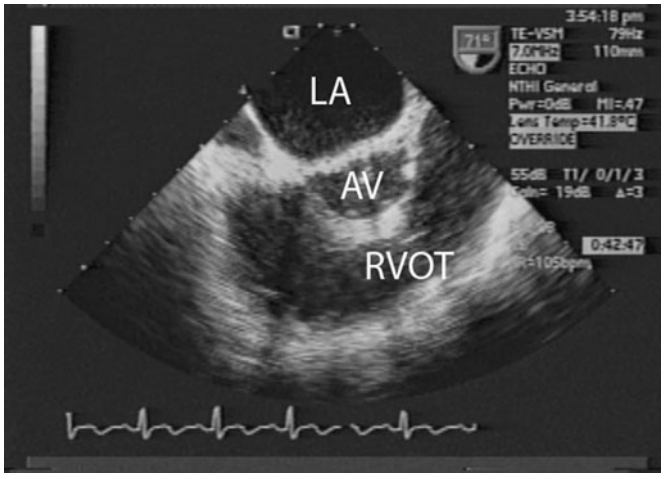


Figure 8.14. RVOT view. The RVOT can be seen "wrapping around" the AV at the base of the heart. Note the initial anterior position of the RVOT followed by a posterior coursing just prior to the bifurcation of the main PA (not seen in this image). AV indicates aortic valve; PA, pulmonary artery; RVOT, right ventricular outflow tract.

PROBE CLEANING

The endoscope should be cleaned according to institutional policy.

GOAL-DIRECTED TEE IN THE ICU

In some clinical situations, a comprehensive TEE examination is indicated. In critical care practice, it is neither necessary nor practical to perform a comprehensive TEE examination in all patients. This is an important consideration when evaluating the acutely ill patient with severe hemodynamic failure in a busy ICU environment and when using TEE as a monitoring tool. Vieillard-Baron et al. have described the use of serial daily TEE examinations in sepsis with circulatory failure using a limited number of views for the purpose of guiding volume resuscitation and inotrope/pressor use.[15]

The number and sequence of views required for a goal-directed TEE exam have not been standardized. Benjamin et al. reported on the use of a monoplane pediatric probe by surgical intensivists.[4] Their protocol required four views with three quantitative assessments. Exams were completed in 12 minutes, and resulted in therapeutic changes in 52% of cases. Vieillard-Baron et al. described three views with four qualitative assessments.[7] These qualitative assessments were similar to results obtained quantitatively in the same patient. These studies support the concept of goal-directed echocardiography and help to define what constitutes an adequate study.

Vieillard-Baron et al. have also reported on the training required to perform a moderately comprehensive TEE (six views, and six quantitative and six qualitative assessments). They compared the proficiency of trainees with expert-level intensivists. Trainees who performed 29 ± 10 TEE examinations over a six-month period had marked improvement in proficiency, although they still had not achieved expert-level skills. After six months of training, trainees required 16 ± 5 minutes, compared with <10 minutes for expert-level echocardiographers, to perform a moderately comprehensive TEE.[8] On the basis of these studies and our own experience, we use an approach that is similar

TABLE 8.1. Goal-directed TEE examination

View	*Qualitative assessment*	*Diagnostic utility*
SVC long axis	SVC respiratory variation (none, minor, major)	Preload responsiveness
Esophageal four-chamber view at 0°	LV function (normal, hyperdynamic, moderately, severely depressed)	LV contractility
	RV size (normal, moderately, severely enlarged)	Acute cor pulmonale
Gastric short axis	Septal shape and function (normal, dyskinetic)	RV pressure overload

LV indicates left ventricular; RV, right ventricular; SVC, superior vena cava. Source: Modified from Vieillard-Baron et al. (Ref. 7)

to that of Vieillard-Baron et al. This is summarized in Table 8.1.[7] Vieillard-Baron has demonstrated that qualitative "eyeball" assessments are of similar accuracy to quantitative assessments in practice.

CONCLUSION

Transesophageal echocardiography is very useful in the ICU. It may be particularly useful in situations where TTE image quality is suboptimal. The intensivist may use TEE to perform a comprehensive examination similar to the approach used in cardiology. This requires mastery of transducer manipulation, cardiac anatomy, and knowledge of all standard views. Alternatively, TEE may be used in a goal-directed fashion for rapid assessment of hemodynamic failure and to guide ongoing therapy of the critically ill patient.

References

1. Vignon P, Mentec H, Terre S, Gastinne H, Gueret P, Lemaire F. Diagnostic accuracy and therapeutic impact of transthoracic and transesophageal echocardiography in mechanically ventilated patients in the ICU. *Chest.* 1994;106:1829–1834.
2. Huttemann E, Schelenz C, Kara F, Chatzinikolaou K, Reinhart K. The use and safety of transoesophageal echocardiography in the general ICU—a minireview. *Acta Anaesthesiol Scand*. 2004;48:827–836.
3. Huttemann E. Transesophageal echocardiography in critical care. *Minerva Anestesiol*. 2006;72:891–913.
4. Benjamin E, Griffin K, Leibowitz AB, et al. Goal-directed transesophageal echocardiography performed by intensivists to assess left ventricular function: comparison with pulmonary artery catheterization. *J Cardiothorac Vasc Anesth*. 1998;12:10–15.
5. Colreavy FB, Donovan K, Lee KY, Weekes J. Transesophageal echocardiography in critically ill patients. *Crit Care Med.* 2002;30:989–996.
6. Poelaert JI, Trouerbach J, De Buyzere M, Everaert J, Colardyn FA. Evaluation of transesophageal echocardiography as a diagnostic and therapeutic aid in a critical care setting. *Chest.* 1995;107:774–779.
7. Vieillard-Baron A, Charron C, Chergui K, Peyrouset O, Jardin F. Bedside echocardiographic evaluation of hemodynamics in sepsis: is a qualitative evaluation sufficient? *Intensive Care Med.* 2006;32:1547–1552.
8. Vieillard-Baron A, Qanadli SD, Antakly Y, Fourme T, Loubieres Y, Jardin F, Dubourg O. Transesophageal echocardiography for the diagnosis of pulmonary embolism with acute cor pulmonale: a comparison with radiological procedures. *Intensive Care Med.* 1998;24:429–433.
9. Krivec B, Voga G, Zuran I, et al. Diagnosis and treatment of shock due to massive pulmonary embolism: approach with transesophageal echocardiography and intrapulmonary thrombolysis. *Chest.* 1997;112:1310–1316.
10. Chandrasekaran K, Bansal RC, Ross JJ, Shah PM, Mintz JS. Impact of transesophageal echocardiographic examinations: comparison between transthoracic and transesophageal approach [abstract]. *J Am Coll Cardiol*. 1989; 13:51A.
11. Charron C, Prat G, Caille V, et al. Validation of a skills assessment scoring system for transesophageal echocardiographic monitoring of hemodynamics. *Intensive Care Med.* 2007;33:1712–1718.
12. Shanewise JS, Cheung AT, Aronson S, et al. ASE/SCA guidelines for performing a comprehensive intraoperative multiplane transesophageal echocardiography examination: recommendations of the American Society of Echocardiography Council for Intraoperative Echocardiography

and the Society of Cardiovascular Anesthesiologists Task Force for Certification in Perioperative Transesophageal Echocardiography. *J Am Soc Echocardiogr.* 1999;12:884–900.

13. Milani RV, Lavie CJ, Gilliland YE, Cassidy MM, Bernal JA. Overview of transesophageal echocardiography for the chest physician. *Chest*. 2003;124:1081–1089.
14. Otto CM. *Textbook of Clinical Echocardiography*. Philadelphia, Pa: WB Saunders Co; 2004:70–93.
15. Vieillard-Baron A, Caille V, Charron C, Belliard G, Page B, Jardin F. Actual incidence of global left ventricular hypokinesia in adult septic shock. *Crit Care Med.* 2008;36:1701–1706.

This page intentionally left blank

CHAPTER 9

Echocardiographic Assessment of Left Ventricular Function and Hydration Status

Balachundhar Subramaniam and Daniel Talmor

APPROACH TO HEMODYNAMIC INSTABILITY IN THE INTENSIVE CARE UNIT (ICU)

Hemodynamic instability (acute and chronic) is a common problem in critically ill patients. Prolonged hypotension may lead to organ ischemia, dysfunction, and poor outcome.[1] Conversely, rapid diagnosis and intervention may prevent this deterioration and improve outcome. Intensivists routinely look for common, immediately treatable problems; however, clinical examination alone may be insufficient to make these immediate therapeutic decisions. Clinical suspicion is the key to building a differential diagnosis and to the intelligent application of technology to aid in therapeutic decision-making. Echocardiography is one such technology, which may make a critical difference in the rapid diagnosis of both common and uncommon, but important, causes of unstable hemodynamics. Used in this way, echocardiography has been found to lead to a change in therapy in at least in a quarter of critically ill patients.[2] The assessment of hemodynamics is commonly approached in terms of preload, afterload, and contractility, all of which may be aided by echocardiologic exam.

When assessing the unstable critically ill patient using echocardiography, the major causes of hemodynamic instability such as pulmonary embolism or pericardial tamponade may be quickly and reliably ruled out. This may be performed by experienced echocardiographers, intensivists, or emergency medicine physicians trained in one of the abbreviated echocardiographic examination protocols, for example, the Focus Assessed Transthoracic Echocardiography (FATE) protocol.[3]

Once major causes are ruled out, the next step in the patient assessment is to assess left ventricular (LV) volume status and function. The most important and commonly used method of assessing LV global and focal wall motion is by a qualitative assessment over multiple views. This method is extremely effective, rapid, and in agreement with nuclear scanning studies when done by an experienced echocardiographer. The result is both an assessment of regional wall motion and an overall assessment of LV function usually expressed in terms of an estimated ejection fraction (EF). Quantitative techniques of ventricular assessment allow for a more measurable and arguably less biased assessment of the ventricle. These techniques have their advantages and their limitations. It is incumbent upon the intensivist echocardiographer to be familiar with these advantages and limitations.

TRANSTHORACIC ECHOCARDIOGRAPHY (TTE) VS. TRANSESOPHAGEAL ECHOCARDIOGRAPHY (TEE) VS. HAND-HELD DEVICES

Transesophageal echocardiography is often considered superior to TTE in the ICU. Transthoracic echocardiography frequently provides poor image quality in postoperative patients due to mechanical ventilation (positive end-expiratory pressure [PEEP] >15 cm of H_2O), inability to position the patient, lack of patient cooperation, chest wall edema and obstructed views due to wound dressings, chest tubes, drains, and an open chest or abdomen. In the critical care setting, TTE leads to a successful exam in 50% of attempts,[4,5] in contrast to 90% with TEE.[6] There are, however, challenges to the routine performance of TEE in the ICU. The TEE examination requires additional time and expertise when compared with the TTE exam. Insertion of the probe

into the esophagus carries with it a risk of loss of the airway. Additionally, TTE carries with it a small but real risk, on the order of 0.01%, of significant complications such as esophageal perforation.

Handheld portable devices are small, simple, and convenient. They can provide a focused qualitative assessment.[7] Handheld devices can be valuable in ultrasound-guided thoracentesis, paracentesis, and central venous cannulation. Newer generations of battery-powered devices are now available. The role and utility of these devices in the assessment of the hemodynamically unstable ICU patient is still evolving.

Regardless of what modality is used to perform the exam, it is important that the exam be complete and as comprehensive as the training of the practitioner allows. If the initial exam is limited based on an ICU protocol and there is doubt as to the findings, the exam should be followed up as soon as possible with a comprehensive exam by a more experienced practitioner. A comprehensive exam is less likely to miss an unexpected diagnosis. With practice, a complete exam may be performed in minutes. A reasonable strategy in performing the exam is to first focus on the areas or structures of interest and as directed by the physical exam. Once the immediate question is answered, this should be followed by a complete examination. Items of interest may then be reexamined in a more leisurely manner. Guidelines exist that standardize the images captured on both the TTE and TEE exams.[8] These are important in assuring that all structures are viewed from multiple angles, allowing each individual structure to be completely and accurately assessed and documented as needed. The standardized views also assure that no structure is missed in the exam, and provide the common language that allows practitioners to communicate their findings with each other.

LEFT VENTRICULAR FUNCTION

Assessment of left and right ventricular systolic and diastolic function and their changes over time is quite helpful in therapeutic decision-making for the unstable critically ill patient. It is important to remember that echocardiography, is largely a two-dimensional (2D) way of looking at a 3D structure. A minimum of two orthogonal views should be performed for each structure of interest before making a diagnostic or a therapeutic decision. New or worsening wall-motion abnormalities may indicate acute ischemia and a causative lesion. Assessment of global ventricular systolic function is also important because many diseases of the critically ill, and in particular sepsis, may lead to global rather than focal ventricular dysfunction.

Ventricular systolic function depends on both preload and afterload. Estimates of systolic function should be performed under different loading conditions to ascertain the true function. Once again, this demonstrates the importance of obtaining serial assessments rather than single snapshot views. Pressure–area relationships are one way of determining left ventricular contractility independent of loading conditions. Qualitative and semiquantitative measures used for assessing global LV systolic function using echocardiography are EF, fractional shortening (FS), fractional area change (FAC), Simpson's method of assessment of left ventricular function, mitral annular motion, dP/dt using mitral regurgitant jet, and segmental wall-motion abnormality assessment using visual identification with a standardized 17-segment model and strain rate imaging. Fractional shortening and FAC are easily assessed with a transgastric midpapillary short-axis view of the LV. The most commonly used methods are discussed first, followed by other modalities.

QUALITATIVE ASSESSMENT OF LV SYSTOLIC FUNCTION

To help interpret LV systolic function, several questions should be asked: (1) Is the ventricle adequately filled? (2) Is the ventricle's contractile function adequate? (3) Is the ventricular contractility uniform throughout the coronary artery distributions?

Visual Assessment with a Standard 17-Segment Model

In an effort to have a uniform nomenclature for the left ventricular function derived from multiple assessment modalities such as cardiac magnetic resonance imaging (MRI), echocardiography, nuclear scanning, and angiography, the American Heart Association (AHA) produced a consensus statement suggesting that the LV be divided into 17 different segments.[9] The LV is divided into basal, midcavity, and apical segments along the long axis of the heart. Each segment is then further divided into six in the basal, six in the midcavity, and four in the apical segments, and an apical cap is included as the 17th segment. The corresponding coronary arterial distribution is shown in Figure 9.1. The left anterior descending coronary artery (LAD) supplies the anterior wall of the heart and anterior two thirds of the interventricular septum. The left circumflex artery (LCx) supplies the lateral wall of the LV. The right coronary artery (RCA) supplies the posterior third of the

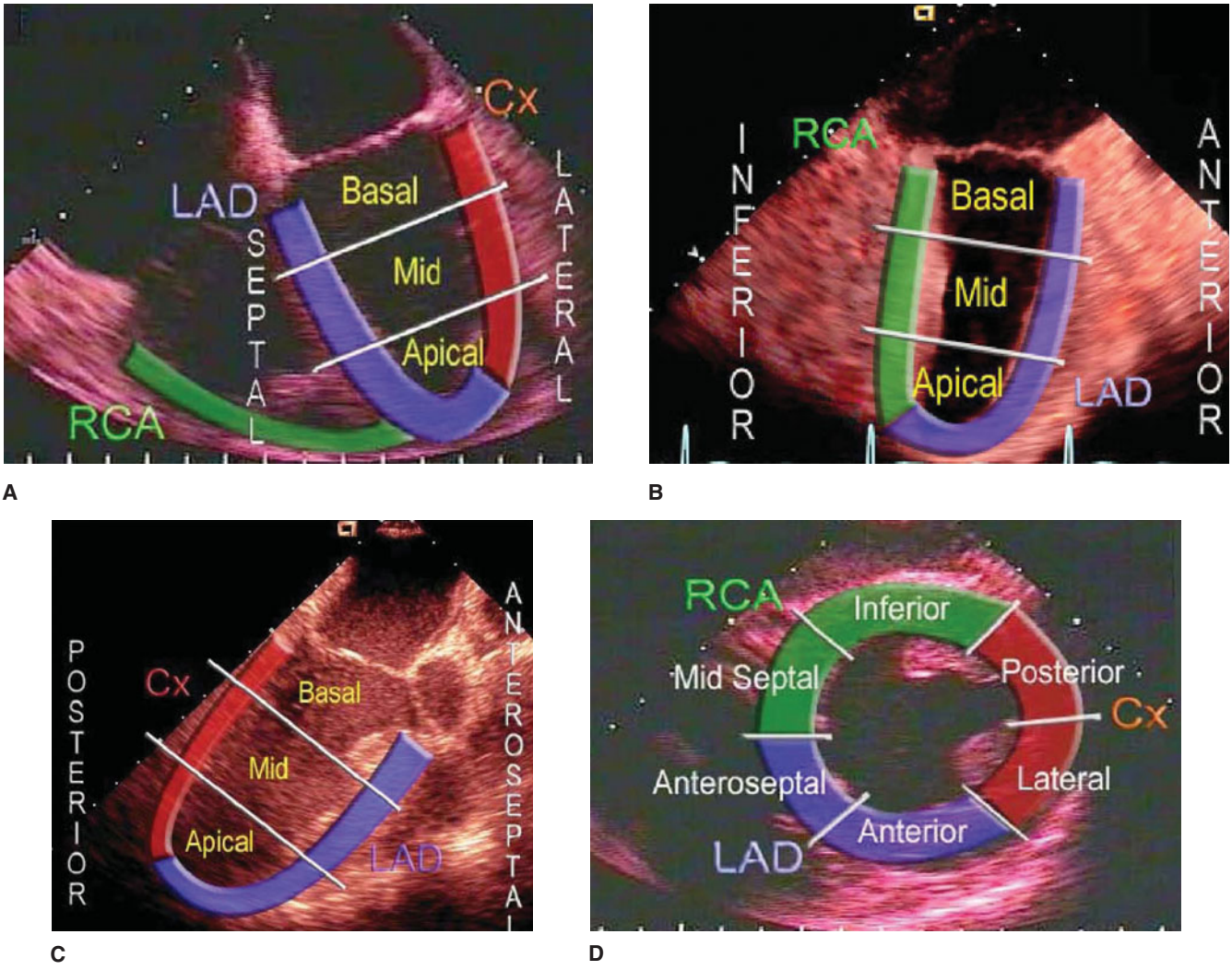


Figure 9.1. (A) Four-chamber view showing the coronary artery distribution and the corresponding LV segments. The septal wall (anterior 1/3) is supplied by the Left Anterior descending artery (LAD); lateral wall is supplied by the circumflex artery (Cx). (B) Two-chamber view showing the coronary artery distribution and the corresponding left ventricular (LV) segments. The base, mid, and apical segments of anterior wall are supplied by the left anterior descending artery (LAD) and the inferior wall is supplied by the right coronary artery. (C) Oblique view of the left ventricle showing the anteroseptal and posterior segments. The base, mid, and apex of the anteroseptal wall and posterior wall are supplied by the left anterior descending (LAD) and left circumflex (Cx) artery, respectively. (D) Midpapillary short-axis view of the LV showing the three arterial distribution and corresponding segments. This is the midpapillary transgastric short-axis view of the LV showing the LAD supplying the midanterior and anteroseptal segments, Cx supplying midlateral and posterior segments of the lateral wall, and RCA supplying midseptal and inferior segments of the LV. Cx indicates left circumflex coronary artery; LAD, left anterior descending artery; LV, left ventricle; RCA, right coronary artery. (Reproduced with permission from Dr. Martin London's Web site. www.ucsf.edu/teeecho)

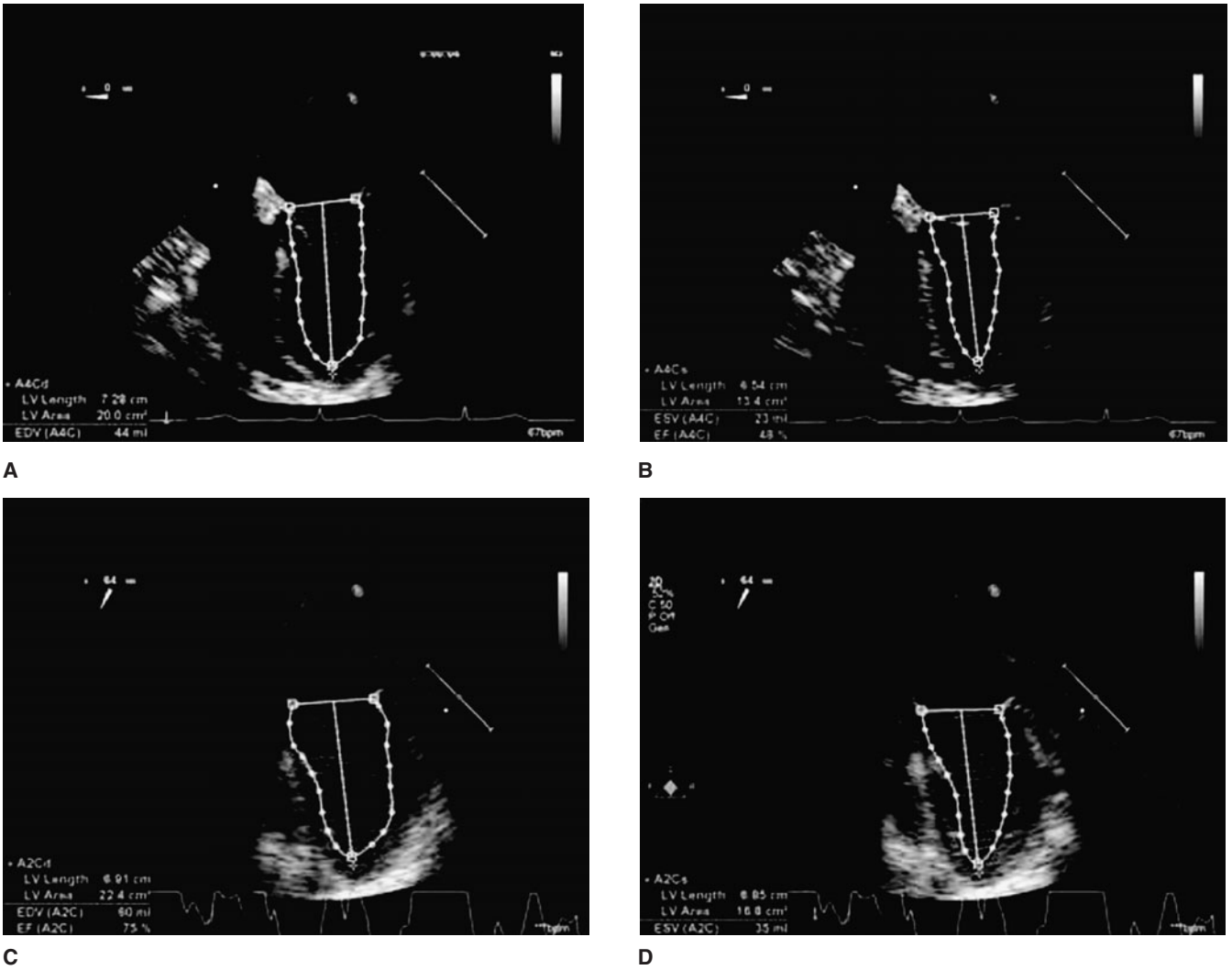
interventricular septum and inferior wall of the LV. A semiquantitative assessment can be performed using a wall-motion score or index. The left ventricular contractility is dependent on movement of the base toward the apex, thickening of the wall segments, and a spiral squeeze or rotational movement of the LV. Thickening of the wall segments and the endocardial excursion of the LV segment are important to assess the wall motion. The wall-motion score is described here:

1. Normal (>30% endocardial excursion and >50% wall thickening)
2. Mild hypokinesis (10–30% endocardial excursion and 30–50% wall thickening)
3. Severe hypokinesis (<20% endocardial excursion and <30% wall thickening)
4. Akinesis (no endocardial excursion and <10% wall thickening)
5. Dyskinesis (moves paradoxically during systole)

The wall-motion score index is defined as the wall-motion score/number of segments. This is a subjective assessment and does not have a true linear relationship. A stunned myocardium without a perfusion defect can exhibit wall-motion abnormality. Multiple views must be obtained to truly define the degree of LV impairment and the arterial distribution involved. Endocardial excursion alone may be due to tethered myocardium, and a change in wall thickness is a precise indicator of ischemia.[10] The reproducibility of wall thickening measurements was evaluated, which led to the following conclusions: (1) it is difficult to obtain consistent data on wall thickening in the longitudinal plane; (2) multiple measurements are necessary to reduce variability; and (3) it is necessary to ensure that borders, locations, and angles are carefully defined.[11]

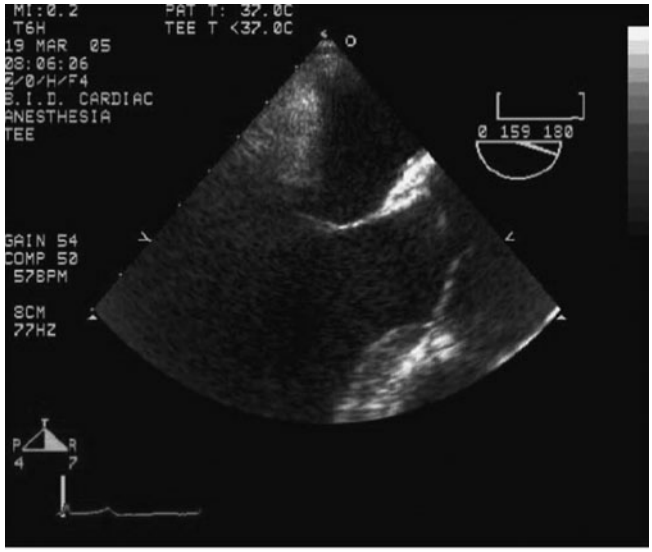
Ejection Fraction

Stroke volume is obtained by calculating the difference between the end-diastolic volume and end-systolic volume. Ejection fraction is defined as stroke volume divided by the end-diastolic volume. The American Society of Echocardiography recommends the modified Simpson's method (Figure 9.2). This method calculates EF in two planes and averages them. When using

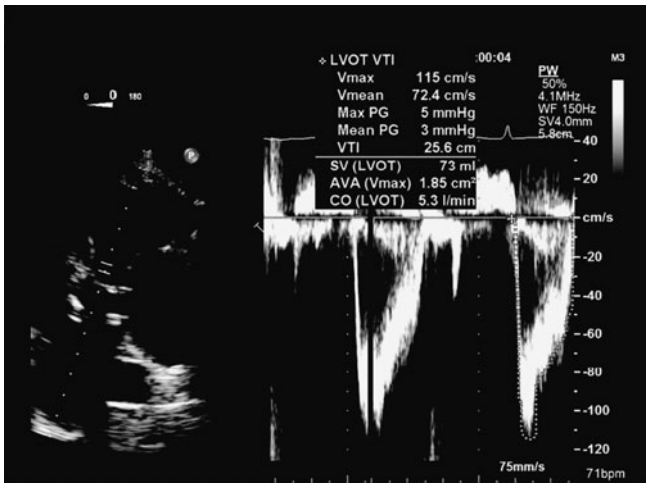


A B C D

Figure 9.2. Simpson's method of left ventricular ejection fraction assessment. (A) Midesophageal four-chamber end-diastolic frame: LV at end-diastole frame is frozen. The endocardial border is traced out to get the end-diastolic dimension. (B) Midesophageal four-chamber end-systolic frame: the LV frame at end-systole is frozen. The endocardial border is tracked in the machine. (C) Midesophageal oblique two-chamber end-diastolic frame: the LV frame at end-diastole is frozen in this oblique view, adding another dimension to the previous measurements. The endocardial border is tracked in the machine. (D) Midesophageal oblique two-chamber end-systolic frame: the LV frame at end-systole is frozen. The endocardial border is tracked in the machine in this two-chamber view, giving an added dimension to the previous calculation.



A



B

Figure 9.3. (A) Stroke volume measurement: calculation of LVOT diameter; decreasing the depth gives an enlarged view of the LVOT. This minimizes error in calculating the LVOT measurements. (B) Calculation of stroke volume: VTI measurement across LVOT. The LVOT is aligned parallel to the Doppler (in this TEE view, it is deep transgastric view); a pulsed-wave Doppler is placed at the same spot as the LVOT measurement to obtain a clean envelope. This is traced to calculate the VTI measurement. LVOT indicates left ventricular outflow tract; TEE, transesophageal echocardiography; VTI, velocity time integral.

TEE, the midesophagus, the four-chamber, and two-chamber planes can be used for this purpose. However, this method has some limitations. Endocardial borders need to be well visualized to trace them out. Mitral annular calcifications commonly interfere with border detection. Lateral wall dropouts can occur in the four-chamber view, as the ultrasound beam is parallel to the lateral wall. The trabeculations in the LV can also interfere with border detection. In those situations, a contrast solution can be used to improve detection of the borders. The LV apex is frequently foreshortened. More commonly, the EF is estimated by the experienced echocardiographer without formal measurement, and this has been shown to have excellent correlation with formal measurements.[12]

QUANTITATIVE ASSESSMENT OF LV SYSTOLIC FUNCTION WITH ECHOCARDIOGRAPHY

Calculation of Cardiac Output

A pulmonary artery catheter can be used to measure cardiac output in the ICU. However, with the current evidence not favoring this modality, echocardiography has a critical role in assessing cardiac output in the ICU. Both the right and left ventricular cardiac outputs can be measured using echocardiography (Figure 9.3). Left ventricular cardiac output measurement is reproducible and accurate.

Cardiac output = Heart rate × stroke volume

LVOT area = (LVOT radius)^2 × 3.14
(LVOT is assumed as a circle and the area of circle is πr^2)

Heart rate can be measured using the electrocardiogram or from one velocity-time integral (VTI) to another VTI. This is automatically stored in the echo machine. Stroke volume is a product of left ventricular outflow tract (LVOT) area and left ventricular aortic valve systolic ejection VTI. When blood is ejected from the LV into the cylindrical aorta, the stroke volume is calculated from the height of this blood column, which is the VTI. The base of this cylindrical output is formed by the LVOT, and LVOT area can be easily calculated. The height of the cylinder is provided by VTI traced with a pulsed-wave Doppler (PWD) assessing the flow at the LVOT through a deep transgastric aortic valve view or transgastric aortic valve long-axis view. Tracing this PWD wave from the LVOT will provide the LVOT VTI. The assumption made here is that the area measured for the calculation of stroke volume is constant

during systole. A small error in radius measurements at LVOT will amplify the error because it is squared. To minimize this error, the depth of the image should be reduced and LVOT amplified. The next assumption is that flow across the LVOT is laminar. The validity of this assumption is demonstrated by the narrow velocity band and smooth spectral signal on pulsed Doppler recordings. The flat velocity profile is confirmed by moving the sample volume across the flowstream in two orthogonal views to demonstrate uniform velocities at the center and at the edges of the flowstream. Importantly, the Doppler beam angle should be parallel to the flow or within 20°. The Doppler signal is recorded at a parallel intercept angle to flow, resulting in an accurate measurement of velocity (Cos 0 = 1). It is important to remember that for Doppler measurements the angle of interrogation should be parallel to flow, while for the 2D or M-mode measurements the interrogating beam should be perpendicular to the structure of interest. The LVOT diameter and the PWD should be evaluated from the same anatomic site to assure that spatial and temporal relationships of interrogating pulsed-wave Doppler are maintained. This error can be minimized by choosing the area proximal to the aortic valve as the point of interest and used as a routine method of measurement. Since there can be dynamic changes in heart rate, the measurements should be performed at the same time and all measures should be repeated when evaluating cardiac output at a different point of time.

Stroke Volume Measurements

With TEE, it is common to utilize LVOT as the primary site, followed by the main pulmonary artery and the right ventricular outflow tract (RVOT). With TTE, stroke volume can be measured at the aortic valve leaflet tips or in the ascending aorta. Ascending aortic diameter is measured from a parasternal long-axis view, and flow from the suprasternal notch or the apical TTE view. Transmitral stroke volume can be measured as well with the pulsed Doppler at the mitral leaflet tips. Because of the complex mitral valve geometry and increasing assumptions, it is uncommon to use this site as a routine for cardiac output evaluation. In the right side of the heart, the tricuspid valve or pulmonary artery can be used to measure stroke volume. Right ventricular cardiac output is possible to measure and the pulmonary valve output can be measured just as described for the LV (Figure 9.4[A]). However, the main pulmonary artery diameter measurements are not always fixed and vary depending upon the view. Also, it is not always possible to achieve the right ventricular output flow parallel to the Doppler beam of interrogation (Figure 9.4[B]).

SEMIQUANTITATIVE METHODS

Fractional Shortening

This is a one-dimensional measurement of left ventricular global systolic function (Figure 9.5).[13] M-mode across the left ventricular midpapillary axis is obtained. A freeze-frame analysis of this M-mode is used to calculate fractional shortening. Ventricular internal dimensions are measured from the leading edge to the leading edge of interest. A fractional shortening is =

(Left ventricular internal diameter in diastole − Left ventricular internal diameter in systole)/ Left ventricular internal diameter in diastole. × 100

Normal >25% (Figure 9.5)

The advantage of this method is that it is quick and fairly reproducible. The M-mode provides a very high time resolution and delineates the endocardial borders well. This is a basic, crude estimate of left ventricular global systolic function, with the normal range being 25–45%. However, this method could easily be prone to error if focal wall-motion abnormalities exist. An oblique cut of the one-dimensional plane can lead to errors in length measurements. It is important to keep in mind these caveats and the potential overestimation or underestimation of this measurement. Adding another dimension to this semiquantitative method may improve the evaluation of global function.

Fractional Area Change

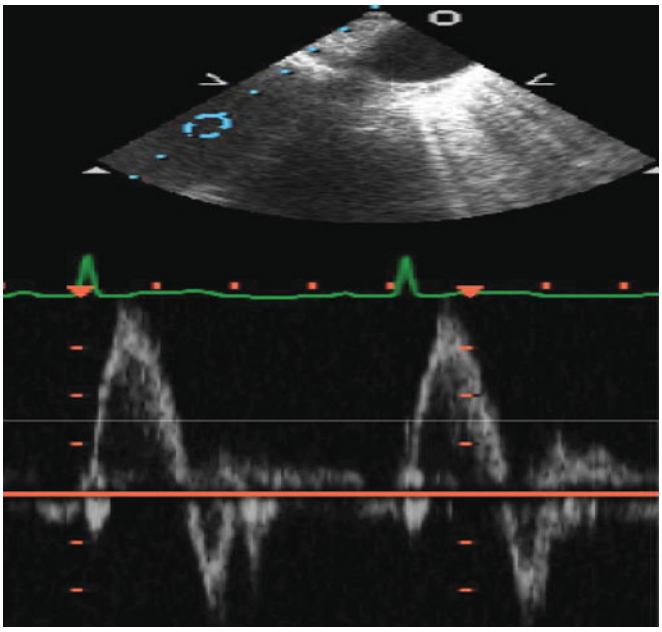
This is a 2D measure of left ventricular systolic function (Figure 9.6). One of the prerequisites for obtaining these values is to have adequate endocardial border definition. Detection of the endocardium throughout the entire view can be time consuming, especially if the borders are not well visualized. Fractional area change can be used to quantify EF.[14]

FAC % = (LVEDA-LVESA)/LVEDA × 100%,

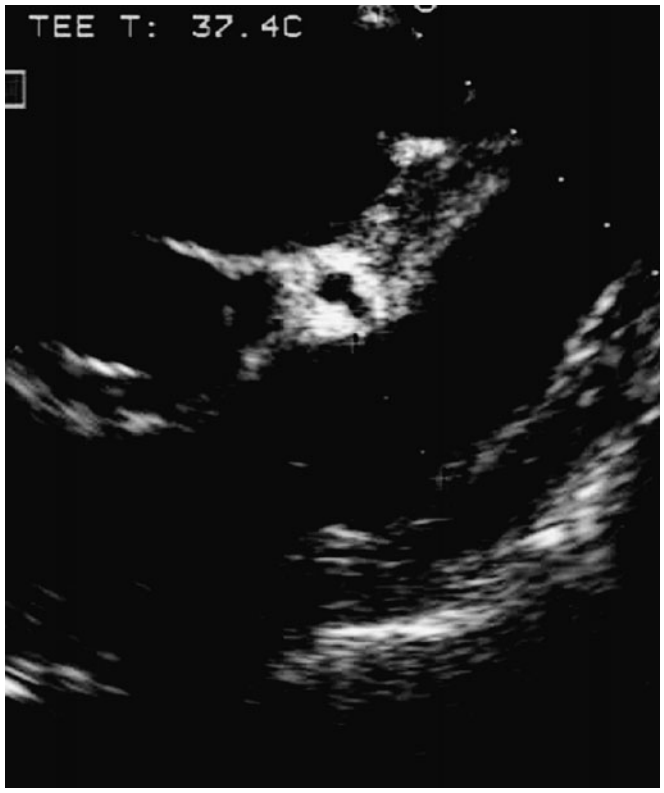
Where LVEDA is left ventricular end-diastolic area, LVESA is left ventricular end-systolic area

Normal >50–75% (Fig. 6a and Fig. 6b)

This measure is heavily dependent on afterload and slightly dependent on preload. A midpapillary, transgastric short-axis view to calculate FAC has demonstrated close correlation to radionuclide angiography and scintigraphy.[15,16]



A



B

Figure 9.4. (A) Stroke volume measurement sites: pulmonary artery from the aortic arch view; in this TEE view, the probe is placed in the aortic arch and rotated 20–40°. The main pulmonary artery appears on the left and aligns parallel to the pulsed-wave Doppler interrogation. (B) Stroke volume measurement sites: pulmonary artery from the midesophageal inflow–outflow view; in this right ventricular view, one can appreciate that the pulmonary artery is not aligned parallel to the pulsed-wave Doppler interrogation. This can lead to error in measurements, as explained in the text. TEE indicates transesophageal echocardiography.

Systolic Index of Contractility (dP/dt):

While ejection phase indices of LV performance are easy to obtain, the load dependency of these units may confound the accurate assessment of true ventricular performance. The rate of pressure increase during the isovolumic contraction phase, dP/dt (max) is sensitive to changes in contractility,[17] insensitive to changes in afterload and wall motion abnormalities, and only mildly affected by changes in preload. Continuous-wave Doppler is used to determine the velocity of the mitral regurgitation jet. Using this jet, the time taken to reach 3 m/sec from 1 m/sec is measured. Applying the simplified Bernoulli equation, [pressure = 4 × (Velocity)^2], contractility index is expressed as

$$dP/dt = 32/\Delta t \text{ (Figure 9.7)}$$

The time for the mitral regurgitant jet to reach a velocity of 3 m/sec from 1 m/sec is estimated. If the left ventricular function is well preserved, this time will be much shorter compared with that of a poorly functioning ventricle. At 3 m/sec, applying the simplified Bernoulli equation, the pressure is 4(3)^2, which is 36 mm Hg. At 1 m/sec, the pressure is 4(1)^2, which is 4 mm Hg. The pressure difference is 32 mm Hg. When 32 mm Hg is divided by the time taken to reach from 1 m/sec, 3 m/sec dP/dt is obtained. A value of >1200 mm Hg/sec, or a time of <27 m/sec, is considered normal and <1000 mm Hg/sec, or a time of >32 m/sec, is considered abnormal. The two caveats to these measurements are that one has to have a mitral regurgitant jet and there is no scale for the intermediate LV contractile states.

Tissue Doppler Imaging

This parameter provides quantitative measurement of global and segmental LV function. Mitral annular

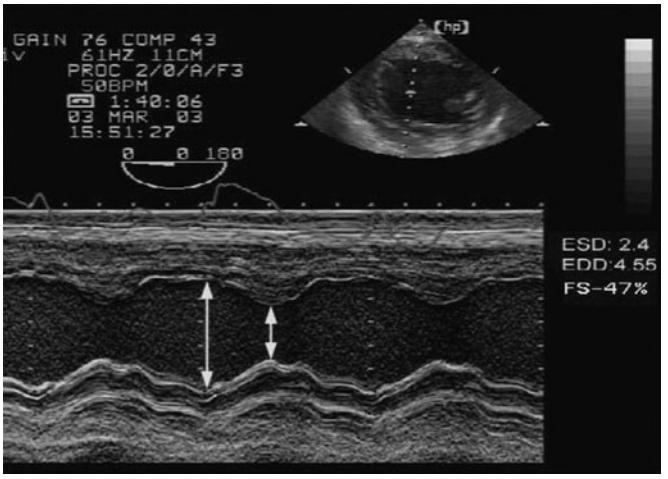


Figure 9.5. Fractional shortening across LV: when an M-mode is sent across the PLAX view of the LV by TTE or midpapillary transgastric short-axis view of the LV, a high-resolution time frame is obtained. When this frame is frozen, end-diastolic and end-systolic lengths can be measured and lead to fractional shortening measurements. EDD indicates end-diastolic diameter; ESD, end-systolic diameter; FS, fractional shortening; LV, left ventricle; PLAX, parasternal long-axis view; TTE, transthoracic echocardiography.

descent as detected by tissue Doppler provides a global estimate of left ventricular contractile function.[18] Myocardial tissue velocity is measured at septal, lateral, inferior, anterior, posterior, and anteroseptal mitral annulus. An equation was derived to predict EF from the average peak mitral annular descent velocity derived from the aforementioned sites.

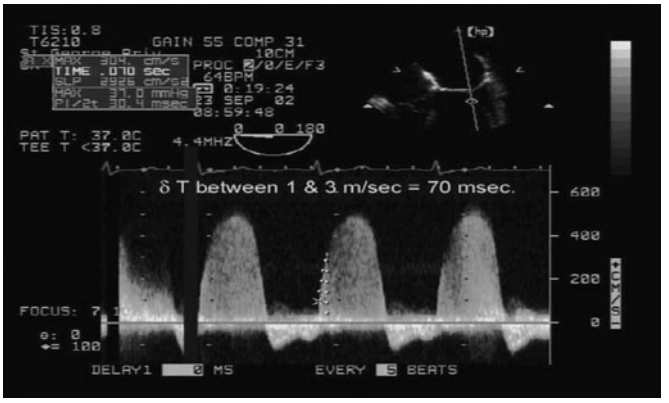
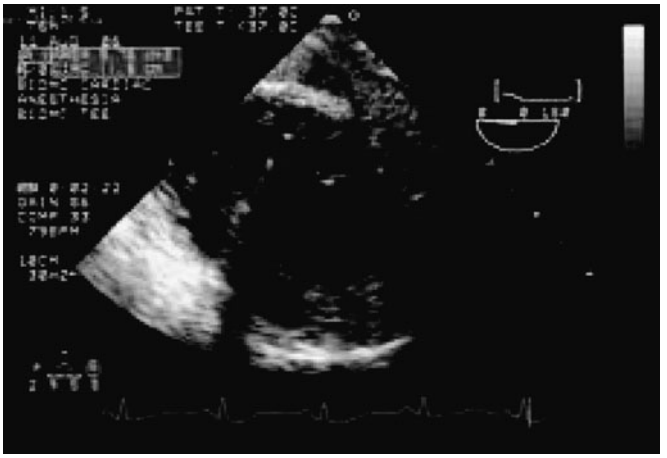


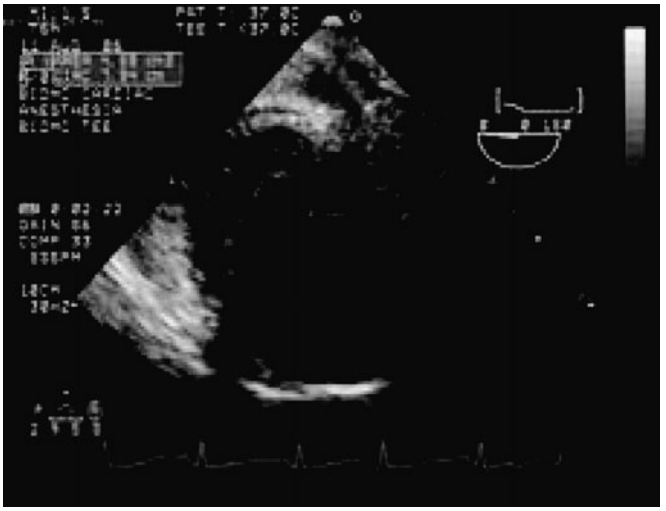
Figure 9.7. dP/dt measurement across mitral valve with a mitral regurgitant jet**:** presence of mitral regurgitant jet is necessary to calculate this measurement. A continuous-wave Doppler across the mitral valve gives the mitral regurgitant jet envelope. This frame is frozen and the points at 1 m/sec and 3 m/sec of the upstroke are marked. The distance between the two points in terms of time will give the dT.

LVEF = 8.2 × (average peak mitral annular velocity) + 3%

This measure has the potential to evaluate global LV function in cases where endocardial border definition is suboptimal for tracing. Myocardial velocity imaging does not have the ability to differentiate tethered myocardial movement or translational movement from true movements. These parameters can be



A



B

Figure 9.6. (A) Fractional area calculation: end-diastole frame. Midpapillary short-axis view of the LV is frozen at the end of diastole. The endocardial border is traced, which gives the end-diastolic area an extra dimension, unlike fractional shortening. (B) Fractional area calculation: end-systole frame. Midpapillary short-axis view of the LV is frozen at the end of systole. The endocardial border is traced. LV indicates left ventricle.

obtained from segmental strain imaging patterns. As of now, they are being vigorously pursued as a research tool.

UNCOMMONLY USED TOOLS

Pressure-Volume (P-V) Loops

The slopes of P-V loops indicate load-independent, true contractile states. With recent advances in technology, continuous measurement of LV area as a surrogate for LV volume is possible. Preload affects ventricular function and this is understood from the P-V loops, where the pressure is represented on the y-axis and volume on the x-axis. An increase in left ventricular systolic function will shift this loop up and to the left, while a decrease in the left ventricular systolic function will shift this loop down and to the right. When the end-systolic pressure points are connected between the different loops, an end-systolic P-V relationship is obtained in the form of a straight line. This is also known as elastance. This measure of left ventricular systolic function is insensitive to changes in loading conditions. This is rarely possible in the busy ICUs, as there is not only lack of time but also a fear of altering loading conditions in a sick patient. The clinical evaluation is performed with the other methods described in this chapter.

Wall Stress and Left Ventricular Mass

Wall stress is the force per unit area exerted on the myocardium. This is dependent on cavity dimensions, pressure, and wall thickness. Wall stress is described as circumferential, meridional, or radial in three dimensions. End-systolic calculations of circumferential and meridional wall stress are used. Multiplying the myocardial volume and the specific density of cardiac muscle provides a derivation of left ventricular mass. Echocardiographic assessment of end-systolic elastance can be assessed by systolic velocity acceleration in the LVOT[19] and systolic strain rate.[20] Myocardial performance index (MPI), obtained by dividing the sum of isovolumic contraction and relaxation with ejection time is another contractility measure.[21] The clinical utility of MPI is controversial.

NEWER MODALITIES

Tissue Doppler, Strain Rate, and Strain

Doppler tissue imaging (DTI) and speckle-tracking imaging (STI) are recently developed and important methods for measuring regional myocardial function. Tissue velocity signals are of low velocity, and by eliminating the wall filter and using low-gain amplification it is possible to display myocardial tissue velocity. A PWD at a specific place in the myocardium, a color map, or an arbitrarily oriented M-mode can be utilized to display the myocardial tissue velocity. The common pitfalls with DTI are: (1) it can only measure the component of motion parallel to the ultrasound beam; (2) translational movements cannot be differentiated; and (3) motion due to tethering of adjacent, normally contracting segments cannot be differentiated. Myocardial peak velocity, strain rate, and strain can identify regional myocardial dysfunction at rest and during stress.[22–24] When there are confusing wall-motion abnormalities with the standard assessments, these techniques can be utilized to confirm the findings. Strain and strain rate measure deformation occurring in the direction of the ultrasound scan line. It is very sensitive to transducer orientation, more so, than to Doppler angular dependence. Speckle-tracking imaging was recently introduced to avoid this angle dependence. This modality measures deformation in two dimensions. It gives an accurate display of tissue velocity, strain rate, and strain. Regional function at rest,[25] at stress,[26] during acute ischemia,[27] and for viability detection[28] are suitable clinical scenarios to utilize strain and strain rate assessment by either DTI or STI. The combination of 3D speckle-tracking with 3D imaging could be an extremely powerful tool for local and global LV function evaluation.[29]

Endocardial border definition is extremely important to assess LV function. In some patients with different conditions such as obesity or emphysema, it is often difficult to define the endocardial borders. Contrast echocardiography[30] plays an important role in such patients. Acoustic quantification[31] differentiates the tissue from blood and automatically outlines the endocardial border. Color kinesis extends this principle by tracking the endocardial border beat-to-beat and provides quantification of LV function.[32] They are useful to follow the trend, and the absolute values still have to be calibrated. It is important to note that in the presence of ventricular aneurysms or other asymmetric abnormalities these modalities may fail and the use of 3D echo may improve the assessment of LV function.

ASSESSMENT OF DIASTOLIC FUNCTION

The ability of the LV to fill at a lower pressure to prevent pulmonary venous congestion and pulmonary edema is equally important to its systolic performance.

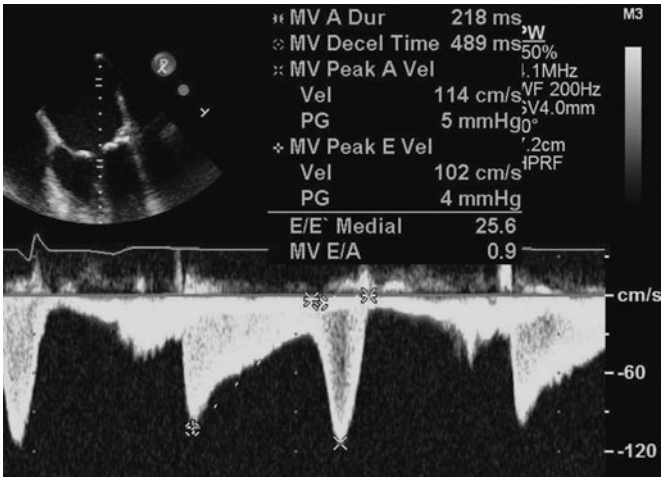


Figure 9.8. Transmitral filling: LV diastolic function assessment**:** pulsed-wave Doppler at the tip of mitral leaflets. A wave indicates atrial contraction; A Dur, atrial wave duration; DT, deceleration time; E wave, early passive filling; LV, left ventricle; MV, mitral valve.

Transmitral filling, pulmonary venous flow pattern, and lateral mitral annular velocities are used in combination to assess the presence and degree of diastolic dysfunction.

Transmitral LV filling

A PWD is placed at the mitral leaflet tips and the early passive filling (E) velocity and late atrial contraction (A) velocities are obtained (Figure 9.8). In normal LV, E velocity is usually higher than A velocity. With LV hypertrophy, aging, the E/A ratio falls <1 and is referred to as impaired relaxation. E wave acceleration is proportional to left atrial pressure divided by τ, the exponential time constant of isovolumic LV pressure fall. To preserve stroke volume, in patients with progressive diastolic dysfunction there is progressive left atrial pressure increase reversing the impaired relaxation pattern to a pseudonormal pattern.[33] At severe LV dysfunction, filling occurs for a short time with extreme rise in left atrium (LA) pressure with a classic fall in deceleration time and a high E/A ratio.[34] These parameters are preload dependent, and certain maneuvers such as valsalva can help to differentiate a pseudonormal pattern from progressive LV diastolic dysfunction. This assessment alone cannot differentiate all the patterns of diastolic dysfunction and may also miss the diagnosis in some cases.

Pulmonary Venous Flow PWD

This is a helpful adjunct to transmitral filling assessment to diagnose diastolic dysfunction.[35] A PWD placed just distal to the pulmonary venous entry into the left atrium obtains a systolic (S) wave, diastolic (D) wave, and atrial (A) wave. The size and morphology of the atrial reversal wave that occurs with atrial contraction is probably the most useful.[36] The difference between the duration of transmitral and pulmonary venous A waves is predictive of LV end-diastolic pressure.[36]

Color M-Mode Flow Propagation Velocity (Vp)

This parameter is obtained by placing a colored-Doppler box in the LV inflow followed by an M-mode across this box. A spatiotemporal map of flow propagating across the mitral valve in diastole is related to LV relaxation and known as Vp (Figure 9.9).[37] Shifting the color baseline to 30–40% of the maximal transmitral velocity and measuring the slope of the red–blue transition is usually helpful to calculate Vp. A Vp <45 cm/sec is predictive of diastolic dysfunction. Unlike transmitral filling, it is not prone to pseudonormalization.[38] The major criticism for this assessment is its low reproducibility. A combination of transmitral filling and pulmonary venous PWD is sometimes equivocal in the diagnosis of diastolic dysfunction. Additional criterion, such as E′ and A′ obtained by DTI at the lateral mitral

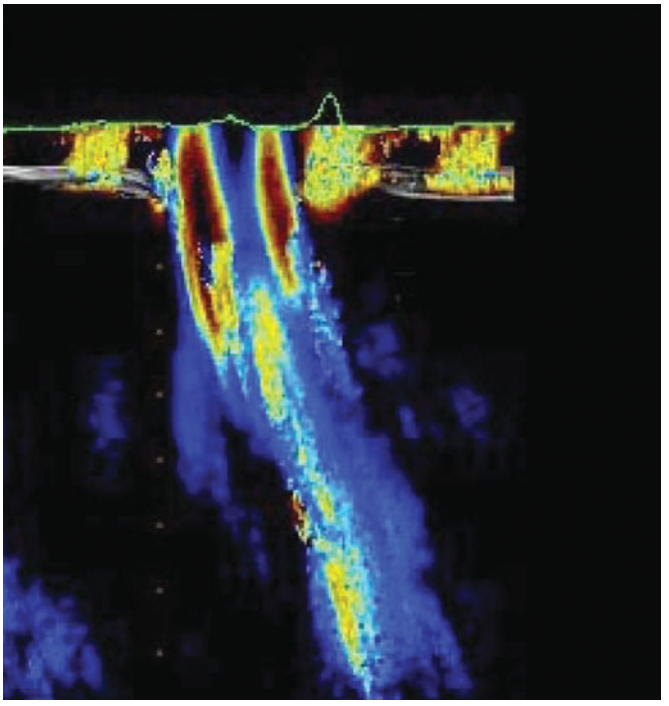


Figure 9.9. Vp of LV to assess diastolic function: an M-mode colored Doppler across the LV filling will yield a color flow map, as depicted. The downslope from the baseline is measured at 4 cm. The slope of this line will yield Vp. LV indicates left ventricle; Vp, flow propagation velocity.

annulus and Vp can help differentiate the degree of diastolic dysfunction.[39] Apart from the qualitative evaluation of LV diastolic dysfunction as described above, several quantitative estimates of diastolic function such as relaxation, compliance, filling pressure, and diastolic suction (intraventricular pressure gradient) have been described.

ASSESSMENT OF LEFT VENTRICULAR FILLING PRESSURE

A pulmonary artery catheter may be used to assess the left ventricular filling pressures. Pulmonary artery occlusion pressure in the absence of any distal obstruction is equivalent to the left ventricular end-diastolic pressure. This is reflective of left ventricular end-diastolic volume and thereby the preload. However, one can assume that this is possible in a compliant LV. With increasing age or hypertension, it is not uncommon to have left ventricular hypertrophy and reduced LV compliance, which leads to altered pressure-volume relationships. It is possible to predict the left ventricular end-diastolic pressures based on the relationship between early passive transmitral fillings of the LV (E velocity) to the corresponding lateral mitral annulus displacement (E' velocity). The decrease in E' velocity is of a larger magnitude compared with the E velocity reductions due to a noncompliant ventricle. If this ratio is >15, it is indicative of left ventricular end-diastolic pressure (LVEDP) >15. If the ratio is <8, it is indicative of LVEDP <15. The intermediate numbers are more difficult to interpret. E' velocity < 5 cm/sec is characteristic of a poorly compliant ventricle.

SEMIQUANTITATIVE LV VOLUME ASSESSMENT

In spite of years of controversy, pulmonary artery catheters are commonly used to assess left ventricular volume using pressure measurements. However, this relationship between filling pressures and filling volumes may not be accurate in any particular patient, and especially in the ventilated patient. A number of methods have been proposed to assess left ventricular volume and pressures using echocardiography. These are used both for one-time assessments and for monitoring the response to fluid challenges. The LV is symmetric, with two relatively equal short axes and with long axis running from the base to the apex. The apex is slightly rounded in the long-axis view and the apical half of the LV resembles a hemi-ellipse. The basal half of the LV is cylindrical, leading to a circular-looking LV in short-axis views. Calculating left ventricular volume is fraught with danger of assumption of these shapes in M-mode or two-dimensional views. This caveat should be remembered while assessing the normal to abnormally shaped ventricles with these parameters.

Left ventricular end-diastolic volume (LVEDV), left ventricular end diastolic area (LVEDA),[40] superior vena cava (SVC) collapsibility,[41,42] inferior vena cava (IVC) size, and fluid responsiveness[43] have all been used for preload assessment. The criteria for diagnosing hypovolemia include an end-diastolic diameter less than 25 mm, systolic obliteration of the LV cavity, and a LV end-diastolic area of less than 55 cm^2.[44] These measures are easily obtained from the transgastric, midpapillary short-axis view (TG, SAX) (Figure 9.6).

Preexisting cardiac disease or poor compliance of the LV will alter the P-V relationships in specific patients, and optimal LVEDA may be even greater than normal. This highlights the usefulness of following trends coupled with stroke volume measurements for a given LVEDA. Rather than basing decision-making on single measurements, serial LVEDV measurements are ideal, but are time consuming and may be difficult to implement in real-time practice. Left ventricular end-diastolic area measurements have been validated to track fluid status changes, and are calculated from tracings of still frames of LV at end-diastole in the aforementioned view. This process can be simplified by using automated acoustic quantification border detection systems.[45] End-systolic and end-diastolic volumes should be inspected, and over time can track changes in the volume status. End-systolic cavity obliteration, or "kissing papillary muscle sign," is a useful sign of hypovolemia. This is a very sensitive predictor of reductions in the end-systolic area (100%) but the specificity for predicting a reduction in preload is low (30%).[46]

Tissue Doppler imaging of mitral annulus (E') combined with transmitral E wave inflow pattern can predict left ventricular mean diastolic pressures. The E/E', when <8, is indicative of a compliant ventricle, and when >15, is indicative of a ventricle with high mean filling pressures and poor compliance.[47] Intermediate ratios should be evaluated using additional parameters such as pulmonary venous inflow and mitral inflow deceleration time.[47]

RECENT TECHNOLOGIES

Three-Dimensional (3D) Assessment of Left Ventricular Function and Volume

Reconstruction of real-time images can provide 3D image of the LV. When reconstruction is performed, a

series of 2D images are obtained at a standard of 3° or 5° with a stationary transducer. The number of planes and the quality of 2D images will determine the quality of 3D image. Development of the matrix-array transducer has led to acquisition of images from many lines of sight simultaneously to reconstruct a volume of ultrasound data in real time. They are still insufficient to display the entire adult LV. To obtain the entire LV, a series of component volumes of the heart are obtained over consecutive cardiac cycles and then integrated to obtain a larger volume.

Left ventricular volume and function can be calculated from the 3D approach and is a more accurate representation. The 3D approach compares favorably with MRI, and is accurate with less interobserver variability.[22,48] This is understandable, as unlike M-mode or 2D, there are fewer assumptions made. Because the volume estimation is more accurate, changes in volume estimates with systole will also be accurate and result in a reliable estimation of LVEF. There are some limitations with this approach. Line-density in a 3D volume is much lower than in a 2D image, and more interpolation is often necessary. When the acquisition is from a fixed transducer position alignment with various structures, it is often not ideal and results in a poor image.[29] Movement of the heart with breathing and arrhythmias can often result in image artifacts. With a progressive reduction in analysis time and the availability of improved technology, 3D is likely to become the preferred method for assessing LV volume and function in critically ill patients in the future.

References

1. Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med*. 2001;345:1368–1377.
2. McLean AS. Transoesophageal echocardiography in the intensive care unit. *Anaesth Intensive Care.* 1998;26:22–25.
3. Jensen MB, Sloth E, Larsen KM, Schmidt MB. Transthoracic echocardiography for cardiopulmonary monitoring in intensive care. *Eur J Anaesthesiol*. 2004;21:700–707.
4. Pearson AC. Noninvasive evaluation of the hemodynamically unstable patient: the advantages of seeing clearly. *Mayo Clin Proc.* 1995;70:1012–1014.
5. Cook CH, Praba AC, Beery PR, Martin LC. Transthoracic echocardiography is not cost-effective in critically ill surgical patients. *J Trauma.* 2002;52:280–284.
6. Pearson AC, Castello R, Labovitz AJ. Safety and utility of transesophageal echocardiography in the critically ill patient. *Am Heart J*. 1990;119:1083–1089.
7. Oh JK, Seward JB, Khandheria BK, et al. Transesophageal echocardiography in critically ill patients. *Am J Cardiol.* 1990;66:1492–1495.
8. Shanewise JS, Cheung AT, Aronson S, et al. ASE/SCA guidelines for performing a comprehensive intraoperative multiplane transesophageal echocardiography examination: recommendations of the American Society of Echocardiography Council for Intraoperative Echocardiography and the Society of Cardiovascular Anesthesiologists Task Force for Certification in Perioperative Transesophageal Echocardiography. *Anesth Analg.* 1999;89:870–884.
9. Cerqueira MD, Weissman NJ, Dilsizian V, et al. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: a statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation.* 2002;105:539–542.
10. Buda AJ, Zotz RJ, Pace DP, Krause LC. Comparison of two-dimensional echocardiographic wall motion and wall thickening abnormalities in relation to the myocardium at risk. *Am Heart J*. 1986;111:587–592.
11. Konstadt SN, Abrahams HP, Nejat M, Reich DL. Are wall thickening measurements reproducible? *Anesth Analg*. 1994;78:619–623.
12. Folland ED, Parisi AF, Moynihan PF, Jones DR, Feldman CL, Tow DE. Assessment of left ventricular ejection fraction and volumes by real-time, two-dimensional echocardiography. A comparison of cineangiographic and radionuclide techniques. *Circulation.* 1979;60:760–766.
13. Vuille C, Malvern WA. Left ventricle I: general considerations, assessment of chamber size and function. In: Malvern WA, ed. *Principles and Practice of Echocardiography.* 2nd ed. Philadelphia, Pa: Lea & Febiger; 1994.
14. Abel MD, Nishimura RA, Callahan MJ, et al. Evaluation of intraoperative transesophageal two-dimensional echocardiography. *Anesthesiology*. 1987;66:64–68.
15. Clements FM, Harpole DH, Quill T, Jones RH, McCann RL. Estimation of left ventricular volume and ejection fraction by two-dimensional transoesophageal echocardiography: comparison of short axis imaging and simultaneous radionuclide angiography. *Br J Anaesth*. 1990;64:331–336.
16. Urbanowicz JH, Shaaban MJ, Cohen NH, et al. Comparison of transesophageal echocardiographic and scintigraphic estimates of left ventricular end-diastolic volume index and ejection fraction in patients following coronary artery bypass grafting. *Anesthesiology.* 1990;72:607–612.
17. Bargiggia GS, Bertucci C, Recusani F, et al. A new method for estimating left ventricular dP/dt by continuous wave Doppler-echocardiography. Validation studies at cardiac catheterization. *Circulation.* 1989;80:1287–1292.
18. Gulati VK, Katz WE, Follansbee WP, Gorcsan J 3rd. Mitral annular descent velocity by tissue Doppler echocardiography as an index of global left ventricular function. *Am J Cardiol.* 1996;77:979–984.

19. Bauer F, Jones M, Shiota T, et al. Left ventricular outflow tract mean systolic acceleration as a surrogate for the slope of the left ventricular end-systolic pressure-volume relationship. *J Am Coll Cardiol*. 2002;40:1320–1327.
20. Greenberg NL, Firstenberg MS, Castro PL, et al. Doppler-derived myocardial systolic strain rate is a strong index of left ventricular contractility. *Circulation*. 2002;105:99–105.
21. Cheung MM, Smallhorn JF, Redington AN, Vogel M. The effects of changes in loading conditions and modulation of inotropic state on the myocardial performance index: comparison with conductance catheter measurements. *Eur Heart J.* 2004;25:2238–2242.
22. Chan J, Jenkins C, Khafagi F, Du L, Marwick TH. What is the optimal clinical technique for measurement of left ventricular volume after myocardial infarction? A comparative study of 3-dimensional echocardiography, single photon emission computed tomography, and cardiac magnetic resonance imaging. *J Am Soc Echocardiogr*. 2006; 19:192–201.
23. Cain P, Baglin T, Case C, Spicer D, Short L, Marwick TH. Application of tissue Doppler to interpretation of dobutamine echocardiography and comparison with quantitative coronary angiography. *Am J Cardiol*. 2001;87:525–531.
24. Abraham TP, Nishimura RA, Holmes DR Jr, Belohlavek M, Seward JB. Strain rate imaging for assessment of regional myocardial function: results from a clinical model of septal ablation. *Circulation.* 2002;105:1403–1406.
25. Zhang Y, Chan AK, Yu CM, et al. Strain rate imaging differentiates transmural from non-transmural myocardial infarction: a validation study using delayed-enhancement magnetic resonance imaging. *J Am Coll Cardiol*. 2005;46: 864–871.
26. Hanekom L, Jenkins C, Jeffries L, et al. Incremental value of strain rate analysis as an adjunct to wall-motion scoring for assessment of myocardial viability by dobutamine echocardiography: a follow-up study after revascularization. *Circulation.* 2005;112:3892–3900.
27. Skulstad H, Urheim S, Edvardsen T, et al. Grading of myocardial dysfunction by tissue Doppler echocardiography: a comparison between velocity, displacement, and strain imaging in acute ischemia. *J Am Coll Cardiol*. 2006;47:1672–1682.
28. Vitarelli A, Montesano T, Gaudio C, et al. Strain rate dobutamine echocardiography for prediction of recovery after revascularization in patients with ischemic left ventricular dysfunction. *J Card Fail*. 2006;12:268–275.
29. Picard MH, Popp RL, Weyman AE. Assessment of left ventricular function by echocardiography: a technique in evolution. *J Am Soc Echocardiogr*. 2008;21:14–21.
30. Mulvagh SL, DeMaria AN, Feinstein SB et al. Contrast echocardiography: current and future applications. *J Am Soc Echocardiogr*. 2000;13:331–342.
31. Yvorchuk KJ, Davies RA, Chan KL. Measurement of left ventricular ejection fraction by acoustic quantification and comparison with radionuclide angiography. *Am J Cardiol*. 1994;74:1052–1056.
32. Mor-Avi V, Vignon P, Koch R, et al. Segmental analysis of color kinesis images: new method for quantification of the magnitude and timing of endocardial motion during left ventricular systole and diastole. *Circulation.* 1997;95: 2082–2097.
33. Thomas JD, Choong CY, Flachskampf FA, Weyman AE. Analysis of the early transmitral Doppler velocity curve: effect of primary physiologic changes and compensatory preload adjustment. *J Am Coll Cardiol*. 1990;16:644–655.
34. Klein AL, Hatle LK, Taliercio CP, et al. Prognostic significance of Doppler measures of diastolic function in cardiac amyloidosis. A Doppler echocardiography study. *Circulation.* 1991;83:808–816.
35. Tabata T, Thomas JD, Klein AL. Pulmonary venous flow by doppler echocardiography: revisited 12 years later. *J Am Coll Cardiol*. 2003;41:1243–1250.
36. Rossvoll O, Hatle LK. Pulmonary venous flow velocities recorded by transthoracic Doppler ultrasound: relation to left ventricular diastolic pressures. *J Am Coll Cardiol.* 1993;21:1687–1696.
37. Brun P, Tribouilloy C, Duval AM, et al. Left ventricular flow propagation during early filling is related to wall relaxation: a color M-mode Doppler analysis. *J Am Coll Cardiol*. 1992;20:420–432.
38. Takatsuji H, Mikami T, Urasawa K, et al. A new approach for evaluation of left ventricular diastolic function: spatial and temporal analysis of left ventricular filling flow propagation by color M-mode Doppler echocardiography. *J Am Coll Cardiol*. 1996;27:365–371.
39. Garcia MJ, Thomas JD, Klein AL. New Doppler echocardiographic applications for the study of diastolic function. *J Am Coll Cardiol*. 1998;32:865–875.
40. Cheung AT, Savino JS, Weiss SJ, et al. Echocardiographic and hemodynamic indexes of left ventricular preload in patients with normal and abnormal ventricular function. *Anesthesiology.* 1994;81:376–387.
41. Vieillard-Baron A, Chergui K, Rabiller A, et al. Superior vena caval collapsibility as a gauge of volume status in ventilated septic patients. *Intensive Care Med.* 2004;30: 1734–1739.
42. Jardin F, Vieillard-Baron A. Ultrasonographic examination of the venae cavae. *Intensive Care Med.* 2006;32:203–206.
43. Barbier C, Loubieres Y, Schmit C, et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responsiveness in ventilated septic patients. *Intensive Care Med*. 2004;30:1740–1746.
44. Sohn DW, Shin GJ, Oh JK, et al. Role of transesophageal echocardiography in hemodynamically unstable patients. *Mayo Clin Proc*. 1995;70:925–931.
45. Perrino AC Jr, Luther MA, O'Connor TZ, Cohen IS. Automated echocardiographic analysis. Examination of serial intraoperative measurements. *Anesthesiology*. 1995;83: 285–292.
46. Leung JM, Levine EH. Left ventricular end-systolic cavity obliteration as an estimate of intraoperative hypovolemia. *Anesthesiology*. 1994;81:1102–1109.

47. Ommen SR, Nishimura RA, Appleton CP, et al. Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: a comparative simultaneous Doppler-catheterization study. *Circulation.* 2000;102:1788–1794.

48. Sugeng L, Mor-Avi V, Weinert L, et al. Quantitative assessment of left ventricular size and function: side-by-side comparison of real-time three-dimensional echocardiography and computed tomography with magnetic resonance reference. *Circulation.* 2006;114:654–661.

CHAPTER 10

Echocardiographic Evaluation of Preload Responsiveness

Michel Slama, Julien Maizel, and Paul H. Mayo

INTRODUCTION

Hemodynamic failure is a common problem in the intensive care unit (ICU). Echocardiography helps the intensivist establish a diagnosis, develop a therapeutic plan, and monitor the results of therapeutic intervention for patients with shock. A common question is whether the patient should receive volume resuscitation. This chapter will review the utility of echocardiography for identification of the volume-responsive patient with hemodynamic failure.

THE PHYSIOLOGY OF HYPOVOLEMIA

In many clinical situations, such as hypotension, shock, functional renal failure, oligoanuria, and clinical or laboratory signs of dehydration, hypovolemia may be suspected. Two types of hypovolemia can be distinguished: absolute and relative. *Absolute* hypovolemia is defined as a reduction of total circulating blood volume, which may be related to blood loss (hemorrhage), or plasma loss (gastrointestinal, renal, cutaneous, extravasation into interstitial tissues). *Relative* hypovolemia is defined as an inadequate distribution of blood volume between the central and peripheral compartments. This is commonly seen during septic shock. Hypovolemia results in a reduction in venous return, which may diminish preload and stroke volume (SV). This response depends on the Frank-Starling relationship, which relates left ventricular (LV) preload to stroke volume. The LV Frank-Starling curve has two parts (Figure 10.1): a steep first part (Segment A), where preload and SV are linearly related and any preload change is accompanied by an SV change (preload-dependent part); and a flat second part (Segment B), in which modifications of ventricular preload do not change SV (preload-independent part).

Significant central hypovolemia reduces preload, SV, mean arterial pressure, and cardiac output. From a hemodynamic perspective, central hypovolemia is clinically relevant when it is of sufficient severity to reduce venous return and ventricular preload to the extent that there is a threat to tissue perfusion. The determination of volume or preload sensitivity is a critical management issue for the patient in shock. If indicated, volume resuscitation may be beneficial; however, inappropriate volume resuscitation in the setting of adequate intravascular volume may be harmful to the patient.

Benefits of Fluid Resuscitation

When central hypovolemia is suspected, the clinical challenge is to assess whether volume resuscitation is needed. The expected benefits of volume expansion are to increase venous return, preload, SV, cardiac output, arterial blood pressure (systolic, mean, and pulse pressure), and tissue oxygen delivery. The rapidity with which these objectives are achieved during the management of hypovolemia constitutes a decisive prognostic element in terms of morbidity and mortality.

Adverse Effects of Fluid Resuscitation

Fluid infusion may be deleterious by increasing hydrostatic pressure, thereby causing pulmonary edema. This complication may be seen in patients with prexisting systolic or diastolic dysfunction. In these patients, LV diastolic pressure is high in basal conditions. Due to LV diastolic dysfunction, the LV pressure–volume relationship is steep. Consequently, a small amount of fluid infusion may increase LV diastolic pressure and precipitate acute pulmonary edema.

Fluid extravasation from the intravascular compartment to the interstitial compartment predisposes to the development of diffuse peripheral edema that

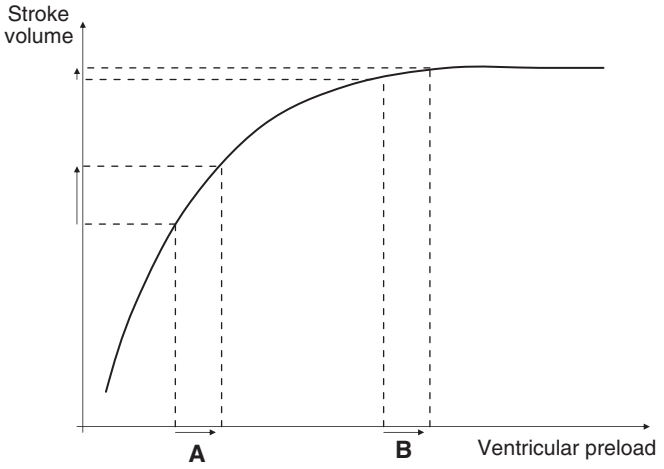


Figure 10.1. (A) The Frank-Starling curve indicating a patient with preload responsiveness; the increase of preload is followed by a significant increase of stroke volume, indicating that the patient is in the ascending part of the Frank-Starling curve. (B) A patient without preload responsiveness; the increase of preload is not followed by a significant increase of stroke volume, indicating that the patient is in the horizontal part of the Frank-Starling curve.

can compromise tissue oxygenation. Other potential complications of volume expansion are cerebral edema, especially in the context of neurological intensive care, and disorders of serum sodium, potassium, or chloride. In the context of uncontrolled hemorrhage, volume expansion can accentuate bleeding by increasing arterial or venous blood pressure. Specific risks inherent to resuscitation fluids are hyperglycemia in the case of glucose solutions, anemia, and clotting disorders due to hemodilution. Blood-derived products carry a risk of transmission of infectious agents, and colloid solutions can cause allergic reactions. In patients with acute respiratory distress syndrome (ARDS) liberal fluid management may be harmful.[1]

Volume resuscitation may also be harmful in patients with shock related to acute cor pulmonale (ACP), as volume infusion causes further right ventricular (RV) enlargement and LV compression, thus worsening the shock state (see Chapter 11). This has particular relevance to the frontline intensivist, as ACP is readily recognized by the clinician with basic-level echocardiography skills.

Given the potential benefits and hazards of volume resuscitation, the intensivist must answer a key question: should the patient receive additional volume infusions? This introduces the concept of the volume-responsive versus volume-nonresponsive patient.

Responders and Nonresponders

Investigators have had to develop a standard definition of volume responsiveness. In the discussion that follows, cited studies were performed using a similar experimental design in patients with hemodynamic failure related primarily to sepsis. Before any volume infusion, one or more parameters were measured that might predict volume responsiveness. After these measurements, patients received fluid expansion. Cardiac output was measured before and after fluid infusion. Most studies considered that an increase >15% of cardiac output defined the volume-responsive patient. In contrast, an increase of <15% defined the nonresponsive patient. The pattern of response can then be correlated with the parameters that were measured before the volume infusion to determine whether a particular parameter predicted volume responsiveness. Investigators have examined parameters of volume responsiveness both in patients who were on mechanical ventilation and completely passive in their interaction with the ventilator and in patients who had spontaneous respiratory effort, whether on mechanical ventilation or breathing without assistance.

The Concept of Fluid Challenge versus Fluid Responsiveness

In resuscitating the patient with hemodynamic failure, the clinician may pursue two different strategies. One approach is to give a fluid challenge to the patient without measuring any parameter that is predictive of fluid responsiveness. This traditional method uses the measures of static pressures such as central venous pressure (CVP) or pulmonary artery occlusion pressure (PAOP) prior to a volume challenge. The clinician then follows these parameters and serial SV and cardiac output measurements after the fluid challenge.

An alternative approach is to provide volume resuscitation guided by parameters that predict positive hemodynamic response. Using this approach, the clinician administers volume only if the patient is determined to be volume responsive. Volume resuscitation may be unnecessary and even harmful to the patient. It is therefore important to determine whether the patient is volume responsive *before* giving a volume challenge. In our view, the determination of volume responsiveness is a key aspect to the management of hemodynamic failure. Investigators have examined a variety of static and dynamic measurements to identify parameters that are useful in predicting volume responsiveness.

THE CONCEPT OF STATIC PARAMETERS

A static parameter is measured under a single ventricular loading condition and is presumed to reliably estimate the preload of one or both ventricles. This estimation of preload can then be used to evaluate the probability of responsiveness to ventricular filling, by assuming that lower preload increases the probability of response to volume expansion. Several static indices of ventricular preload have been analyzed in the ICU, such as PAOP, CVP, and right and left ventricular end-diastolic volume size.

Static Parameters Measured While on Mechanical Ventilation

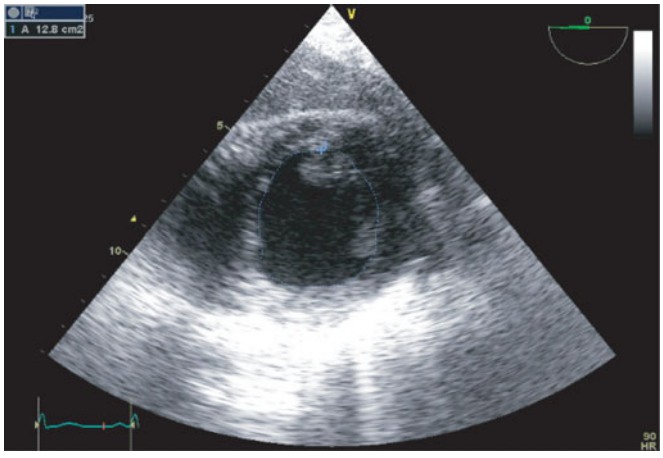
The CVP may be assessed by measuring the size of inferior vena cava (IVC) by transthoracic echocardiography. If the patient is on mechanical ventilation, the correlation between IVC size and CVP is low.[2–5] Feissel et al. demonstrated that, in general, absolute IVC size failed to predict fluid responsiveness in patients under mechanical ventilation in septic shock; however, an IVC diameter <10 millimeters (mm) may predict a positive response to fluid infusion.[5] Direct measurement of CVP is easily performed if a central venous catheter is in place. However, CVP is a poor predictor of volume responsiveness.[6]

The PAOP may be assessed by use of a pulmonary artery catheter. Alternatively, echocardiography may be used to determine PAOP noninvasively by Doppler indices derived from mitral flow (E/A ratio), pulmonary venous flow, tissue Doppler (E/Ea ratio), and color-coded Doppler (E/Vp ratio).[7–11] In general, PAOP fails to demonstrate any relationship with increases in cardiac output after fluid expansion and is therefore not useful to predict fluid responsiveness.[12] The only exception is when the PAOP is <5 mm Hg; this is an uncommon event in ICU practice.

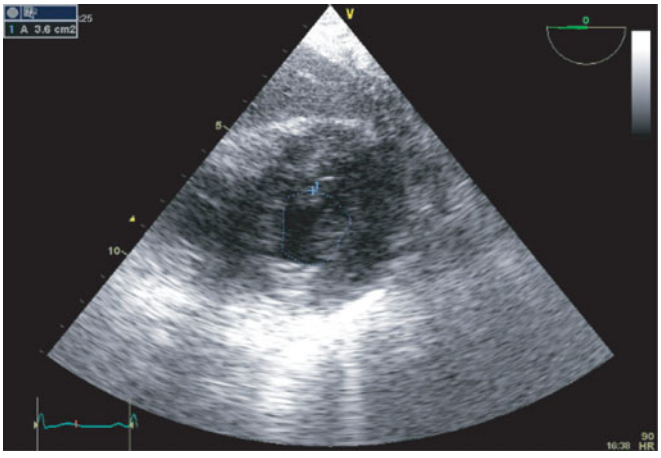
The RV and LV diastolic diameter, area, or volume are reliable parameters of preload. Tavernier et al. and Feissel et al. have demonstrated that LV size is a useful predictor of fluid responsiveness in patients on mechanical ventilation, particularly if the LV is very small and hyperkinetic.[13,14] This corresponds to a low preload and therefore predicts a positive response to fluid infusion (Figure 10.2).

Static Parameters Measured with Spontaneous Breathing

In spontaneously breathing patients, there is a relationship between the size of the IVC and CVP. Moreover, respiratory variations of IVC >50% correspond to CVP <10 mm Hg. However, CVP, PAOP, left ventricular end-diastolic volume (LVEDV), and right ventricular end-diastolic volume (RVEDV) do not predict volume responsiveness. In a study by Kumar et al. on healthy subjects, static indices of ventricular preload (CVP, PAOP, LVEDV index, RVEDV index) and cardiac performance indices (cardiac index, stroke volume index) were measured before and after 3 liters of normal saline loading.[6] There was no correlation between changes in



A



B

Figure 10.2. Measurements of left ventricular end-diastolic (LVEDA)(A) and end-systolic area (LVESA)(B) with transesophageal echocardiography in a short-axis view. The quasi virtual LVESA (3.6 cm^2) is indicative of decreased left ventricular filling pressure.

CVP and PAOP and variations of cardiac performance indices (cardiac index, stroke volume index). Similarly, no correlation was observed between baseline measurements of static indices and variations of cardiac performance indices after fluid loading. Coudray et al. reviewed five studies on mixed populations of patients (mechanically ventilated and spontaneously breathing) and demonstrated the absence of correlation between the initial PAOP and crystalloid infusion (an average of 1 liter).[15] The cardiac index was increased by >15% in nine subjects. No correlation was demonstrated between PAOP measured at baseline and the variation of the cardiac index.

In conclusion, standard indices of preload are not generally useful in predicting volume responsiveness. The exception occurs in those patients with very low parameters of preload. Unfortunately, low values are uncommon in the ICU. Rather than relying on determination of preload as a static measurement, the intensivist should have a more effective strategy to identify the patient who is volume responsive. Dynamic parameters of volume responsiveness are the best available alternative.

THE CONCEPT OF DYNAMIC PARAMETERS

Dynamic parameters are used to determine whether the patient is situated on the ascending portion of the Frank-Starling curve (where a variation of preload induces a variation of SV, i.e., a preload-dependent situation) or on the plateau portion (where a variation of preload does not induce a variation of stroke volume, i.e., a preload-independent situation) (Figure 10.1). Several approaches can be used to determine on what portion of the preload/stroke volume relationship the ventricle is functioning to establish the diagnosis of preload dependence or independence. The effect of ventilator cycling on intrathoracic pressures and stroke volume is the basis for many of the dynamic measurements that are useful in determining volume responsiveness.

The pathophysiology of cardiorespiratory interactions has been studied in sedated, ventilated patients. During insufflation, increased intrathoracic pressure and transpulmonary pressure (alveolar pressure–pleural pressure) results in a reduction in preload and an increase of RV afterload. These two phenomena lead to a reduction of RV stroke volume at the end of insufflation only if the ventricle is working on the steep part of the Frank-Starling relationship. This reduction is responsible, 2–3 cardiac cycles later (pulmonary transit time), for a reduction of the LV preload and LV stroke volume during exsufflation only if this ventricle is working on the steep part of the Frank-Starling relationship.

Alternatively, in spontaneously breathing patients, on inspiration the reduction of intrathoracic pressure and the decrease of transpulmonary pressure are responsible for an increase of RV preload and a moderate decrease of RV afterload, respectively. The increase of the RV stroke volume at the end of inspiration, 2–3 cardiac cycles later, results in an expiratory increase of left ventricular preload and stroke volume. These phenomena are accentuated when the ventricles are in a preload-dependence situation.

During mechanical ventilation, variations of intrathoracic pressure induced by insufflation decrease the biventricular preload and decrease the stroke volume when the ventricles are functioning on the steep portion of the Frank-Starling relationship. This is reflected by cyclic respiratory changes of stroke volume (Figure 10.3). The presence of these cyclic variations indicates that any variation of preload would induce a variation of stroke volume, and that volume expansion would increase SV and cardiac output (i.e., a preload-dependent situation). The absence of these variations indicates that any variation of preload would not modify stroke volume; and that consequently, volume expansion would not induce a significant increase in cardiac output (i.e., a preload-independent situation). This principle is the basis for the use of arterial pulse pressure variation to determine volume responsiveness.[16] Heart–lung interactions form the basis for some of the echocardiographic methods that are useful in determining preload dependence.

Echocardiography: Dynamic Parameters Measured in Patients on Mechanical Ventilation

Analysis of the respiratory changes of LV stroke volume during mechanical ventilation provides a dynamic, biventricular evaluation of preload dependence. The respiratory changes of stroke volume can be estimated by esophageal Doppler, by transesophageal echocardiography (TEE, or by transthoracic echocardiography (TTE). Slama and colleagues demonstrated in an animal study that progressive blood withdrawal is closely related to increased respiratory variation of aortic blood flow.[17] In clinical studies, maximal aortic blood flow velocity or velocity-time integral (VTI) variation

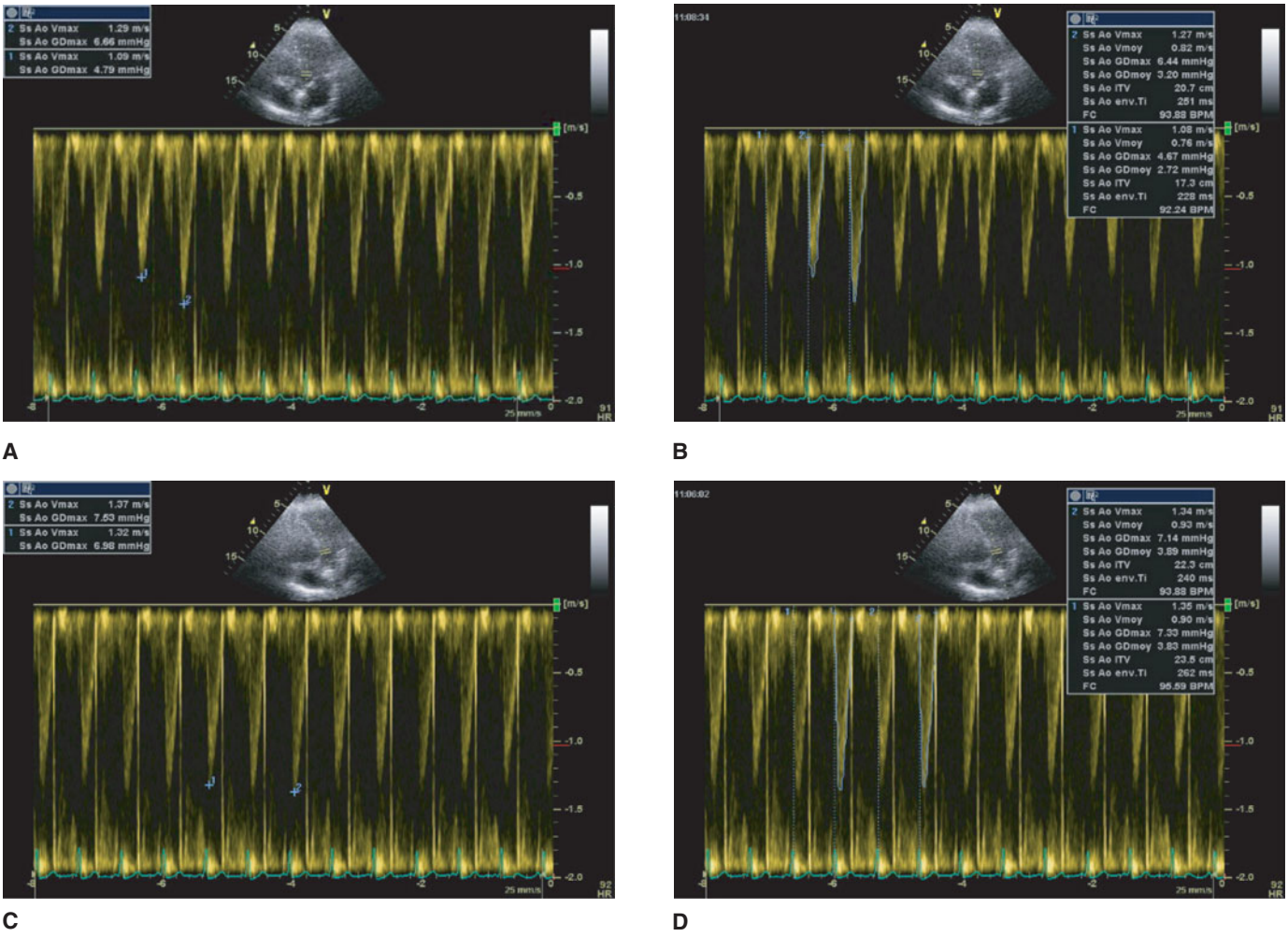
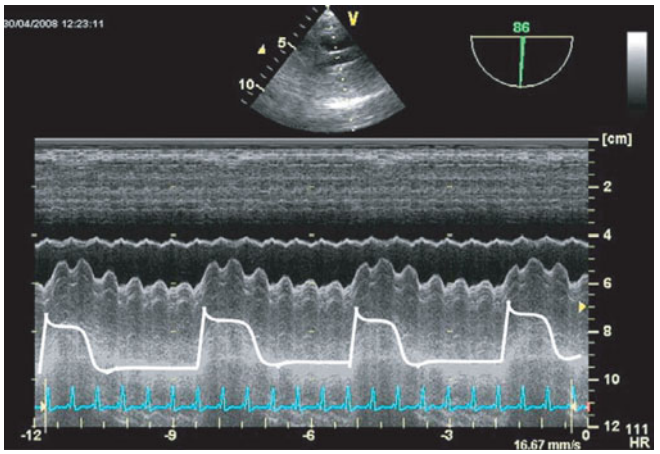


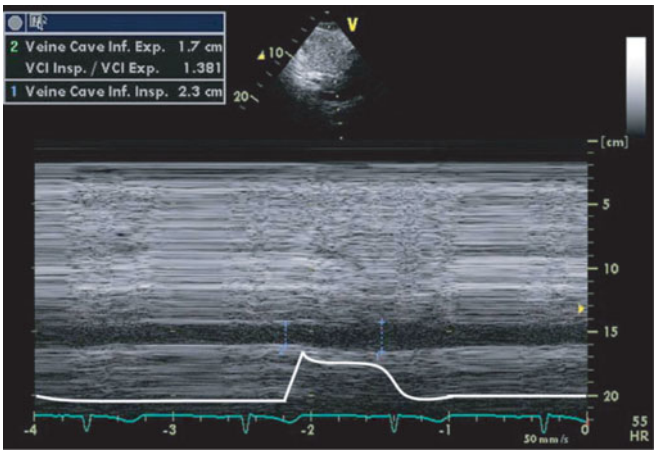
Figure 10.3. Respiratory variations of maximal velocity (Vmax) (A and C) and VTI (B and D) of aortic blood flow recorded with a pulsed Doppler transthoracic echocardiography in a mechanically ventilated patient. (A and C) Presence of significant respiratory variations of Vmax (Vmax − Vmin/[Vmax + Vmin/2]; 1.29 − 1.09/1.19 = 17%) and VTI (VTImax − VTImin/[VTImax + VTI min/2]; 20.7 − 17.3/19 = 18%). (B and D) Same patient after volume expansion, regression of the respiratory variations: Vmax (1.37 − 1.32/1.34 = 4%), VTI (23.5 − 22.3/22.9 = 5%).

measured with TEE predict, with high sensitivity, specificity, and predictive value, increases in cardiac output after fluid infusion in patients with shock. A cutoff value of 12% for maximal velocity and 20% for respiratory cycle changes of aortic VTI discriminated responder from nonresponder patients.[14,18] The same values were found by using an esophageal Doppler probe to assess aortic blood flow in the descending aorta.[19] Another approach to identify volume responsiveness used 2D echocardiography. Cannesson et al. assessed LV diastolic area (LVDA) changes by TEE from short-axis view.[20] They found that a 16% respiratory variation of LVDA between inspiration and expiration predicted fluid responsiveness with a sensitivity of 92% and a specificity of 83%. The change of area at baseline was related to the percentage increase in cardiac output in response to volume expansion. Using the same principle, IVC and superior vena cava (SVC) diameter changes during mechanical ventilation were measured to predict fluid responsiveness. Inferior vena cava diameter was analyzed from a longitudinal subcostal view and recorded by using M-mode (Figure 10.4). Superior vena cava was recorded from TEE longitudinal view at 90–100°. Cutoff values of 12% (by using max–min/mean value) and 18% (by using max–min/min) for IVC (distensibility index) and 36% for SVC (sensitivity 90%, specificity 100%) (collapsibility index) were found to accurately separate responders and nonresponders.[5,21,22]

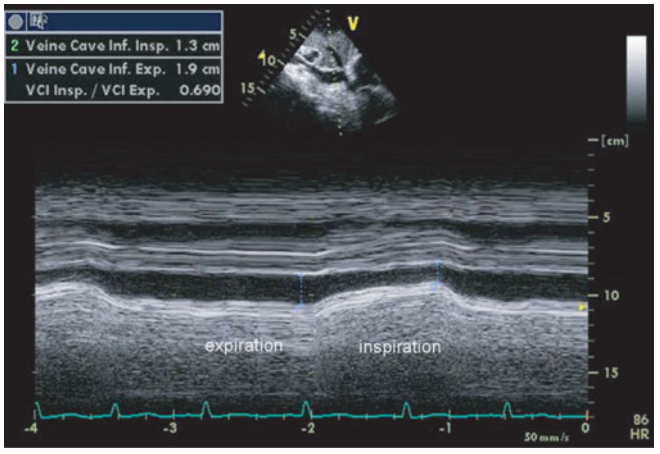
The described methods have value in the determination of volume responsiveness using echocardiography. However, they have significant methodological limitations:



A



B



C

Figure 10.4. Respiratory vena cava variations in different circumstances. (A) Significant superior vena cava collapsibility recorded with transesoephageal echocardiography. (B) significant inferior vena cava distensibility recorded with transthoracic echocardiography in a mechanically ventilated patient. (C) Significant vena cava collapsibility recorded with transthoracic echocardiography in a spontaneously breathing patient.

1. All require that the patient be on mechanical ventilation and passive in their interaction with the ventilator.
2. The patient can make no spontaneous breathing effort during the measurement and must be in a regular heart rhythm.
3. The degree of respiratory variation is contingent on the change of intrathoracic pressure.
4. Tidal volume and positive end-expiratory pressure (PEEP) levels are known to influence pulse pressure variation, so they will clearly have a similar effect on echocardiographic measurements of stroke volume independent of preload dependence.

These methods have been studied in patients with sepsis, but they have not been extensively examined in patients with coexisting heart disease. This is of particular concern in patients with RV dysfunction. Patients with dilation of the RV may have false-positive respiratory changes of SV assessed by arterial pulse pressure variations. In these patients, respiratory changes of SV seem to be due to RV afterload effect and do not reflect a need for fluid resuscitation. Therefore, RV size should be assessed before any fluid challenge in patients with respiratory variations of SV, aortic VTI, or maximal velocity of aortic blood flow. Finally, esophageal Doppler measurement of flow in the descending aorta is limited by the fact that the diameter of the aorta changes according to the volume status of the patient. Changes in aortic flow velocity or VTI must be adjusted for any concomitant change in aortic diameter that may have occurred due to changes in volume status.[23]

There is a close relationship between all of the described parameters and increase in cardiac output after fluid infusion. Therefore, large respiratory changes predict a large increase in cardiac output after fluid infusion. All these parameters were validated on a selected population of patients who were mechanically ventilated without spontaneous breathing effort. It is necessary to consider whether there may be dynamic parameters that predict preload dependence in patients making spontaneous breathing effort.

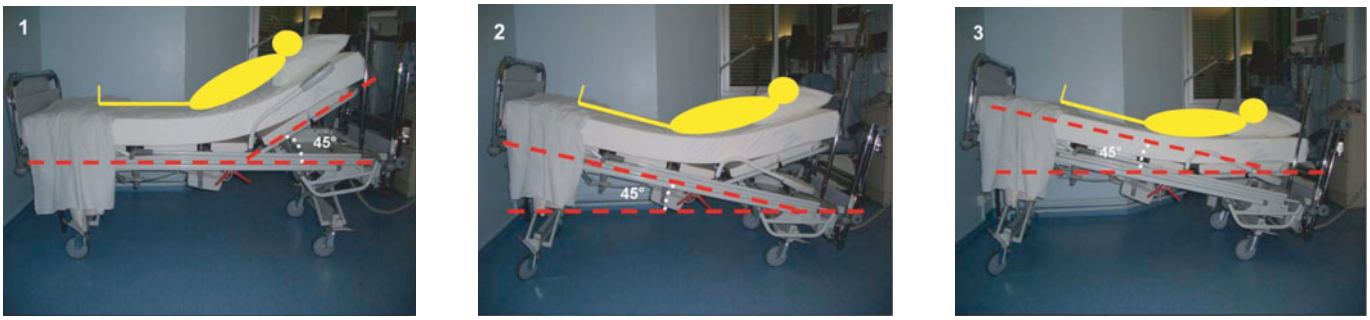


Figure 10.5. The realization of a passive leg raising maneuver in three steps: step 1, at baseline the patient is laying in a semirecumbent position, the trunk of the patient at 45° up to the horizontal; step 2, the entire bed is pivoted to obtain a head down tilt at 45°; and step 3, the head of the bed is adjusted to obtain a strictly horizontal trunk.

Echocardiography: Dynamic Parameters with Spontaneous Breathing

Recent publications have proposed the passive leg raising (PLR) test as an alternative to a fluid challenge test to predict preload dependence (Figure 10.5). This maneuver rapidly mobilizes about 300 mL of blood from the lower limbs to the intrathoracic compartment and reproduces the effects of volume expansion. It is reversible and devoid of any risks of volume expansion. The test consists of raising both legs of the supine patient to an angle of 45° in relation to the bed while measuring SV and cardiac output before and approximately one minute following the PLR maneuver. This is readily accomplished by measuring the VTI of the aortic outflow velocity envelope with either TTE (five-chamber view) or TEE (deep-gastric view).

Boulain et al. studied the hemodynamic effects of PLR in 15 sedated and mechanically ventilated ICU patients with acute circulatory insufficiency.[24] In this study, PLR induced a significant increase of pulse pressure measured at the radial artery, PAOP, and SV. The effects of volume expansion on SV were related to those of PLR on pulse pressure. The intensity of the effect of PLR on SV was correlated with the effect of PLR on pulse pressure. Volume expansion was performed with 300 mL of macromolecular solution over 20 minutes.

Lafanechère et al. analyzed 22 patients by examining esophageal Doppler aortic blood flow (ABF) changes on descending aorta induced by PLR in patients under mechanical ventilation.[25] In responders, the increase in ABF induced by PLR was similar to that induced by a 250 mL volume expansion. A PLR-induced increase in ABF of more than 8% predicted fluid responsiveness with a sensitivity of 90% and a specificity of 83%. Corresponding positive and negative predictive values were 82% and 91%, respectively.

In another study using esophageal Doppler to measure aortic blood flow, Monnet et al. showed that, when PLR induced an increase of aortic flow of >10%, it was predictive of an increase of aortic flow of >15% in response to volume expansion (sensitivity: 97%; specificity: 94%).[26] The patients included in this study presented signs of acute circulatory insufficiency (hypotension, tachycardia, oliguria, mottled skin). Volume expansion was performed with 500 mL of isotonic saline over 10 minutes. Thirty-seven (52%) of the 71 patients included in this study responded to volume expansion, and 22 subjects had spontaneous breathing movements (spontaneous breathing mode with inspiratory assistance). The authors showed that respiratory cyclic variations of pulse pressure ≥12% in response to the PLR test were predictive of an increase of aortic flow by >15% in response to volume expansion (sensitivity: 88%; specificity: 93%).

In two recent studies, aortic VTI, stroke volume and cardiac output were recorded by using transthoracic echocardiography in spontaneously breathing patients during PLR. Lamia et al. demonstrated that in 24 patients PLR-induced increase in stroke volume of 12.5% or more predicted an increase in stroke volume of 15% or more after volume expansion, with a sensitivity of 77% and a specificity of 100%.[27] In this study, static indices of preload such as left ventricular end-diastolic area or E/Ea did not predict volume responsiveness. Patients were intubated with spontaneous breathing movements. In the study of Maizel et al., 34 patients were spontaneously breathing without tracheal tube.[28] An increase of cardiac output or stroke volume by >12% during PLR was highly predictive of central hypovolemia. Sensitivity and specificity values were 63% and 89% for cardiac output

and 69% and 89% for stroke volume, respectively. A close correlation was observed between cardiac output changes during PLR and changes in cardiac output after fluid expansion. Of particular note is that these studies have demonstrated that PLR may be used to predict volume responsiveness in patients with atrial fibrillation.

The PLR approach to determination of volume responsiveness is an elegant solution to a difficult problem. It answers the question of whether the patient will have augmentation of cardiac output following volume resuscitation in a direct and unambiguous fashion. Approximately 300 cc of blood is delivered to the thoracic compartment very rapidly, while the effect of this rapid volume challenge is measured with real-time echocardiography. The test avoids many of the problems found with dynamic parameters measured during ventilator cycling, and does not expose the patient to the risks of inappropriate volume resuscitation.

Bedside Application of Echocardiographic Parameters

Echocardiography is a very useful tool for the evaluation of the patient with hemodynamic failure. In treating the patient with shock, the intensivist echocardiographer will first evaluate for major cardiac disease that requires prompt intervention (e.g., pericardial tamponade, severe valve failure, acute cor pulmonale pattern, segmental wall dysfunction suggesting acute myocardial infarction). If these are excluded, the focus of the echocardiographer may then shift to assessment and management of hemodynamic function. Should the patient in shock receive volume resuscitation, inotropes, pressors, or some combination thereof? Once a decision has been made, echocardiography may then be used to monitor the results of therapeutic intervention, the course of the disease, and to look for new problems. How should the frontline intensivist use dynamic parameters of volume responsiveness to guide fluid therapy?

An important issue to consider is that echocardiography is only useful within the overall clinical context of the case. Normal individuals without any hemodynamic compromise are volume responsive, as normal subjects are positioned on the steep preload dependent part of the Frank-Starling curve. Patients who exhibit echocardiographic evidence of preload dependence should receive volume resuscitation only if they have evidence of clinically significant hemodynamic failure. Echocardiography should always be integrated into the overall clinical picture. Another example of this principle is that the presence of severe LV failure on echocardiography does not necessarily require therapeutic response if the patient is otherwise stable hemodynamically. Therefore, echocardiographic evidence of volume responsiveness does not warrant volume resuscitation unless the clinician identifies hemodynamic failure that might improve by augmenting cardiac output.

There are clinical situations where the need for immediate volume resuscitation is obvious, such as massive hemorrhage, severe dehydration due to gastrointestinal diseases, major 3rd space losses, or obvious septic shock. Clinical context and physical examination allow the recognition of severe central hypovolemia, where immediate volume resuscitation is appropriate. Initial resuscitation is generally accomplished to some extent before transfer to the ICU. The intensivist must then answer the question as to whether further volume resuscitation should continue. It is our opinion that echocardiographic indices of volume responsiveness are particularly useful where there is clinical ambiguity as to whether the patient should have further volume resuscitation in the ICU.

By definition, intensivists with proficiency in basic critical care echocardiography have limited Doppler capability; they cannot measure variation of SV with ventilator cycling nor changes in SV or cardiac output before and after PLR. However, respiratory variation of IVC size (limited to the completely passive patient on mechanical ventilation in a regular heart rhythm) is a validated method of determining volume responsiveness that is easily mastered by the basic critical care echocardiographer. There are pitfalls to this method. In addition to the need to obtain a good quality image, translational artifact may be a problem. As the ventilator cycles, it displaces the liver and adjacent IVC. The IVC may appear to change in size, when it simply is moved out of the ultrasound beam plane. In this case, there is no actual change is IVC size, even though there is the appearance of such. The intensivist with basic critical care echocardiography skill level can also identify a small hyperdynamic LV (with effacement of the end-systolic LV cavity) or a very small IVC diameter ($>$10 mm). Both patterns strongly suggest volume responsiveness in the patient with shock.

The intensivist with proficiency in advanced critical care echocardiography has full training in Doppler measurements as well as TEE capability. Regarding the latter, respiratory variation of SVC diameter has similar application to IVC variation. It is easy to obtain with TEE and is well validated. The intensivist should consider it as a straightforward method of determining volume

responsiveness in the passive mechanically ventilated patient.

The intensivist may also choose to measure respiratory variation of aortic outflow velocity to determine volume responsiveness. The major problem with this approach relates to the possibility of inadequate image quality with TTE (easily remedied with TEE), and translational artifact related to respiratory cycling. In this case, respiratory cycling may alter cardiac position to the extent that there is significant change in the angle or position of the Doppler interrogation that varies during the respiratory cycle. Changes in velocity or VTI may be ascribed to changes in SV, when they actually derive from changes in the angle of interrogation. Fortunately, the left ventricular outflow tract (LVOT) diameter does not change during the respiratory cycle nor is it influenced by the volume status of the heart (unlike the descending aorta). Its being a constant value is a strength of the method, as changes in velocity or VTI reflect true changes in SV.

Passive leg raising is an attractive alternative for the advanced-level critical care echocardiographer. The pulsed-wave Doppler box is placed in the LVOT from the TTE apical five-chamber view (or deep-gastric view with TEE), and VTI is recorded for several heart beats. Both legs are raised to 45°, and a minute later the measurement is repeated. Appropriate calculations using the LVOT measured with 2D technique yield SV and cardiac measurements before and after the reversible volume challenge engendered by the PLR maneuver. Alternatively, as LVOT diameter is a constant, the percent change in VTI or maximal outflow velocity may be used in order to simplify calculations. The main problem with the PLR relates to the time required to perform the test. An unresolved technical detail of PLR is that the original reports describe the initial position of the patient to be in a semirecumbent 45° position. The bed design then allowed the patient to be shifted to a supine position with the legs at a 45° elevation fully supported by the structure of the bed and mattress. Lacking a specialized bed, an alternative method is to start the patient in supine position, and then to manually lift the legs to a 45° angle. This requires one person to be assigned to lift each leg. This method has not been validated, but appears to be a practical bedside approach in the absence of sophisticated bed design.

CONCLUSION

Echocardiography provides the intensivist with several methods to determine volume responsiveness in patients with hemodynamic failure. The clinician with basic skills in critical care echocardiography may use respiratory variation of IVC diameter, a small IVC, or a small hyperdynamic LC to identify preload dependent patients. The intensivist with a more advanced skill level may use respiratory variation of SV determined by echocardiography and changes in SV following the PLR maneuver to identify volume responsiveness. In general, dynamic parameters determined by echocardiography are superior to static measurements of preload for the determination of volume responsiveness.

References

1. National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network, Wiedemann HP, Wheeler AP, et al. Comparison of two fluid-management strategies in acute lung injury. *N Engl J Med.* 2006;354:2564–2575.
2. Nagueh SF, Kopelen HA, Zoghbi WA. Relation of mean right atrial pressure to echocardiographic and Doppler parameters of right atrial and right ventricular function. *Circulation.* 1996;93:1160–1169.
3. Simonson JS, Schiller NB. Sonospirometry: a new method for noninvasive estimation of mean right atrial pressure based on two-dimensional echographic measurements of the inferior vena cava during measured inspiration. *J Am Coll Cardiol.* 1988;11:557–564.
4. Kircher BJ, Himelman RB, Schiller NB. Noninvasive estimation of right atrial pressure from the inspiratory collapse of the inferior vena cava. *Am J Cardiol.* 1990;66:493–496.
5. Feissel M, Michard F, Faller JP, et al. The respiratory variation in inferior vena cava diameter as a guide to fluid therapy. *Intensive Care Med.* 2004;30:1834–1837.
6. Kumar A, Anel R, Bunnell E, et al: Pulmonary artery occlusion pressure and central venous pressure fail to predict ventricular filling volume, cardiac performance, or the response to volume infusion in normal subjects. *Crit Care Med.* 2004;32:691–699.
7. Appleton CP, Galloway JM, Gonzalez MS, et al. Estimation of left ventricular filling pressures using two-dimensional and Doppler echocardiography in adult patients with cardiac disease. Additional value of analyzing left atrial size, left atrial ejection fraction and the difference in duration of pulmonary venous and mitral flow velocity at atrial contraction. *J Am Coll Cardiol.* 1993;22:1972–1982.
8. Giannuzzi P, Imparato A, Temporelli PL, et al. Doppler-derived mitral deceleration time of early filling as a

strong predictor of pulmonary capillary wedge pressure in postinfarction patients with left ventricular systolic dysfunction. *J Am Coll Cardiol.* 1994;23:1630–1637.

9. Kuecherer HF, Muhiudeen IA, Kusumoto FM, et al. Estimation of mean left atrial pressure from transesophageal pulsed Doppler echocardiography of pulmonary venous flow. *Circulation.* 1990;82:1127–1139.
10. Combes A, Arnoult F, Trouillet JL. Tissue Doppler imaging estimation of pulmonary artery occlusion pressure in ICU patients. *Intensive Care Med.* 2004;30:75–81.
11. Gonzalez-Vilchez F, Ares M, Ayuela J, et al. Combined use of pulsed and color M-mode Doppler echocardiography for the estimation of pulmonary capillary wedge pressure: an empirical approach based on an analytical relation. *J Am Coll Cardiol.* 1999;34:515–523.
12. Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. *Chest.* 2002;121:2000–2008.
13. Tavernier B, Makhotine O, Lebuffe G, et al. Systolic pressure variation as a guide to fluid therapy in patients with sepsis-induced hypotension. *Anesthesiology.* 1998;89:1313–1321.
14. Feissel M, Michard F, Mangin I, et al. Respiratory changes in aortic blood velocity as an indicator of fluid responsiveness in ventilated patients with septic shock. *Chest.* 2001;119:867–873.
15. Coudray A, Romand JA, Treggiari M, et al. Fluid responsiveness in spontaneously breathing patients: a review of indexes used in intensive care. *Crit Care Med.* 2005; 33:2757–2762.
16. Michard F, Boussat S, Chemla D, et al. Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. *Am J Respir Crit Care Med.* 2000;162:134–138.
17. Slama M, Masson H, Teboul JL, et al. Respiratory variations of aortic VTI: a new index of hypovolemia and fluid responsiveness. *Am J Physiol Heart Circ Physiol.* 2002; 283:H1729–H1733.
18. Charron C, Fessenmeyer C, Cosson C, et al. The influence of tidal volume on the dynamic variables of fluid responsiveness in critically ill patients. *Anesth Analg.* 2006; 102:1511–1517.
19. Monnet X, Rienzo M, Osman D, et al. Esophageal Doppler monitoring predicts fluid responsiveness in critically ill ventilated patients. *Intensive Care Med.* 2005;31:1195–1201.
20. Cannesson M, Slieker J, Desebbe O, et al. Prediction of fluid responsiveness using respiratory variations in left ventricular stroke area by transoesophageal echocardiographic automated border detection in mechanically ventilated patients. *Crit Care.* 2006;10:R171.
21. Barbier C, Loubières Y, Schmit C, et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responsiveness in ventilated septic patients. *Intensive Care Med.* 2004;30:1740–1746.
22. Vieillard-Baron A, Augarde R, Prin P, et al. Influence of superior vena caval zone condition on cyclic changes in right ventricular outflow during respiratory support. *Anesthesiology.* 2001;95:1083–1088.
23. Monnet X, Chemla D, Osman D, et al. Measuring aortic diameter improves accuracy of esophageal Doppler in assessing fluid responsiveness. *Crit Care Med.* 2007;35:477–482.
24. Boulain T, Achard JM, Teboul JL, et al. Changes in BP induced by passive leg raising predict response to fluid loading in critically ill patients. *Chest.* 2002;121:1245–1252.
25. Lafanechère A, Pène F, Goulenok C, et al. Changes in aortic blood flow induced by passive leg raising predict fluid responsiveness in critically ill patients. *Crit Care.* 2006; 10:R132.
26. Monnet X, Rienzo M, Osman D, et al. Passive leg raising predicts fluid responsiveness in the critically ill. *Crit Care Med.* 2006;34:1402–1407.
27. Lamia B, Ochagavia A, Monnet X, et al. Echocardiographic prediction of volume responsiveness in critically ill patients with spontaneously breathing activity. *Intensive Care Med.* 2007;33:1125–1132.
28. Maizel J, Airapetian N, Lorne E, et al. Diagnosis of central hypovolemia by using passive leg raising. *Intensive Care Med.* 2007;33:1133–1138.

CHAPTER 11

Echocardiographic Diagnosis and Monitoring of Right Ventricular Function

Adolfo Kaplan

INTRODUCTION

Right ventricular (RV) dysfunction is common in critically ill patients.[1–3] It is associated with multiple clinical scenarios frequently encountered by the intensivist, including acute cor pulmonale, acute RV dysfunction of sepsis, and acute RV infarction. In addition, the assessment of RV function is essential to determination of a patient's preload responsiveness. Echocardiography is the best available method to diagnose and monitor RV function at the bedside and provides the critically ill patient a prompt, accurate, noninvasive, and serial method to monitor the function of the right heart and its responsiveness to different clinical interventions. This chapter describes a variety of echocardiographic methods to assess RV function that are particularly relevant to critical care practice for both the novice and experienced echocardiographer. While the assessment of RV function in the noncritically ill patient is beyond the purview of this chapter, the techniques described here are also applicable to the assessment of RV function in the ambulatory patient.

NORMAL RV ANATOMY AND FUNCTION

The RV comprises two anatomically and functionally different cavities separated by the crista supraventricularis: an inflow region (the sinus) and an outflow tract (the cone or infundibulum). The tricuspid valve (TV) and its apparatus plus heavily trabeculated myocardium form the sinus. Smooth myocardium and the pulmonic valve form the infundibulum. The sinus generates pressure during systole while the infundibulum modulates this pressure and prolongs its duration. Right ventricular contraction occurs serially in three different phases: (a) contraction of the sinus along its longitudinal axis, (b) radial contraction of the RV free wall toward the interventricular septum (IVS), and (c) torsion of the left ventricle (LV) (clockwise rotation of the LV base with counterclockwise rotation of apex) pulling the RV in similar manner. Overall, LV contraction contributes 25% of its own stroke work to the generation of RV stroke work via the IVS. In pulmonary hypertension, this contribution increases to 35%.[4]

The normal RV is less muscular than the LV and has a free wall thickness 3.3 millimeters (mm). As a consequence, it is easily affected by its surroundings and the effects of increased afterload. Unlike the LV, it is able to acutely dilate. Relative to the LV, the RV is a lower pressure chamber, with normal pressures of approximately 30/10 centimeters of water (cm/H_2O) with the patient in the resting state. When afterload increases acutely, the RV is unable to correspondingly generate higher pressures. However, when subjected to chronic loading conditions, the RV compensates with a hypertrophic response that is suggested by a thickened RV free wall on echocardiography. In this situation, the RV can generate up to systemic-level pressures, such as is seen with advanced pulmonary arterial hypertension.

Acute Cor Pulmonale

Acute cor pulmonale (ACP) is defined as the clinical setting in which the RV experiences a sudden increase in afterload.[5] This may occur in the context of previously normal RV function or in the RV that is already impaired. Sudden increases in RV afterload occur frequently in critically ill patients (Table 11.1). Acute cor pulmonale is synonymous with acute right heart failure. It is characterized by the combination of systolic and diastolic overload, both of which have characteristic echocardiographic features.

TABLE 11.1. Causes of acute cor pulmonale

Acute left heart failure (ischemic, myocardial, or valvular origin)
Acute pulmonary embolism
Acute respiratory distress syndrome (ARDS)
Inappropriately adjusted ventilatory support
Respiratory and metabolic acidosis[3]
Fat emboli
Gas emboli
Low PaO2

Echocardiographic Features of RV Systolic Overload

Septal dyskinesia is the echocardiographic hallmark of a sudden elevation of RV systolic afterload and occurs because of ventricular interdependence. When RV afterload is increased, its contraction is prolonged, requiring a longer time for completion than the left ventricular systole. Because the RV is still contracting at the end of systole when the LV is beginning to relax, the right intraventricular pressure becomes transiently greater than the left intraventricular pressure, and the interventricular septum is displaced leftward.[6] When due to pulmonary vascular processes (e.g., acute respiratory distress syndrome [ARDS], pulmonary embolism), septal dyskinesia develops rapidly but, when due to LV dysfunction, occurs later in the course of disease. Septal dyskinesia can be assessed qualitatively and quantitatively. The qualitative evaluation includes the observation of paradoxical septal motion (Videos 11.1 and 11.2 in enclosed DVD). In most clinical situations, this approach will suffice. M-mode echocardiography is another useful method for examining septal movement (Figure 11.1).

Quantification of systolic RV overload can also be achieved by direct measurement of the systolic eccentricity index (EI). To do this, the short-axis view of the mid-LV (when both papillary muscles are displayed) is obtained via a transthoracic (TTE) or transesophageal (TEE) echocardiography. At end-systole, D1 is measured as the diameter that bisects the papillary muscles, and D2 is the orthogonal diameter to D1. The systolic eccentricity index = D2/D1. A normal systolic EI is 1. Septal dyskinesia will result in an EI >1 (Figure 11.2).[7] An atrial septal defect could, however, result in a falsely elevated EI.[8] Therefore, a search for intracardiac shunting should always be considered when the EI is >1 and the right clinical context appropriate.

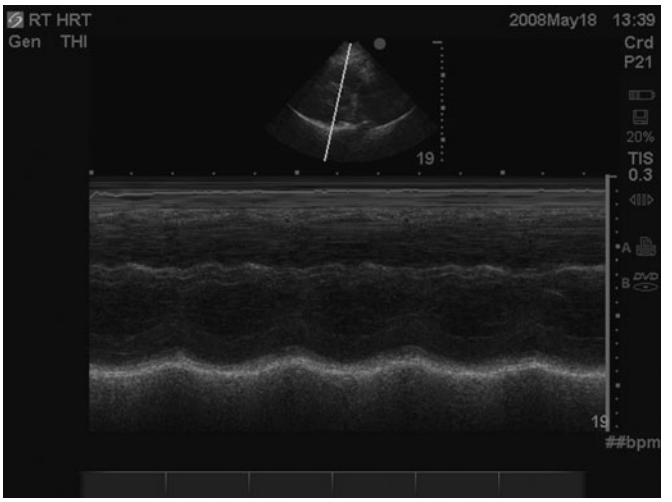


Figure 11.1. M-mode at the parasternal long-axis midventricular level showing paradoxical septal motion.

Systolic RV overload can also be indirectly identified by Doppler assessment of the right ventricular outflow tract (RVOT). RV systolic overload results in increased RV output impedance. It can be assessed by both TTE and TEE using pulsed-wave (PW) Doppler. The PW interrogation box is placed either above the pulmonic valve (by TTE or by TEE, using a transgastric view) or just below the valve (view of the great vessels by TEE). A spectral Doppler signal is obtained, allowing both qualitative and quantitative assessments of increased pulmonary vascular impedance. The normal RVOT spectral Doppler signal is easily discriminated from the biphasic pattern that occurs due to the reduction of velocity during midsystole when impedance is increased (Figure 11.3). Multiple quantitative measurements can be obtained. Table 11.2 summarizes the normal and

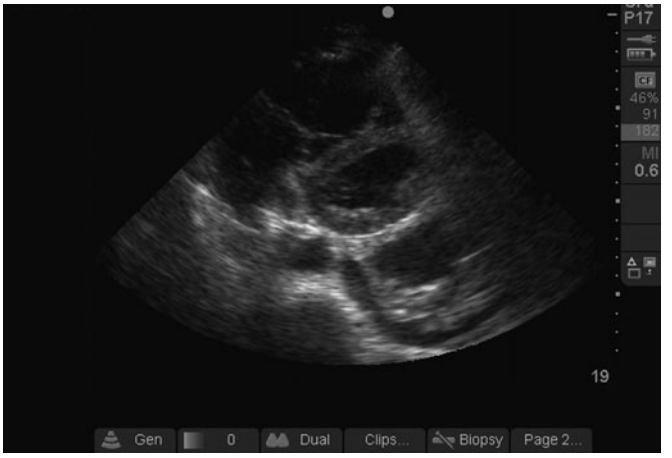


Figure 11.2. Parasternal short-axis midventricular view showing EI ratio >1. EI indicates eccentricity index.

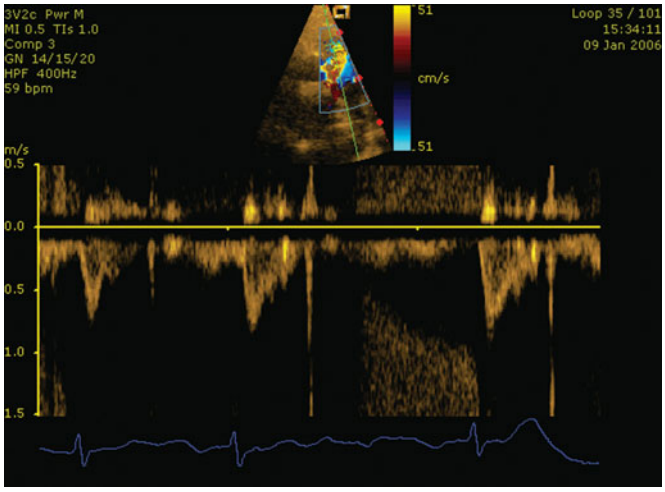


Figure 11.3. Pulsed-wave Doppler tracing of the RV outflow tract showing a biphasic pattern systolic velocity envelope. RV indicates right ventricle.

pathological measurements derived from analysis of the RVOT spectral Doppler signal. Unfortunately, these measurements have several limitations that frequently lead to high interobserver and intraobserver variability. These include:

- Poor TTE image quality
- Misalignment between the ultrasound beam and RVOT jet. Even with color Doppler guidance, the spectral Doppler signal may not reflect the spatial distribution of the pulmonary artery jet, which is higher along the inner edge of its natural curvature

TABLE 11.2. Quantitative RV output measurements

	Normal	*RV systolic overload*
Decreased stroke volume	70–100 mL	<70 mL
Decreased RVOT velocity-time integral (VTI)	18 ± 3 cm	<15 cm
Decreased acceleration time	≥120 m/sec	<80 m/sec
Decreased ejection time	304 ± 23 m/sec	<281 m/sec
Decreased acceleration time/ ejection time	>0.34[2]	<0.34

RV indicates right ventricular; RVOT, right ventricular outflow tract; VTI, velocity-time integral.

- Misinterpretation of the spectral Doppler signal; only the outer edge of the dark spectral envelope should be used
- Misinterpretation of the very short time intervals being measured

The assessment of RV contractile function can also be performed qualitatively and quantitatively. Frequently, the evaluation of RV systolic function by the intensivist is based upon the qualitative analysis of the RV free wall and IVS endomyocardial thickening and contraction. This is similar to the qualitative assessment of LV function. The contractile function of the RV is graded as normal, mild, moderate, or severely reduced. This approach is dependent upon the skills and experience of the interpreter. There are several quantitative techniques to assess RV contractile function. These include ejection fraction; fractional area of contraction; systolic tissue annular motion by 2D, M-Mode, or tissue Doppler; myocardial performance index; and the measurement of dP/dt. These measurements are not practical for use by the bedside critical care echocardiographer.

Echocardiographic Features of RV Diastolic Overload

Right ventricular diastolic overload is synonymous with RV dilation. There are multiple definitions of right heart dilation. Ideally, RV volume should be measured. However, 2D echocardiographic methods cannot accurately estimate RV volumes because of a failure of geometric models to appropriately reflect the complicated RV anatomy. To circumvent this problem, Jardin and colleagues have proposed a semiquantitative assessment of right heart diastolic overload.[1] They measured both ventricular areas at end-diastole by tracing the endomyocardium from the apical four-chamber view. When the endomyocardium was not well visualized, the area was traced to the epicardium. They found that the RV end-diastolic area to left ventricular end-diastolic area (RVEDA:LVEDA) ratio correlated well with RV dilation. The normal RVEDA:LVEDA ratio was found to be between 0.36 and 0.6 (0.48 ± 0.12). They defined moderate dilation as a ratio = 0.7–0.9, and severe dilation as a ratio ≥1 (Figure 11.4). Recently, another group confirmed the prognostic value of end-diastolic area (measured at the beginning of the QRS complex) RVEDA:LVEDA in patients with acute pulmonary embolism.[9] In their registry of 1416 hospitalized patients with acute pulmonary embolism, 31 subjects (3.3%) died. Among patients with systolic blood pressure ≥90 mm Hg, the mortality rate was 3.3% for those

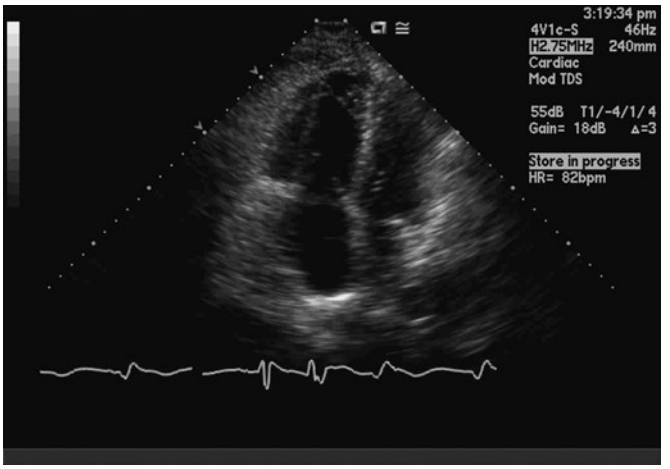


Figure 11.4. Apical four-chamber view showing a severely dilated right ventricle (RV/LV ratio >1). LV indicates left ventricle; RV, right ventricle.

with a RV/LV ratio ≥0.6, and 1.1% for those with a ratio <0.6. Using mortality receiver operating curves (ROC) curves, an RV/LV ratio ≥0.9 had the best sensitivity (72%) and specificity (58%) to discriminate between those patients with the highest risk of dying: 6.6% if the ratio ≥0.9, 1.9% if <0.9. Interestingly, Fremont and colleagues measured chamber size in the parasternal or subcostal views, which is important because the subcostal plane is frequently the most acceptable TTE view in critically ill patients on ventilatory support (Figure 11.5, and Video 11.3 in enclosed DVD).

The assessment of RV size in comparison with LV size has many advantages, including its simplicity, avoidance of individual variations in cardiac size, and the reduced interobserver and intraobserver variability introduced by quantitative methods.[1] Vieillard-Baron et al. recently evaluated the accuracy of qualitative versus quantitative assessments of several echocardiographic parameters by TEE, as performed by intensivist-echocardiographers.[10] The authors subjectively classified RV end-diastolic size in comparison with the LV end-diastolic size in the apical four-chamber view as normal, moderately enlarged, and markedly enlarged. The subjective qualitative observation of chamber size correlated well with the quantitative assessment of the RV/LV ratio. In addition, the interobserver variability was very good (K = 0.74, 95%; CI 0.54–0.94). Their results indicate that a trained echocardiographer can readily identify RV diastolic overload with an "eyeball" assessment.

A sudden increase in RV diastolic afterload will not only result in its dilation, but also in a change of its normal configuration. When enlarged, the RV loses its triangular shape and becomes more rounded in the apical views. An oval, instead of half-moon–like, shape is observed both on short- and long-axis views (Fig ure 11.6).

Right heart dilation results in reduced LV filling. Because the pericardium encloses the heart within a relatively stiff envelope, the right heart can only dilate at the expense of the space normally occupied by the larger LV. This leads to an impairment of LV filling. Pulsed-wave Doppler interrogation of mitral valve inflow displays a spectral signal that reverses when impaired filling is present (E/A <1).

The critical care echocardiographer should be aware that there are published guidelines on RV chamber quantification by the American Society of Echocardiography (ASE) in conjunction with the European Association of Echocardiography (EAE).[11] They are summarized in Table 11.3. These measurements have limited clinical utility in critical care practice.

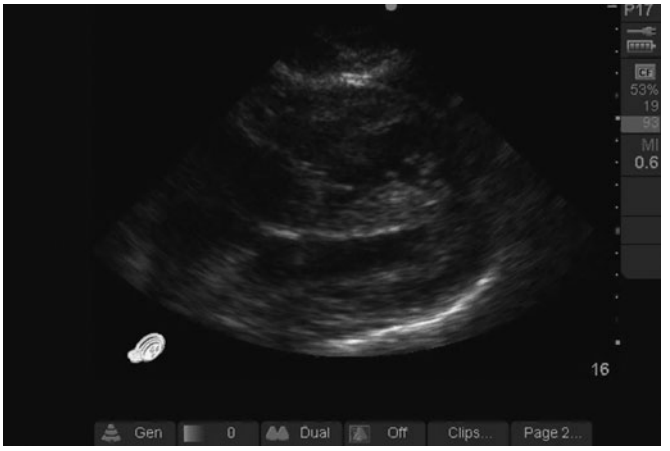


Figure 11.5. Subcostal long-axis view showing a severely dilated right ventricle (RV/LV ratio >1). LV indicates left ventricle; RV, right ventricle.

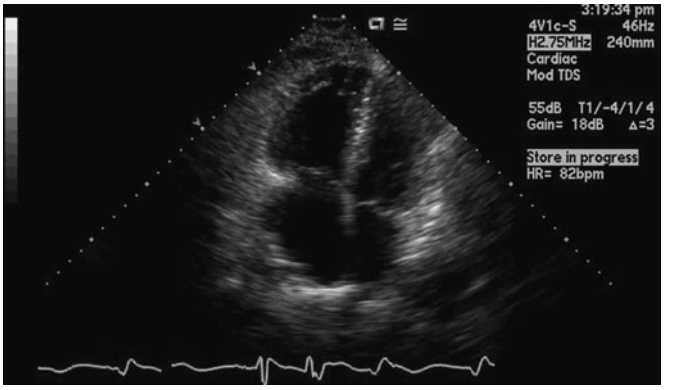


Figure 11.6. Apical four-chamber view showing a severely dilated RV with a rounded apex. RV indicates right ventricle.

TABLE 11.3. Recommended ASE/EAE guidelines on RV chamber quantification

	Normal (cm)	*Severe dilation (cm)*
Basal diameter	≤2.8	≥3.9
Midventricular diameter	≤3.3	≥4.2
Base-to-apex length	≤7.9	≥9.2
RVOT diameter above aortic valve	≤2.9	≥3.6
RVOT diameter above pulmonic valve	≤2.3	≥3.2

ASE indicates American Society of Echocardiography; EAE, European Association of Echocardiography; RV, right ventricular; RVOT, right ventricular outflow tract.

Acute Versus Subacute/Chronic Cor Pulmonale

There are no definitive criteria to distinguish between acute cor pulmonale and chronic right heart failure. To further complicate this assessment, acute cor pulmonale may occur in a patient with chronic right heart failure. The causes of chronic right heart failure include left heart failure, pulmonary arterial hypertension of any etiology, and a variety of pulmonary processes. If right heart impedance is chronically elevated, the RV wall becomes hypertrophic relatively rapidly. The RV free wall thickness is best evaluated on the subcostal view at the end of diastole. The normal thickness is 3.3 ± 0.6 mm. After only 48 hours of a sudden increase in afterload, the RV free wall thickness may increase to double this width.[5] In chronic cor pulmonale, the RV free wall may thicken to as much as 10–11 mm. In addition to wall thickening, there may be increased intracavitary muscle trabeculations, and frequently there may be LV hypertrophy.[12] The level of pulmonary artery pressure calculated by Doppler techniques can also provide a clue that the process is chronic. While the pulmonary arterial systolic pressure (PAPs) in acute conditions is generally <60 mm Hg, it can be higher in chronic cor pulmonale. Finally, the confirmation of acute cor pulmonale can be made retrospectively. Acute cor pulmonale reverses once the baseline disorder is corrected. The lack of complete reversibility of acute cor pulmonale indicates the presence of chronic cor pulmonale.

When faced with a chronic cor pulmonale pattern, the echocardiographer must always rule out common left-side pathologies including:

1. Significant valvular and subvalvular dysfunction
2. Dilated left-sided chambers with wall motion abnormalities, indicating ischemic heart disease
3. Dilated left-sided chambers with wall motion abnormalities suggestive of transient apical ballooning syndrome (TABS) or broken-heart syndrome (reversible aneurismal dilation of LV and/or RV apex, hypo/akinesis of midventricular segments, and hyperkinesis of basal segments)
4. Signs of dynamic LV intracavitary pressure gradient with or without systolic anterior motion of the anterior leaflet of the mitral valve
5. LV hypertrophy with evidence of diastolic dysfunction and signs of elevated LV end-diastolic pressure.

Cor Pulmonale and Pulmonary Hypertension

Cor pulmonale is usually associated with an elevation of pulmonary arterial pressures. High pulmonary arterial pressures in a patient with findings of ACP correlate with increased impedance. However, if the RV pump function is severely impaired, it may not be able to generate significant cardiac output, resulting in pseudo-normalization of pulmonary artery pressures. Alternatively, the clinician will frequently encounter clinical scenarios where elevated pulmonary arterial pressures are present in the absence of ACP. This finding should prompt the physician to rule out "intracardiac" and "intrapulmonary" causes of pulmonary arterial hypertension. Intracardiac etiologies include: left-to-right shunts, pulmonary embolism (PE), unsuspected pulmonary valve stenosis, subpulmonic stenosis, and constrictive pericarditis. Pulmonary etiologies of pulmonary hypertension include chronic thromboembolic pulmonary hypertension and lung diseases such as chronic obstructive pulmonary disease and a variety of interstitial lung processes.

Measurements of Pulmonary Arterial Pressure

Pulmonary artery systolic, diastolic, and mean pressures can be estimated from the tricuspid and pulmonic regurgitant jets, using the modified Bernouilli equation. The tricuspid regurgitant (TR) jet is used to measure PAPs as follows:

$$\text{PAPs} = 4 \times (\text{tricuspid regurgitant jet}_{\text{peak velocity}})^2 + \text{right atrial pressure (RAP)}$$

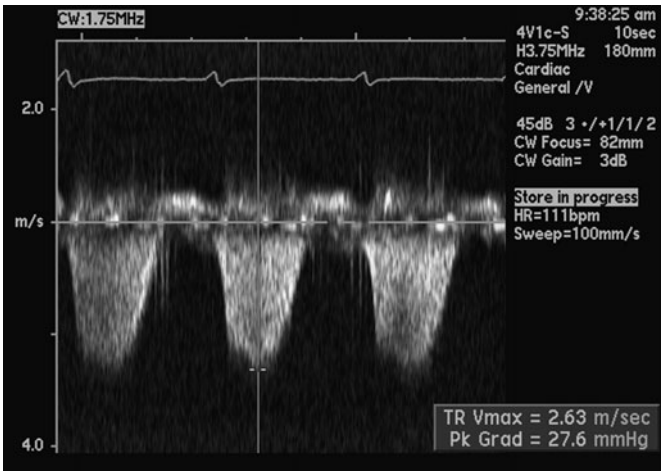


Figure 11.7. Continuous-wave Doppler tracing of a tricuspid regurgitation jet with measurement of the systolic pressure gradient across the valve.

The TR jet should be interrogated from multiple acoustic windows (apical and parasternal), with careful transducer angulation to obtain a parallel intercept angle between the ultrasound beam and TR jet (Figure 11.7). Numerous factors can affect the accuracy of the measurement. Technically, it may be difficult to obtain an adequate spectral Doppler envelope or adequate intercept between the ultrasound beam and the TR jet. In this case, a low-intensity TR jet may be augmented by injection of agitated saline contrast. Using estimates of right atrial pressure instead of actual measurements can lead to over- or underestimations of PAPs. It is important to use an accurate RAP in the PAPs equation. Inferior vena cava (IVC) size and changes with respiratory cycles may be used to estimate RAP (Table 11.4). Right atrial pressure can also be measured directly if the patient has a central venous catheter in place.

In addition to PAPs, pulmonary artery diastolic pressure (PAPd) and mean pulmonary artery pressure (PAPm) can be measured from the pulmonic valve regurgitant jet, when present, as follows:

$$\text{PAPd} = 4 \times (\text{pulmonic regurgitant}_{\text{end-diastolic velocity}})^2 + \text{RAP}$$

$$\text{PAPm} = 4 \times (\text{pulmonic regurgitant}_{\text{peak velocity}})^2, \text{ or } 80 - \text{RVOT acceleration time}/2.^{13-15}$$

TABLE 11.4. RAP as estimated by IVC size and dynamic

RAP (mm Hg)	*IVC size*	*IVC contraction (inspiration)*	*Hepatic vein*
0–5	<20 mm	>50%	Normal
10	<20 mm	<50%	Normal
15	>20 mm	<50%	Normal
20	>20 mm	<50%	Dilated

IVC indicates inferior vena cava; RAP, right atrial pressure.

SPECIFIC CLINICAL SCENARIOS LEADING TO ACUTE COR PULMONALE

Massive Pulmonary Embolism

Pulmonary embolism may be associated with right ventricular dysfunction. Several authors have defined ACP in the context of acute PE using different criteria.[16–18] Because of its simplicity, the RV/LV ratio is the most practical for a bedside application. Using this ratio, Vieillard-Baron et al. reported the incidence of ACP as 61% in 161 subjects with anatomically massive PE.[19] Whether ACP is an independent prognostic factor for mortality and its influence on treatment (e.g., thrombolysis) remains under investigation. Occasionally, a thrombus in transit will be identified in the right heart (Figure 11.8, and Video 11.4 in enclosed DVD). These are typically mobile and serpiginous; they are of grave clinical concern. Thrombus may be visible in the main and proximal pulmonary arteries in a parasternal short-axis view with TTE (Video 11.5 in enclosed DVD). The main and proximal pulmonary artery (PA) are readily visualized with TEE, and this is a means of establishing rapid diagnosis of PE in the critically ill patient who is not able to undergo contrast study.[20,21]

Other indirect echocardiographic signs of acute PE include:

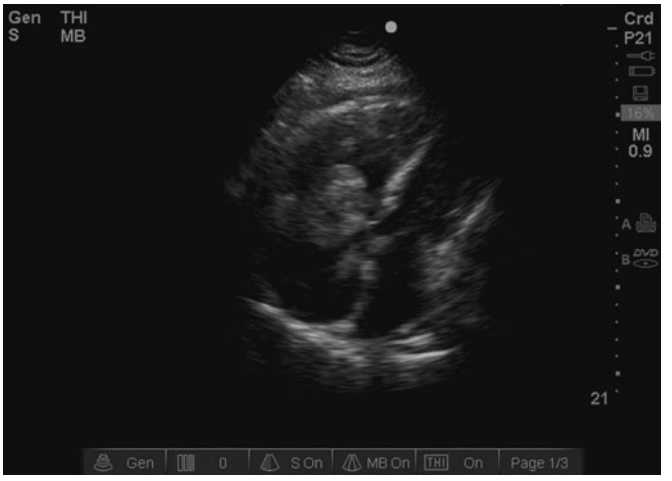


Figure 11.8. Off-axis apical four-chamber view showing a thrombus in transit in right atrium and right ventricle.

1. McConnell sign: diffuse hypokinesis of the RV free wall sparing the apex (Video 11.6 in enclosed DVD)
2. 60/60 sign: RVOT acceleration time <60 m/sec in association with pulmonary artery systolic pressure <60 mm Hg (as estimated by TR regurgitant jet)[23]

Both of these signs were described retrospectively. When tested in a prospective study of 100 consecutive patients with proven PE, the sensitivity of McConnell and 60/60 signs ranged from 19% to 36%.[23] This is likely due to the subjectivity inherent in determining segmental wall motion abnormality and the high margin of error when measuring the acceleration time.

Acute Respiratory Distress Syndrome

In patients with ARDS on ventilatory support, the RV can become easily afterloaded due to factors intrinsic to the disease and factors associated with mechanical ventilation. These include:

1. Occlusion of the pulmonary vascular bed (microthrombi, inflammation, interstitial edema, and atelectasis)[24]
2. High transpulmonary pressures that result in pulmonary capillary compression (tidal volume and positive end-expiratory pressure [PEEP] effect)[25,26]
3. Acidosis and hypoxemia, with pulmonary vascular constriction[27]
4. Intra-abdominal hypertension[28]

Acute cor pulmonale is frequent in ARDS. Vieillard-Baron et al. reported an incidence of 25% in patients with ARDS submitted to protective ventilation, defined as a plateau pressure <30 cm H_2O with a mean PEEP of 6–7 cm H_2O.[29] Echocardiography can identify patterns of ventilatory support that may result in ACP. Serial echocardiography can alert the physician to this possibility and allow adjustment of therapies in a sequential and effective manner.

Recruitment Maneuvers

Typically a recruitment maneuver involves applying a high level of PEEP for a defined period of time. A simple recruitment maneuver consists of 40 cm H_2O of PEEP for 40 seconds. Higher pressures or longer times have also been described.[30,31] Echocardiography demonstrates ACP during the recruitment maneuver.[32–34] This occurs as a result of acute augmentation of RV afterload due to pulmonary vascular derecruitment from increased transpulmonary pressures and results in reduced cardiac output and hemodynamic instability. The efficacy of a recruitment maneuver is questionable.

Acute Right Ventricular Failure of Sepsis

Function of both LV and RV can become depressed in sepsis.[3,35–38] In one series, 32% of patients had evidence of RV dysfunction.[38] The cardiomyopathy is usually compensated, and cardiac output is maintained within normal limits with adequate fluid resuscitation. It is maximal on the second day after onset of sepsis and recovers completely in seven to 10 days.[39] However, RV failure with ACP can become manifest when ventilatory support is applied. By increasing pulmonary vascular resistance, mechanical ventilation can result in ACP in an RV that is already dysfunctional from sepsis. If ACP develops, the clinician should adjust the ventilator settings to minimize alveolar distension, hypoxemia and acidosis, restrict intravenous fluid resuscitation, and add vasoactive therapy to maintain adequate coronary artery perfusion pressure.[5,40]

Right Ventricular Infarction

Right ventricular infarction occurs predominantly due to occlusion of the right coronary artery. In addition to perfusing the RV free wall, this artery also supplies blood to the inferior aspect of the LV and the inferior interventricular septum through the posterior descending artery. As a result, RV infarction is frequently accompanied by LV inferior wall infarction with corresponding segmental wall abnormality.

Echocardiography allows an assessment of RV segmental wall abnormality. When myocardial ischemia is present, there is reduced endomyocardial thickening and wall motion abnormalities (hypokinesis, akinesis, or dyskinesis). Using TTE, the parasternal long view allows examination of the RV infundibulum; while the parasternal, short midventricular-level view permits assessment of the anterior, lateral, and portion of the inferior RV walls. The anterior and inferior free RV walls are also seen in a different plane on inspection of the RV inflow tract axis, while the apical four-chamber view demonstrates the lateral wall and apex. When RV infarction is suspected, the echocardiographer must assess the subcostal views. Its long-axis plane shows the RV inferior free wall, while its short-axis plane brings up part of the inferior wall and the RV outflow tract. Importantly, 20% of RV infarctions may be missed if this plane is not interrogated and the right coronary artery (RCA) occlusion is distal.[41]

With proximal RCA occlusion, severe acute RV failure may occur. Unlike ACP, PA pressures may not be

elevated, as the cause of the RV failure is acute failure of pump function. A clue to the diagnosis of ischemia is the presence of abnormal endomyocardial thickening and segmental wall motion abnormality of the inferior left ventricular wall and inferior septum. Dilation of the tricuspid annulus secondary to RV dilation results in acute TR, elevation of RAP, right atrial enlargement, and IVC dilation.

PRELOAD, VOLUME RESPONSIVENESS AND THE RIGHT HEART

For a complete discussion of echocardiographic indices of preload or volume responsiveness, the reader is referred to Chapter 10. The dynamic parameters of volume responsiveness should always be evaluated within the context of right heart function. While preload responsiveness is best assessed with dynamic parameters, all described indices (e.g., change in right atrial pressure with respiration, ∆down, pulse pressure variation, change in peak aortic velocity or stroke volume with respirator cycling or leg elevation) assume a normal RV function. Right ventricular failure itself can result in pulse pressure variation during tidal positive pressure ventilation, independently of the patient's volume status. If RV function is not evaluated, the "blinded" clinician could erroneously conclude, by relying on dynamic parameters alone, that more volume expansion is necessary. The presence of ACP with septal dyskinesia provides a contraindication to volume resuscitation, even if the dynamic parameters indicate otherwise.

BASIC AND ADVANCED LEVEL ECHOCARDIOGRAPHY FOR THE ASSESSMENT OF RV FUNCTION

The intensivist with competence in basic critical care echocardiography has a limited ability to perform Doppler-based assessment of RV function. The limited or goal-directed echocardiogram emphasizes 2D examination of a few key image planes. Of these, the apical four-chamber and subcostal views are the most important for identification of RV dysfunction. A necessary and diagnostic feature of acute cor pulmonale is RV dilation. Septal dyskinesia can also be identified qualitatively by the basic-level echocardiographer. The finding of a dilated, hypokinetic RV in a patient with shock has major diagnostic implications (e.g., acute PE, inappropriate ventilator settings, RV infarction, etc.). It also has important implications for management because volume resuscitation may have adverse consequences, leading the clinician to favor pressors and inotropes while simultaneously considering means of reducing RV afterload.

In addition to the standard 2D imaging, Doppler examination allows sophisticated assessment of RV hemodynamic function, while M-mode quantitates septal kinetics and RV wall thickness. In a busy ICU, it may be both impractical and unnecessary to make a very wide range of measurements of RV function. The intensivist will have to decide which are relevant to the clinical situation. Our practice is to obtain the appropriate 2D images, followed by measurement of TR jet velocity from as many angles as possible. Pulmonary arterial systolic pressure is calculated as previously reviewed. Although no specific PAPs level will change our clinical management, an elevated PAPs within the context of ACP is theoretically consistent with maintained RV pump function.

CONCLUSION

Right ventricular failure is frequent in critically ill patients. Echocardiography is the best bedside diagnostic tool available to identify RV dysfunction. Careful assessment of RV size and function is a key part of the critical care echocardiographic examination that is performed to render immediate diagnosis and to guide therapy at the bedside.

References

1. Jardin F, Dubourg O, Bourdarias JP. Echocardiographic pattern of acute cor pulmonale. *Chest*. 1997;111:209–217.
2. Ozier Y, Gueret P, Jardin F, Farcot JC, Bourdarias JP, Margairaz A. Two-dimensional echocardiographic demonstration of acute myocardial depression in septic shock. *Crit Care Med.* 1984;12:596–599.
3. Etchecopar-Chevreuil C, François B, Clavel M, Pichon N, Gastinne H, Vignon P. Cardiac morphological and functional changes during early septic shock: a transesophageal echocardiographic study. *Intensive Care Med*. 2008; 34: 250–256.
4. Hoffman D, Sisto D, Frater RW, Nikolic SD. Left-to-right ventricular interaction with a noncontracting right ventricle. *J Thorac Cardiovasc Surg.* 1994;107:1496–1502.
5. Vieillard-Baron A, Prin S, Chergui K, Dubourg O, Jardin F. Echo-Doppler demonstration of acute cor pulmonale

at the bedside in the medical intensive care unit. *Am J Respir Crit Care Med.* 2002;166:1310–1319.

6. Elzinga G, Pienne H, De Jong J. Left and right ventricular pump function and consequences of having two pumps in one heart: a study on isolated cat heart. *Circ Res*. 1980;46:564–574.
7. Ryan T, Petrovic O, Dillon JC, Feigenbaum H, Conley MJ, Armstrong WF. An echocardiographic index for separation of right ventricular volume and pressure overload. *J Am Coll Cardiol*. 1985;5:918–927.
8. Nielsen JC, Kamenir SA, Ko HS, Lai WW, Parness IA. Ventricular septal flattening at end-systole falsely predicts right ventricular hypertension in patients with ostium primum atrial septal defects. *J Am Soc Echocardiogr*. 2002; 15:247–252.
9. Frémont B, Pacouret G, Jacobi D, Puglisi R, Charbonnier B, de Labriolle A. Prognostic value of echocardiographic right/left ventricular end-diastolic diameter ratio in patients with acute pulmonary embolism: results from a monocenter registry of 1,416 patients. *Chest.* 2008;133: 358–362.
10. Vieillard-Baron A, Charron C, Chergui K, Peyrouset O, Jardin F. Bedside echocardiographic evaluation of hemodynamics in sepsis: is a qualitative evaluation sufficient? *Intensive Care Med*. 2006;32:1547–1552.
11. Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr.* 2005;18: 1440–1463.
12. Jardin F, Gueret P, Prost JF, Farcot JC, Ozier Y, Bourdarias JP. Two-dimensional echocardiographic assessment of left ventricular function in chronic obstructive pulmonary disease. *Am Rev Respir Dis*. 1984;129:135–142.
13. Otto CM, ed. *Textbook of Clinical Echocardiography*. 3rd ed. Philadelphia, Pa: Elsevier Saunders; 2004:259–269.
14. Oh JK, Seward JB, Tajik AJ, eds. *The Echo Manual.* 2nd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 1999:181–190.
15. Feigenbaum H, Armstrong WF, Ryan T, eds. *Feigenbaum's Echocardiography*. 6th ed. Philadelphia, Pa: Lippincott Wiliams & Wilkins; 2005:255–260.
16. Kasper W, Meinertz T, Kerstin F, Löllgen H, Limbourg P, Hanjörg J. Echocardiography in assessing acute pulmonary hypertension due to pulmonary embolism. *Am J Cardiol*. 1980;45:567–572.
17. Ribeiro A, Lindmaker P, Juhlin-Dannfelt A, Johnsson H, Jorfeldt L. Echocardiography Doppler in pulmonary embolism: right ventricular dysfunction as a predictor of mortality rate. *Am Heart J.* 1997;134:479–487.
18. Goldhaber S, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Embolism Registry (ICOPER). *Lancet.* 1999;353:1386–1389.
19. Vieillard-Baron A, Page B, Augarde R, et al. Acute cor pulmonale in massive pulmonary embolism: incidence, echocardiographic pattern, clinical implications and recovery rate. *Intensive Care Med.* 2001;27:1481–1486.
20. Pruszczyk P, Torbicki A, Pacho R, et al. Noninvasive diagnosis of suspected severe pulmonary embolism: transesophageal echocardiography vs spiral CT. *Chest.* 1997; 112:722–728.
21. Vieillard-Baron A, Qanadli SD, Antakly Y, et al. Transesophageal echocardiography for the diagnosis of pulmonary embolism with acute cor pulmonale: a comparison with radiological procedures. *Intensive Care Med.* 1998;24:429–433.
22. McConnell MV, Solomon SD, Rayan ME, Come PC, Goldhaber SZ, Lee RT. Regional right ventricular dysfunction detected by echocardiography in acute pulmonary embolism. *Am J Cardiol*. 1996;78:469–473.
23. Kurzyna M, Torbicki A, Pruszczyk P, et al. Disturbed right ventricular ejection pattern as a new Doppler echocardiographic sign of acute pulmonary embolism. *Am J Cardiol*. 2002;90:507–511.
24. Penuelas O, Aramburu JA, Frutos-Vivar F, Esteban A. Pathology of acute lung injury and acute respiratory distress syndrome: a clinical-pathological correlation. *Clin Chest Med.* 2006;27:571–578.
25. Jardin F, Delorme G, Hardy A, Auvert B, Beauchet A, Bourdarais JP. Reevaluation of hemodynamic consequences of positive pressure ventilation: emphasis on cyclic right ventricular afterloading by mechanical lung inflation. *Anesthesiology.* 1990;2:966–970.
26. Howell JB, Permutt S, Proctor DF, Riley RL. Effect of inflation of the lung on different parts of the pulmonary vascular bed. *J Appl Physiol*. 1961;16:71–76.
27. Balanos GM, Talbot NP, Dorrington KL, Robbins PA. Human pulmonary vascular response to 4 h of hypercapnia and hypocapnia measured using Doppler echocardiography. *J Appl Physiol*. 2003;94:1543–1551.
28. Malbrain ML, Cheatham ML, Kirkpatrick A, et al. Results from the international conference of experts of intra-abdominal hypertension and abdominal compartment syndrome. Definitions. *Intensive Care Med*. 2006;32:1722–1732.
29. Vieillard-Baron A, Schmitt JM, Augarde R, et al. Acute cor pulmonale in acute respiratory distress syndrome submitted to protective ventilation: incidence, clinical implications, and prognosis. *Crit Care Med*. 2001;29:1551–1555.
30. Toth I, Leiner T, Mikor A, Szakmany T, Bogar L, Molnar Z. Hemodynamic and respiratory changes during lung recruitment and descending optimal positive end-expiratory pressure titration in patients with acute respiratory distress syndrome. *Crit Care Med.* 2007;35:961–963.
31. De Matos G, Borges J, Meyer E, et al. Maximal recruitment strategy guided by thoracic CT scan in severe acute respiratory distress syndrome patients: a case series report. *Critical Care*. 2007;11(Suppl 2):P188.

32. Jardin F, Vieillard-Baron A. Is there a safe plateau pressure in ARDS? The right heart only knows. *Intensive Care Med*. 2007;33:444–447.
33. Nielsen J, Østergaard M, Kjaegaaard J, et al. Lung recruitment maneuver depresses central hemodynamics in patients after cardiac surgery. *Intensive Care Med*. 2005;31: 1189–1194.
34. Jardin F. Acute leftward septal shift by lung recruitment maneuver. *Intensive Care Med*. 2005;31:1148–1149.
35. Parker MM, McCarthy KE, Ognibene FP, Parrillo JE. Right ventricular dysfunction and dilation, similar to left ventricular changes, characterize the cardiac depression of septic shock in humans. *Chest*. 1990;97:126–131.
36. Jardin F, Brun-Ney D, Auvert B, Beauchet A, Bourdarais JP. Sepsis-related cardiogenic shock. *Crit Care Med*. 1990;18:1055–1060.
37. Schneider AJ, Teule GJ, Groeneveld AB, Nauta J, Heidendal GA, Thijs LG. Biventricular performance during volume loading in patients with early septic shock, with emphasis on the right ventricle: a combined hemodynamic and radionuclide study. *Am Heart J*. 1988;116:103–112.
38. Vieillard-Baron A, Schmitt JM, Beauchet A, et al. Early preload adaptation in septic shock? A transesophageal echocardiographic study. *Anesthesiology*. 2001;94:400–406.
39. Rabuel C, Mebazaa A. Septic shock: a heart story since the 1960s. *Intensive Care Med*. 2006;32:799–807.
40. Vieillard-Baron A, Prin S, Chergui K, Dubourg O, Jardin F. Hemodynamic instability in sepsis: bedside assessment by Doppler echocardiography. *Am J Respir Crit Care Med*. 2003;168:1270–1276.
41. Gemayel CY, Fram DB, Fowler LAA, Kiernan FJ, Kelsey AM, Gillam LD. The importance of using multiple windows for the echocardiographic identification of right ventricular infarction. *JACC*. 2001;37(Suppl 2):1110–1147.

CHAPTER 12

Echocardiographic Diagnosis of Cardiac Tamponade

Daniel A. Sweeney and Dorothea McAreavey

INTRODUCTION

Cardiac tamponade is a life-threatening condition in which the accumulation of fluid or gas in the pericardial sac prevents adequate filling of the heart and compromises cardiac output. Prompt diagnosis and treatment is imperative if the patient is to survive. Unfortunately, the signs and symptoms can be subtle and easily missed. This is especially true in the intensive care unit (ICU), where mechanical ventilation and sedation can limit physical examination and prevent the patient from vocalizing symptoms. Echocardiography is an important tool in the diagnosis of cardiac tamponade because ultrasound equipment is readily available, portable, and can confirm or refute a clinical diagnosis. The intensivist with the ability to perform a basic cardiac ultrasound has the opportunity to make a timely diagnosis of cardiac tamponade and sometimes even save the life of a critically ill patient.

THE PHYSIOLOGY OF THE PERICARDIUM

The pericardial sac surrounds the heart and is formed by two layers—the outer, pleural pericardium, and the inner, visceral pericardium. Histologically, the pericardium is composed of collagen and elastin fibers that combine to give the structure both its stiffness and elastic quality.[1] Normally, the pericardial sac contains 30–50 milliliters (mls) of a low protein, transudative fluid that originates from the visceral pericardium and is thought to act as a lubricant surrounding the beating heart. Typically, the pressure within the pericardial sac is equivalent to the pleural pressure or approximately 5 millimeters of mercury (mm HG) below the central venous pressure.[2] Under normal conditions, the pericardium may prevent excessive motion of the heart and reduces friction between moving organs. The pericardium also serves to prevent acute cardiac dilatation and contributes to ventricular interdependence, the mechanism whereby the filling of one ventricle affects the filling of the other. However, cardiac function remains normal in rare individuals with a congenital absence of pericardium.

THE PATHOLOGY OF PERICARDIAL EFFUSION AND CARDIAC TAMPONADE

Pericardial effusion is defined as an accumulation of fluid in the pericardial sac; this fluid can either be transudative 2° to impaired lymphatic drainage, or more commonly exudative (serous, hemorrhagic, or purulent). On rare occasions, chylous fluid (2° to pancreatitis) or air (2° to esophageal rupture) in the pericardial sac has also been described. The physiologic effects of a pericardial effusion depend on both the amount of fluid and the rate of accumulation (Figure 12.1). When a pericardial effusion accumulates gradually, the compliant pleural pericardium is able to stretch and the pressure in the pericardial sac can remain relatively low until late in the disease course. Accordingly, pericardial effusions of 2 liters have been described in relatively asymptomatic patients with chronic disease. Once the pericardial reserve volume is exhausted, however, small incremental increases in pericardial fluid will result in large increases in pericardial pressure. When pericardial effusions develop rapidly, the pleural pericardium is unable to adapt and the pericardial reserve volume is relatively small. In these cases, very small increases in volume can cause rapid increases in pericardial pressure.[3]

Cardiac tamponade occurs when the pressure associated with a pericardial effusion compresses the chambers of the heart, preventing adequate filling and resulting in a reduction of cardiac output. For the right and left ventricles to fill, the pressure within each cavity during diastole must be greater than the pericardial pressure, otherwise the chambers collapse. Classically, cardiac tamponade progresses along a continuum with

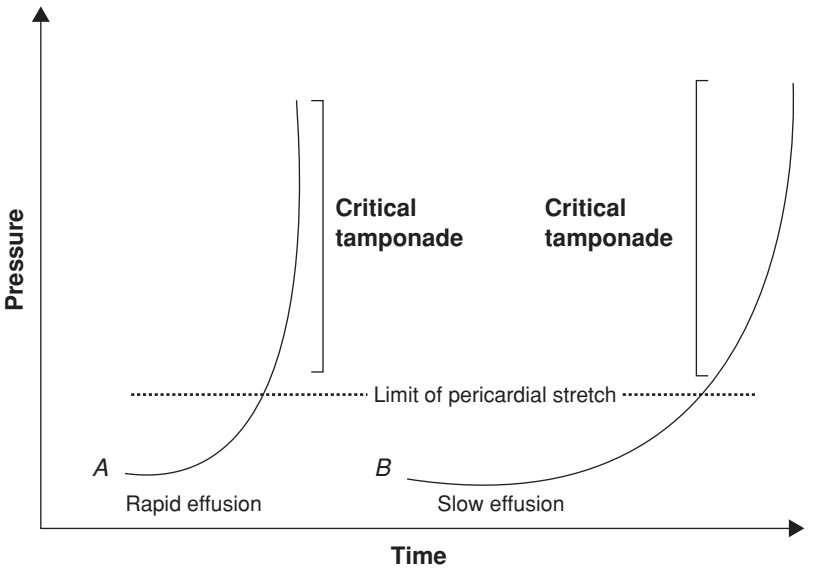


Figure 12.1. Pressure–volume curves in rapid and slow effusions in cardiac tamponade. (A) In cases of rapid accumulation of pericardial fluid, the cardiac reserve (the initial flat segment on the curve) is quickly exceeded and pressure thereafter rises with small increases in pericardial fluid. (B) When a pericardial effusion occurs gradually, the pericardium is able to adapt and stretch; with time, however, the limit of pericardial stretch is met and additional fluid accumulation results in increasing intrapericardial pressure. (From Ref. 3; Copyright © 2007 Texas Heart Institute.)

the pericardial pressure first equilibrating with the right ventricular end-diastolic pressure and then with the left ventricular end diastolic pressure (as estimated by the pulmonary capillary wedge pressure).[4] Adaptive mechanisms, including increasing blood volume, heart rate, and vasoconstriction, can temporarily maintain cardiac output in the face of progressive cardiac tamponade, but eventually shock ensues.

EPIDEMIOLOGY AND CAUSES OF CARDIAC EFFUSION AND TAMPONADE

The decision about whether to perform a cardiac ultrasound in an individual patient can be guided, in part, by knowledge of both the clinical settings and the frequent causes of pericardial effusion and tamponade. In the largest review of patients with an ultrasound-proven cardiac effusion (n = 322), the most commonly associated medical conditions were: recent cardiac surgery or endovascular procedure (14%); cancer (9%); recent myocardial infarction (9%); and chronic renal failure (7%).[5] Other, less frequently associated diseases included cardiac failure, aortic dissection, hypothyroidism, and human immunodeficiency virus infection. The most commonly identified causes of tamponade were acute idiopathic pericarditis (23%), malignancy (22%), and iatrogenic effusion (18%). Notably, 17 % of all cases of tamponade were in patients without any risk factors at the time of presentation.

The epidemiology and prevalence of pericardial tamponade in the ICU has not been systematically studied. A handful of studies involving intensivist-initiated echocardiograms found cardiac tamponade in 2–10% of patients.[6–8] There are a number of ICU patient subgroups who should be considered to be at especially increased risk for cardiac tamponade (Table 12.1). Procedures including the placement of central venous catheters, pulmonary artery catheters, and endovenous pacemakers are potential causes of cardiac tamponade unique to the ICU.

SYMPTOMS AND SIGNS, CHEST RADIOGRAPHY, AND EKG FINDINGS

Multiple lines of evidence can assist in identifying patients who have a pericardial effusion and may be progressing toward florid cardiac tamponade. A recent metaanalysis identified dyspnea as the most sensitive

TABLE 12.1. ICU patients at increased risk for cardiac tamponade

Postcardiac surgery, endovascular procedure, or catheter-based intervention
Recent myocardial infarction
Known or suspected malignancy
Chronic renal failure
Collagen vascular diseases
HIV, risk factors for tuberculosis
Blunt or penetrating chest trauma

HIV indicates human immunodeficiency virus.

symptom (87–88%), with fever, chest pain, and cough occurring in 25% or less of patients with cardiac tamponade.[2] This same study went on to evaluate physical examination findings in the diagnosis of cardiac tamponade and found tachycardia and elevated jugular venous pressure (JVP) to have sensitivities of 77% and 76%, respectively. Hypotension, on the other hand, was insensitive (26%).

During normal respiration, venous return to the right heart is augmented in inspiration by the fall in intrathoracic pressure. Conversely, blood pools in the pulmonary vasculature, reducing flow to the left heart. Normally, the right ventricle expands symmetrically with the free wall bowing outward and the septum bowing toward the left, resulting in a subtle decrease in left ventricular filling. During expiration, the effect is reversed; with increase of flow to the left side of the heart, the septum bows rightward and there is a decrease in venous return to the right ventricle. Thus, the respiratory cycle alternately favors filling of the right and the left side of the heart. The result is a small (<10 mm Hg) variation in systolic blood pressure with inspiration and expiration.

By contrast, pulsus paradoxus is the classic physical finding associated with cardiac tamponade. In cardiac tamponade, the effect of the respiratory cycle on systolic blood pressure variation is exaggerated. The increase in pericardial pressure prevents the right ventricular free wall from moving outward during diastole. Thus, increased venous return during inspiration causes an asymmetric expansion of the right ventricle with the septum bowing deeper into the left ventricle. Pulsus paradoxus is said to be present when the systolic pressure during inspiration is at least 10 mm Hg less than the systolic pressure during expiration. This difference can be measured by two different techniques. Using a sphygmomanometer, the examiner inflates the cuff and listens with his stethoscope for Korotkoff sounds as the cuff deflates. The examiner notes the pressure at which the Korotkoff sounds are first heard intermittently (this corresponds with the patient's expiration phase) and when these sounds are heard throughout the respiratory cycle; if the difference is ≥10 mm Hg, then pulsus paradoxus is present (pulsus paradoxus has also been defined as a percentage reduction [10%] of expiratory systolic pressure pulsus paradoxus).[9] In the ICU setting, the presence of pulsus paradoxus can be identified by using either an arterial line or pulse oximeter tracing and simply calculating the difference between the peak systolic pressure during expiration and the trough systolic pressure during inspiration. Measurement of pulsus paradoxus has many applications in the setting of possible cardiac tamponade. For example, it is a reasonable screening test, as the sensitivity in cardiac tamponade is 88%. The degree of pulsus paradoxus (i.e., 12 mm Hg versus 25 mm Hg) has also been shown to correlate with the severity of hemodynamic compromise in cardiac tamponade.[10] Conversely, the absence of pulsus paradoxus is also very helpful in ruling out cardiac tamponade (likelihood ratio [LR], 0.03; 95% confidence interval [CI], 0.01–0.24).[2] Limitations of this physical finding should also be appreciated; namely, pulsus paradoxus can be diminished in the setting of hypotension, right ventricular hypertrophy, left ventricular hypertrophy, or increased pulmonary wedge pressure. In addition to cardiac tamponade, pulsus paradoxus is routinely present in conditions associated with increased intrathoracic pressures including chronic obstructive pulmonary disease, acute asthma, and massive pulmonary embolism.

Chest radiography and electrocardiography can also be abnormal in cardiac tamponade. An enlarged cardiac silhouette, often described as "boot-shaped," is a sensitive test (88%) for cardiac tamponade.[2] The electrocardiogram, in contrast, is a less sensitive test for cardiac tamponade, with either a low QRS voltage or electrical alternans (associated with a "swinging" heart within the pericardial sac on echocardiographic examination) being uncommon findings.

In the ICU, the clinical features of tamponade may be masked and it is in this situation that additional techniques can be most useful. One diagnostic procedure has been right heart catheterization to demonstrate equalization (within 5 mm Hg) of the mean right atrial, right ventricular end-diastolic, and mean pulmonary arterial occlusion pressures However, the widespread availability of echocardiography makes it an attractive

and easy noninvasive method to detect pericardial effusions and determine their characteristics.

GOAL-DIRECTED CARDIAC ULTRASOUND TO DIAGNOSE CARDIAC TAMPONADE

When pericardial tamponade is suspected, echocardiographic assessment should not be delayed. With the development of handcarried ultrasound (HCU) technology, multiple published reports have demonstrated that intensivists and other noncardiologists can be trained to effectively perform a limited cardiac ultrasound exam at the bedside.[8,11,12] In terms of equipment, two dimensional (2D) ultrasound evaluation of the heart should be performed using a 2.0–5.0 MHz transducer. It is worth noting that the frequency (MHz) determines the performance characteristics of the transducer: the lower the sound frequency, the greater the distance that the ultrasound beam penetrates the body; the higher the frequency, the better the resolution.[13] Also, transducers that feature harmonic imaging can improve picture resolution. In the adult population, a 3.5-MHz transducer is generally satisfactory, whereas in small children, a 5–7-MHz probe is used, depending on size. The patient should be placed in the left lateral decubitus position, if possible. The examiner should proceed to examine the heart from four different transducer positions (Figure 12.2).[14] Beginning with the parasternal position, the transducer is placed in the third or fourth left intercostal space. The parasternal long-axis view is performed with the transducer aimed in the direction of the right shoulder. Once this window is achieved, the parasternal short-axis view ("bread loaf" view) is obtained by turning the transducer in a clockwise direction approximately 90° so that the transducer points in the direction of the left shoulder. To attain a four-chamber apical view, the transducer is placed at the apex of the heart in an intercostal space near the point of maximal impulse; the ultrasound beam is then directed toward the right scapula. For the subcostal examination, the patient should be positioned flat on the back. The transducer is then placed beneath the middle or the right border of the xiphoid process. The ultrasound beam is directed slightly downward and toward the patient's left scapula. The subcostal view is especially useful in the ICU, where patients are frequently receiving mechanical ventilation or have surgical chest wounds that preclude examining the heart from the other transducer positions. Regardless of transducer positions, the quality of the "window" and the delineation of tissue planes can be improved with subtle alterations in patient positioning or adjustment of the gain (creating either a darker or grainer image), or by changing the depth of the ultrasound beam.

When a pericardial effusion is present, the pericardial sac appears as an echo-free space. Small effusions (<25 mL) are usually located posteriorly and are visible only during systole. As the size of the effusion increases, an echo-free space surrounds the heart and persists throughout the cardiac cycle. Occasionally, the heart can appear to be "swinging" within the pericardial sac; this finding is typically associated with the electrical alternans appearance on the electrocardiogram (EKG). Additional evidence supporting the diagnosis of cardiac tamponade can also be gleaned by examining the inferior vena cava (IVC) from the subcostal view. Failure of the IVC to collapse with inspiration can be suggestive of cardiac tamponade.[15] Nonetheless, cardiac chamber collapse is the most characteristic sign of tamponade, and the ability to recognize this finding is crucial to making the diagnosis (Figure 12.3). Right atrial collapse occurring during early diastole is the most sensitive (70–100%) echocardiographic sign of tamponade (Video 12.1 in enclosed DVD); it is also extremely specific for cardiac tamponade when collapse persists for greater than one third of the cardiac cycle.[16,17] Right ventricular collapse, in contrast, is specific but insensitive for cardiac tamponade (Video 12.2 in enclosed DVD). Finally, left atrial collapse is also described in cardiac tamponade. For more advanced practitioners, Doppler findings include increased tricuspid velocity and decreased mitral forward flow (Figure 12.4) during inspiration.

There are a few pitfalls to avoid when performing an ultrasound exam in the setting of suspected cardiac tamponade. The pericardial space does not extend behind the left atrium beyond the atrioventricular ring, so an echo-free space behind the left atrium is not a pericardial effusion (Video 12.3 in enclosed DVD). Similarly, an echo-free space limited to the anterior surface often represents an epicardial fat pad and not an effusion. A pleural effusion can be mistaken for a pericardial effusion, but pleural effusions lie posterior to the descending thoracic aorta, which is typically seen in cross-section on the parasternal long-axis view. Unlike a pericardial effusion, pleural effusions will never surround the heart on all sides; moreover, a pericardial effusion tends to be motionless, whereas a pleural effusion will frequently slide back and forth with the respiratory cycle. Lastly, if there is low output

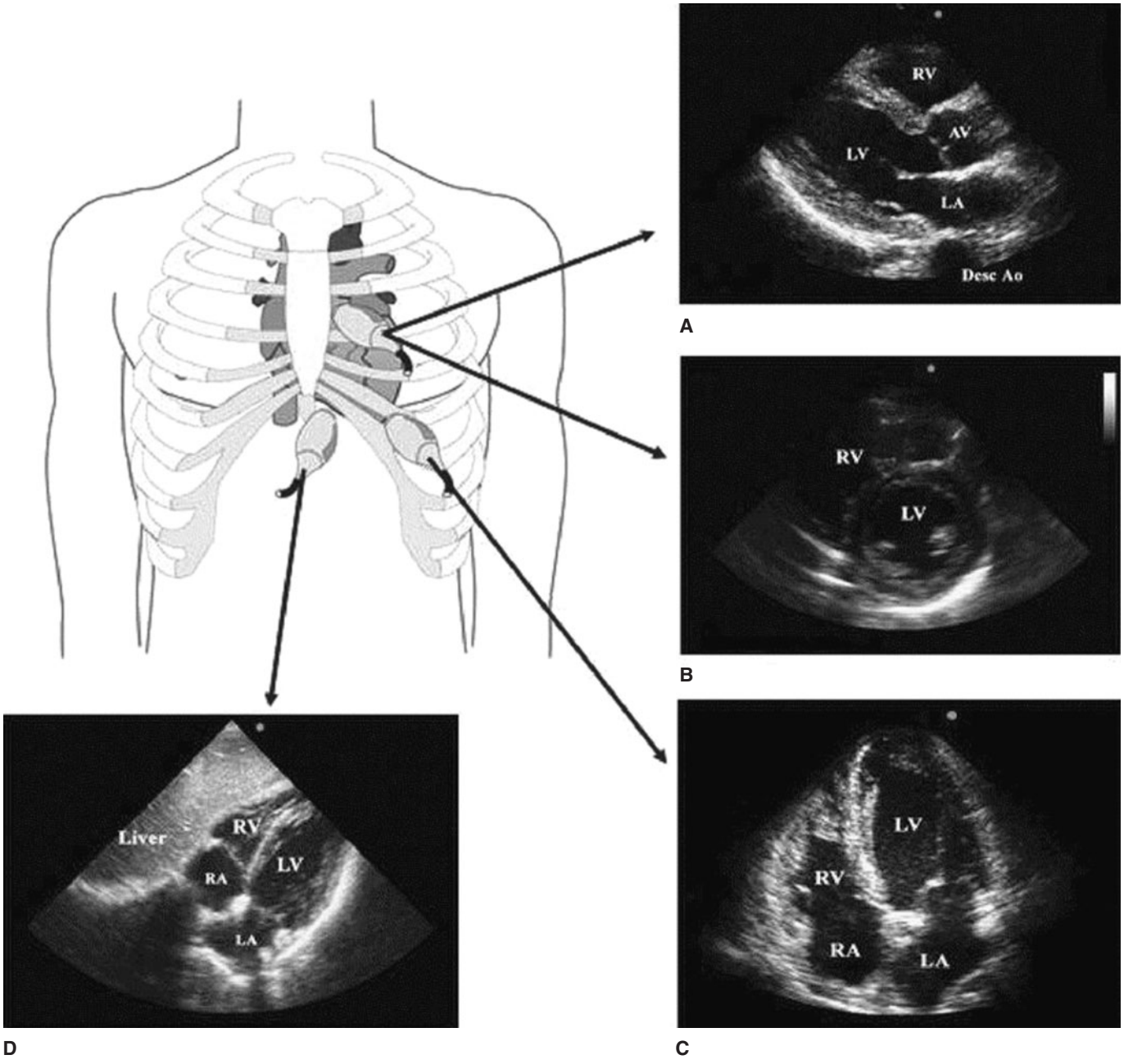
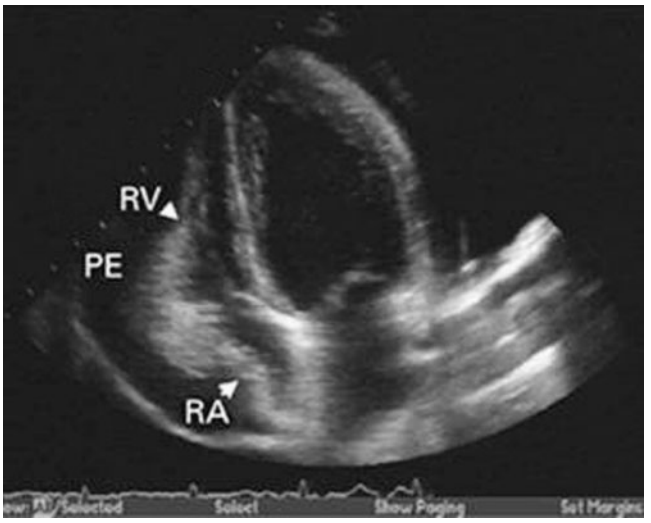


Figure 12.2. Tranducer placement for goal-directed 2D echocardiography with corresponding echocardiographic images. Parasternal long-axis (A) and short-axis (B) views; apical four-chamber view (C); subcostal four-chamber view (D). AVindicates aortic valve; Desc Ao, descending thoracic aorta; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle. (From Ref. 14.)

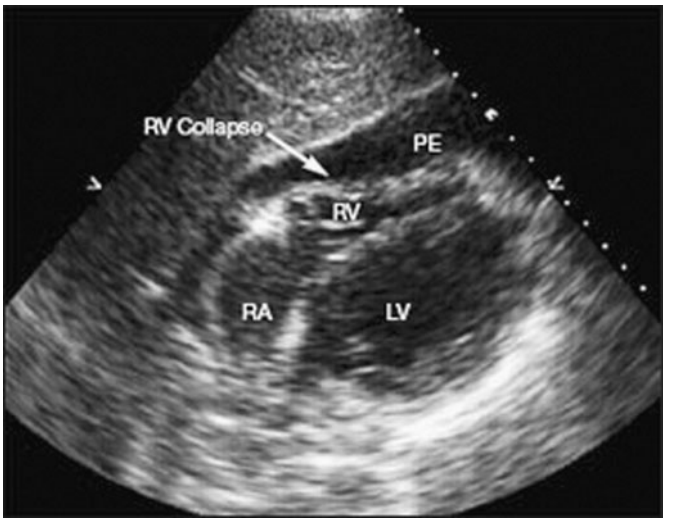
state or hemodynamic instability postcardiac surgery, cardiac tamponade must be suspected. In this setting, the pericardial effusion may be loculated and can overlie a single cardiac chamber rather than circumscribing the heart. To exclude a loculated effusion, it is very important to obtain multiple views of the heart from different locations, especially the subxiphoid view.

TREATMENT: ULTRASOUND-GUIDED PERICARDIOCENTESIS

Whether a pericardial effusion requires drainage depends not only on the echo findings but also on the clinical features and clinical judgment. For example, an effusion might be monitored carefully if there is no pulsus



A



B

Figure 12.3. (A) Apical four-chamber view showing marked right atrial and ventricular collapse indicative of cardiac tamponade. (From Ref. 20.) (B) Subxiphoid view showing right ventricular collapse secondary to cardiac tamponade. PE indicates pericardial effusion; RA, right atrium; RV, right ventricle. (From Ref. 21.)

paradoxus and no hypotension, even if the echo appearances suggest early tamponade.

The treatment for acute life threatening cardiac tamponade is pericardiocentesis. Ultrasonographic guidance of pericardiocentesis (Figure 12.5) is the best method of performing the procedure, and has major advantages when compared to fluoroscopic guidance. Technical aspects of pericardiocentesis are discussed in chapter 27. The use of ultrasound guidance has largely superseded electrocardiographic monitoring during aspiration. If the effusion can be visualized in the subcostal window, a 16–18 gauge needle with applied negative pressure should be inserted between the xiphoid process and the left costal margin.[18] The needle should be advanced with real-time ultrasound guidance and visualized as it enters into the pericardial

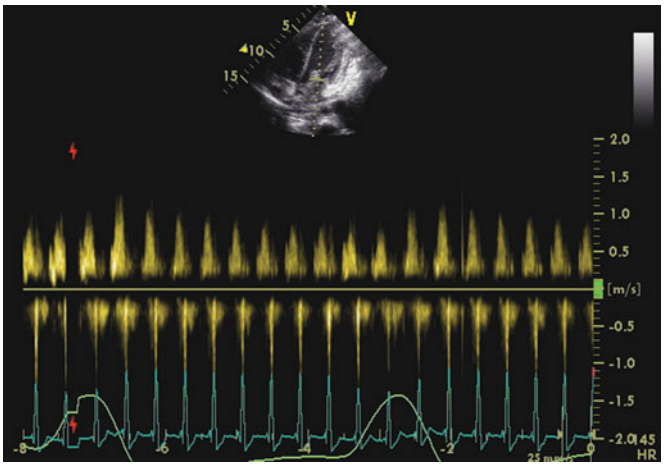


Figure 12.4. Doppler flow across mitral valve in cardiac tamponade showing a fall in flow in inspiration.

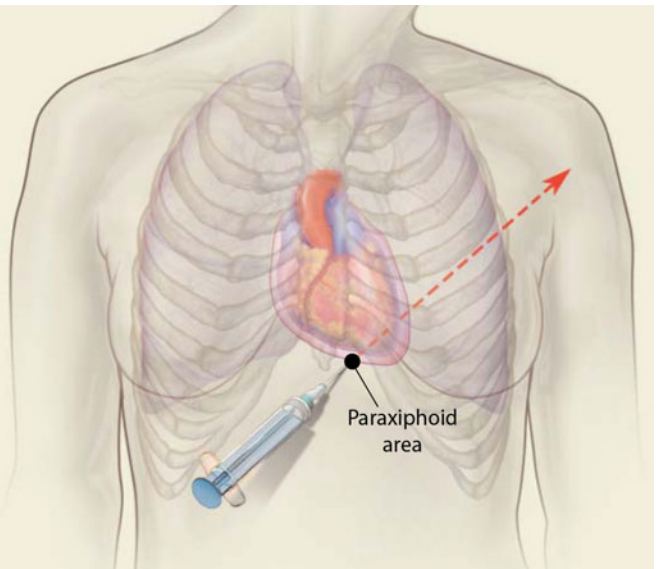


Figure 12.5. Ultrasound-guided pericardiocentesis using a subxiphoid approach. After obtaining an adequate subcostal window, a 16–18 gauge needle is inserted between the xiphoid process and the left costal margin. Using real-time echocardiography, the needle is advanced and visualized as it enters the pericardial sac. (From Ref. 19.)

sac. The aspiration of even a small amount of fluid (50 mL) can restore the patient's hemodynamic status. Typically, a multihole catheter is placed in the pericardial space to allow complete drainage of the pericardial fluid. In some situations, surgical drainage may be preferred, for example, for loculated or small but hemodynamically significant effusions, or for malignant effusions. Adjunctive medical therapy for pericardial tamponade, including the use of dopamine and fluid boluses, is generally only a temporizing maneuver and should not delay the definitive treatment by drainage of the effusion. In patients who are mechanically ventilated, however, high positive airway pressure, including positive end-expiratory pressure, should be minimized, as this will further compromise right heart filling and, in turn, cardiac output.[19]

SUMMARY

Cardiac tamponade is a medical emergency requiring prompt treatment. The signs and symptoms can be subtle and especially difficult to recognize in the confines of the intensive care unit. Traditionally, the intensivist only needed to have an understanding of the pathophysiology of cardiac tamponade to be able to identify those critically ill patients at an increased risk for cardiac tamponade. With the development of HCU devices, a very important tool for diagnosing cardiac tamponade is now widely available. The ability to perform a focused bedside ultrasound exam and either rule in or rule out cardiac tamponade in a matter of minutes represents a life-saving skill that should be part of every critical care doctor's armamentarium.

References

1. Hoit BD. Pericardial disease and pericardial tamponade. *Crit Care Med.* 2007;35(suppl 8):S355–S364.
2. Roy CL, Minor MA, Brookhart MA, Choudhry MK. Does this patient with a pericardial effusion have cardiac tamponade? *JAMA*. 2007;297:1810–1818.
3. Ariyarajah V, Spodick DH. Cardiac tamponade revisited: a postmortem look at a cautionary case. *Tex Heart Inst J.* 2007;34:347–351.
4. Reddy PS, Curtiss EI, Uretsky BF. Spectrum of hemodynamic changes in cardiac tamponade. *Am J Cardiol*. 1990;66:1487–1491.
5. Sagrista-Sauleda J, Merce J, Permanyer-Miralda G, Soler-Soler J. Clinical clues to the causes of large pericardial effusions. *Am J Med*. 2000;109:95–101.
6. Joseph MX, Disney PJ, Da Costa R, Hutchison SJ. Transthoracic echocardiography to identify or exclude cardiac cause of shock. *Chest.* 2004;126:1592–1597.
7. Vignon P, Frank MP, Lesage J, et al. Hand-held echocardiography with Doppler capability for the assessment of critically-ill patients: is it reliable? *Intensive Care Med.* 2004;30:718–723.
8. Vignon P, Dugard A, Abraham J, et al. Focused training for goal-oriented hand-held echocardiography performed by noncardiologist residents in the intensive care unit. *Intensive Care Med.* 2007;33:1795–1799.
9. Reddy PS, Curtiss EI, O'toole JD, Shaver JA. Cardiac tamponade: hemodynamic observations in man. *Circulation.* 1978;58:265–272.
10. Curtiss EI, Reddy PS, Uretsky BF, Cecchetti AA. Pulsus paradoxus: definition and relation to the severity of cardiac tamponade. *Am Heart J.* 1988;115:391–398.
11. Kobal SL, Atar S, Siegel RJ. Hand-carried ultrasound improves the bedside cardiovascular examination. *Chest*. 2004;126:693–701.
12. Manasia AR, Nagaraj HM, Kodali RB, et al. Feasibility and potential clinical utility of goal-directed transthoracic echocardiography performed by noncardiologist intensivists using a small hand-carried device (SonoHeart) in critically ill patients. *J Cardiothorac Vasc Anesth*. 2005;19:155–159.
13. Oh JK, Seward JB, Tajik AJ, eds. *The Echo Manual*. 2nd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 1999:278.
14. Beaulieu Y. Bedside echocardiography in the assessment of the critically ill. *Crit Care Med.* 2007;35(suppl 5):S235–S249.
15. Himelman RB, Kircher B, Rockey DC, Schiller NB. Inferior vena cava plethora with blunted respiratory response: a sensitive echocardiographic sign of cardiac tamponade. *J Am Coll Cardiol.* 1988;12:1470–1477.
16. Gillam LD, Guyer DE, Gibson TC, King ME, Marshall JE, Weyman AE. Hydrodynamic compression of the right atrium: a new echocardiographic sign of cardiac tamponade. *Circulation.* 1983;68:294–301.
17. Reydel B, Spodick DH. Frequency and significance of chamber collapses during cardiac tamponade. *Am Heart J.* 1990;119:1160–1163.
18. Vayre F, Lardoux H, Pezzano M, Bourdarias JP, Dubourg O. Subxiphoid pericardiocentesis guided by contrast two-dimensional echocardiography in cardiac tamponade: experience of 110 consecutive patients. *Eur J Echocardiogr.* 2000;1:66–71.
19. Spodick DH. Acute cardiac tamponade. *N Engl J Med*. 2003; 349:684–690.
20. (Gyger M, Rosenberg A, Shamy A, et al. Vascular leak syndrome and serositis as an unusual manifestation of chronic graft-versus-host disease in nonmyeloablative transplants. *Bone Marrow Transplant.* 2005;35:201–203.
21. Starley BQ, Rohrer MJ, Harrison SA. Cardiac tamponade: a rare complication of interferon-induced hypothyroidism. *Resident and Staff.* 2007;53(8).

This page intentionally left blank

CHAPTER 13

Echocardiographic Diagnosis and Monitoring of Acute Myocardial Infarction and Associated Complications

Rodney W. Savage

INTRODUCTION

Despite all the effort and expense, acute myocardial infarction (MI) remains a major cause of death and disability. Ultimate progress in dealing with this plague will depend not on emergency departments, cardiac care units, and cardiac catheterization laboratories, but on the prevention of atherosclerosis. Thus, diet, exercise, and smoking avoidance coupled with early identification of atherosclerosis with effective targeted medical therapy will stabilize vulnerable plaque, reduce atherosclerotic burden, and prevent acute thrombotic events.

To the individual in the grip of acute MI, however, different and pressing priorities exist. Symptoms must be ameliorated, lethal arrhythmias identified and treated, arteries opened, and complications identified and managed. In many cases, little is needed beyond a targeted history and physical, 12-lead electrocardiogram (ECG), and simple, rapid blood work with prompt thrombolysis or emergency coronary arteriography and balloon angioplasty with or without stenting. In such straightforward cases, point-of-care echocardiography will prove interesting and perhaps helpful if potential complications are identified early. Such study, however, should never delay needed efforts at reperfusion. In other cases,[1–5] the history and physical examination may be confusing, or ECG and enzymatic data may be conflicting, misleading, or delayed. These situations include: (1) typical symptoms but normal or equivocal lab studies, (2) atypical symptoms with equivocal or abnormal lab studies, (3) pacemaker therapy, (4) left bundle branch block on ECG, (5) presence of new systolic murmur, (6) shock, including right ventricular myocardial infarction, (7) late clinical presentation, including post-MI pericarditis, (8) large, non-Q-wave MI, (9) true posterior MI, and (10) suspected LV thrombus. In these instances, point-of-care ultrasonography is not only beneficial, it may be critical for improving the understanding of the patient's condition and selecting appropriate treatment.

TECHNICAL AND ADMINISTRATIVE ISSUES

For point-of-care echocardiography to prove helpful in the acute MI setting, a simple, rapidly activated and portable machine must be present in the proximate clinical area. This machine must provide good quality two-dimensional and colored Doppler images on a wide variety of challenging patients (chronic obstructive pulmonary disease [COPD], obesity). In most situations, a full, formal follow-up echocardiogram should be obtained later with results correlated to the point-of-care echocardiography findings. Point-of-care operators require training in theory and hands-on techniques plus proctored imaging and interpretation experience. These providers will need to work closely with institutional credentialing bodies to ensure that standards of initial training, ongoing training, and quality assurance are identified and met.

The three standard windows should be interrogated in each patient with and without color-flow Doppler (Figure 13.1[A,B]). Apical views should be examined first because the two-chamber, four-chamber, and five-chamber views are often readily obtained and identify all left ventricular myocardial segments in addition to the right ventricle (Figure 13.1[A]). Aortic, mitral, and tricuspid valves are easily identified. Color-flow

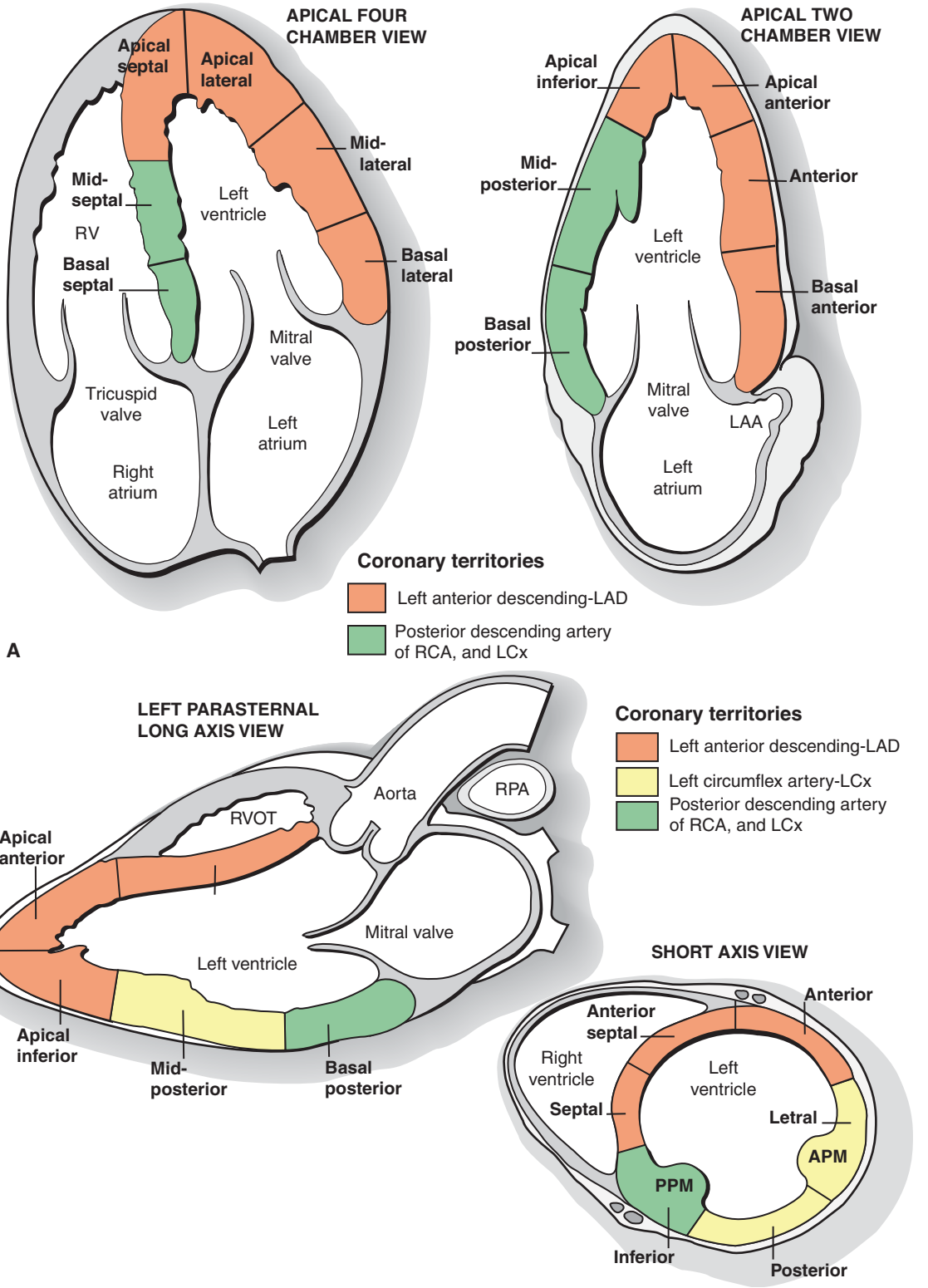


Figure 13.1. Standard views and left ventricular segments. (Reproduced with permission from Yale University Echocardiography laboratory educational website. http://www.med.yale.edu/intmed/cardio/echo_atlas/contents/index.html Left ventricular myocardial segments as seen in the standard echocardiographic windows and views with corresponding coronary artery territories.)

interrogation in the apical views readily identify ventricular septal defects and mitral insufficiency. Left parasternal short-axis views should be obtained next with expected good visualization of the apex, mid left ventricle, and left ventricle at base plus aortic and mitral valves en face (Figure 13.1[B]). The pulmonic valve is often best seen as the aortic valve is brought into view. The left parasternal long-axis views usually miss the left ventricular apex and may provide suboptimal visualization of the inferior and posterior walls even with the far gain turned up. Aortic valve and mitral valve are usually well seen and color-flow Doppler provides good visualization of aortic and mitral insufficiency. Although difficult to obtain in obese patients and in patients with severe chest pain or congestive heart failure, subcostal views prove helpful in visualizing left and right ventricular endocardium in addition to the aortic, mitral, and tricuspid valves. Sometimes, adequate visualization of the left ventricular apex will only be available in these views, making operator experience and proficiency mandatory. Having the acutely ill patient bend at the knees will often help in obtaining subcostal views, but this may be difficult in many circumstances. Sometimes the images are only obtainable after initial medical therapy with oxygen, nitrates, morphine, and diuretics.

During each study, every reasonable effort should be made to identify each major myocardial segment[6,7] and right ventricular free wall, left atrium, right atrium, aortic, mitral, pulmonic, and tricuspid valves with and without color interrogation. Aortic root, transverse aorta, and descending aorta should be identified. Pericardial effusion, ventricular septal defect, and a left ventricular thrombus should be sought. Structures not adequately visualized cannot be described. If the endocardium is not seen, a wall motion abnormality cannot be identified. After the study, findings must be recorded, even when incomplete or limited for later correlation and quality assurance. Never make bad decisions based on bad images. The point-of-care echocardiographer must always be ready to get assistance from another echocardiographer who may be able to acquire better images, or help from another technology such as cardiac computerized tomography, transesophageal echocardiography, or cardiac catheterization.

TYPICAL SYMPTOMS AND NORMAL/EQUIVOCAL LABS

Patients may present acutely with typical chest discomfort with symptom onset <1 hour. The initial ECG may show only peaked T waves or anterior ST-segment depressions. Initial bedside cardiac enzymes may well be negative. With continued ischemia more straightforward, ST-segment elevations may be seen on subsequent ECGs prompting thrombolysis or emergent catheter-directed intervention. Continued symptoms and positive enzymes might similarly prompt a trip to the catheterization laboratory even without classic ST-segment elevations. During the time between presentation and decision, muscle loss will continue.

When initial diagnostic confusion and uncertainty might lead to costly delays, immediate bedside echocardiography can result in diagnostic clarity and timely reperfusion. In patients presenting very early with acute MI, enzymes will be normal and ECG may show only peaked (hyper acute) T waves. A true posterior MI may show only anterior ST-segment depressions, mirror images of ST-segment elevations.[8] These changes may be unrecognized, subtle, or, in the case of peaked T waves, due to hyperkalemia. Occasionally, similar T waves may occur in young, healthy athletes with or without symptoms. Waiting for ECG evolution or abnormal cardiac enzymes can lead to delay with unnecessary myocardial muscle loss. Bedside echocardiography demonstrating clear segmental wall motion abnormality will prompt earlier decision, treatment, and myocardial salvage.

ATYPICAL SYMPTOMS WITH EQUIVOCAL OR ABNORMAL STUDIES

A minority of patients with acute ischemia lack typical chest discomfort. Atypical presentations include shortness of breath, diaphoresis, nausea, vomiting, abdominal pain, syncope, profound weakness, and feeling of doom. Diabetic and postoperative patients are often asymptomatic or nearly so. Appropriately, emergency department physicians, intensive care physicians, and internal medicine and cardiology consultants cast a broad diagnostic net in these situations. Minor cardiac enzyme abnormalities and nonspecific ECG changes frequently result. Should aggressive treatment await further testing hours down the line? Should the risks of intervention be undertaken in high comorbidity patients without better information? For example, no one likes to take a fresh postoperative patient to the catheterization laboratory for urgent angioplasty with or without stent placement. Immediate echocardiography may show little or no segmental wall motion abnormality. Such a patient should do well with a conservative approach. If a large segmental wall motion

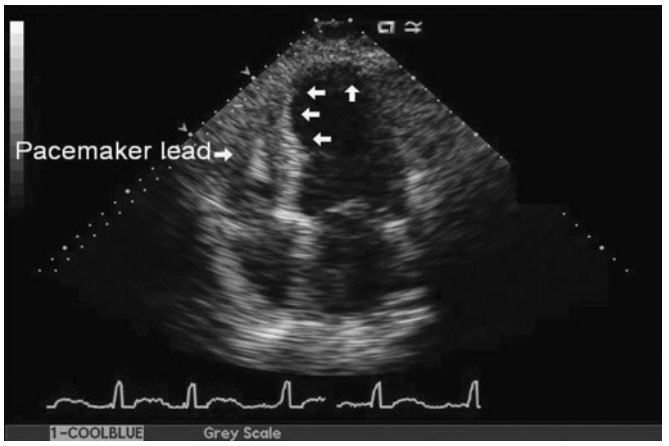


Figure 13.2. Apical four chamber views in systole showing akinesis of mid and distal anterior septal segments and apex. Note the pacemaker lead in the right heart.

abnormality were found,[9,10] the more aggressive invasive approach might well be worth an increased risk of bleeding in an effort to achieve significant myocardial salvage. Even if catheter-directed intervention did not follow, a large defect might prompt intensive care unit monitoring and more intensive medical therapy.

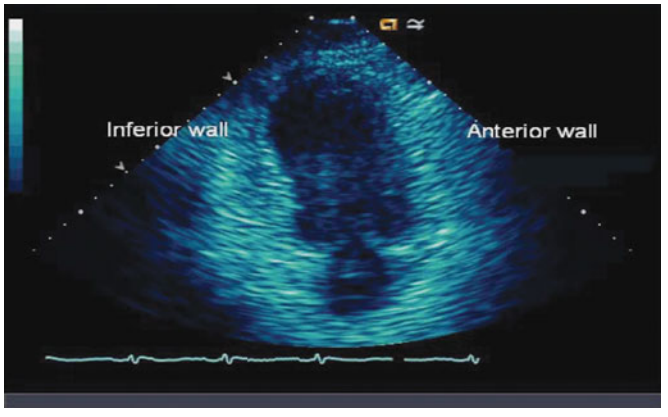
Pacemaker Therapy

Ventricular pacing may certainly mask an acute transmural MI.[11] Echocardiography will often demonstrate a new segmental wall motion abnormality over and above the expected left bundle branch block (LBBB) contraction timing abnormality seen with right ventricular (RV) pacing (Figure 13.2). A nontransmural event may or may not lead to a noticeable segmental wall motion abnormality. Following a fresh pacemaker lead implantation, a lead tip perforation may cause pericardial irritation with or without a pericardial effusion. Rarely, cardiac tamponade may result. Imaging may avert inappropriate anticoagulation with possible dire consequences. Alternatively, a large segmental wall motion abnormality without effusion would tip the scales toward immediate reperfusion attempts. This differential, established by echocardiography is less than 5 minutes, can prove potentially life saving.

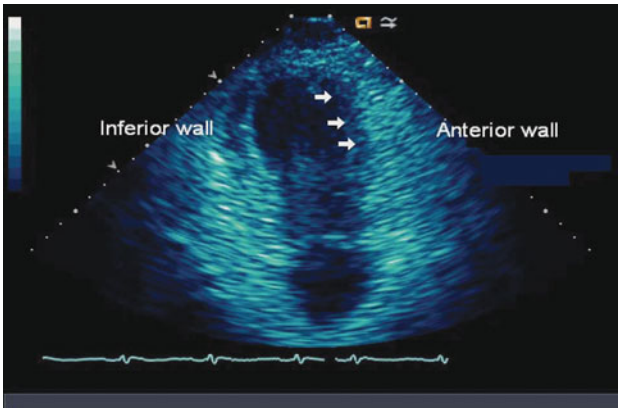
Left Bundle Branch Block (LBBB)

Approximately 3.7% of acute MIs present with new or age-undetermined LBBB.[12] Even preexisting LBBB does not exclude acute MI in the symptomatic patient.[13] With an early clinical presentation, a normal troponin provides little reassurance and help. Without early revascularization, patients may well suffer large infarctions and either death or disability due to congestive heart failure while waiting for subsequent cardiac enzymes to point the way. Yet, a patient's atypical story may make decision-making tough. Admission, observation, and further enzymes can follow with resulting myocardial loss.

An LBBB causes a contraction timing abnormality of the interventricular septum and anterior wall of left ventricle. The muscle exhibits appropriate thickening when not ischemic or infarcting. Left bundle branch block with MI shows the expected segmental wall motion abnormality without myocardial thickening in the infarcting segment (Figure 13.3[A,B]), making the clinical decision much more timely, confident, and straightforward.



A



B

Figure 13.3. Apical two chamber views in diastole (A) and systole (B) showing akinesis of anterior myocardial segments. Of special note, there is no myocardial thickening. Similar two chamber views in diastole (2c) and systole (2d) from a Patient with LBBB without MI demonstrate systolic movement and thickening.

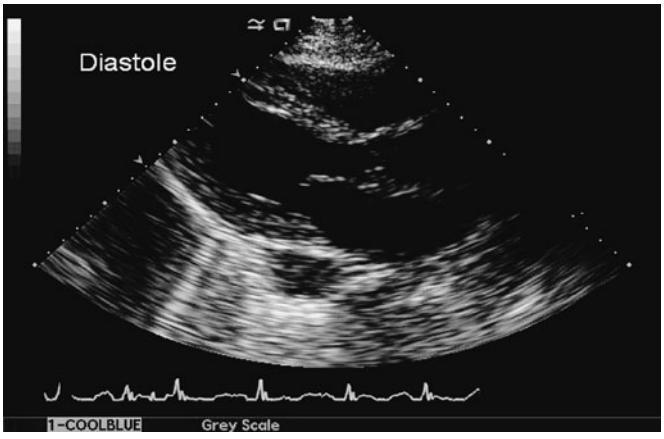
MYOCARDIAL INFARCTION AND NEW MURMUR

Myocardial rupture following MI can involve: (1) a papillary muscle or papillary muscle head, causing acute mitral regurgitation,[14,15] (2) the interventricular septum leading to an acute muscular ventricular septal defect,[16] (3) the left ventricular free wall leading rapidly to cardiac tamponade and death,[17,18] and (4) contained rupture causing pseudoaneurysm.[19] All of these entities, except free wall rupture, include a new systolic murmur. Unidentified and untreated, all carry a poor prognosis that worsens as time passes.

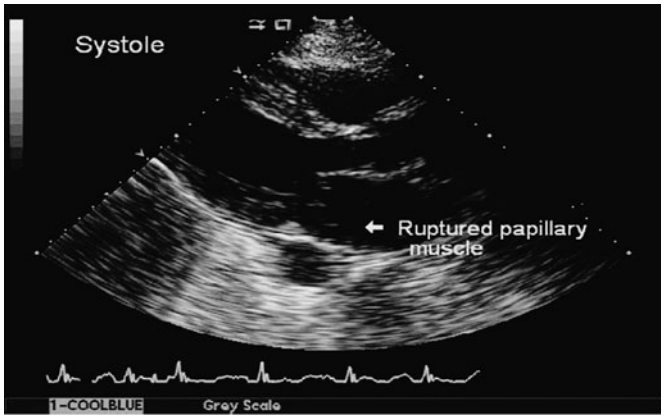
Rupture of a papillary muscle usually occurs 3–5 days after MI. Posteromedial papillary muscle rupture, associated with inferior MI (Figure 13.4) occurs more frequently than rupture of the anterolateral papillary muscle, associated with anterolateral myocardial infarction. Complete transection leads to rapid hemodynamic deterioration and death due to torrential mitral regurgitation.[15] Rupture of a tip or head of the muscle is more common and usually results in severe, but not overwhelming mitral regurgitation. Treated medically, 90% of patients succumb. However, with a timely diagnosis and surgical intervention, this can be reduced to 40–90%, depending on timing of treatment, severity of left ventricular damage, and severity of shock on presentation.[14]

Rupture of the interventricular septum in MI occurs 3–5 days after the inciting event and causes a new systolic murmur that may include a thrill. Anterior MI is more common than inferior MI. Although the diagnosis can be made with an oxygen saturation stepup on right heart catheterization, echocardiography with colored Doppler provides more complete and timely diagnosis. With prompt surgical repair, 90% mortality drops to 50%.[16]

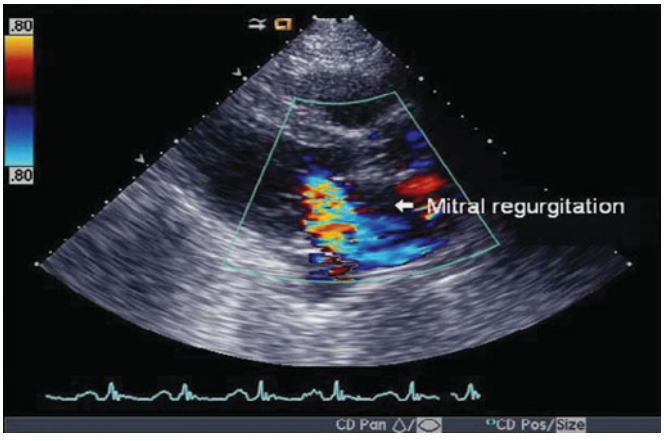
Survival with free wall rupture occurs rarely only with immediate diagnosis and surgical relief. Only with immediate echocardiography can rapid cardiac tamponade, cardiac arrest, and death be averted by heroic surgical intervention. Most cases occur 3–6 days after initial infarction, usually an anterolateral event. Up to 10% of autopsy series find this situation.[16] Rare case reports of success[20] may become more common as rapid diagnosis becomes more available. At the other end of the clinical spectrum, post-MI pseudoaneurysm of the left ventricle usually occurs with an inferoposterior



A

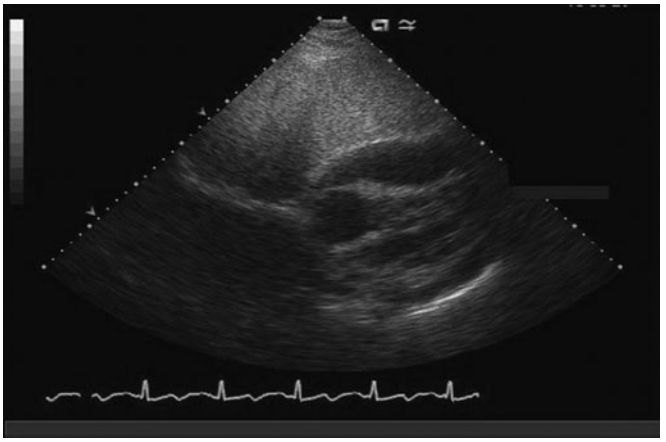


B

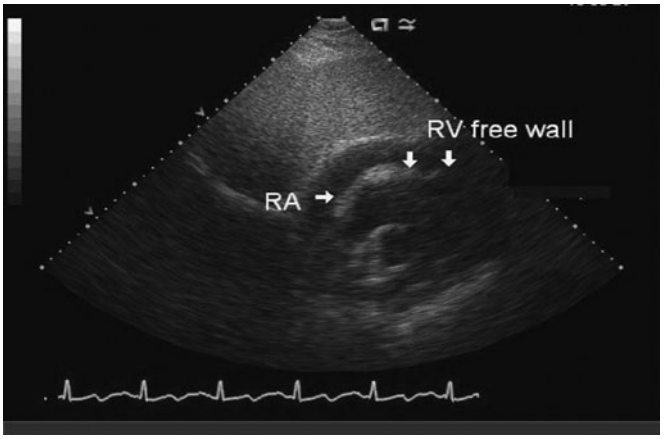


C

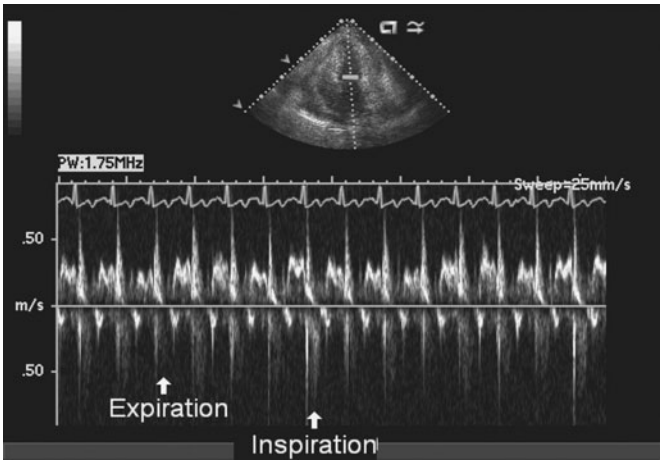
Figure 13.4. Left parasternal long axis views in diastole (A) and systole (B) reveal LAD coronary artery distribution akinesis with (using color flow Doppler interrogation) severe mitral regurgitation in systole.



A



B



C

Figure 13.5. Subcostal four chamber views in diastole (A) and systole (B) show a moderate pericardial effusion with collapse (arrows) of RV and RA free walls in diastole strongly suggestive of cardiac tamponade. Pulsed Doppler interrogation of LV inflow track with respiration (C) shows significant variation in diastolic frequency shift amplitudes inspiration (higher) and expiration (lower), confirming tamponade physiology. Caution must be high when calling wall motion abnormalities in the presence of significant pericardial effusion as both false positive and false negative situations occur.)

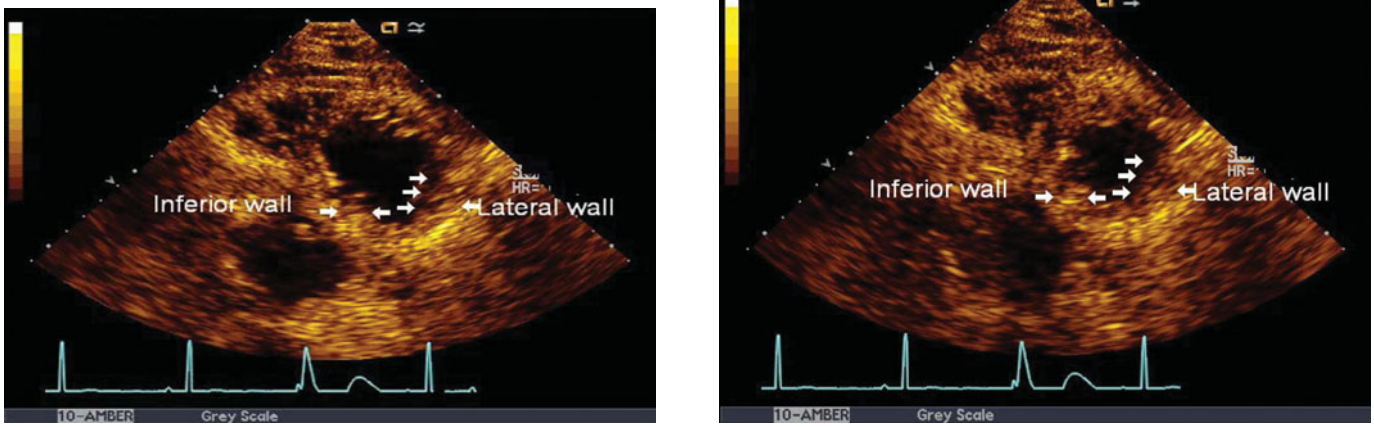
event and may be associated with a new systolic murmur, arrhythmia, thromboemboli, and congestive heart failure. Surgical repair is recommended even when asymptomatic, as late rupture presents a real and unpredictable risk.[19]

SHOCK, INCLUDING RIGHT VENTRICULAR MYOCARDIAL INFARCTION

Cardiogenic shock continues to occur in acute MI patients. Usually, large infarctions with late or no reperfusion can be blamed. As noted above, ventricular septal defect, acute mitral regurgitation, and free wall rupture need to be considered. Sometimes, a new infarction of moderate or even small size superimposed on old infarction will lead to shock. With an inferior wall infarction, right ventricular involvement may well lead to shock even with limited left ventricular damage with or without reperfusion.[21] In such patients, aggressive intravenous (IV) fluids, vasopressors, intraaortic balloon pump (IABP) support and, rarely, atrioventricular (AV) sequential pacing may be needed. Finally, pericardial effusion with cardiac tamponade[22,23] may lead to a shock state (Figure 13.5). This may occur after an inadvertent and unrecognized guide wire micro perforation during attempts at catheter-directed reperfusion. Timely, accurate diagnosis will lead to appropriate lifesaving treatment ranging from emergency angioplasty with or without stenting to coronary artery bypass surgery and ventricular septal defect or mitral regurgitation repair. In the case of cardiac tamponade, anticoagulation must be avoided and drainage promptly undertaken.

LATE PRESENTATION, INCLUDING POSTMYOCARDIAL INFARCTION PERICARDITIS

Patients who present to medical attention late in the course of MI may offer varied and, at times, confusing clinical pictures. Some suffer a stuttering and increasingly severe course. Others experience postinfarction angina, arrhythmia, or congestive heart failure. A



A B

Figure 13.6. Left parasternal short axis views in diastole (A) and systole (B) show a large area of akinesis involving lateral and inferolateral segments (arrows).

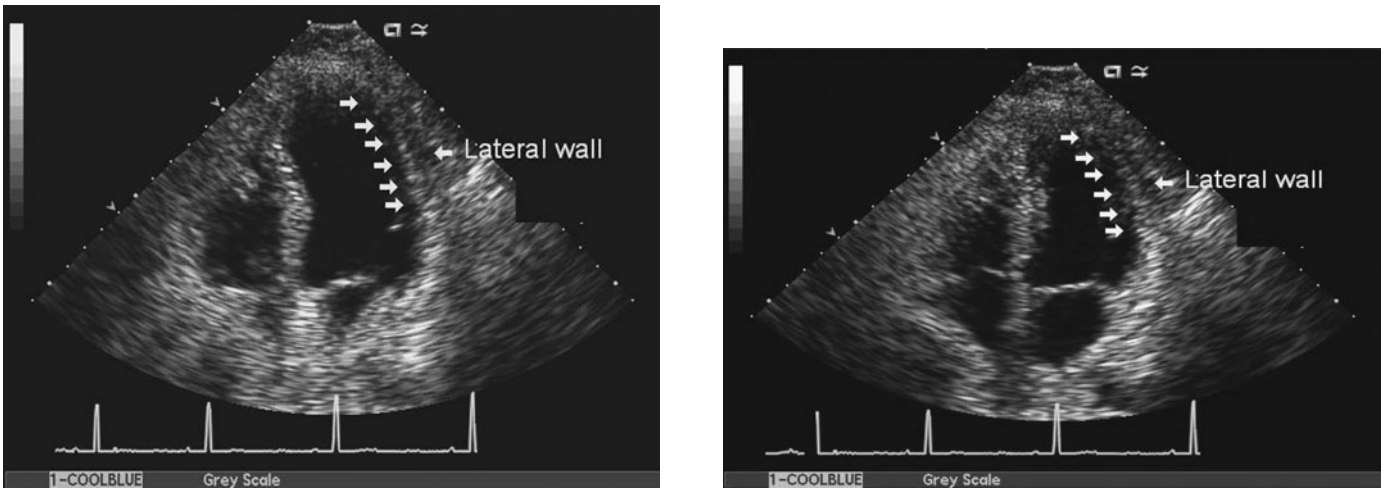
well-tolerated infarction may be followed a short time later by a second infarction with increased pain and/or congestive heart failure. Finally, post-infarction pericarditis may cause more pain than the initial infarction, prompting the patient to seek attention. For example, a patient gave a story of moderate pain and nausea five days ago. Recurrent, but more severe pain then prompted an emergency department visit with minimal elevation of creatine kinase (CK) muscle and brain (CK-MB) and moderate elevation of troponin. Electrocardiogram demonstrated normal sinus rhythm, inferior Q waves, and diffuse ST-T wave changes and 1 millimeter (mm) ST elevations in V1 and V2. The bedside echocardiogram followed (Figure 13.6). This study did not show a pericardial effusion. Post-MI pericarditis could certainly exist without one, but diffuse ST-segment elevations and PR depression in a VR were not seen. There was no pericardial friction rub. The study did show expected inferoposterior akinesis. This alone could have led to the V1 and V2 ST-segment elevations, the equivalent of posterior ST-segment depression.[8] There was no evidence of pending myocardial rupture. Of importance, akinesis also appeared in the mid-to-distal left anterior descending (LAD) artery distribution, strongly implicating a second acute event likely to lead to congestive heart failure and, potentially, cardiogenic shock without timely reperfusion. Like this situation, late-presentation MI patients commonly provide confusing clinical stories and unclear clinical pictures when only history, physical, ECG, and enzymes are considered. Bedside echocardiography should be used liberally to provide further definition and more timely decisions.

LARGE, NON–ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

Most non–ST-segment elevation MIs can be stabilized medically with coronary arteriography pursued electively, but soon after presentation.[24] Many are small events. In patients who stabilize nicely with limited myocardial damage, a more conservative approach can be elected with coronary arteriography pursued later, or reserved for recurrent symptoms, poor left ventricular function on noninvasive evaluation, and/or significant ischemia on stress imaging study. Early in the clinical course, separating small from large events can prove difficult. A bedside echocardiogram may reveal a large segmental wall motion abnormality (Figure 13.7). Even with improving symptoms, a large area of involvement[10] might prompt an aggressive approach with immediate invasive study with anticipated early revascularization.

TRUE POSTERIOR MYOCARDIAL INFARCTION

With true posterior MIs, the ECG may show only anterior ST depressions which, in fact, represent posterior ST elevations.[8] Other potential diagnoses such as acute pulmonary embolism or pleural effusion with pain may lead to delay in correct diagnosis and myocardial loss. Immediately obtained bedside echocardiography (Figure 13.8) can readily show posterobasal and inferolateral wall motion abnormalities leading to revascularization. Alternatively, normal wall motion in



A B

Figure 13.7. Apical four chamber views in diastole (A) and systole (B) show akinesis of the lateral segments extending to apex (arrows).

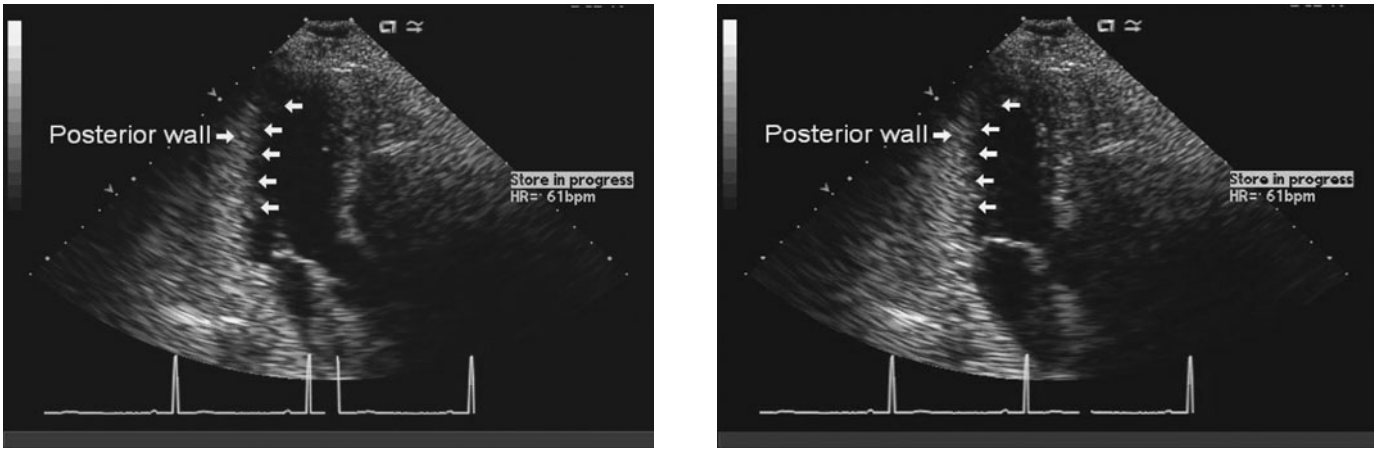
this setting would point to a pulmonary cause of similar life-threatening potential, but requiring a different therapeutic approach.

LEFT VENTRICULAR THROMBI

Left ventricular thrombi continue to occur after acute MI. These may result in potentially catastrophic emboli to heart, brain, kidneys, gut, and extremities.[25,26] Left ventricular thrombi are distinctly more common with anterior location, large extent, and late presentation and/or revascularization (Figure 13.9). Usually seen in the left ventricular apex, thrombi may be absent with early imaging even in the presence of apical akinesis or dyskinesis, but easily seen three to five days later. If full dose anticoagulation is withheld, daily bedside imaging can detect thrombus development and suggest timely change in treatment. In case of unexplained thromboembolic event, prompt bedside echocardiography can lead to emergency intervention such as thrombolysis or embolectomy.

CONCLUSION

Efficacious treatment of acute MI requires fast, accurate diagnosis and rapid reperfusion. Given the expense and attendant risks of thrombolytic therapy or catheter-directed intervention, overtreatment must be



A B

Figure 13.8. Apical two chamber views in diastole (A) and systole (B) reveal inferior akinesis plus additional mid to distal anterior and apical akinesis. An apical thrombus is not seen in this patient with a recent inferior MI and an acute anterior MI.

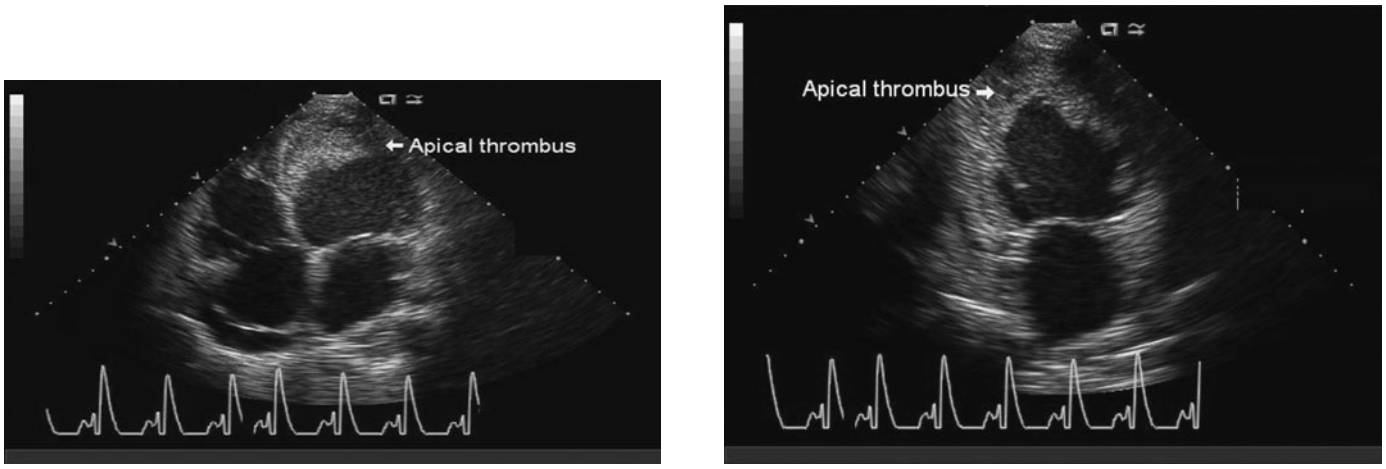


Figure 13.9. Apical four (A) and two (B) chamber views in diastole show a definite apical thrombus.

minimized without missing opportunities to save muscle, preserve function, and prolong life. Similarly, major complications require timely diagnosis if surgical intervention, pericardiocentesis, and repeat coronary intervention are to be effective. Echocardiography has long been recognized as helpful in the acute MI environment. Wide deployment of this powerful imaging technology, however, lacked portable equipment and adequately trained and experienced operators/interpreters. New, reasonably priced machines provide easy portability and availability. Emergency department (ED) physicians, ICU physicians, hospitalists, cardiologists, and even some physician extenders are acquiring needed training and experience. These professionals will use their skills and expertise with increasing frequency to foster expedited, more accurate diagnosis and, when needed, targeted, aggressive treatment. The examples above will serve only as a starting point, as providers with readily available bedside imaging will identify new and important applications.

References

1. Gibson RS et al. Value of early two dimensional echocardiography in patients with acute myocardial infarction. *Am J Cardiol.* 1982;49:1110.
2. Horowitz RS et al. Immediate diagnosis of acute myocardial infarction by two-dimensional echocardiography. *Circulation.* 1982;65:323.
3. Reimold SC, Antman EM. Noninvasive cardiac imaging in chest pain syndromes. *J Thrombosis Thrombolysis.* 1998; 6:239.
4. Peels CH et al. Usefulness of two-dimensional echocardiography for immediate detection of myocardial ischemia in the emergency room. *Am J Cardiol.* 1990;65:687.
5. Sabia P et al. Importance of two-dimensional echocardiographic assessment of left ventricular systolic function in patients presenting to the emergency room with cardiac-related symptoms. *Circulation.* 1991;84:615.
6. Cerqueira MD et al. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart. A statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation.* 2002;105:539.
7. Bourdillon PD et al. Regional wall motion index for infarct and non-infarct regions after reperfusion in acute myocardial infarction: comparison with global wall motion index. *J Am Soc Echocardiogr.* 1989;2: 398.
8. Zimetbaum PJ, Josephson ME. Use of the electrocardiogram in acute myocardial infarction. *N Engl J Med.* 2003; 348:933.
9. Romano S et al. Usefulness of echocardiography in the prognostic evaluation of non-Q-wave myocardial infarction. *Am J Cardiol*. 2000;86:43G.
10. Horowitz RS, Morganroth J. Immediate detection of early high-risk patients with acute myocardial infarction using two-dimensional echocardiographic evaluation of left ventricular regional wall motion abnormalities. *Am Heart J.* 1982;103:814.
11. Sparbossa EB, Pinski SL, Gates KB, et al. Early electrocardiographic diagnosis of acute myocardial infarction in the presence of ventricular paced rhythm. GUSTO-1 investigators. *Am J Cardiol.* 1996;77:423.
12. Shlipak MG, Go AS, Frederick PD, et al. Treatment and outcomes of left bundle branch block patients with

myocardial infarction who present without chest pain. *J Am Coll Cardiol.* 2000;36:706.

13. Madias JE, Sinha A, Ashitani R, et al. A critique of the new ST-segment criteria for the diagnosis of acute myocardial infarction in patients with left bundle branch block. *Clin Cardiol.* 2001;10:652.
14. Birnbaum Y, Chamoun AJ, Conti VR, et al. Mitral regurgitation following acute myocardial infarction. *Coron Artery Dis.* 2002;13:337.
15. Bias B, Graba J, Siu S, et al. Papillary muscle rupture complicating an acute myocardial infarction. *Can J Cardiol.* 2001;17:722.
16. Birnbuam Y, Fishbein MC, Blanche C, et al. Ventricular septal rupture after acute myocardial infarction. *N Eng J Med.* 2002;347:1426.
17. Sugiura T, Nagahama Y, Nakamura S, et al. Left ventricular free wall rupture after reperfusion therapy for acute myocardial infarction. *Am J Cardiol.* 2003;92: 282.
18. Spodick DH. Acute cardiac tamponade. *N Engl J Med.* 2003;349:684.
19. March KL et al. Current concepts of left ventricular pseudoaneurysm: pathophysiology, therapy, and diagnostic imaging methods. *Clin Cardiol.* 1989;12:531.
20. Renyen K, Strasser RH. Images in clinical medicine. Impending rupture of the myocardial wall. *N Engl J Med.* 2003;348:e3.
21. Mehta SR, Eikelboom JW, Natarajan MK, et al. Impact of right ventricular involvement on mortality and morbidity in patients with inferior myocardial infarction. *J Am Coll Cardiol*. 2001;37:37.
22. Picard LA et al. Incidence and significance of pericardial effusion in acute myocardial infarction as determined by two-dimensional echocardiography. *J Am Coll Cardiol.* 1986;8:517.
23. Figueras J et al. Nature and progression of pericardial effusion in patients with a first myocardial infarction: relationship to age and free wall rupture. *Am Heart J.* 2002; 144:251.
24. Anderson JL, Adams CD, Antman EM, et al. ACC/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction-executive summary; a report of the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non-ST-Elevation Myocardial Infarction). *J Am Coll Cardiol*. 2007;50:652.
25. Jugdutt BI, Sivaram CA. Prospective two-dimensional echocardiographic evaluation of left ventricular thrombus and embolism after acute myocardial infarction. *J Am Coll Cardiol.* 1989;13:554.
26. Keren A et al. Natural history of left ventricular thrombi: their appearance and resolution in the post hospitalization period of acute myocardial infarction. *J Am Coll Cardiol.* 1990;15:790.

CHAPTER 14

Echocardiographic Diagnosis of Cardiomyopathies

Narinder P. Bhalla, Amitabh Parashar, and Marguerite Underwood

INTRODUCTION

In its simplest form, cardiomyopathy (CMP) can be defined as a cardiac disorder involving myocardial dysfunction. Though there are various formal definitions of CMP in the literature, the two major ones are from the World Health Organization (WHO) and from the American Heart Association (AHA). The WHO definition is more clinical, while the AHA definition is more molecular and scientific in its delineation. For the purpose of this chapter, we will follow the simpler definition by WHO as it lends itself well to the echocardiographic evaluation of cardiomyopathies.

Cardiomyopathies can also be defined by their etiology, as was done also by the WHO task force in 1995. Since the etiology may not always be obvious, such as in many cases of dilated CMP, this classification is also less useful when discussing the general principles of echocardiographic diagnosis of CMP. The various types of cardiomyopathies, as classified by WHO, are listed in Table 14.1. Each type of CMP can have various etiologies, the discussion of which is beyond the scope of this chapter (see suggested reading list for further information). The various etiologies are nonetheless listed in Table 14.2. Table 14.3 demonstrates the key elements in the echocardiographic evaluation of a patient with a CMP.

As we describe the echocardiographic features of the various cardiomyopathies, we will incorporate the elements described above. The reader should recognize that this chapter assumes familiarity with the basic echocardiographic views and how to obtain those views. It also needs to be emphasized that the two-dimensional image and M-mode interrogations need to be of good quality to make reliable interpretations.

DILATED CMP

Dilated cardiomyopathies have various etiologies, and the dilated state usually represents the response of the myocardium to the various insults. Though the dilated state may represent a common response to multiple types of insults, every effort should be made to identify a potentially remediable cause, as this may significantly influence the prognosis. Dilated cardiomyopathies generally are classified as either ischemic or nonischemic, the latter category being rather broad and inclusive of those caused by valvular diseases (Table 14.2).

Echocardiography is a critical tool in assessing patients with a dilated CMP, and, in some cases, can help elucidate the etiology of the CMP and gauge prognosis and response to treatment. The most common clinical presentation of a patient with a dilated CMP is congestive heart failure, associated with dyspnea and a fluid overload state. Echocardiographic evaluation of a patient presenting with dyspnea, with or without a fluid overload state, should be undertaken early in the course of management if a clear etiology is not evident on presentation, such as symptoms or signs of coronary ischemia, electrocardiograpahic changes suggestive of myocardial ischemia, a history of known CMP, or a recent echocardiogram. If a remediable etiology is suspected, like coronary ischemia and mitral regurgitation (MR), every effort should be made to alleviate the condition. Echocardiography should still be carried out in such a case, and the timing should depend on the availability of the ultrasound equipment and the delay it might cause in treatment. However, the current availability of good-quality portable equipment can expedite such evaluations.

The echocardiographic diagnosis of a dilated CMP rests solely on the demonstration of a dilated, hypofunctional left ventricle. Though various secondary features are frequently evident on the echocardiogram, the diagnosis can only be made by the demonstration of left ventricular dilatation.

Features of Dilated CMP

Left Ventricular Dilatation (Figure 14.1)

Left ventricular (LV) dilatation is best evaluated in the parasternal long-axis view (Figure 14.1A). The

TABLE 14.1. The WHO classification of cardiomyopathies

Dilated CMP
Hypertrophic CMP
Restrictive CMP
Arrhythmogenic right ventricular CMP (dysplasia)
Other unclassified cardiomyopathies

CMP indicates cardiomyopathy; WHO, World Health Organization.

spherical change in the LV cavity with dilatation is more apparent in this view, and is represented by an increasing vertical axis when compared with the horizontal axis (Figure 14.1B). Global dilatation of all four chambers, however, is better appreciated in the apical four-chamber view. The LV dilatation may be mild, moderate, or severe, and accurate measurements are important to grade the degree of dilatation. The short-axis view at the level of the papillary muscles is also a good view to assess LV dilatation. If there is difficulty in visualizing the endocardium, echocardiographic contrast can be used; the risk/benefit ratio of the use of such agents should be assessed in each individual patient (we recommend avoiding the agents in acute ischemia and in acute heart failure).

Left Ventricular Wall Motion and Thickening (Figure 14.2[A–C])

Left ventricular wall motion is universally impaired in patients with a dilated CMP (Figure 14.2[A,B]). The wall motion abnormalities may well be regional in patients with an ischemic etiology. These regional abnormalities correlate well with impairment of vascular supply to the dysfunctional wall. However, basal wall motion may be better preserved than other regions in some patients with a nonischemic, dilated CMP, leading to occasional confusion with ischemic CMP. This should be kept in mind when trying to distinguish between ischemic and nonischemic etiologies. Another potential issue may arise in patients with a dilated CMP and a left bundle branch block (LBBB) or a dilated CMP and previous cardiac surgery. The bundle branch abnormality may be from the increased mass from the cardiac dilatation or from an ischemic injury in the left anterior descending territory. A dyssynchronous septum is seen is such patients, and is evident in the parasternal long-axis and the apical four-chamber views. In such cases, one should concentrate on looking at other walls to make the distinction between regional and diffuse wall motion abnormalities. In patients with a previous anterior wall myocardial infarction, other wall motion abnormalities will be apparent, such as apical and/or anterior lateral hypokinesis, akinesis, or dyskinesis.

TABLE 14.2. Partial listing of etiologies of cardiomyopathies

Type of CMP	*Etiologies (examples)*
Dilated CMP	Idiopathic (mostly genetic)
	Familial
	Myocardial ischemia/infarction
	End-stage valvular heart disease
	End-stage hypertensive heart disease
	Infectious (viral myocarditis, Chagas, bacterial, etc.)
	Toxic/metabolic (chemotherapy, alcoholic, cocaine, etc.)
	Tachycardia-induced
	Peripartum
	Rheumatologic (SLE, scleroderma)
	Endocrine disorders (diabetes, thyroid disease, etc.)
	Neuromuscular diseases (Duchenne's, myotonic dystrophy, etc.)
	Electrolyte abnormalities (hypocalcemia, hypophosphatemia, etc.)
	Nutritional deficiencies (thiamine, carnitine, etc.)
	Infiltrative diseases (usually end-stage)
Hypertrophic CMP	Idiopathic/familial (asymmetric)
	Concentric
	Apical
Restrictive CMP	Idiopathic/familial
	Diabetes
	Infiltrative (amyloidosis, sarcoidosis, etc.)
	Storage diseases (hemochromatosis, glycogen storage diseases, etc.)
	Endomyocardial fibrosis
	Rheumatologic (scleroderma)
	Radiation
Unclassified Cardiomyopathies	Isolated LV noncompaction
	LV apical ballooning syndrome
	Endocardial fibroelastosis

CMP indicates cardiomyopathy; LV, left ventricular; SLE, systemic lupus erythematosus.

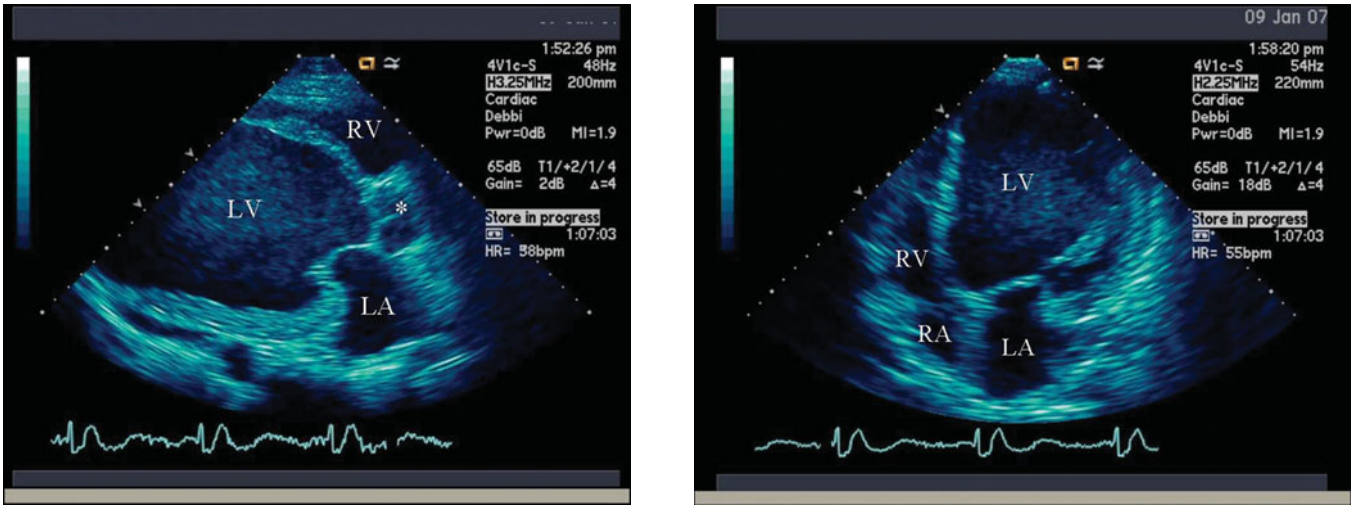
TABLE 14.3. The key elements in the echocardiographic evaluation of a patient with a CMP

Focused assessment	*Findings*
Assessment of chamber sizes and mass	Left ventricular and left atrial dilatation
	Right ventricular dilatation, either primarily or from pulmonary hypertension
	Increased cardiac mass and hypertrophy
Assessment of valvular function and valve apparatus	Tricuspid regurgitation, usually secondary from pulmonary hypertension
	Mitral valve regurgitation, either due to primary dysfunction of the valvular apparatus or secondary to annular dilatation
	A change in the position of the papillary muscles
Assessment of systolic and diastolic function	Doppler flow patterns across the mitral valve
	Tissue Doppler interrogation
	Wall motion abnormalities, either segmental or global
	Grading the systolic and diastolic dysfunction
Assessment of right heart pressures, as this will influence treatment and prognosis	
Assessment of other features	Assessment of other features
	Left atrial or left ventricular thrombus
	Appearance of the endocardium
	Assist in the diagnosis of amyloidosis or LV noncompaction, for example

CMP indicates cardiomyopathy; LV, left ventricular.

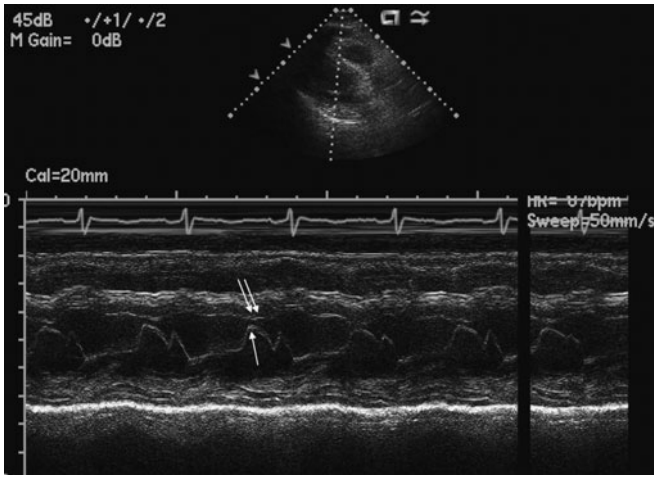
Furthermore, infarcted tissue will appear thinned on two-dimensional imaging (Figure 14.2C).

Though two-dimensional imaging is excellent at assessing wall motion, the importance of M-mode interrogation is often overlooked. M-mode interrogation may allow a better assessment of wall motion than two-dimensional imaging in circumstances where the endocardium is not well visualized by the two-dimensional approach. M-mode imaging is also important in gauging wall thickness. During normal

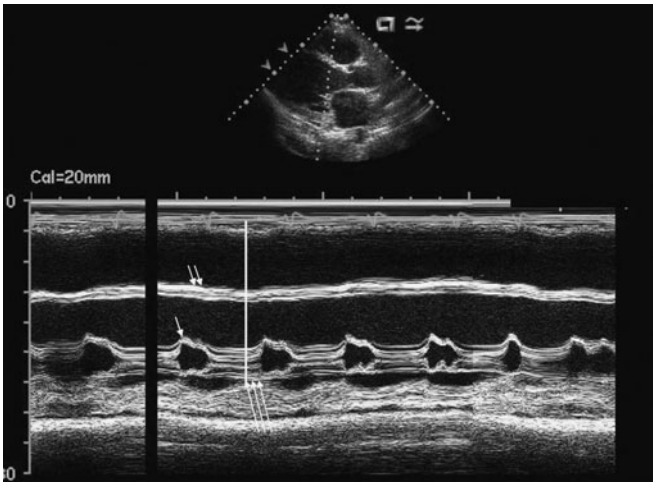


A B

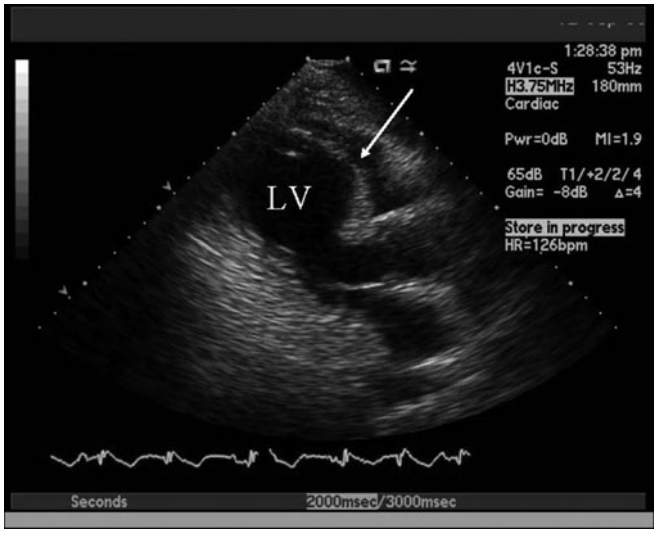
Figure 14.1. (A) A two-dimensional image of a parasternal long-axis view in a patient with a dilated cardiomyopathy. Note the dilated LV and the increased long axis of the LV. (B) Two-dimensional image of an apical four-chamber view of a patient with a dilated cardiomyopathy. Note the spherical change in the LV cavity with the dilatation. *Aortic root and aortic valve. LA indicates left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.



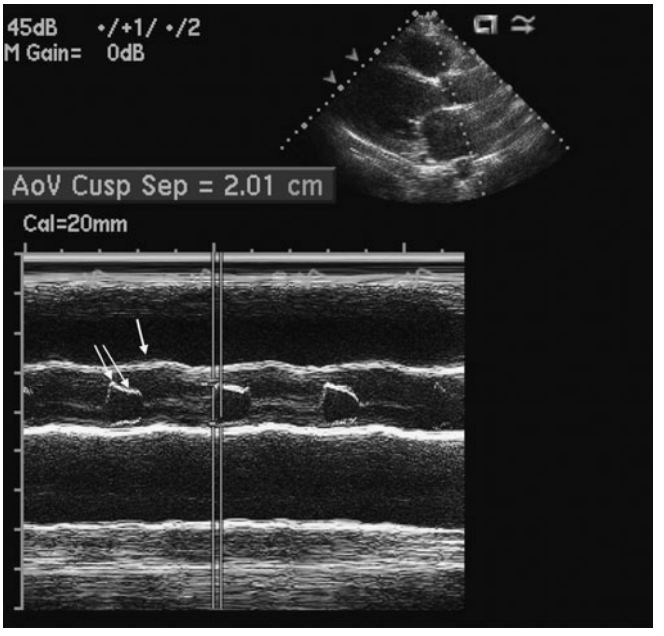
A



B



C



D

Figure 14.2. (A) M-mode through the mitral valve in a normally contractile heart. Note the close relationship of the mitral E wave (single arrowhead) with the septal M-mode (double arrowheads). This is called the EPSS (E point septal separation). (B) M-mode recording through the mitral valve in a patient with a dilated cardiomyopathy. Note the significantly abnormal separation between the mitral valve E wave (single arrowhead) and the septal M-mode (double arrowheads). The EPSS is abnormal in this patient. Also note the lack of motion and thickening with systole in the M-mode of the septum and the posterior wall (triple arrowheads). Systole is marked by the vertical solid white line. (C) Two-dimensional imaging in the parasternal long axis in a patient with a previous myocardial infarction. Note the thinned-out septum that is akinetic (arrowhead). The septum bows into the right ventricle with systole. (D) M-mode imaging in the parasternal long-axis view through the aortic root and aortic valve. Note the relatively "flat" motion of the aortic root during the cardiac cycle (single arrowhead) and the "trapezoidal" pattern of aortic valve motion (double arrowheads). See text for more information.

contractility of a myocardial segment, there is motion inward toward the ventricular cavity and associated thickening. M-mode imaging can exhibit the presence or absence of both these phenomena quite well.

Other Associated Features of Dilated CMP

Various other features (also called secondary features) may also be evident in patients with a dilated CMP.

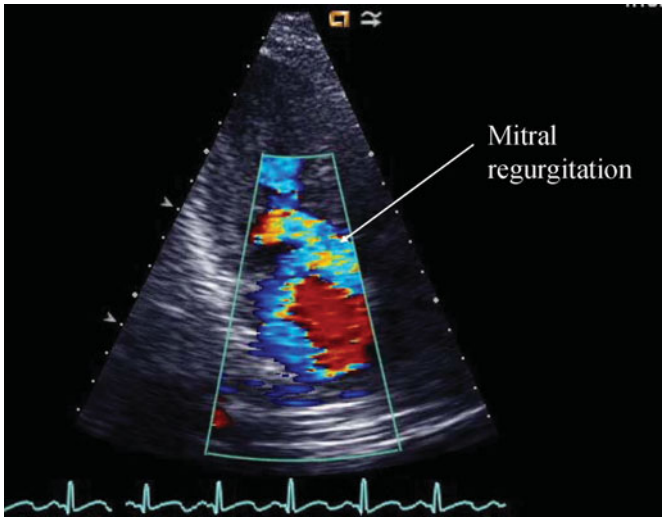


Figure 14.3. Two-dimensional imaging with color-flow showing mitral regurgitation in a patient with dilated cardiomyopathy. Note the central jet location. The mitral regurgitation in these patients is often due to dilatation of the mitral valve annulus.

Mitral Regurgitation (Figure 14.3)

A common secondary feature is mitral regurgitation (MR) (also called functional MR). This occurs due to a relative dilatation of the mitral annulus. As the left ventricle dilates and approaches a spherical shape, the mitral valve apparatus is pulled apically. This results in a relative dilatation of the mitral annulus, leading to MR. One has to make an effort, clinically, to sort out whether the MR is a primary phenomenon resulting in the dilated CMP or whether it is the result of the dilated CMP. A transesophageal echocardiogram may help in resolving the issue. If the mitral valve apparatus appears structurally normal (absence of leaflet or chordal redundancy, absence of valve thickening, absence of annular calcification, absence of papillary muscle dysfunction), and an ischemic cause of the regurgitation is ruled out, then one should consider the etiology of the regurgitation to be related to the dilated CMP. The best determination of etiology is important as it may influence the treatment approach and the choice of surgical repair technique for a particular patient. Furthermore, etiology, along with hemodynamic status and left ventricular mechanical status, also influences prognosis with or without surgical treatment. When present secondary to a CMP, the regurgitant jet is usually central (the cause is relative annular dilatation) (Figure 14.3). Various criteria have been established to judge the severity of the regurgitant jet, and though some are better accepted than others, demonstration of flow reversal in the pulmonary veins during systole and a high peak inflow velocity across the mitral valve are good correlates of severe regurgitation. A standard transesophageal echocardiogram or a three-dimensional study may be superior to standard two-dimensional imaging for determining the severity of the regurgitation. Transesophageal echocardiography, particularly, also helps in deciding the appropriate surgical approach (valve repair/reconstruction vs. replacement).

M-Mode Features

In addition to the M-mode features described above, there are other features that should be reviewed when performing an echocardiographic examination of a patient with a dilated CMP.

E Point Septal Separation (EPSS) (Figure 14.2A,B)

This is best assessed in the parasternal longaxis view with the ultrasound beam through the mitral leaflets. The E point of the mitral valve opening is quite close to the septal wall on M-mode imaging in normal individuals. However, this distance is significantly increased in patients with a dilated CMP.

Aortic Root Motion (Figure 14.2D)

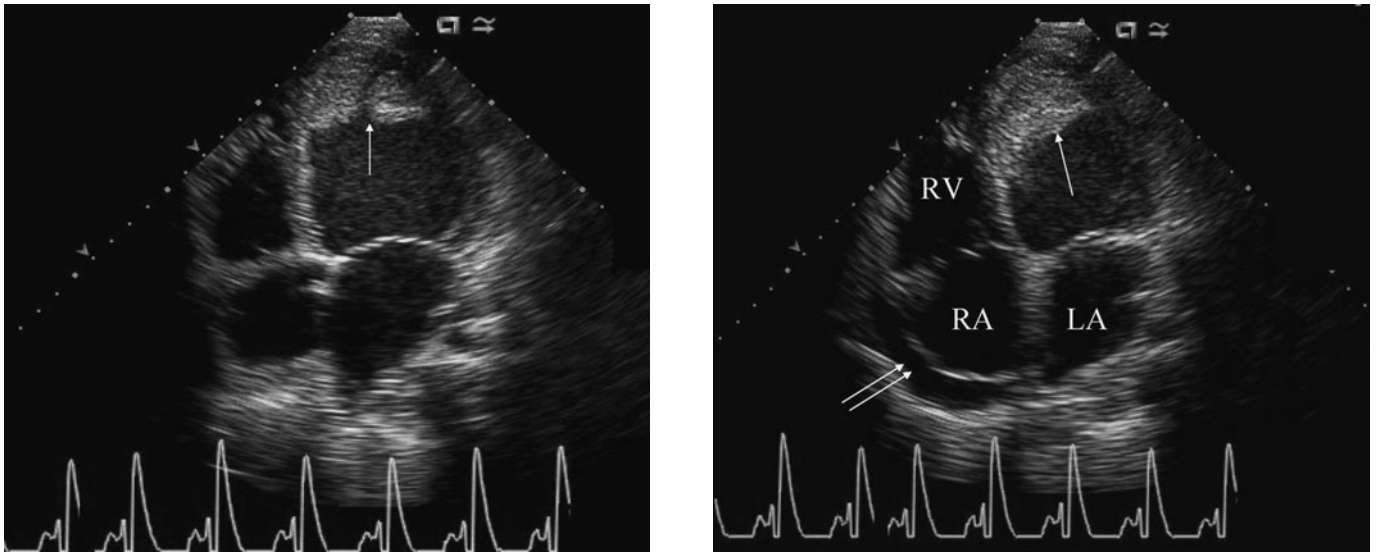
In normal circumstances, there is a sinusoidal pattern of motion exhibited by the aortic root with anterior motion during systole. This is best seen in the parasternal long axis, with the ultrasound beam focused through the aortic root at the level of the aortic leaflets. In patients with a dilated CMP, the normal sinusoidal pattern is replaced by a relatively flat pattern of motion.

Changes in Aortic Valve Opening and Closing (Figure 14.2D)

The normal pattern of aortic valve opening and closing appears as a rectangular box on M-mode imaging performed in the parasternal long-axis view through the aortic valve. In cases of reduced stroke volume, as is the case in dilated CMP, there is poor opening of the aortic valve and the valve tends to start closing earlier in systole. The pattern changes from that of a rectangular box to a more “trapezoidal” box.

Left Ventricular Thrombus (Figure 14.4)

The presence of a mural thrombus in the left ventricular cavity on two-dimensional imaging can be seen in a dilated CMP. In cases of ischemic CMP, the thrombus is most often seen in an akinetic and/or aneurysmal



A B

Figure 14.4. (A) Two-dimensional imaging in the apical four-chamber view demonstrating a left ventricular thrombus in the apex (arrowhead). (B) Apical four-chamber view demonstrating a left ventricular thrombus in the apex (single arrowhead). The thrombus occupies most of the apex. Incidentally, also note the pericardial fluid around the right atrium (double arrowheads). LA indicates left atrium; RA, right atrium; RV, right ventricle.

segment. The presence of thrombus must be distinguished from the endocardium. Though this is usually straightforward, there are helpful techniques that can be utilized if confusion exists. Contrast enhancement can be used to delineate the endocardium. Usually, the contrast will outline the thrombus by circumventing it. Color-flow can also be used in a similar fashion. A color sector focused on the area of concern will outline the thrombus, as color will not go into thrombus. Care should be taken to not have too much gain on the color jet, as this may well obscure the delineation between the thrombus and the endocardium.

Left Atrial Dilatation

Left atrial dilatation is a universally associated finding in dilated CMP. Left atrial size and volume correlate well with the severity and duration of the dilated CMP. The left atrial dilatation is caused by multiple reasons, including the secondary MR, increased diastolic pressures, and potential involvement of the left atrium itself by the myopathic process. Spontaneous echo contrast can be seen commonly in the left atrium because of impaired of blood flow, and may be more prominent if there is associated atrial fibrillation.

Right Heart Findings (Figure 14.5)

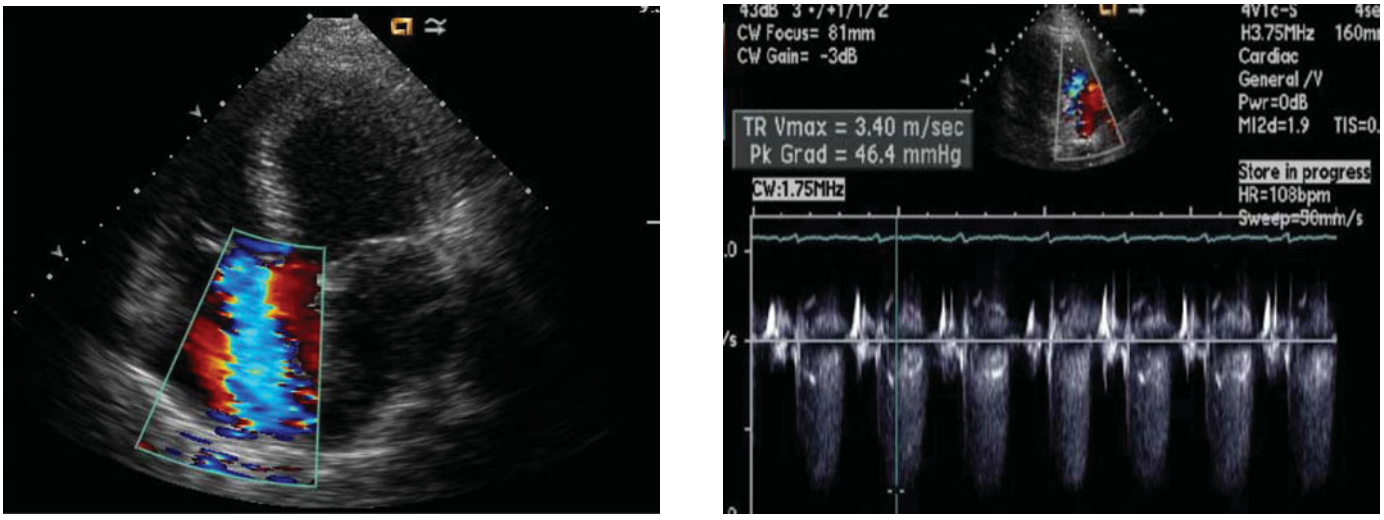
There are various right heart–related findings that can be seen in patients with a dilated CMP. These include tricuspid regurgitation, right ventricular dilatation, and pulmonary hypertension. The right ventricular dilatation is either secondary to pulmonary hypertension, which is commonly seen in such patients, or due to involvement by the myopathic process. The tricuspid regurgitation, in most cases, is a secondary phenomenon from the pulmonary hypertension and right ventricular dilatation, the latter resulting in tricuspid annular dilatation.

Assessment of Systolic and Diastolic Performance

Assessment of systolic and diastolic performance should be done in all patients with a CMP, regardless of type. The relative impairment in these indices is predictive of prognosis and clinical course. Though the evaluation of systolic and diastolic function is described in this section with reference to dilated CMP, similar principles apply with other types of cardiomyopathies.

Doppler Assessment

Doppler interrogation of all patients with a dilated CMP is important, as it can assist with the assessment of systolic and diastolic function, which helps predict prognosis. The most important predictors of survival in dilated CMP are end-diastolic and systolic volumes and ejection fraction.



A B

Figure 14.5. (A) Apical four-chamber view with color-flow showing tricuspid regurgitation. (B) Continuous-wave Doppler recording through the tricuspid valve during systole. There is tricuspid regurgitation present, and the peak gradient across the right ventricle and the right atrium is 46.4 mm Hg. If one assumes a right atrial pressure of 10 mm Hg and the absence of pulmonic valve stenosis, then the estimated pulmonary artery pressure is 56.4 mm Hg, consistent with moderate pulmonary hypertension.

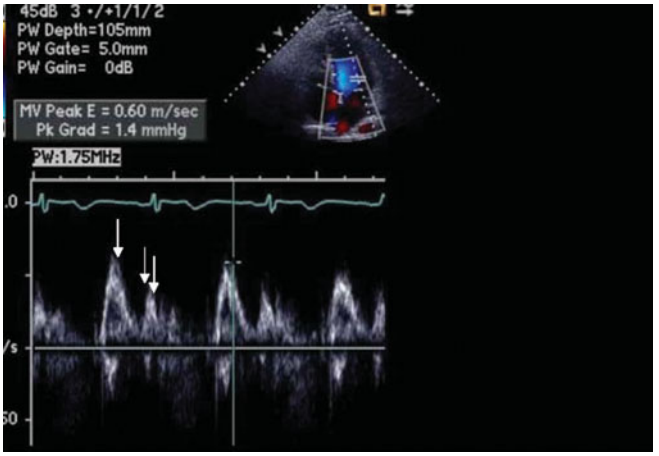
Systolic Function Assessment

The principles of laminar flow are used in determining the various indices of systolic function. This includes an assessment of stroke volume and cardiac output. The pulsed Doppler can be used to interrogate the flow in the left ventricular outflow tract (LVOT) over a period of time. This generates the time velocity integral (TVI) (sometimes also referred to as velocity time integral [VTI]). The mechanical status of the left ventricle is a key component in determining the TVI. Assuming the principles of laminar flow, the stroke volume (SV) is a product of the LVOT cross-sectional area (CSA) and the TVI (SV = TVI × LVOT CSA). Cardiac output (CO), in turn, is defined as the product of the SV and the heart rate (HR). Because the LVOT CSA can be assumed to be stable for a given patient, and hence a constant, the change in TVI is directly proportional to the change in the left ventricular mechanical status. Hence an increase in TVI can be used as gauge for improvement in left ventricular systolic function when a therapy is undertaken, such as initiation of inotropic support, alleviation of coronary ischemia, treatment of valvular insufficiency with afterload reduction, placement of an intraaortic balloon pump, or a combination thereof. With appropriate therapy, not only is there an increase in the TVI but, the shape of the envelope also changes from a more parabolic and somewhat blunted one to a more triangular one. Measurements of the acceleration and deceleration times of the mitral regurgitant jet have been identified as predictive of prognosis. These times correlate well with dP/dt (a measurement of left ventricular contractility) obtained at cardiac catheterization in similar patients. An acceleration time (dP/dt) of <600 mm Hg/sec and a deceleration time (−dP/dt) of <450 mm Hg/sec identifies a significantly higher-risk group, with a decreased event-free survival.

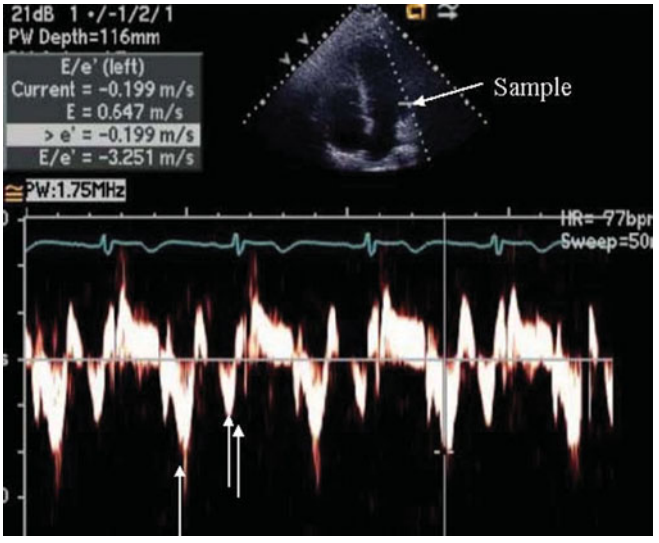
Diastolic Function Assessment (Figure 14.6)

Doppler assessment of diastolic function relies on interrogation techniques and evaluation of a few key parameters: mitral valve inflow patterns, tissue Doppler interrogation techniques, the isovolumic relaxation time, mitral flow deceleration time, and pulmonary vein flow patterns. It is important to utilize a combination of the above findings to arrive at a decision regarding the diastolic function of a patient. These parameters tend to be the most predictive with the presence of systolic dysfunction, and can be altered by various other clinical issues, such as valvular disease, heart rates, and therapeutics. Though a detailed discussion of these parameters is beyond the scope of this chapter, basic principles of the use of Doppler in evaluating diastolic dysfunction are discussed.

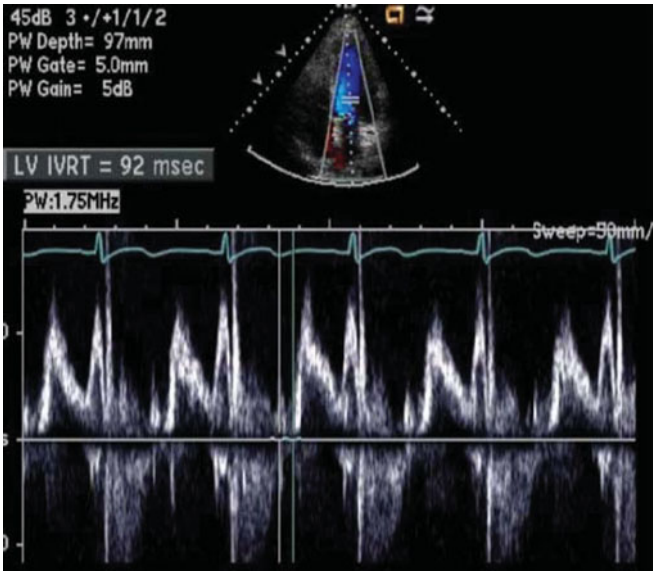
Mitral Inflow Pattern. This evaluation should be performed in the apical view with the sample volume placed at the mitral valve tips. The two major components of the inflow pattern are the E wave and the A



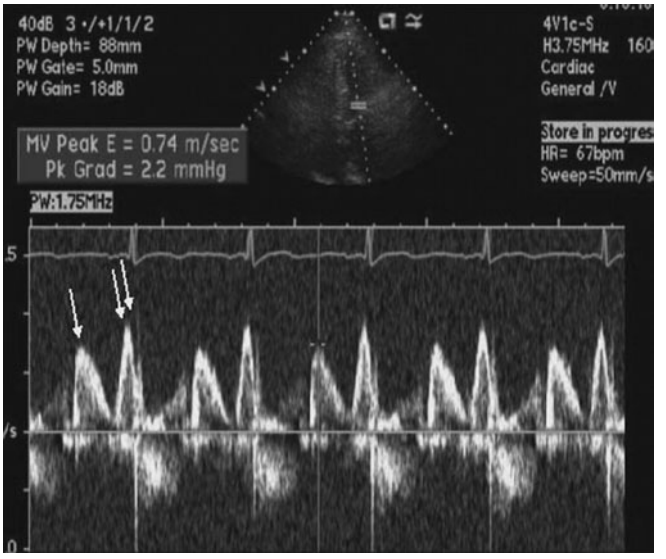
A



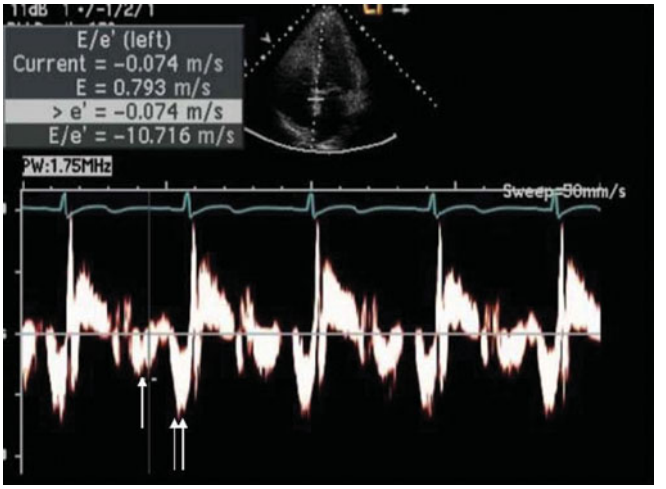
CL



D



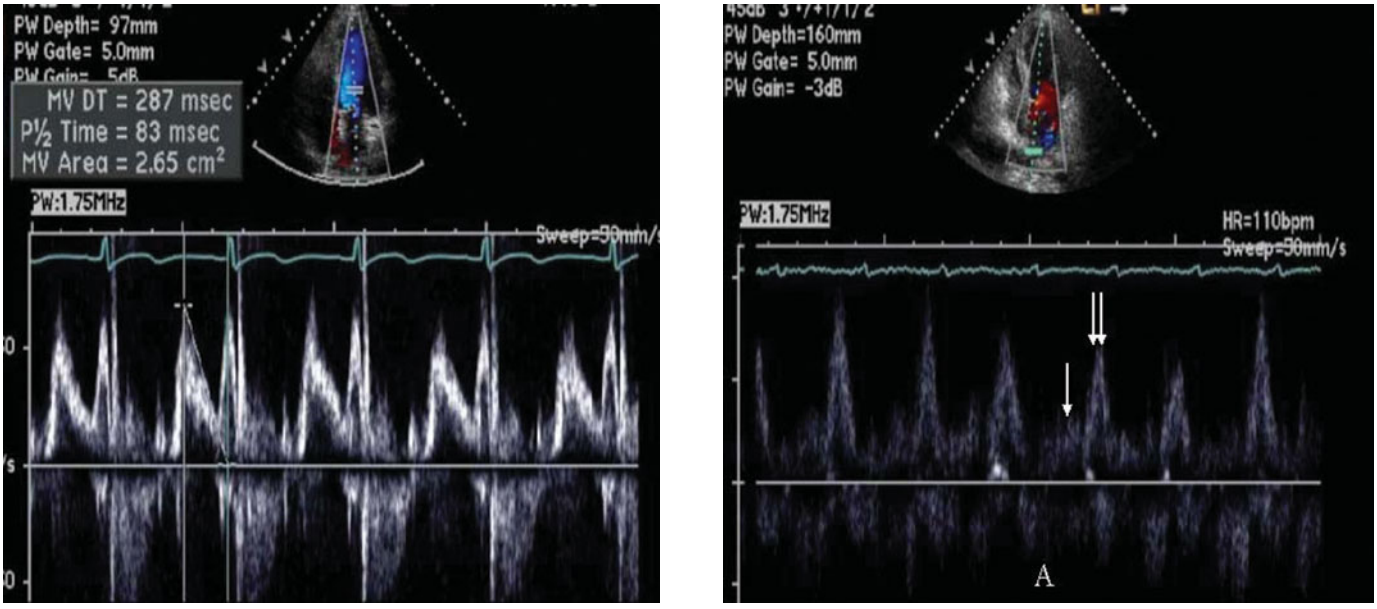
B



CR

Figure 14.6. (A) The normal pulsed Doppler pattern seen with mitral valve inflow. Note the E/A >1 (E wave: single arrowhead; A wave: double arrowheads). The best flow patterns are recorded with the sample volume placed at the mitral valve tips in the apical four-chamber or apical two-chamber views. (B) Mitral valve inflow recording with pulsed-wave Doppler showing evidence of mild diastolic relaxation abnormality, with E/A <1 (E wave: single arrowhead; A wave: double arrowheads). (C) (Left): A normal tissue Doppler pattern recorded at the mitral valve leaflet. The Ea/Aa >1. Note the placement of the sample volume at the lateral aspect of the mitral leaflet (Ea: single arrowhead; Aa: double arrowheads). (Right): Tissue Doppler recording of the mitral valve in a patient demonstrating a diastolic relaxation abnormality. Note the reversal of the Ea/Aa to <1. Because the annular velocity is not volume dependent, obtaining a tissue Doppler avoids the

(*continued*)



E F

Figure 14.6. (*Continued*) issue with pseudonormalization seen with mitral valve inflow velocity pulsed-wave Doppler measurements in patients with mild diastolic dysfunction (Ea: single arrowhead; Aa: double arrowheads). (D) Normal isovolumic relaxation time (IVRT) is demonstrated here. The IVRT decreases with progressive diastolic dysfuction. (E) This demonstrates the normal deceleration time (DT) on pulsed-wave Doppler for mitral valve inflow (287 m/sec). The deceleration time decreases with progressive diastolic dysfunction, approaching values <140 m/sec. (F) Pulsed-wave Doppler recording on a transthoracic echocardiogram demonstrating forward flow in the pulmonary veins. Note the predominance of diastolic flow (double arrowheads) over systolic flow (single arrowhead). This demonstrates a significant amount of diastolic dysfunction. Also note the wide pulmonary "a" wave (labeled A).

wave (Figure 14.6A). The rapid early filling phase correlates with the E wave and the latter phase, prompted by the atrial contraction, yields the A wave. In a normal circumstance, the E wave amplitude and velocity and duration are greater than the A wave. Because the E wave amplitude is greater, in normal individuals the E/A ratio is >1. However, with progressive worsening of diastolic function, there is a change in both the volume and velocity of the E and A waves. The spectrum extends from a mild form (Grade 1), where there is abnormal relaxation, to a severe form (Grade 4), where the compliance is compromised irreversibly.

Grade 1: Abnormal relaxation, with reversal of the E/A ratio and attenuated deceleration of the E wave (Figure 14.6B).

Grade 2: A pseudonormalization pattern, as the ventricle becomes more noncompliant. Use of the Valsalva maneuver (which decreases preload by diminishing venous return) may help unmask the pathology here by showing a reversed E/A ratio (reminiscent of the pattern in Grade 1).

Grade 3: A markedly increased E/A ratio, with a diminished A wave and an E velocity that almost touches the baseline prior to the A wave.

Grade 4: An irreversible phase, where the E/A ratio is markedly increased and the A wave is diminutive. Furthermore, there is a separation of the E and A waves, with the A wave velocity not always returning to baseline.

Tissue Doppler Interrogation (TDI). Tissue Doppler interrogation has the advantage of not being influenced by atrial rhythm disturbances or by increased heart rates. The recommended way is to sample the lateral mitral annulus in the apical view. The velocity of the mitral annulus in diastole correlates with systolic and diastolic function. During diastole, the mitral annulus has two phases: E_a and A_a. These phases are similar to those discussed above in that E_a occurs in early diastole and A_a occurs in late diastole and correlates with the atrial contraction. Similar to the mitral inflow pattern, the E_a is greater than the A_a under normal circumstances. As diastolic abnormalities progress (toward an abnormal relaxation pattern), the early mitral annular velocity decreases, making the E_a/A_a <1. Because the annular velocity is not volume dependent, the potential issue with pseudonormalization (seen with mitral inflow) is avoided, and the ratio remains

reversed. As diastolic dysfunction progresses to an irreversible stage, the E_a and A_a waves progressively become lower in amplitude, but the ratio reversal is maintained (Figure 14.6C). A predictor of increasing pulmonary capillary filling pressures is the ratio between the mitral inflow E wave and the Ea wave. An increasing ratio (particularly greater than 15) correlates well with adverse outcomes in both ischemic and nonischemic CMP.

Isovolumic Relaxation Time and Deceleration Time (IVRT and DT). Isovolumic relaxation time occurs when systolic flow across the aortic valve ceases (marking the end of systole), but before the mitral valve opens to allow ventricular filling (marking the beginning of diastole). Normal values in adults for IVRT range between 65 m/sec and 90 m/sec. Isovolumic relaxation time will decrease progressively with worsening diastolic function, as the rising left atrial pressure results in earlier opening of the mitral valve. Furthermore, as the left ventricle becomes more noncompliant, it takes on a restrictive filling pattern with a decrease in the DT (<140 m/sec), as most of the LV filling occurs in early diastole (Figure 14.6D,E). This gives rise to the S3 gallop in dilated CMP, and predicts a higher mortality in patients with symptomatic heart failure. Similar adverse prognosis is seen in patients who have this filling pattern postmyocardial infarction.

Pulmonary Venous Flow Patterns. The left upper pulmonary vein (posteriorly) can be well interrogated by transthoracic echocardiography. This is accomplished in the apical view, and contrast enhancement can be used, if necessary. Under normal circumstances, pulmonary venous flow is triphasic. During systole, there is forward flow in the pulmonary veins due to atrial relaxation (the "x" descent) and movement of the base of the heart during the contraction phase. During early diastole, there is a second phase of forward flow through the pulmonary veins due to the open mitral valve (the "y" descent). During late diastole, in sinus rhythm, there is a brief period of reversal of flow in the pulmonary veins because of increasing pressure in the left atrium during atrial systole. When left ventricular dynamics are normal, the amplitude of the systolic phase of pulmonary flow is greater than the amplitude during the diastolic phase. With increasing left ventricular noncompliance, there is incomplete emptying of the left atrium during diastole, raising left atrial pressures. This results in attenuation of pulmonary vein flow during the systolic phase. Conversely, there is an increase in the pulmonary vein flow during diastole, as most of the filling occurs when the mitral valve is open and there is direct emptying into the left ventricle. This increases the amplitude of the diastolic phase of the pulmonary vein flow. In essence, there is a reversal of the systolic and diastolic ratio (Figure 14.6F). Because the left atrial pressure is increased, the flow reversal seen during atrial contraction is more pronounced and lasts longer. There has been some suggestion that if this flow reversal period exceeds the A wave period of the mitral inflow, then the left ventricular end-diastolic pressure exceeds 15 mm Hg in the majority of cases.

Myocardial Performance Index

Myocardial performance index (MPI) is a measurement that is obtained in the apical five-chamber view, as mitral flow and left ventricular outflow tract flow need to be obtained simultaneously (similar to obtaining IVRT). The measurement allows assessment of both systolic and diastolic performance using a single Doppler approach. The concept of MPI relies on interrogating the entire systolic period, which occurs from the closure of the mitral valve of one beat to the opening of the mitral valve of the next beat. This complete systolic period includes in it the isovolumic contraction time (the period between the closure of the mitral valve and the opening of the aortic valve), the ejection period (during which the aortic valve is open and blood is ejected into the aorta), and the isovolumic relaxation time, which has been described earlier. The measurement is given by (IVRT + IVCT)/ET. A normal MPI is 0.40, and increasing values suggest worsening left ventricular function.

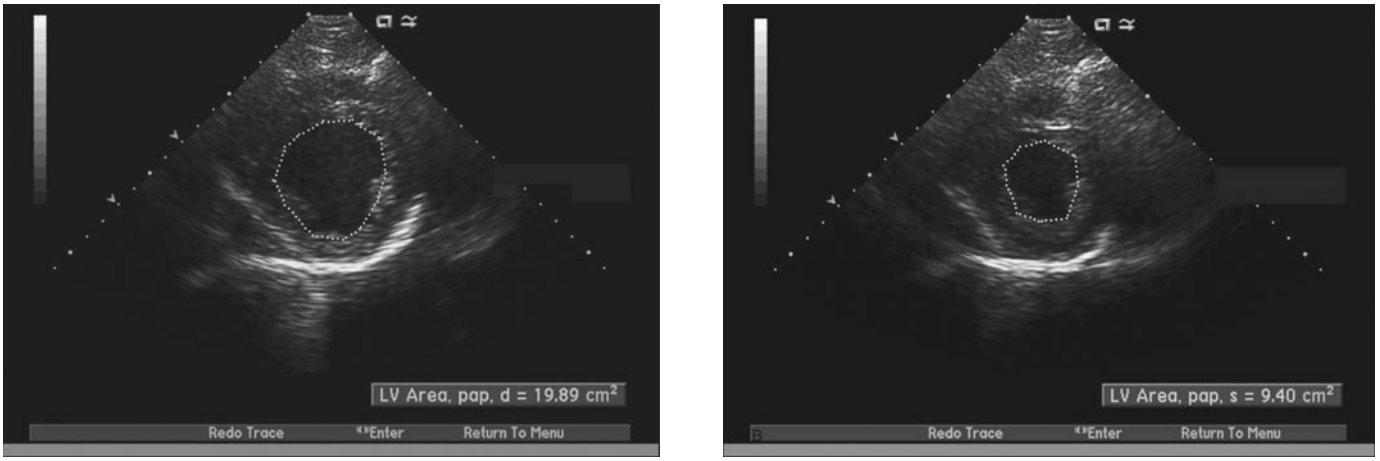
Two-Dimensional and M-Mode Assessment

Two-dimensional imaging can also be used to assess systolic and diastolic dysfunction.

Assessment of Systolic Function (Figures 14.7)

Evaluation of systolic function with two-dimensional and M-mode echocardiography uses some of the methods already described earlier, such as wall motion, left ventricular dilatation on linear measurements, and wall thickening. However, two other features for systolic function assessment that use two-dimensional echocardiography, are fractional area change (sometimes referred to as fractional shortening) and volume measurements.

Fractional Area Change. Fractional area change looks at the relative change in area, in the short-axis view, between diastole and systole. As mentioned earlier, the



A B

Figure 14.7. Short axis view demonstrating relative change in ventricular area between systole and diastole.

decrease in area during systole is due to the inward motion and thickening of the ventricular wall. The formula is given by (area in diastole – area in systole)/area in diastole. The normal values range from 0.35 to 0.60. With significant dilatation and dysfunction of the left ventricle in dilated CMP, values in the 0.15 range can be seen. The limitation with this measurement has to do with regional wall motion abnormalities. For example, in a patient with a left ventricular apical infarction, fractional area change in the midcavity region may well be within the normal range, and will not account for the decrease in ejection fraction from a large akinetic apex. Hence, much like any measurement in echocardiography, the interpreter has to make a conclusion based on multiple measurements and parameters.

Volume Measurements. Volume measurements are usually made using various area and length measurements. Many assumptions go into such measurements regarding the shape of the ventricle, assumed either to be conical or spherical, depending on where the measurement is made and the view used to make the measurement. The most commonly used method for determination of left ventricular volumes is Simpson's rule. In simple terms, this method involves stacking disks with a fixed height along the longitudinal axis of the left ventricle in the apical four-chamber or apical two-chamber view, in end-systole and end-diastole. The volume of each disk is calculated and all the volumes are added together to yield the total volume. Care needs to be taken to make sure that the entire ventricular cavity is visualized along its endocardial surface. If there are regional wall motion abnormalities, evaluating volumes in two planes is recommended. Once the end-diastolic and end-systolic volumes have been gained, the difference between the two is the stroke volume. Stroke volume multiplied by the heart rate yields the cardiac output. Recognize that the cardiac output represents the entire flow from the left ventricle when calculated with this technique, hence it will include the regurgitant volume if mitral or aortic regurgitation is present, and may not necessarily represent the true output into the aorta. Ejection fraction can also be calculated using Simpson's rule as (end-diastolic volume – end-systolic volume)/end-diastolic volume.

HYPERTROPHIC CMP

Though the disease pattern of hypertrophic CMP was described over a century ago, the true characterization of the disease was not realized until the 1950s. Various features have been described in association with the disease, but the only consistent finding is that of inappropriate left ventricular hypertrophy with relation to the hemodynamic load. Hypertrophic CMP has a familial predilection, and various genetic mutations have been associated with this autosomal dominant pattern of inheritance. There is, however, varied penetrance, leading to different types of phenotypic expression of the disease. The various forms of primary hypertrophic CMP include, hypertrophic CMP (with or without obstruction), apical hypertrophic CMP, and hypertrophic CMP of the elderly. Secondary hypertrophic CMP, the hallmark of which is a pattern of concentric hypertrophy, is usually a late result of hypertension and characterizes hypertensive heart disease. Echocardiography

is extremely important in characterizing the various forms of hypertrophic cardiomyopathies, and this is discussed below with reference to each of the above entities. The most common imaging views that are used for discerning the type of hypertrophic CMP include parasternal long-axis, short-axis, and apical four-chamber. All interrogation modalities are important, though M-mode is probably the least utilized.

Hypertrophic CMP (with or without Obstruction)

This particular entity has had various names associated with it, including idiopathic hypertrophic subaortic stenosis (IHSS) and muscular subaortic stenosis. Because obstruction may not always be present, hypertrophic CMP is a more appropriate term. Clinically speaking, timely diagnosis of this disorder in the critical care setting can be potentially lifesaving by avoiding critical treatment errors. An example is a 44-year-old male patient who presents with precordial discomfort and dyspnea. The chest discomfort description has some typical anginal features, and the patient has dyslipidemia and a family history of sudden death and premature cardiovascular disease. The discomfort is ongoing and on exam a systolic murmur is identified at the base of the heart. The remainder of the exam is relatively unremarkable, and the echocardiogram (ECG) shows T wave inversions in the precordial leads. A diagnosis of unstable angina or acute coronary syndrome and its treatment could be potentially problematic in such a patient. This patient may well have hypertrophic CMP with an obstructive gradient. Administering nitroglycerine to control the chest discomfort may have the paradoxical effect of worsening the discomfort and precipitating hypotension and heart failure by worsening the obstructive gradient and MR. Hence a good history and exam along with echocardiography may well avoid such pitfalls in management.

M-Mode and Two-Dimensional Assessment (Figure 14.8)

M-mode findings were the original way of diagnosing hypertrophic CMP, and used a septal wall–to–posterior wall thickness ratio at the end of diastole of greater than 1.3. This finding can be seen in other scenarios as well, such as right ventricular hypertrophy with pulmonary hypertension. Two-dimensional echocardiography has aided in solidifying the diagnosis by eliminating confusion, and the septal thickness is usually at least 15 millimeters (mm). The asymmetrical septal hypertrophy is most prominent in the mid portion between

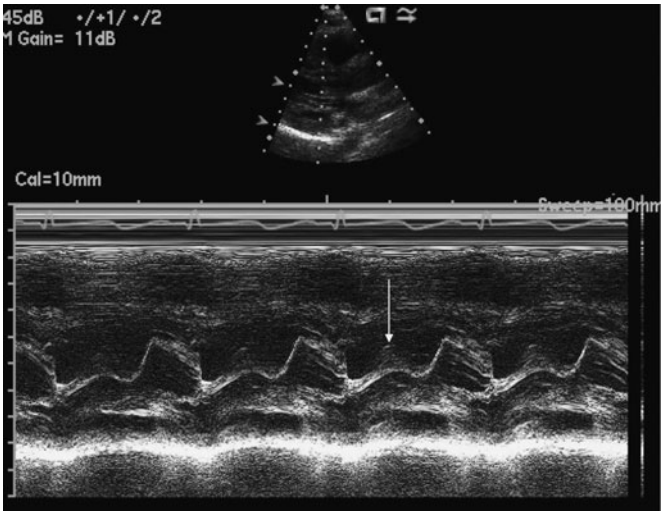
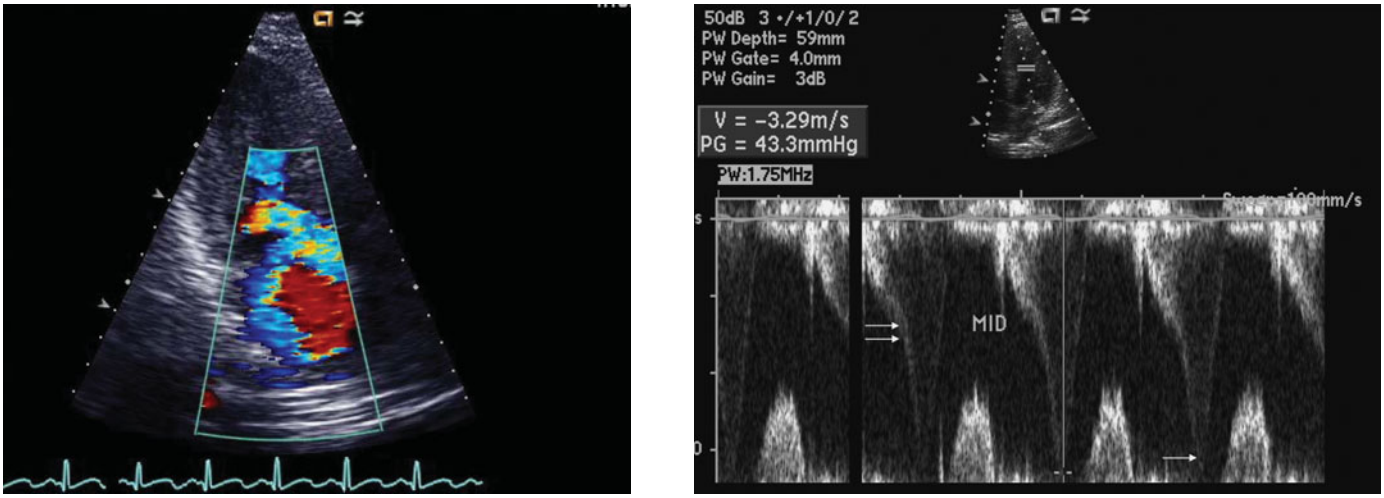


Figure 14.8. M-mode recording in a patient with hypertrophic cardiomyopathy. Note the systolic anterior motion of the mitral valve (arrowhead).

the base and the apex in the parasternal long-axis view, and a variety of thicknesses can be seen from mild hypertrophy to massive hypertrophy (septal thickness of 60 mm). In a recent study, the septal hypertrophy pattern on echocardiography was used to predict the potential for myofilament mutations. The mid septal thickness pattern (also termed the reverse septal pattern) was significantly associated with an abnormal genetic pattern in 79% of those who had this septal morphology. Left ventricular outflow tract obstruction is another important finding in this type of hypertrophic CMP (though it does not have to be present to make the diagnosis). The thickened septum and an elongated anterior mitral leaflet create the outflow tract obstruction. This abnormal geometry also leads to mitral valve regurgitation and systolic anterior motion of the mitral valve. The degree of MR is directly related to the degree of the outflow tract obstruction and the anterior displacement of the mitral valve. Other two-dimensional features include a small left ventricular cavity, normal or increased motion of the posterior wall, with abnormal motion of the septum, and fluttering of the aortic valve due to turbulent flow in the outflow tract in systole. All of these features can occur to varying degrees, and may be made more apparent by various provocative maneuvers (see Doppler section below).

Doppler Assessment (Figure 14.9)

Doppler assessment, both with color and spectral analysis, is critical to the diagnosis of hypertrophic CMP. Colored Doppler assessment is important for characterizing the degree of mitral valve regurgitation. The



A B

Figure 14.9. (A) Color-flow Doppler recording in a patient with hypertrophic cardiomyopathy demonstrating severe mitral regurgitation. The regurgitant jet is usually central and peaks later in systole. (B) Pulsed-wave Doppler recording during systole in the left ventricular outflow tract in a patient with hypertrophic cardiomyopathy. Note the late peaking pattern (single arrowhead) and the jagged knifelike pattern (double arrowheads).

MR jet is usually central and late peaking, coinciding with the greatest anterior motion during systole. The later peak of the mitral valve regurgitation in patients with outflow tract gradients distinguishes it from cases of structural mitral valve regurgitation. Hence the MR is not holosystolic in patients with dynamic outflow tract gradients. Furthermore, there can often be confusion when interrogating the MR and the outflow tract velocity on continuous-wave Doppler. The onset of the MR signal is usually later than that of the outflow tract signal. The shape of the outflow tract signal is also different, usually in the shape of a dagger, because of the late-peaking velocity. Spectral analysis during systole and diastole provides information on the severity of the outflow tract obstruction and the degree of diastolic dysfunction. Provocative maneuvers include any physiologic (such as a Valsalva maneuver) or pharmacologic manipulation (such as amyl nitrate) that results in decreased left ventricular volume, or increased contractility. In the critical care setting, use of inotropes, volume depletion, tachycardia, and use of nitrates can all serve as provocative maneuvers, and can worsen the outflow tract gradient. Certain inhalation anesthetics and regional anesthetic agents can also worsen the outflow gradient, and should be avoided in patients with hypertrophic CMP. Doppler interrogation of the outflow tract velocity can be used to measure the therapeutic effect of an intervention, whether pharmacologic (use of negatively chronotropic calcium channel blockers or beta blockers) or invasive (ablative therapy of the first septal perforator). A change in the degree of mitral valve regurgitation on color Doppler can also be used to assess response to therapy. Diastolic dysfunction is present in the majority of the patients with hypertrophic CMP, and it does not have to be associated with an outflow tract obstruction. Furthermore, the degree of diastolic dysfunction does not always correlate with the degree of hypertrophy.

Apical Hypertrophic CMP

This disorder is seen mostly in the Japanese population, and accounts for up to 25% of Japanese patients with hypertrophic CMP. It is distinctly uncommon in the Western world. Angiography usually defines the typical features of the disease with a spade-shaped ventricle on left ventricular injection. The ECG reveals deeply inverted precordial T waves. The usual course is benign in such patients, and they tend to be minimally symptomatic. On echocardiography, there is no outflow tract pressure gradient. On two-dimensional imaging, the base of the heart has normal thickness, and there is an increase in wall thickness toward the apex. This essentially reduces the size of the apical cavity. Contrast enhancement during the echocardiographic study can help define the endocardial border at the apex. Care should be taken to visualize the apex in its full longitudinal view, as foreshortening can mimic the hypertrophy. Occasional diastolic dysfunction can be seen in these patients, particularly with changes in

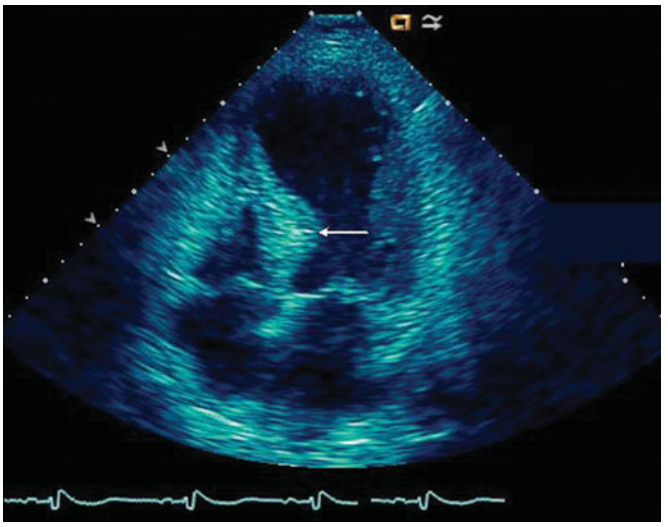


Figure 14.10. Two-dimensional image of the apical four-chamber view in an elderly patient with eccentric hypertrophic cardiomyopathy. Note the hypertrophied septum represented as a septal "knuckle" (arrowhead).

volume status. Atrial fibrillation is the most common clinical complication in such patients.

Hypertrophic CMP of the Elderly (Figure 14.10)

This is usually seen in patients who have been hypertensive; however, the hypertrophy tends to be eccentric rather than concentric. The septum tends to hypertrophy to a greater degree, and this, coupled with the change in angulation of the septum accompanied with aging, results in an outflow tract gradient. Systolic anterior motion of the mitral valve is also seen and can result in mitral valve regurgitation. Genetic abnormalities distinct from those seen in patients with the early onset familial form of hypertrophic CMP (described above) have been defined in such patients. The echocardiographic features are similar to those described above in the hypertrophic CMP section, and outflow tract obstructive gradients can achieve a severity seen in the familial forms.

Secondary Hypertrophic CMP (Table 14.4)

The most common etiologies include hypertension and aortic stenosis. There is usually concentric hypertrophy of the left ventricle. Systolic function tends to be normal, but if these disorders progress untreated, the final result is a dilated CMP with all its attendant features and clinical prognosis. Diastolic dysfunction is common in these patients and a depleted volume status can potentially provoke midcavitary obstruction of the left ventricle with a gradient. Echocardiography can be used to measure and index the left ventricular mass in such patients, and there are defined criteria for the same (Table 14.4). The emphasis in treating these patients should be on the underlying disease state and keeping them euvolemic. Hemodynamics of hypertrophic CMP can also be seen in patients who have a history of hypertension, and have hyperdynamic left ventricular wall motion because of volume depletion. This situation is further augmented in the critical care setting when such a patient is hypotensive and is put on inotropic support. A dynamic outflow tract obstruction is created, and MR can occur due to the systolic anterior motion of the mitral valve. Repletion of volume and weaning of the inotropic support will usually return the hemodynamics toward normal.

TABLE 14.4. American Society of Echocardiography criteria for grading left ventricular hypertrophy (LVH)

	Mild LVH	*Moderate LVH*	*Severe LVH*
Male	103–116 g/m^2	117–130 g/m^2	>130 g/m^2
Female	89–100 g/m^2	101–112 g/m^2	>112 g/m^2

Modified from: Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr. 2005;18:1440–1463.

Athlete's Heart

Steady endurance and strength training results in physiologic and electrical changes in the cardiovascular system. These changes are also accompanied by changes in left ventricular volume, wall thickness, and mass. Such changes need to be distinguished from pathological changes, as they are reversible on cessation of the training, and usually do not alter prognosis (unless accompanied by other cardiac pathology or with use of pharmacologic agents, such as anabolic steroids). Echocardiography can help in distinguishing the two subsets. The left ventricular hypertrophy is usually symmetric in athletes, and wall thickness is usually $< / =$ 12 mm. Though series looking at trained athletes have described wall thicknesses of up to 16 mm,

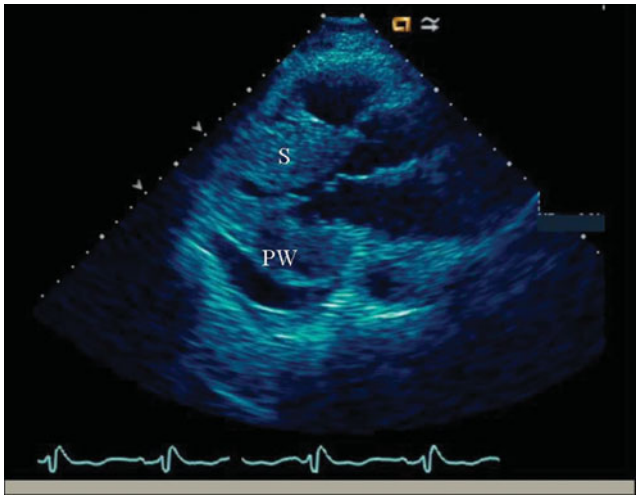
they are typically less than 16 mm. A wall thickness of greater than 16 mm should prompt an investigation to rule out a primary hypertrophic CMP. The left ventricular diastolic dimension is typically greater than 50 mm in trained athletes, while it is less than that in hypertrophic CMP (usually less than 45 mm). On Doppler evaluation of diastolic function, the patterns are normal in the athletic heart, as is the left atrial dimension on two-dimensional imaging.

RESTRICTIVE CMP

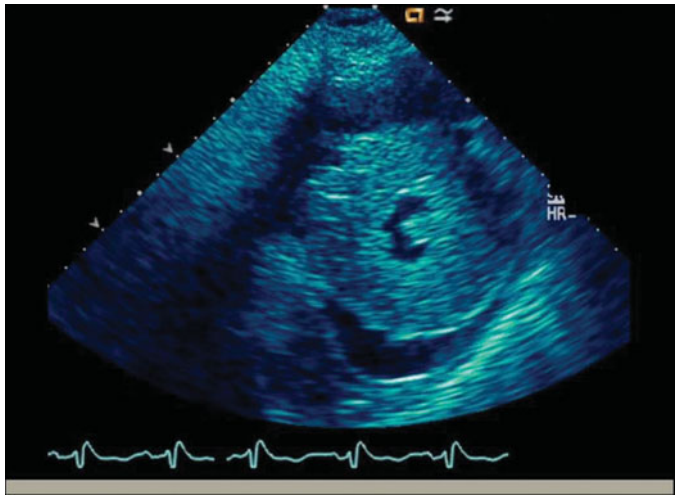
Restrictive CMP is characterized by normal ventricular chamber sizes with impaired filling during diastole. Though hypertrophy of the ventricles is typically absent, certain acquired forms, such as amyloidosis, can cause left ventricular wall thickening. Amyloidosis remains the classic association with infiltrative restrictive CMP, though it is uncommon as a disease entity. Because the cause of heart failure in restrictive CMP is impaired diastolic filling and progressive diastolic dysfunction, various other forms of heart disease can result in a "restrictive physiology." These include hypertrophic CMP (discussed earlier), concentric hypertrophy from long-standing hypertension, hypertrophic heart disease of the elderly, and idiopathic restrictive CMP. Glycogen storage diseases and hemochromatosis are examples of storage diseases that lead to a restrictive CMP. Various other important causes include radiation therapy, endomyocardial fibrosis (particularly in tropical regions), anthracycline toxicity, and metastatic cancers. Restrictive physiology can be confused at times with constrictive physiology. This issue can usually be resolved with careful history taking, a good echocardiographic study, and evaluation of the pericardium. On some occasions, an endomyocardial biopsy may be necessary to sort the issue. Because the main feature of restrictive CMP is diastolic dysfunction, a careful Doppler analysis of diastolic function is important in these patients. The Doppler principles of diastolic function assessment have been discussed earlier in this chapter, and aspects will be highlighted below. Increased left ventricular noncompliance results in substantially elevated left atrial pressures, and significant atrial dilatation is the rule in patients with restrictive CMP. Needless to say, atrial arrhythmias can be quite common in these patients, particularly atrial fibrillation. Pulmonary venous pressures are also increased, giving rise to the symptoms of heart failure. Secondary pulmonary hypertension is commonly seen in these patients.

Two-Dimensional and M-Mode Assessment (Figure 14.11)

The major findings on two-dimensional imaging and M-mode imaging include normal ventricular chamber size and preserved systolic function. As the disease progresses, the left ventricle may develop some systolic dysfunction, although initially, this is without dilation.



A



B

Figure 14.11. (A) Two-dimensional imaging in parasternal long axis of a patient with amyloidosis. Note the thickened septum (S) and posterior wall (PW). Although this is a systolic frame, there is severe hypertrophy present. (B) Two-dimensional imaging in the short-axis view of the same patient represented in (A). Note the severe concentric hypertrophy with a somewhat refractile myocardium, typical of amyloidosis.

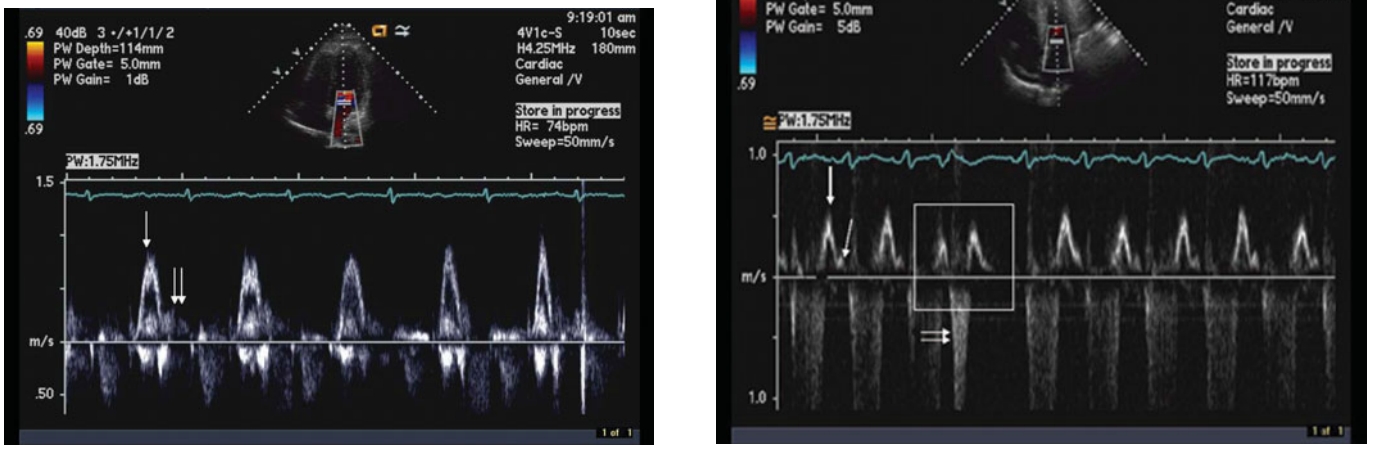


Figure 14.12. (A) Mitral valve inflow Doppler pattern showing a restrictive filling pattern. Notice the predominant E wave (single arrowhead) and the lack of any significant A wave (double arrowheads). (B) Mitral valve inflow Doppler pattern showing a severely restrictive filling pattern. The diastolic filling occurs almost entirely during the E wave (thick arrowhead). The A wave is quite diminutive (thin arrowhead). There is an incidental finding of a ventricular premature beat during the recording (rectangle). Note the interruption of the diastolic filling by the premature beat and the occurrence of "diastolic mitral regurgitation" seen below the baseline (double arrowheads).

Though left ventricular hypertrophy is absent in the idiopathic form, it is commonly seen in infiltrative diseases such as amyloidosis and in chronic hypertension. Biatrial enlargement is also seen.

Doppler Assessment (Figure 14.12)

Doppler assessment remains the hallmark in diagnosing the diastolic dysfunction accompanying restrictive CMP. Mitral valve inflow velocities, mitral annular tissue Doppler interrogation, and assessment of the pulmonary venous flow patterns should be performed routinely in such patients. Since restrictive CMP is a global process, there is simultaneous involvement of the right ventricle. This can be used to an advantage from the standpoint of echocardiographic diagnosis. Similar patterns, as seen on the left side, are seen on the right side with tricuspid valve inflow velocities and hepatic vein flows.

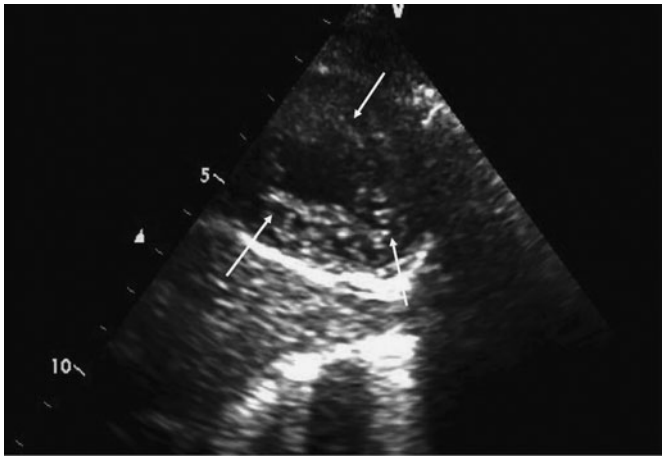
As mentioned earlier in this section, it may become necessary to distinguish restrictive physiology from constrictive physiology in some patients. Echocardiographic features can be used to distinguish between the two scenarios. The left atrial size is normal in constriction, but is enlarged as a rule in restriction. The septal motion is abnormal in constriction and normal in restriction. The septum becomes a physiologic part of the right ventricle in constriction (particularly in constrictive effusive physiology). Hence the septal position varies with respiration in constriction, with increased right-sided inflow during inspiration pushing the septum into the left ventricle and compromising diastolic filling of the left ventricle. Evaluation of transmitral inflow reveals a significant respiratory variation in the E wave velocity (usually $\geq$25%). The E wave velocity of the transmitral flow is impaired during inspiration and increases greatly during expiration. This is contrasted by a lack of such variation in restriction. Though the E/A ratio is increased in both constriction and restriction (usually $\geq$2), the A wave is relatively well preserved in constrictive effusive physiology. Much of the diastolic filling in constrictive effusive states occurs during the atrial contraction. Hence a patient who is hemodynamically compromised from such physiology may experience complete cardiovascular collapse if the rhythm changes to atrial fibrillation, particularly with a rapid ventricular response. Another Doppler feature distinguishing constriction from restriction is the respiratory variation in isovolumic relaxation time that occurs in constriction and is unchanged (albeit decreased) in restriction. On tissue Doppler investigation of the lateral mitral annulus, the E wave velocity is normal in constriction and diminished in restriction.

ARRHYTHMOGENIC RIGHT VENTRICULAR DYSPLASIA (ARVD)

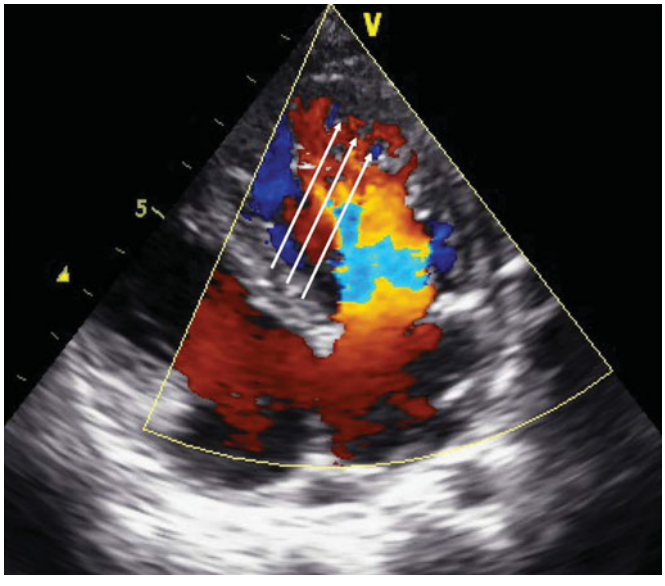
This is a somewhat rare entity, and is characterized by abnormal morphology of the right ventricle. Though echocardiography has been used extensively for the diagnosis of this disorder, cardiac magnetic resonance imaging is also becoming an important tool. The myocardium of the right ventricular free wall is replaced with fibrous and/or fatty tissue. Malignant ventricular arrhythmias and sudden death are the major concerns in patients with this disorder. The echocardiographic diagnosis is based primarily on two-dimensional and M-mode imaging. Echocardiographic features include hypokinesis of the right ventricular (RV) free wall, occasional aneurysmal dilatation of the RV free wall, and increased echogenicity of the RV free wall from the deposition of collagen and/or adipose tissue. Though the disease has been linked with right ventricular issues, progressive LV dysfunction has been described. In one pathologic study, 76% of the hearts with ARVD had left ventricular involvement. The involvement in this study was more pronounced, with a longer history of the disease, and was associated with more significant cardiomegaly, inflammatory infiltrates, and heart failure. The left ventricular abnormalities were also linked to clinical arrhythmias.

UNCLASSIFIED CARDIOMYOPATHIES

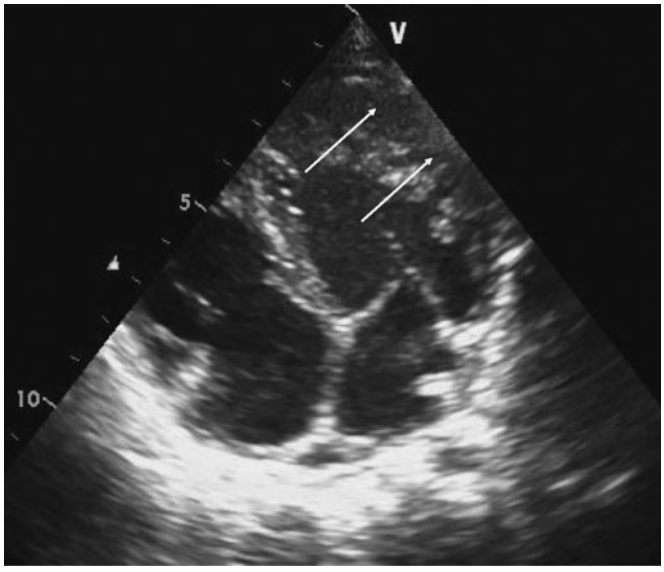
Three major syndromes will be discussed in this section. These include, endocardial fibroelastosis, left



A



C



B

Figure 14.13. (A) A cross-sectional view of the left ventricle in a patient with LV noncompaction. Notice the "sinusoids" (arrowheads) that make up the noncompacted layer. (B) An apical four-chamber view demonstrating LV noncompaction. The arrowheads point to the noncompacted layer. (C) An apical four-chamber view of a noncompacted left ventricle with color-flow into the noncompacted areas (arrowheads). The color-flow signifies blood flow into the noncompacted "sinusoids." LV indicates left ventricular.

ventricular noncompaction, and the transient left ventricular apical ballooning syndrome (also called broken heart syndrome, stress-induced CMP, or Takotsubo CMP in Japan). Though these syndromes have features of dilated CMP or restrictive CMP, they do not neatly fall into the respective categories.

Endocardial Fibroelastosis

This disease is seen primarily in infants and fetuses. It is characterized by deposition of collagen and elastin in the ventricular walls. It is associated with hypertrophy and endocardial thickening. The disease has an association with viral infections, autoimmune diseases, and congenital left-sided obstructive lesions. In late stages, it presents as a dilated CMP with restrictive physiology. Treatment is usually unsuccessful in infants with the primary form of the disease, and transplantation offers the only recourse.

Left Ventricular Noncompaction (LVNC) (Figure 14.13)

This is a rare disorder associated with heart failure, thromboembolism, and ventricular arrhythmias. It is caused by a developmental arrest of compaction of the left ventricular myocardial trabecular network. This results in sinusoidal recesses that communicate with the endocardium and are filled with blood. There is, however, no communication to the epicardium. Echocardiographic diagnosis is based primarily on two-dimensional and colored Doppler imaging. On two-dimensional imaging, particularly in the apical views, there is a distinction between the compacted and the noncompacted portion of the myocardium, appearing as two layers. A ratio of noncompacted to compacted layer of $\geq$2:1 in end-systole is considered diagnostic of noncompaction. The myocardium in most of the noncompacted segments is hypokinetic, and is seen in the apical, midinferior and midlateral regions. On color-flow Doppler, blood flow can be seen into the sinusoidal recesses. Other associated echocardiographic features include left ventricular dilatation, Doppler evidence of diastolic dysfunction, left ventricular thrombus, and abnormal papillary muscle structure.

Transient Left Ventricular Apical Ballooning Syndrome (Figure 14.14)

This syndrome was first described in Japan (called Takotsubo CMP there), but it has since been seen in other populations, including the United States. It presents with signs and symptoms of a myocardial infarction, and is associated with ST-segment elevation and apical wall motion abnormalities but without evidence of obstructive coronary artery disease on coronary angiography. The onset is triggered by either significant emotional or physical stress (sudden loss of a family member, sudden life-threatening accident, medical illness, physical abuse). A recent study looked prospectively at patients admitted to the intensive care unit (ICU) with serial echocardiography to see how many developed the apical ballooning. Of 92 patients, 26 had the pathology on echocardiography with a mean ejection fraction of 33 $\pm$ 8%. Compared with the patients without the pathology, those with apical ballooning more frequently had sepsis as the admission diagnosis for the ICU (62% vs. 14%), a higher incidence of developing hypotension on admission and use of inotropes, and higher frequency of cardiomegaly and pulmonary congestion. Sepsis was the only associated feature with the cardiac pathology. The findings resolved in 20 of the 26 patients (77%) within two to 25 days (mean 7.4 $\pm$ 5.6 days). The two-month survival was lower in the group with apical ballooning compared with those without the pathology (52% vs. 71%, respectively). The echocardiographic features include apical akinesis or dyskinesis on two-dimensional imaging; presence of left ventricular thrombus has been seen resulting in a cerebrovascular accident; the

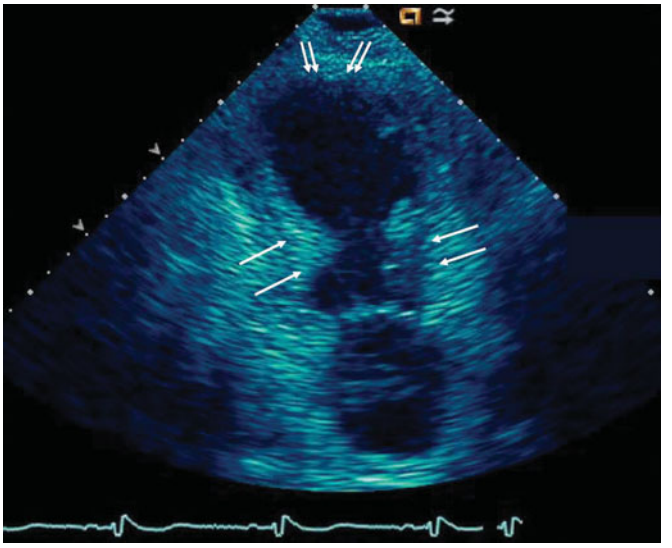


Figure 14.14. An apical two-chamber view of the left ventricle from a patient with transient apical ballooning syndrome. This systolic frame demonstrates normal inward motion and thickening of the inferior base and the anterior base (single arrowheads) and the lack of motion and ballooning of the apex (double arrowheads).

basal wall may exhibit hyperkinesis and generate a left ventricular outflow tract gradient similar to that seen in hypertrophic CMP, with resultant MR. The ejection fraction is reduced, though not always severely (mostly because of hyperkinesis of other ventricular segments).

Suggested Reading

Abergel E, Chatellier G, Hagege AA, et al. Serial left ventricular adaptations in world-class professional cyclists: implications for disease screening and follow-up. *J Am Coll Cardiol*. 2004;44:144–149.

Aurigemma GP, Gottdeiner JS. Predictive value of systolic and diastolic function forincident congestive heart failure in the elderly: the Cardiovascular Health Study. *J Am Coll Cardiol*. 2001;37:1042–1048.

Binder J, Ommen SR, Gersh BJ, et al. Echocardiography-guided genetic testing in hypertrophic CMP: septal morphological features predict the presence of myofilament mutations. *Mayo Clin Proc*. 2006;81:459–467.

Braunwald E, Seidman CE, Sigwart U. Contemporary evaluation and management of hypertrophic CMP. *Circulation.* 2002;106:1312–1316.

Chen C, Rodriguez L, Lethor JP, et al. Continuous wave Doppler echocardiography for noninvasive assessment of left ventricular dP/dt and relaxation time constant from mitral regurgitant spectra in patients. *J Am Coll Cardiol*. 1994;23:970–976.

Corrado D, Fontaine G, Marcus FI, et al. Arrhythmogenic right ventricular dysplasia/CMP: need for an international registry. *Circulation*. 2000;101:e101–e106.

Dec, GW. Recognition of the apical ballooning syndrome in the United States.*Circulation.* 2005;111:388–390.

Devereux RB, Roman MJ, Paranicas M, et al. A population-based assessment of left ventricular systolic dysfunction in middle-aged and older adults: the Strong Heart Study. *Am Heart J*. 2001;141:439–446.

Dujardin KS, Tei C, Yeo TC, et al. Prognostic value of Doppler index combining systolic and diastolic performance in idiopathic dilated CMP. *Am J of Cardiol*. 1998;82:1071–1076.

Fans R, Coats AJ, Henein MY. Echocardiography derived variables predict outcome in patients with non-ischemic dilated CMP with or without a restrictive filling pattern. *Am Heart J.* 2002;144:343–350.

Feigenbaum H, Armstrong WF, Ryan T. *Feigenbaum's Echocardiography*. 6th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2004.

Felker GM, Thompson RE, Hare JM, et al. Underlying causes and long-term survival in patients with initially unexplained CMP. *N Engl J Med*. 2000;342:1077–1084.

Frischknecht BS, Jost CH, Oechslin EN, et al. Validation of noncompaction criteria in dilated CMP, and valvular and hypertensive heart disease. *J Am Soc Echocardiogr*. 2005;18:865–872.

Garcia MJ, Thomas JD, Klein AL. New Doppler echocardiographic applications for the study of diastolic function. *J Am Coll Cardiol*. 1998;32:865–875.

Gemayel C, Pelliccia A, Thompson PD. Arrhythmogenic right ventricular dysplasia. *J Am Coll Cardiol.* 2001;38:1773–1781.

Groote P, Millaire A, Foucher-Hossein C, et al. Right ventricular ejection fraction is an independent predictor of survival in patients with moderate heart failure. *J Am Coll Cardiol*. 1998;32:948–954.

Kjaergaard J, Hastrup Svendsen J, Sogaard P, et al. Advanced quantitative echocardiography in arrhythmogenic right ventricular CMP. *J Am Soc Echocardiogr*. 2007;20:27–35.

Koelling TM, Aaronson KD, Cody RJ, et al. Prognostic significance of MR and tricuspid regurgitation in patients with left ventricular systolic dysfunction. *Am Heart J*. 2002;144:524–529.

Kolias TJ, Aaronson KD, Armstrong WF. *Doppler-derived dP/dt and −dP/dt predict survival in congestive heart failure. J Am Coll Cardiol*. 2000;36:1594–1599.

Kushwaha SS, Fallon JT, Fuster V. Restrictive CMP. *N Eng J Med*. 1997;336:267–276.

Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiography.* 2005;18:1440–1463.

Maddukuri PV, Vieira ML, DeCastro S, et al. What is the best approach for the assessment of left atrial size? Comparison of various unidimensional and 2-dimensional parameters with three-dimensional echocardiographically determined left atrial volume. *J Am Soc Echocardiography*. 2006;19:1026–1032.

Maron BJ, Gardin JM, Flack JM, et al. Prevalence of hypertrophic CMP in a general population of young adults. Echocardiographic analysis of 4111 subjects in the CARDIA Study. Coronary Artery Risk Development in (Young) Adults. *Circulation*.1995;92:785–789.

Maron BJ, Towbin JA, Thiene G, et al. Contemporary definitions and classification of the cardiomyopathies: an American Heart Association Scientific Statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. *Circulation.* 2006;113:1807–1816.

Maron MS, Olivotto I, Betocchi S, et al. Effect of left ventricular outflow tract obstruction on clinical outcome in hypertrophic CMP. *N Engl J Med.* 2003;348:295–303.

Niimura H, Patton KK, McKenna WJ, et al. Sarcomere protein gene mutations in hypertrophic CMP of the elderly. *Circulation.* 2002;105:446–451.

Oechslin EN, Jost CHA, Rojas JR, et al. Long-term follow-up of 34 adults with isolated left ventricular noncompaction: a distinct CMP with poor prognosis. *J Am Coll Cardiol*. 2000;36:493–500.

Palka P, Lange A, Donnelly JE, Nihoyannopoulos P. Differentiation between restrictive CMP and constrictive pericarditis by early diastolic Doppler myocardial velocity gradient at the posterior wall. *Circulation*. 2000;102:655–662.

Pelliccia A, Maron BJ, Spataro A, et al. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. *N Engl J Med*. 1991;324:295–301.

Richardson P, McKenna W, et al. Report of the 1995 World Health Organization/International Society and Federation of Cardiology Task Force on the Definition and Classification of Cardiomyopathies. *Circulation.* 1996;93:841–842.

Rihal CS, Nishimura RA, Hatle LK, Bailey KR, Tajik AJ. Systolic and diastolic dysfunction in patients with clinical diagnosis of dilated CMP. Relation to symptoms and prognosis. *Circulation.* 1994;90:2772–2779.

Sakamoto T. Apical Hypertrophic CMP (apical hypertrophy): An overview. *J Cardiol*. 2001;37(suppl 1):161.

Shapiro LM, McKenna WJ. Distribution of left ventricular hypertrophy in hypertrophic CMP: a two dimensional echocardiographic study. *J Am Coll Cardiol*. 1983;2:437–444.

Sharkey SW, Lesser JR, Zenovich AG, et al. Acute and reversible CMP provoked by stress in women from the United States. *Circulation*. 2005;111:472–479.

Siqueira-Filho AG, Cunha CL, Tajik AJ, et al. M-mode and 2-dimensional echocardiographic features in cardiac amyloidosis. *Circulation*. 1981;63:188–196.

Sun JP, James KB, Yang XS, et al. Comparison of mortality rates and progression of left ventricular dysfunction in patients with idiopathic dilated CMP and dilated versus non-dilated right ventricular cavities. *Am J Cardiol*. 1997;80:1583–1587.

Tabata T, Thomas JD, Klein AL. Pulmonary venous flow by Doppler echocardiography: revisited 12 years later. *J Am Coll Cardiol*. 2003;41:1243–1250.

Temporelli PL, Corra U, Imparato A, et al. Reversible restrictive left ventricular diastolic filling with optimized oral therapy predicts a more favorable prognosis in patients with chronic heart failure. *J Am Coll Cardiol*. 1998;31:1591–1597.

Ward RP, Weinert L, Spencer KT, et al. Quantitative diagnosis of apical CMP using contrast echocardiography. *J Am Soc Echocardiography*. 2002;15:316–322.

Zipes DP, ed. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*. 7th ed. Philadelphia, Pa: Elsevier Health Sciences; 2004.

CHAPTER 15

Echocardiographic Evaluation of Septic Shock

Marc Mikulski, Olivier Axler, and Paul H. Mayo

INTRODUCTION

Septic shock is a common cause for admission to the intensive care unit (ICU).[1–3] It is defined by a systemic inflammatory response syndrome (SIRS) coupled with complex hemodynamic failure. By traditional definition, the term sepsis implies the presence of infection. In this chapter, the term sepsis will be used in a more inclusive way. Septic shock follows the activation of the inflammatory response at the systemic level, most commonly as a result of infection, but on occasion due to other initiating factors. The molecular pathophysiology of sepsis is complex, and beyond the scope of this chapter. However, the clinical presentation is well known to the frontline intensivist. Hemodynamic failure is a dominant feature of septic shock, so that echocardiography has a major application in the bedside management of the condition.[4] This chapter will review the use of echocardiography in characterizing and managing the cardiovascular dysfunction associated with septic shock.

BACKGROUND

The pathophysiological consequences of sepsis include hypovolemia, left ventricular (LV), systolic, and diastolic dysfunction, and right ventricular (RV) systolic dysfunction. Echocardiography allows the intensivist to identify these processes, to monitor their progression, and to direct therapeutic interventions.

HYPOVOLEMIA AND SEPTIC SHOCK

Absolute hypovolemia is defined as a reduction of total circulating blood volume. Absolute intravascular hypovolemia is common in septic shock on initial presentation and requires prompt correction; there are several causes that depend on the underlying disease process. They may include the following:

1. Insensible losses (e.g., skin and respiratory) augmented by fever, sweating, and hyperventilation.
2. Gastrointestinal losses (e.g., diarrhea and vomiting)
3. Third-space losses (e.g., pancreatitis, burns, soft tissue injury, capillary leak, low oncotic pressure, ascites, pleural effusion)
4. Lack of fluid intake (e.g., mental status change, physical weakness, inadequate volume resuscitation when in hospital)

Relative hypovolemia is caused by an abnormal distribution of blood volume between the peripheral and central compartments. Relative hypovolemia is common in septic shock and may persist as a manifestation following initial volume resuscitation. The total blood volume may be normal, but the distribution of the blood volume is away from the central vascular compartment. The vasodilatation arises from failure of peripheral vasoconstrictor mechanisms with an inappropriate activation of vasodilator mechanisms.

Whether hypovolemia is absolute, relative, or combined, its effect is the same. Significant central hypovolemia reduces venous return to the heart, preload, stroke volume (SV), mean arterial pressure, and cardiac output. Volume resuscitation benefits the patient with septic shock by increasing venous return, preload, stroke volume, cardiac output, arterial blood pressure (systolic, mean, and pulse pressure), and tissue oxygen delivery. Identification and correction of hypovolemia is a major goal in the treatment of septic shock.

Left Ventricular Systolic Dysfunction and Septic Shock

Myocardial contractile dysfunction occurs in septic shock. Both experimental and clinical studies have demonstrated the presence of circulating substances that depress myocardial function in septic shock through a variety of mechanisms. Myocardial edema,[5] cardiomyocyte apoptosis,[6] cytokine effects (in

particular IL1, IL6, and TNF-α), and nitric oxide have all been invoked.[7,8] Elevations of cardiac troponin levels without myocardial infarction occur commonly in septic shock,[9] although coronary perfusion and myocardial energy metabolism are preserved in septic shock.[7,8]

The identification of LV systolic dysfunction may be difficult using echocardiography, as traditional echocardiographic indices of LV systolic function may be load dependent. Hypovolemia has a major influence on preload, while vasodilation may have profound effect on afterload. The hypovolemic ventricle in the vasodilated hypotensive patient may have a normal ejection fraction. Following volume resuscitation and vasopressor use, the repeat echocardiogram may only then show depressed contractile activity. Clinical and experimental studies show an early and reversible reduction in LV function in septic shock with rightward displacement of the LV pressure volume curve.[7,10–12] A reduction in ejection fraction is a common finding in septic shock, and should alert the clinician to the possibility of a need for manipulation of afterload and inotropic support. Likewise, a normal ejection fraction in septic shock does not exclude LV dysfunction. Alterations of preload and afterload with volume and vasopressors may alter the echocardiographic findings. There is controversy as to whether the presence of LV systolic dysfunction in septic shock might be associated with improved survival. Parker et al. were the first to describe this association.[13] However, Vieillard-Baron and colleagues reported no survival difference between patients with and without LV systolic dysfunction.[14] The hypothesis that LV dilatation associated with depression of systolic function is adaptive and maintains cardiac output was not confirmed by echocardiography.[15]

Left Ventricular Diastolic Dysfunction and Septic Shock

Left ventricular diastolic dysfunction has also been reported in septic shock[11,16,17] and has been associated with increased mortality.[18] It is associated with elevated cardiac troponin levels, increased cytokine activity (TNF, IL-8, IL-10), and frequently occurs in conjunction with systolic dysfunction, but also occurs as an isolated abnormality in 20% of cases.[19]

Right Ventricular Dysfunction and Septic Shock

An impairment of RV contractile function is common in septic shock. It may occur in the absence of increased pulmonary artery pressure[20–23] or LV dysfunction,[24] and is probably caused by the same circulating factors that result in LV dysfunction. Septic shock is frequently associated with complications that may indirectly affect RV function. Lung disease such as severe pneumonia or acute respiratory distress syndrome (ARDS), hypoxic pulmonary vascular constriction, and high ventilator pressures may all cause elevation of RV afterload with a resultant acute cor pulmonale pattern (see Chapter 16). Septic shock may cause RV dysfunction by both direct and indirect depression of RV function.

Echocardiography in the Management of Septic Shock

Because significant hypovolemia is so common in septic shock, initial volume resuscitation should never be delayed by waiting for echocardiography. Clinical assessment in the prehospital and emergency department suffices to make a decision to initiate vigorous volume resuscitation of the septic patient, as early volume resuscitation has been associated with improved outcomes.[25] However, the intensivist frequently receives the patient in the ICU following some degree of volume resuscitation. The question then becomes whether the patient should receive further volume resuscitation, vasopressors, or inotropic support. In this situation, echocardiography is an ideal tool for the rapid assessment of the septic patient, as it allows identification of hypovolemia, LV systolic and diastolic dysfunction, and RV dysfunction. Echocardiography is performed by the intensivist at the bedside of the patient with hemodynamic failure. The results of the initial assessment allow for the formulation of a therapeutic plan, while follow-up study allows ongoing assessment for the effect of therapy, progression of disease, and identification of new problems.

Echocardiography in the Management of Septic Shock: Evaluation of Preload Sensitivity

Volume resuscitation of the septic patient is an important component of the initial resuscitation. However, excessive volume resuscitation may have adverse consequences (see Chapter 10). One approach to this problem is to use bedside echocardiography to assess for preload sensitivity. In using echocardiography, the intensivist is asking a straightforward question: Will further volume resuscitation improve

cardiac output? Echocardiography allows the intensivist to answer this question at the bedside of the patient. The echocardiographic measurement of volume status uses dynamic measurements of preload sensitivity; the type depends upon the proficiency of the echocardiographer. Static measurements have been shown to be unreliable.[26]

The intensivist with competence in basic critical care echocardiography has, by definition, limited skill at Doppler measurement and must rely on 2D imaging to guide decision-making. Respiratory variation of inferior vena cava (IVC) size is a validated method of determining volume responsiveness that is easily performed by the basic critical care echocardiographer (see Chapter 10).[27,28] The patient must be on mechanical ventilatory support and completely passive in interaction with the ventilator. In addition, pattern recognition of a small hyperdynamic LV (with effacement of the end-systolic LV cavity),[29] or a small IVC diameter (<10 millimeter [mm]) suggests volume responsiveness in the patient with septic shock. While pattern recognition by the echocardiographer of a small hyperkinetic LV and RV in septic shock strongly suggests hypovolemia, these findings have somewhat limited predictive value for response to volume resuscitation.

Competence in advanced critical care echocardiography allows the intensivist to make a variety of Doppler-based measurements to identify the need for further volume resuscitation in the patient with septic shock. If the patient is on mechanical ventilatory support and lacking any spontaneous breathing effort, respiratory variation of superior vena cava diameter measured with transesophageal echocardiography (TEE) is a simple method of determining volume sensitivity.[30] Alternatively, respiratory variation of stroke volume measured with Doppler is useful in identifying preload sensitivity (see Chapter 10).[31] Like the variation in IVC and superior vena cava (SVC) size, the patient must be passive in interaction with the ventilator and in sinus rhythm. The measurement of SV and cardiac output before and after passive leg raising using Doppler is another method of identifying preload sensitivity.[32,33] This method has the major advantage of having utility in patients who are breathing spontaneously and with irregular heart rhythm.

Echocardiography allows the intensivist to guide volume resuscitation. The initial examination allows determination of whether the patient is in a preload sensitive state. If volume resuscitation is indicated, serial examination is useful in determining whether to continue with volume resuscitation.

Echocardiography in the Management of Septic Shock: Evaluation of Left Ventricular Systolic Function

In the initial phase of septic shock, LV function is often described as "hyperkinetic" and cardiac output is classically considered to be augmented (hyperkinetic phase of sepsis). In fact, the study of cardiac function indices that are load independent demonstrate impairment of contractile function even though cardiac output and ejection fraction are normal or elevated. The reduction in LV contractile function occurs early in the course of septic shock and often resolves completely 7–10 days following resolution of the sepsis. Volume resuscitation and the use of vasopressors alter the load conditions of the LV.

On initial presentation, echocardiography may demonstrate a hyperdynamic LV contraction pattern as the LV is underfilled and afterload is low. Following volume resuscitation and elevation of blood pressure, echocardiographic findings may then change to show reduced LV systolic function.

Form a practical perspective, the evaluation of LV systolic function relies on the measurement of ejection fraction (EF). The intensivist with basic critical care echocardiography skills has a limited ability to measure EF. Visual estimates of EF can be quite accurate if performed by a skilled echocardiographer. However, the basic echocardiographer should not attempt a numerical estimate, but rather classify the LV function as severely impaired, moderately impaired, normal, or hyperdynamic. Alternatively, the intensivist with competence in advanced critical care echocardiography may use a variety of methods to determine EF. M-mode measurements rely on diameter measurements. The Teichholz method requires that the diameter of the LV be measured at the midventricular level in the parasternal long-axis view. This method requires careful attention to technical detail, and has not been validated in critically ill patients on mechanical ventilation. It cannot be used in patients with segmental wall abnormality, and requires that the M-mode interrogation be perpendicular to the LV wall. This is often difficult to accomplish in the critically ill, as the heart may be difficult to orientate for adequate measurement. Coupled with the translational artifacts that occur with machine cycling and the geometric assumptions intrinsic to using a diameter measurement to define a complex three-dimensional structure, M-mode EF methodology may not be a reliable means of measuring EF in the critically ill. The

alternative is to use an area measurement method. A simple approach is to measure the surface area of the LV cavity in the parasternal short-axis view at the papillary muscle level (using transthoracic echocardiography [TTE] or TEE) at end-diastole and at end-systole. These values are used to derive the EF (end-diastolic surface area–end-systolic surface area/end-diastolic surface area). While conceptually superior to diameter-based measurements, the surface area method is still susceptible to segmental wall abnormality and translational artifact. An accurate estimate of EF may be derived using Simpson's method. The surface area of the LV at end-diastole and end-systole is measured from the apical view in two orthogonal planes (apical four-chamber and apical two-chamber views). Simpson's method yields an accurate measurement of EF, but may not be practical for the frontline intensivist because it requires significant time to perform, clear endomyocardial border definition, careful attention to technical detail (good axis and avoidance of translational artifact), and a high-quality machine.

The determination of EF is helpful in assessing LV contractile function, but it does not address the issue of SV or cardiac output (these values are best indexed to body surface area). The underfilled hyperdynamic LV may exhibit an excellent EF, and yet the SV and cardiac output may be inadequate. Likewise, the dilated hypocontractile LV with low EF may have an adequate SV and cardiac output. Measurement of SV and a cardiac output require the use of Doppler. In the apical five-chamber view (TTE) or deep-gastric view (TEE), the pulsed-wave Doppler interrogation box is placed in the LV outflow tract (LVOT) with parallel intercept to the direction of blood flow. The area under the systolic outflow velocity curve (VTI = velocity time integral) is directly proportional to the SV. The VTI is multiplied by the area of the LVOT to yield the SV and cardiac output. These values may be indexed to body surface area and used to derive other parameters of hemodynamic function. The knowledge of EF gives the intensivist information about LV contractile function, while the measurement of SV and cardiac output may be used to determine whether oxygen delivery is at acceptable level. Measurement of cardiac output and EF will vary according to the evolution of the sepsis state and in response to therapeutic interventions. This favors an approach of serial echocardiographic examination. An excellent example of the importance of repeated examination occurs in the recovery phase from septic shock. In this case, the study done early in the course of septic shock might demonstrate a severe reduction of EF. Several weeks later, a repeat study might show completely normal LV function. This is very important information for the clinical management of the patient. Absent the second examination, the patient could be labeled as having chronic LV failure and receive inappropriate long-term therapy for a self-limited condition.

Echocardiography in the Management of Septic Shock: Evaluation of Left Ventricular Diastolic Function

Septic shock alters LV diastolic function. Diastolic relaxation is an active energy requiring process; this is probably disrupted by the same circulating factors that cause systolic dysfunction. Traditional methods of measurement rely on Doppler analysis of mitral valve inflow. These are load dependent. Alternative load independent methods measure tissue Doppler velocity of the longitudinal movement of the mitral valve annulus (E').[34] Assessment of diastolic function has interest to the intensivist because it allows estimation of LV diastolic pressures and left atrial pressures. Elevation of left-sided diastolic pressures result in an elevation of pulmonary capillary pressure. This, in turn, augments risk of hydrostatic pulmonary edema. Current recommendations suggest that patients with septic shock should receive vigorous volume resuscitation. However, excessive volume resuscitation runs the risk of causing pulmonary edema. Patients with septic shock are at risk for acute lung injury or acute respiratory distress syndrome (ALI/ARDS). Hydrostatic pulmonary edema compounds this problem. It is prudent, following the initial volume resuscitation phase and particularly if the patient has ALI/ARDS, to obtain echocardiographic estimates of left-sided diastolic pressures. The identification of elevated pressure can then lead to therapeutic interventions such as fluid restriction or diuretics. The goal is to reduce left-sided filling pressures to reduce the risk of hydrostatic pulmonary edema. Doppler measurements allow the intensivist to estimate pulmonary capillary occlusion pressure (PAOP) in several ways.

Transmitral diastolic flow velocity is readily measured using pulsed-wave Doppler from the apical four-chamber view. An E/A >2 is associated with a PAOP >18 mm Hg with a positive predictive value of 100%.[35] Pulmonary venous inflow is also useful in estimating PAOP. A ratio of the VTI of the systolic anterior velocity envelope to the total VTI of the systolic and diastolic velocity envelope $<45\%$ predicts a PAOP >12 mm Hg with a positive predictive value of $>100\%$.[35] A

duration of the pulmonary venous retrograde A wave that is greater than the duration of the mitral inflow A wave is associated with a PAOP >15 mm Hg with a positive predictive value of 83%.[35] Tissue Doppler of the lateral mitral annulus allows measurement of the velocity of early diastolic annular velocity. The ratio of the mitral E wave velocity to E' (E/E') >9 suggests a PAOP >15 mm Hg.[36] Measurement of mitral valve inflow and annular velocities are usually obtainable with TTE. Good quality pulmonary venous inflow velocities are often difficult to obtain with TTE and may require TEE examination.

Echocardiography in the Management of Septic Shock: Evaluation of Right Ventricular Function

Right ventricular function may be compromised as a direct effect of circulation factors of sepsis, as is the case of the LV. In addition, RV function may be compromised by factors that occur as a complication of sepsis. Acute lung injury, hypoxic pulmonary vascular vasoconstriction, and positive pressure ventilation may all increase RV afterload to the extent of causing acute cor pulmonale. Echocardiography allows the intensivist to identify acute cor pulmonale (see Chapter 16). Identification of acute cor pulmonale allows the intensivist to take specific steps to reduce RV afterload, and reduce RV dilatation.

Echocardiography in the Management of Septic Shock: Clinical Applications

Hypovolemia and hypotension are cardinal features of septic shock. In addition to the immediate use of broad spectrum antibiotics[37] with the control of localized infection, initial management will generally include vigorous volume resuscitation and the assurance of perfusion with vasoactive medications (typically norepinephrine). This standard approach is usually initiated in the emergency department. The intensivist receives the patient in the ICU following initial stabilization, and must then develop a management plan. Early echocardiography serves two purposes. First, it excludes alternative or coexisting causes for the shock state such as unrecognized pericardial tamponade, severe valve failure, segmental wall abnormality raising concern for ischemic heart disease, or pulmonary embolism. Second, it allows the intensivist to answer some key questions related to the ongoing hemodynamic management of septic shock. These are as follows:

1. Will the patient benefit from further volume resuscitation? The echocardiographer may answer this question by pattern recognition alone: a small IVC or a hyperdynamic LV with end-systolic cavity obliteration suggests a need for further volume resuscitation. If the patient is on a ventilator without spontaneous breathing effort, the presence of significant respiratory IVC diameter variation indicates the need for continued volume resuscitation, while its absence suggests no further need. The advanced-level echocardiographer may answer this question by the PLR test in the patient who is making spontaneous respiratory effort. Alternatively, If the patient is on a ventilator without spontaneous breathing effort and in sinus rhythm, significant respiratory variation of SV (measured with echocardiography) indicates a need for further volume resuscitation, while its absence suggests no further need. The decision to continue volume resuscitation is a critical one, as inappropriate volume resuscitation may be harmful to the patient.
2. Should the patient receive inotropic support like dobutamine or epinephrine? Echocardiography allows the evaluation of LV function. The basic-level echocardiographer is able to recognize but not quantitate a reduced EF, while the advanced-level echocardiographer may accurately estimate the EF or choose to perform a quantitative determination. This may be supplemented by the measurement of the SV and cardiac output. The finding of reduced LV systolic function is common in septic shock. However, this does not necessarily warrant the use of inotropic agents. Direct measurement of SV and cardiac output, if available, are helpful in this situation. If the SV and cardiac output are in normal range, there is no purpose served in using inotropic support. Targeting supranormal levels of oxygen delivery is not indicated in septic shock.[46] On the other hand, if SV and cardiac output are sufficiently low to be associated with a significant reduction in oxygen delivery, inotropes are then indicated. If quantitative SV and cardiac output measurement are not available, the intensivist may need to rely on clinical indicators in making a decision to initiate inotropic support. In summary, the finding of reduced LV systolic function does not by itself require initiation of inotropic support. Measurement of SV and cardiac output is helpful in making the decision, as is careful assessment of the clinical status of the patient.

3. Is there an indication of elevated PAOP with risk of hydrostatic pulmonary edema? The advanced-level echocardiographer may be able to identify the patient with elevation of PAOP. If this is present, treatment directed at this problem may improve lung function in patients who have concomitant ALI/ARDS.
4. Is acute cor pulmonale present? The intensivist with competence in basic critical care echocardiography is able to recognize RV dilatation and septal dyskinesia that is diagnostic of acute cor pulmonale. Acute cor pulmonale may be multifactorial. A direct effect of sepsis on RV function may contribute, but secondary factors are also likely, such as when the patient is on ventilatory support with ALI/ARDS. Identification of acute cor pulmonale allows the intensivist to take steps to reduce RV afterload.

The team of Vieillard-Baron et al. has fully integrated serial echocardiography into the management of septic shock on a routine basis.[14,38] Their approach can be readily adapted to TTE, and serves as a motivating example for the utility of critical care echocardiography.

CONCLUSION

Critical care echocardiography is a useful method for establishing the diagnosis of hemodynamic failure in the ICU. It offers the frontline intensivist a bedside tool to guide the ongoing management of the patient. It is particularly useful for the management of hemodynamic failure related to sepsis, as it can guide the use of volume resuscitation, vasopressor, and inotropic agents.

References

1. Martin GS, Mannino DM, Eaton S, Moss M. The epidemiology of sepsis in the United States from 1979 through 2000. *N Engl J Med.* 2003;348:1546–1554.
2. Vincent JL, Sakr Y, Sprung CL, et al. Sepsis in European intensive care units: results of the SOAP study. *Crit Care Med*. 2006;34:344–353.
3. Annane D, Aegerter P, Jars-Guincestre MC, Guidet B. Current epidemiology of septic shock: the CUB-Rea Network. *Am J Respir Crit Care Med*. 2003168:165–172.
4. Axler O. Evaluation and management of shock. Seminars in respiratory and critical care medicine 2006;27:230-240. In "Non-pulmonary complications in ICUs". Curt Sessler, Guest Editor. Joseph Lynch. Editor-in-Chief.)
5. Yu P, Boughner DR, Sibbald WJ, keys J, Dunmore J, Martin CM. Myocardial collagen changes and edema in rats with hyperdynamic sepsis. *Crit Care Med.* 1997;25:657–662.
6. Neviere R, Fauvel H, Chopin C, Formstecher P, Marchetti P. Caspase inhibition prevents cardiac dysfunction and heart apoptosis in a rat model of sepsis. *Am J Respir Crit Care Med.* 2001;163:218–225.
7. Krishnagopalan S, Kumar A, Parrillo JE. Myocardial dysfunction in the patient with sepsis. *Curr Opin Crit Care*. 2002;8:376–388.
8. Kumar A, Krieger A, Symeoneides S, Parrillo JE. Myocardial dysfunction in septic shock: Part II. Role of cytokines and nitric oxide. *J Cardiothorac Vasc Anesth*. 2001;15:485–511.
9. Ammann P, Fehr T, Minder EI, et al. Elevation of troponin I in sepsis and septic shock. *Intensive Care Med.* 2001;27:965–969.
10. Ognibene FP, Parker MM, Natanson C, Shelhamer JH, Parrillo JE. Depressed left ventricular performance. Response to volume infusion in patients with sepsis and septic shock. *Chest*. 1988; 93:903–10.
11. Poelaert J, Declerck C, Vogelaers D, Colardyn F, Visser CA. Left ventricular systolic and diastolic function in septic shock. *Intensive Care Med*. 1997;23:553–560.
12. Robotham JL, Takata M, Berman M, Harasawa Y. Ejection fraction revisited. *Anesthesiology*. 1991;74:172–183.
13. Parker MM, Shelhamer JH, Bacharach SL, et al. Profound but reversible myocardial depression in patients with septic shock. *Ann Intern Med*. 1984;12:596–599.
14. Vieillard-Baron A, Caille V, Charron C, Belliard G, Page B, Jardin F. Actual incidence of global left ventricular hypokinesia in adult septic shock. *Crit Care Med*. 2008;36: 1701–1706.
15. Vieillard-Baron A, Schmitt JM, Beauchet A, et al. Early preload adaptation in septic shock? A transesophageal echocardiographic study. *Anesthesiology*. 2001;94:400–406.
16. Etchecopar-Chevreuil C, Francois B, Clavel M, Pichon N, Gastinne H, Vignon P. Cardiac morphological and functional changes during early septic shock: a transesophageal echocardiographic study. *Intensive Care Med*. 2008;34:250–256.
17. Jafri SM, Lavine S, Field BE, Bahorozian MT, Carlson RW. Left ventricular diastolic function in sepsis. *Crit Care Med*. 1990;18:709–714.
18. Munt B, Jue J, Gin K, Fenwick J, Tweeddale M. Diastolic filling in human severe sepsis: an echocardiographic study. *Crit Care Med*. 1998;26:1829–1833.
19. Bouhemad B, Nicolas-Robin A, Arbelot C, Arthaud M, Feger F, Rouby JJ. Isolated and reversible impairment of ventricular relaxation in patients with septic shock. *Crit Care Med*. 2008;36:766–774.

20. Parker MM, McCarthy KE, Ognibene FP, Parrillo JE. Right ventricular dysfunction and dilatation, similar to left ventricular changes, characterize the cardiac depression of septic shock in humans. *Chest*. 1990;97:126–131.
21. Hoffman MJ, Greenfield LJ, Sugerman HJ, Tatum JL. Unsuspected right ventricular dysfunction in shock and sepsis. *Ann Surg*. 1983;198:307–319.
22. Dhainaut JF, Lanore JJ, de Gournay JM, et al. Right ventricular dysfunction in patients with septic shock. *Intensive Care Med*. 1988;14(suppl 2):488–491.
23. Lambermont B, Ghuysen A, Kolh P, et al. Effects of endotoxic shock on right ventricular systolic function and mechanical efficiency. *Cardiovasc Res.* 2003;59:412–418.
24. Kimchi A, Ellrodt AG, Berman DS, Riedinger MS, Swan HJ, Murata GH. Right ventricular performance in septic shock: a combined radionuclide and hemodynamic study. *J Am Coll Cardiol.* 1984;4:945–951.
25. Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med*. 2001;345:1368–1377.
26. Axler O, Tousignant CP, Thompson CR, et al. Small hemodynamic effect of typical rapid volume infusions in critically ill patients. *Crit Care Med*. 1997;25:965–970.
27. Feissel M, Michard F, Faller JP, Teboul JL. The respiratory variation in inferior vena cava diameter as a guide to fluid therapy. *Intensive Care Med*. 2004;30:1834–1837.
28. Barbier C, Loubieres Y, Schmit C, et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responsiveness in ventilated septic patients. *Intensive Care Med*. 2004;30:1740–1746.
29. Leung JM, Levine EH. Left ventricular end-systolic cavity obliteration as an estimate of intraoperative hypovolemia. *Anesthesiology*. 1994;81:1102–1109.
30. Vieillard-Baron A, Chergui K, Rabiller A, et al. Superior vena caval collapsibility as a gauge of volume status in ventilated septic patients. *Intensive Care Med*. 2004;30: 1734–1739.
31. Feissel M, Michard F, Mangin I, Ruyer O, Faller JP, Teboul JL. Respiratory changes in aortic blood velocity as an indicator of fluid responsiveness in ventilated patients with septic shock. *Chest.* 2001;119:867–873.
32. Monnet X, Rienzo M, Osman D, et al. Passive leg raising predicts fluid responsiveness in the critically ill. *Crit Care Med*. 2006;34:1402–1407.
33. Lamia B, Ochagavia A, Monnet X, Chemla D, Richard C, Teboul JL. Echocardiographic prediction of volume responsiveness in critically ill patients with spontaneously breathing activity. *Intensive Care Med*. 2007;33:1125–1132.
34. Vignon P, Allot V, Lesage J, et al. Diagnosis of left ventricular diastolic dysfunction in the setting of acute changes in loading conditions. *Crit Care.* 2007;11:R43.
35. Boussuges A, Blanc P, Molenat F, Burnet H, Habib G, Sainty JM. Evaluation of left ventricular filling pressure by transthoracic Doppler echocardiography in the intensive care unit. *Crit Care Med*. 2002;30:362–367.
36. Combes A, Arnoult F, Trouillet JL. Tissue Doppler imaging estimation of pulmonary artery occlusion pressure in ICU patients. *Intensive Care Med*. 2004;30:75–81.
37. Kumar A, Roberts D, Wood KE, et al. Duration of hypotension before initiation of effective antimicrobial therapy is the critical determinant of survival in human septic shock. *Crit Care Med*. 2006;34:1589–1596.
38. Vieillard-Baron A, Charron C, Chergui K, Peyrouset O, Jardin F. Bedside ehocardiographic evaluation of hemodynamics in sepsis: is a qualitative evaluation sufficient? *Intensive Care Med*. 2006;32:1547–1552.

This page intentionally left blank

CHAPTER 16

Echocardiographic Evaluation of Valve Function and Endocarditis

Paul H. Mayo

INTRODUCTION

Echocardiography is an effective means of assessing cardiac valve function. It is useful for a rapid qualitative assessment or a more comprehensive assessment for all forms of valve function using transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE). Doppler assessment allows an accurate quantitative measurement of the severity of stenotic and regurgitant lesions. The extent to which the critical care echocardiographer applies the sophisticated tools of the cardiologist to assess valvular heart disease is highly variable. By training, background, and interest, cardiologists often take the lead in this aspect of echocardiography. However, the intensivist who performs echocardiography should have competence in assessing valve function, as many patients in the intensive care unit (ICU) may have valve dysfunction that adversely impacts their cardiopulmonary status.

In general, the intensivist will be interested in the identification of catastrophic valve failure or valve dysfunction that is sufficiently severe to impact the hemodynamic functioning of the patient. Conversely, the identification of lesser degrees of valve disease or normal valve function are also of interest, as the intensivist may then determine that valve failure is not a contributing factor to the patient's critical illness. This chapter will review the echocardiographic assessment of valve function from the perspective of the bedside intensivist.

LEVEL OF TRAINING

Intensivists who perform critical care echocardiography will either have competence in basic critical care echocardiography (several standard 2D views without comprehensive training in Doppler) or competence in advanced critical care echocardiography (see also Chapter 4). The latter is equivalent to level 2 training by standard cardiology criteria.[1] Intensivists who have basic training in echocardiography have a limited ability to assess valve function. The standard 2D views of basic critical care echocardiography generally include the parasternal short- and long-axis, the apical four-chamber, and the subcostal views. Detailed examination of the tricuspid valve (TV) is not part of basic echocardiographic assessment, as the focus of the basic examination is to assess for left ventricular (LV) and right ventricular (RV) size and function, for pericardial effusion, and for evidence of preload sensitivity. It is not to perform a detailed valvular assessment. Without training in Doppler measurements, the basic-level examiner can identify obvious mechanical failure of the mitral valve (MV) (e.g., a flail leaflet, ruptured chordae, or ruptured papillary muscle) or obvious aortic valve (AV) disruption. Severe stenosis of these valves may also be apparent. By definition, intensivists with training in basic echocardiograpy do not have comprehensive Doppler training and often lack the ability to perform quantitative measurements of valve function.

Color Doppler may be used for a qualitative assessment of valve function. The basic-level critical care echocardiographer should be cautious in assuming to have the skills in the use of color Doppler. One of the problems with the technique is that it appears to be straightforward, when it is actually not. The pitfalls of color Doppler include gain settings, wall jet effects,[2] angle effects, and shadowing by surrounding structures, such as prosthetic valve apparatus or a calcified annulus, and are not intuitively obvious. The problem is that the echocardiographer may miss a severe valvular lesion due to misinterpretation of the color Doppler image. A key cognitive skill for the basic critical care echocardiographer is to recognize when to call for a consultation from a more experienced echocardiographer. If there is the possibility of significant valve dysfunction, a comprehensive study should be performed by an echocardiographer with advanced training.

Intensivists with proficiency in advanced critical care echocardiography have, by definition, the

capability to assess valve function that is similar to that of a fully trained cardiology echocardiographer. This proficiency may be particularly useful when immediate cardiology echocardiography services are not available. The advanced intensivist echocardiographer, who is directly involved at the patient's bedside, is also able to immediately perform the study. Patients with severe valve failure may have life-threatening hemodynamic failure requiring prompt intervention such as vasoactive medication, mechanical assist devices, or valve replacement. This cannot wait for the convenience of a delayed echocardiogram.

A common indication for bedside echocardiography in the ICU is to identify a cause for severe cardiopulmonary failure. The assessment of valve function is a key part of the bedside examination for several reasons. First, severe valve failure may be the primary cause of the shock state or respiratory failure. Early recognition of severe valve failure may allow for lifesaving interventions. Second, significant valve dysfunction may combine with another disease process to worsen cardiopulmonary failure. It is essential that the coexisting valve lesion be identified, as this may have a major influence on management. Finally, the absence of significant valvular dysfunction is useful information in a patient with hemodynamic failure.

The focus here is on the identification and quantification of major valve failure, as this is the most immediate concern of the bedside critical care clinician. Comprehensive assessment of valve function requires training in Doppler analysis, so some of the following discussion assumes that the reader has background in Doppler measurements.

TRICUSPID VALVE

The evaluation of the TV focuses on the assessment of tricuspid regurgitation (TR), as tricuspid stenosis is an uncommon lesion. The echocardiographic examination of the TV starts with 2D imaging of the valve and support structures. Lack of coaptation, flail leaflet, vegetation or mass, RV or right atrial (RA) dilation, tricuspid annular calcification, or the presence of hardware (pacer wire, valve repair/replacement), all suggest the possibility of significant TR. The 2D study includes examination of the valve from multiple sites: parasternal long- and short-axis, apical four-chamber, and subcostal. The severity of TR by color Doppler is classed as mild, moderate, and severe. It is important to properly set the color gain and to interrogate the jet at multiple angles. The severity of TR may be measured semiquantitatively by measuring the color jet area.[3] Minimal TR

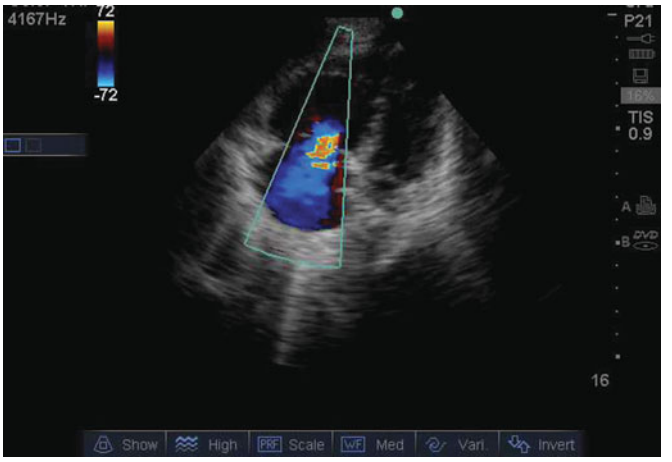


Figure 16.1. Apical four-chamber view of the heart. Color Doppler examination shows severe tricuspid regurgitation.

may often be detected in normal individuals.[4] A simple "eyeball" method of judging severity is to consider that TR is severe when the jet hits the back wall of the RA or occupies the entire RA (Figure 16.1, and Video 16.1 in enclosed DVD). Severe TR is often associated with volume overload of the right ventricle, although this finding is not specific to TR. Spectral Doppler of the TR jet may provide additional information of severity. If the intensity of the continuous-wave (CW) Doppler TR signal is greater than that of the inflow signal, or if the CW TR jet has a truncated downslope, the TR is likely to be severe. Measurement of the CW jet velocity permits estimation of pulmonary arterial systolic pressure (PASP). However, PASP does not necessarily correlate with the severity of TR. In theory, the severity of TR is amenable to quantitative measurements using the continuity principle or proximal isovelocity surface area (PISA) method. In practice, this is seldom performed.

PULMONIC VALVE

The evaluation of the pulmonic valve (PV) generally focuses on assessment for pulmonic valve regurgitation (PR), as PV stenosis is uncommon. Trace PR is often detected in normal individuals. The echocardiographic examination of the PV starts with 2D imaging of the valve. Rarely, a vegetation may involve the valve, or a leaflet prolapse may be identified. The PV is difficult to image with 2D technique even using TEE. The 2D TTE examination of the valve is usually limited to the parasternal short-axis view and a short-axis view from the subcostal region. Color Doppler is the primary means of detecting PR (Figure 16.2, and Video 16.2 in enclosed DVD). The subcostal approach is frequently a

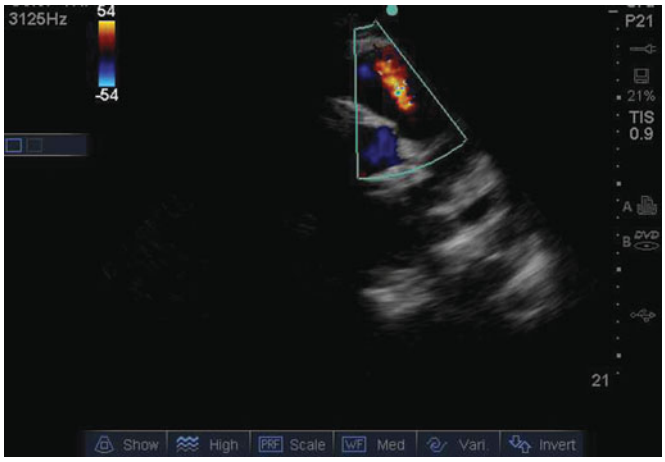


Figure 16.2. Right ventricular outflow view of the heart. Color Doppler examination shows moderate pulmonary regurgitation.

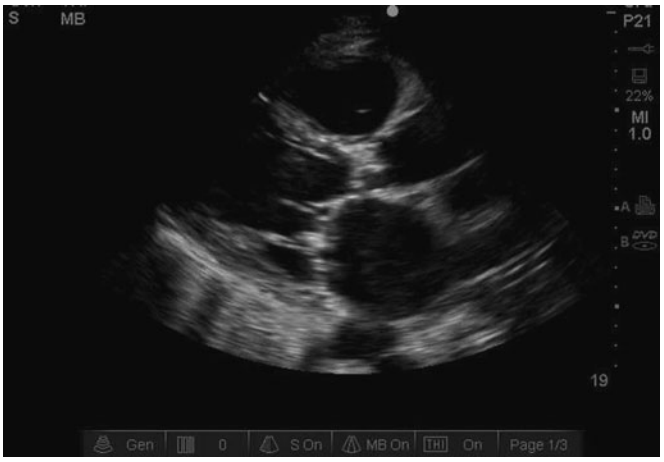


Figure 16.3. Parasternal long-axis view of the heart showing severe aortic stenosis.

superior angle for Doppler analysis because the interrogation line may be placed parallel with the blood flow through the PV and proximal pulmonary artery. Regurgitation is detected by color Doppler and is classed as trace, mild, moderate, or severe.[3] Because PR usually has minimal hemodynamic consequence, clinical echocardiographers often use an "eyeball" approach to grading the severity of PR. Spectral Doppler analysis of valve function is not generally used to assess the severity of PR. However, spectral Doppler of the PV area is important for other reasons such as the measurement of cardiac shunts, pulmonary artery diastolic pressure, and indirect evidence for pulmonary arterial hypertension.

AORTIC VALVE: AORTIC STENOSIS

Aortic stenosis (AS) is a common valvular lesion in the critical care unit. When it is severe, it has major hemodynamic consequences. When it coexists with other causes of shock, such as distributive shock, it complicates hemodynamic management. Many patients come to the ICU with the diagnosis already established. However, in others, it is unknown at the time of presentation. The intensivist with basic-level echocardiography training may suspect the diagnosis by 2D scanning. The valve is often hyperechoic or heavily calcified with obviously reduced movement (Figure 16.3, and Video 16.3 in enclosed DVD).

Advanced level training is required for a complete assessment of the AV. This begins with 2D study to rule out sub- or supravalular stenosis or a stenotic bicuspid valve, which lack the characteristic 2D features of typical AS. These entities have the same physiological consequence. Included in the 2D examination is planimetry of the valve area in short axis both by TTE and TEE.[5]

Three methods for identifying severe AS exist. Doppler analysis is the most reliable method of identifying severe AS. As the aortic valve area narrows, the velocity of blood flow through it rises. The peak velocity across the valve is measured with CW Doppler and reliably reflects the pressure gradient across the valve according to the modified Bernoulli equation.[6] A second method for quantification of AS is the continuity equation.[7] This is based on the principle that the stroke volume measured at one point in the heart should equal the stroke volume at another point in the heart (barring shunt or valvular regurgitation). A final method compares the velocity (or velocity time integral [VTI]) of the left ventricular outflow track to that of the aortic valve.[8]

Commonly accepted values indicating severe aortic stenosis include[9]:

1. Peak aortic valve velocity >4.5 m/sec
2. Mean pressure gradient across the aortic valve >50 mm Hg
3. Aortic valve area <0.75 cm^2
4. LVOT VTI/aortic VTI <0.25 (LVOT: left ventricular outflow tract)

Accurate assessment of AS requires skill in advanced echocardiography. Common pitfalls include underestimation of the peak velocity and mean pressure gradient due to poor CW Doppler interrogation angle. The AS jet may also be eccentric; therefore, the examiner must use multiple points of measurement, including the suprasternal and right parasternal sites. This may require the use of a small, nonimaging CW transducer.

Accurate measurement of the LVOT diameter is required, as any error is squared in the calculation of the LVOT area. The LVOT VTI may be overestimated if the PW interrogation box is placed too close to the AV with resulting measurement of flow acceleration near the valve orifice. Patients with poor LV function and AS may have pseudosevere AS that improves when the measurements are made during dobutamine infusion.[10]

AORTIC VALVE: AORTIC REGURGITATION

Aortic regurgitation (AR) is a common valvular lesion in the ICU. Acute severe AR may be immediately life threatening because the LV has no time to adapt to the sudden volume overload, resulting in fulminant pulmonary edema compounded by a low-flow state. Aortic balloon pump is specifically contraindicated with severe AR, and urgent valve replacement may be life saving. Chronic severe AR is often well tolerated because the LV has had time to dilate; and, with the maintenance of good LV function, forward flow is maintained without elevation of hydrostatic pressure in the lung. However, chronic severe AR may eventually lead to hemodynamic failure. When it coexists with other causes of shock, it may complicate hemodynamic management. The degree of regurgitation is afterload sensitive, so that the identification of significant AR has an important influence on acute hemodynamic management.

The evaluation for AR begins with the 2D examination. Dilation of the aortic root, proximal aortic dissection, abnormal valve architecture, noncoaptation or prolapse of valve leaflets, or vegetation, all suggest the possibility of significant AR. M-mode findings of severe AR include anterior MV diastolic fluttering, early closure of the MV, and the presence of a B wave on M-mode of the MV. Aortic regurgitation may occur due to dilation of the aortic ring with valvular incompetence from nonapposition of the leaflets. Valve anatomy may be normal in this situation. Alternatively, AR may occur due to structural failure of the valve itself from such factors as endocarditis, rheumatic heart disease, Marfan's syndrome, congenitally abnormal valves, degenerative calcific changes, or aortic dissection. None of these anatomic abnormalities allow determination of the severity of AR.

Doppler studies allow several methods to determine the severity of AR. Color Doppler performed preferentially in the parasternal long- and short-axis view with TTE or TEE is one method of determining the severity of AR (Figure 16.4, and Video 16.4 in enclosed DVD).

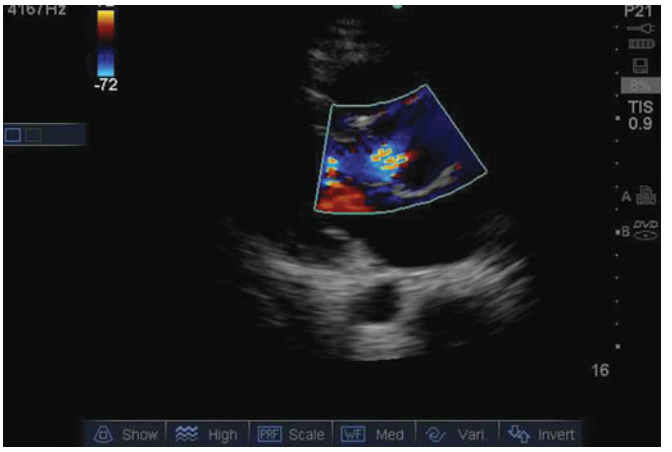


Figure 16.4. Parasternal long-axis view of the heart. Color Doppler examination shows severe aortic regurgitation.

The length of the regurgitant jet does not correlate well with severity. The jet area in a short-axis view, relative to the LVOT area, or the width of the jet at its origin, relative to the LVOT width in long axis, is considered a good index of severity.[3] The width of the vena contracta,[11] the smallest width of the jet below flow convergence, is another index of severity that relies on color Doppler. The pressure half time (PHT) of the AV regurgitant jet measured with CW Doppler falls in proportion to the severity of the AR, although the PHT is load dependent. Severe AR results in a high mitral E wave velocity as well as diastolic retrograde flow in the descending aorta.[12]

The continuity principle and PISA method[13] may be used to quantify the severity of AR. These methods are laborious, so the bedside echocardiographer frequently relies on color Doppler and PHT.

Commonly accepted values indicating severe AR are as follows[3]:

1. Regurgitant jet width/LVOT diameter ratio >65%
2. Regugitant jet area/LVOT area ratio >60%
3. Vena contracta width >6 mm
4. PHT <200 m/sec
5. Holodiastolic flow reversal in the descending aorta
6. Effective regurgitant orifice >0.30 cm^2
7. Regurgitant volume >60 mL
8. Regugitant fraction >50%

MITRAL VALVE: MITRAL STENOSIS

Compared with AS, mitral stenosis (MS) is uncommon in the ICU. When it is severe, it has major hemodynamic

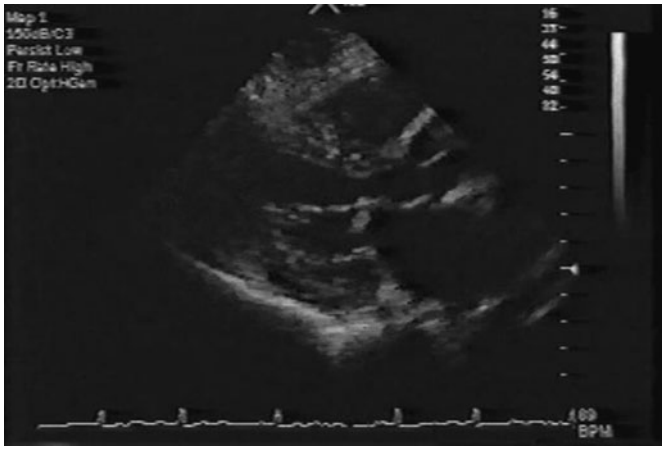


Figure 16.5. Parasternal long-axis view of the heart showing mitral stenosis.

consequences. When it coexists with other causes of shock, it complicates hemodynamic management. For example, tachycardia has an adverse effect in patients with MS, so the prompt identification of severe MS allows the intensivist to intervene to reduce heart rate. Most patients with MS arrive in the ICU with the diagnosis already established. However, in others, it will be unknown at time of presentation. Most cases are caused by rheumatic heart disease, although severe mitral annular calcification may also cause the condition. In addition to the typical immobility of the valve tips ("hockey stick configuration") (Figure 16.5, and Video 16.5 in enclosed DVD), 2D imaging may demonstrate calcified leaflets and subvalvular structures, immobility of the posterior MV leaflet, and "fish mouth" valve orifice in short axis. Planimetry of the MV area is possible from the parasternal short-axis view. Heavy calcification and poor gain settings may result in inaccurate planimetry results. Significant MS results in reduced E-F slope on M-mode.

Definitive identification of severe MS requires Doppler measurements. Severity of MS may be estimated from the mean diastolic pressure gradient across the valve using CW Doppler from an apical view. This Doppler envelope is also used to measure the PHT of the diastolic inflow to apply the following formula:

$$\text{Mitral valve area (MVA)cm}^2 = 220/\text{PHT}$$

Pressure half time is load dependent. It may be prolonged by factors independent of the MV area such as abnormal LV relaxation, decreased LV compliance, elevated LV diastolic pressures, coexisting mitral regurgitation (MR), or low cardiac output. The MVA may also be measured using the continuity principle and PISA. Atrial fibrillation is common with MS. This complicates quantitative measurements of MS using Doppler techniques because 5–10 cardiac cycles must be measured, making the process very laborious.

Commonly accepted values indicating severe MS are as follows[14]:

1. Resting mean pressure gradient >10 mm Hg
2. Mitral valve area <1.0 cm^2
3. PHT >220 m/sec

MITRAL VALVE: MITRAL REGURGITATION

Mitral regurgitation is common in the ICU. Functional MR refers to MR that is caused by LV remodeling without structural abnormalities of the valve itself. Conceptually, the annulus of the valve is enlarged so that valve closure is incomplete. On 2D echocardiography, the valve leaflets appear normal. The MR is only detectable by Doppler analysis. Structural abnormalities of the valve apparatus may also cause MR. These include MV prolapse, rheumatic heart disease, mitral annular calcification, endocarditis, flail leaflet, ischemic papillary muscle dysfunction, and ruptured chordae. Acute failure of the valve apparatus may cause life-threatening cardiopulmonary failure, while chronic severe MR may complicate hemodynamic management of the critically ill. The advanced critical care echocardiographer should be able to perform a comprehensive evaluation of MV function to identify patients with severe MR.

The 2D examination serves to identify structural abnormalities associated with MR. The basic-level critical care echocardiogrpaher may be able to identify flail chordae (Figure 16.6, and Video 16.6 in enclosed

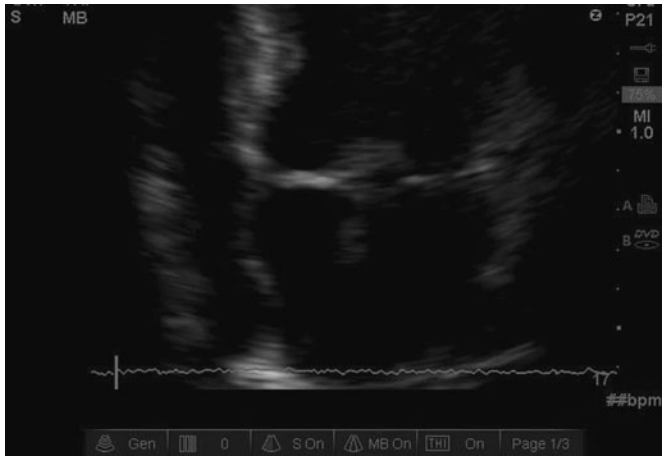


Figure 16.6. Apical four-chamber view of the heart showing a flail mitral valve chordae.

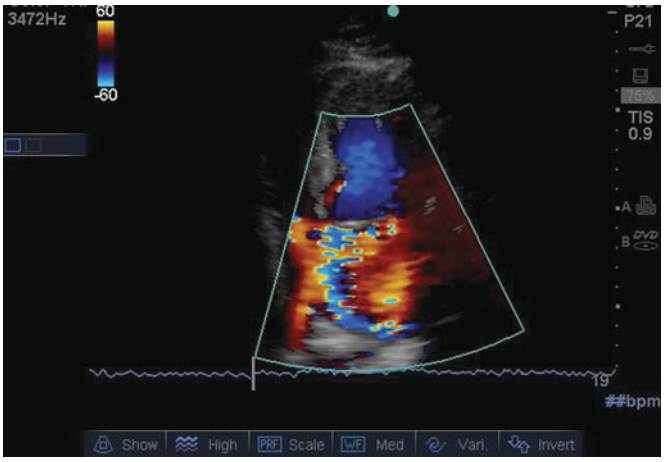


Figure 16.7. Apical four-chamber view of the heart. Color Doppler examination shows severe mitral regurgitation (color Doppler of Figure 16.5).

DVD), noncoapting valves, or valve destruction by endocarditis, but Doppler analysis is required for a definitive assessment of severity. Color Doppler is a practical means of assessing severity of MR. The area of the color-flow jet of MR relative to the left atrial (LA) size is a simple means of assessing severity of MR (Figure 16.7, and Video 16.7 in enclosed DVD).

However, as discussed previously, the assessment of MR by color Doppler has limitations that may not be obvious to the basic-level echocardiographer. Spectral Doppler may demonstrate characteristics suggesting severe MR. The CW Doppler MR jet has increased intensity with severe MR. Severe MR may cause reversal of systolic flow in the pulmonary vein. The width of the vena contracta is also an index of severity, as a width >7 mm is associated with severe MR. When vena contracta measurement and Doppler color-flow signal yield ambiguous results, the continuity principle or PISA allow quantitative measurement of MR. The PISA method is effective for a central LA jet, while the continuity method is required for an eccentric jet.

Commonly accepted values indicating severe MR are as follows[6]:

1. 2D evidence of severe MV apparatus failure
2. Effective regurgitant orifice >0.40 cm^2
3. Regurgitant volume >60 mL
4. Regurgitant fraction >50%
5. Vena contracta >7 mm
6. Pulmonary vein systolic flow reversal
7. MR color Doppler jet >10 cm^2 or occupying >40% of the LA area

Mitral regurgitation is load dependent. For example, a patient with hypertension in association with cardiogenic pulmonary edema may have severe MR on initial echocardiographic examination. Following treatment, when the patient is normotensive and improved from a clinical point of view, the MR may be much improved. Mitral regurgitation in association with cardiac chamber enlargement may show improvement following diuresis, as the mitral annulus is smaller in size, so the MV leaflets can coapt more effectively.

THE ROLE OF TEE IN EVALUATION OF VALVE FUNCTION

Transthoracic echocardiography is very effective in evaluating valve function in the ICU. However, if image quality is poor, TEE may be necessary to fully evaluate valve function. Transesophageal echocardiography is particularly effective for imaging the MV and performing Doppler evaluation of the MV. The AV is also well situated for 2D imaging and color Doppler evaluation with TEE, but is not well oriented for use of spectral Doppler. The PV and TV are often not well seen for 2D imaging, and reliable Doppler analysis of these valves is difficult with TEE. Transesophageal echocardiography has special utility in the evaluation of prosthetic valve function, as discussed below.

EVALUATION OF PROSTHETIC VALVE FUNCTION

Evaluation of prosthetic valve function is a demanding part of echocardiography. Without special commitment that includes performance of a high volume of prosthetic valve cases, it is unlikely that the intensivist will be able maintain a high degree of competence in the field. For a definitive evaluation of prosthetic valve function, the intensivist will serve patients well by requesting cardiology consultation. If there is a delay in performing the definitive study, the intensivist should perform a screening echocardiogram, particularly if the patient is hemodynamically unstable. Generally, the evaluation of a mechanical prosthetic AV and MV requires TEE for adequate visualization. The AV may be difficult to completely visualize with TTE, if a mechanical valve is in the aortic position. A mechanical valve in the aortic position frequently blocks the view of the LA and MV when using the parasternal approach, while an MV prosthesis will block adequate visualization of the LA from both the parasternal and apical approaches. In this situation, the use of TEE will allow better

visualization of posterior structures. The screening 2D examination includes a search for valve instability, thrombus, vegetation, and paravalvular abscess or regurgitation. If visualized by a screening study, they may require urgent intervention.

EVALUATION FOR INFECTIVE ENDOCARDITIS

The evaluation of valvular function by the critical care echocardiographer includes assessment for infective endocarditis. Typical echocardiographic findings of infective endocarditis are as follows[15]:

1. The presence of an oscillating intracardiac mass on a valve or valve support structure, on a foreign device in the heart, or in the path of a regurgitant jet
2. New valvular regurgitation
3. New dehiscence of a mechanical valve particularly
4. Cardiac abscess

Typically, infected vegetations occur on the upstream side of the valve. TV vegetations are generally larger that left-sided lesions. Pulmonic valve endocarditis is rare. It has been associated with the use of the pulmonary artery catheter. The intensivist with basic-level training may be able to recognize obvious vegetations (Figures 16.8, 16.9, and 16.10, and Videos 16.8, 16.9, and 16.10 in enclosed DVD).

However, smaller vegetations may be difficult to recognize, other valves may be affected to a subtle extent, and there may be significant failure of valve or perivalvular function that will not be apparent to the basic-level echocardiographer. If the intensivist with basic skill identifies vegetations, it is advisable to proceed with a comprehensive study. Clinical suspicion combined with nondiagnostic screening studies mandates a comprehensive examination by an echocardiographer with advanced training. In general, echocardiography to evaluate for the possibility of native valve endocarditis should be performed by a skilled echocardiographer because the findings may be subtle. Fully trained echocardiographers with a background in TEE are qualified for this level of evaluation. Evaluation for prosthetic valve endocarditis presents a difficult challenge. In the case of a nondiagnostic study of a prosthetic valve, the intensivist should seek consultation with a cardiology echocardiographer with specific experience in the field.

Findings consistent with endocarditis are a major criteria for the diagnosis of the disease; however, the diagnosis depends on the clinical context. The patient with positive blood cultures for *S. aureus* and a

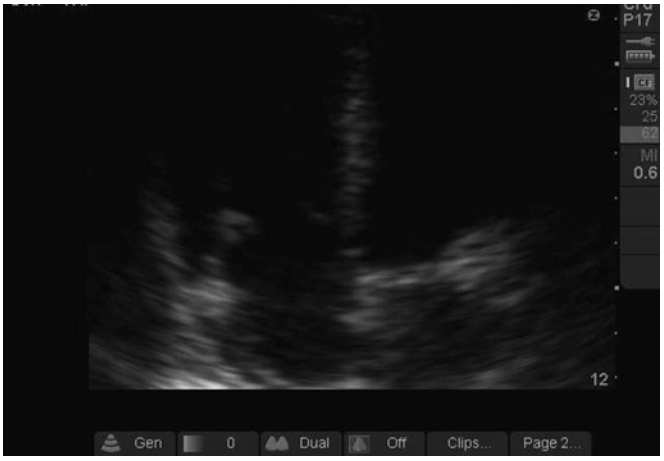


Figure 16.9. Apical four-chamber view of the heart showing tricuspid valve endocarditis.

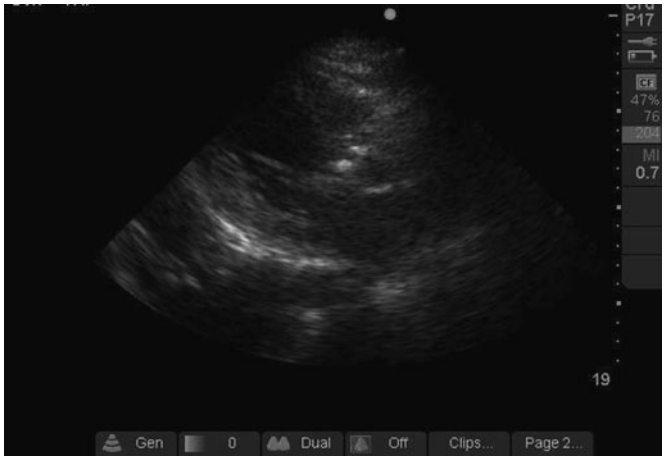


Figure 16.8. Parasternal long-axis view of the heart showing aortic valve endocarditis.

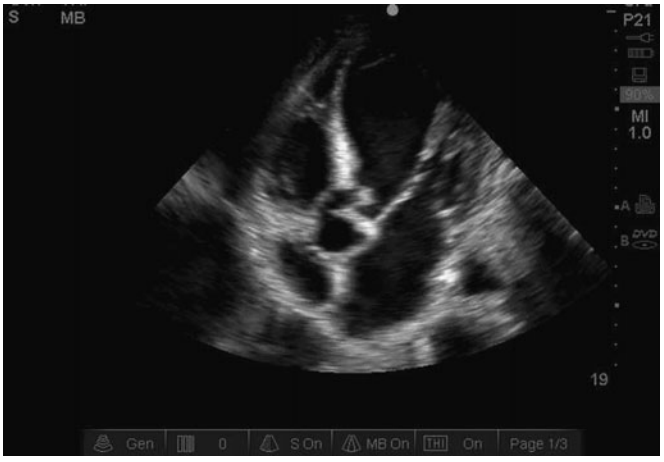


Figure 16.10. Apical four-chamber view of the heart showing aortic valve endocarditis.

large vegetation on the MV is not subtle. However, the difficulty arises when the pretest probability is low, yet there are findings consistent with endocarditis on echocardiography, particularly if the findings are minimal. This may occur when an abnormality that might be consistent with endocarditis is detected as an incidental finding in a patient with critical illness and other explanation for signs of infection.

False-positive findings for endocarditis include the following:

1. Persistent abnormality of the valve from previously treated infective endocarditis such as a persistent vegetation or scarring of the valve. One hint that the process is no longer active is that, as the vegetation "ages," it may become more echo dense, or even calcify. The presence of a vegetation does not necessarily mean that there is an active infection at that site. Clinical correlation is required.
2. Nonbacterial thrombotic endocarditis. Platelet fibrin deposition on damaged valve endothelium may cause nonbacterial thrombotic vegetations that have the appearance of infective endocarditis on echocardiography. The basal portion of the MV is most often affected, but the process may extend to the chordae and papillary muscles. The cause is unknown, but the disease is associated with antiphospholipid syndrome, systemic lupus erythematosus (SLE), and malignancy (marantic endocarditis).
3. Other mimickers. Lambl's excresences are an incidental finding that may resemble small vegetations. They are idiopathic linear mobile structures that are found most commonly on the upstream side of the aortic valve. Papillary fibromelastoma is a small benign tumor that occurs on either surface of the AV or MV and occasionally on the other valves or endomyocardial surface. They may have a "sea anemone" appearance with mobile fingerlike projections. Rarely, metastatic tumors may adhere to heart valves, and mimic infective endocarditis. Sterile thrombus may adhere to intracardiac devices such as pacemaker wires or on the endomyocardium of the RA or RV, and have the appearance of infected vegetation.

It is not always possible to distinguish an infected vegetation from one of these abnormalities based on the morphologic pattern alone. Combining clinical assessment with the echocardiographic results is essential for establishing the diagnosis of infective endocarditis. Formal criteria for the diagnosis of infective endocarditis are available.[15] Echocardiography plays a major role in establishing the diagnosis, but other criteria are also important.

Good image quality is particularly important when the echocardiographic examination is being performed for possible infective endocarditis. Patients in the ICU may have poor image quality due to obesity, edema, chest wall wounds or dressings, subcutaneous air, and hyperinflation due to mechanical ventilator support degrade TTE image quality. Transesophageal echocardiography has superior resolution when compared to TTE. Transesophageal echocardiography is more sensitive than TTE in detecting vegetations and the complications of infective endocarditis such as involvement of periaortic structures, annular abscess, or disruption of the MV apparatus.

When should the intensivist use TEE in evaluation for endocarditis? A reasonable approach is to use TEE when the TTE is nondiagnostic and the clinical suspicion of infective endocarditis is high. Suspicion of infective endocarditis involving a prosthetic valve is a strong indication for TEE. Echocardiography of prosthetic valve endocarditis may require an echocardiographer with specific expertise in that application.

Echocardiography has major application in the identification and stratification of complications of endocarditis. Echocardiography is the primary imaging modality used to identify anatomic complications of infective endocarditis. These include valve leaflet failure, perforation of valve leaflet or adjacent structure, abscess, aneurysm, fistula, and prosthetic valve dehiscence, all of which may lead to life-threatening hemodynamic failure. Serial echocardiography may demonstrate progression of anatomic complications predictive of catastrophic valve failure. The echocardiographer should work in close cooperation with the cardiac surgeon to determine the timing and type of surgical intervention that may be required for progressive valve failure.

Embolic events are another major complication of infective endocarditis. Echocardiography is useful in determining risk of embolism because the risk is largely determined by the size of the vegetation. Vegetations >10 mm in size, have substantial risk of embolism.

The intensivist with proficiency in echocardiography provides an important role in the diagnosis and management of infectious diseases involving the heart. Consultation should be sought for those with less experience or if questions remain. Echocardiography for prosthetic valve endocarditis often requires an echocardiography performed by a cardiologist with specific expertise in that application, and will generally require TEE.

CONCLUSION

For the intensivist, echocardiographic assessment of valve function is useful to answer three key questions:

1. Is there valvular failure that is catastrophic and that requires urgent surgical consultation?
2. Is there severe but noncatastrophic valve failure that requires specific medical therapy or that will complicate management of coexisting critical illness?
3. Is there mild or moderate valve dysfunction that is only incidental to the primary critical illness?

The intensivist with basic critical care echocardiography skills can identify valvular abnormalities on 2D imaging, particularly if they are severe, but otherwise has limited capability to assess valvular function. The use of color Doppler to assess the severity of valvular abnormality is sufficiently challenging that the basic-level echocardiographer may need consultation to assist with diagnosis. The basic-level echocardiographer may screen for major valve failure, but will need a definitive study if the clinical situation suggests the possibility of significant valve failure. The intensivist with advanced critical care echocardiography skills is able to fully evaluate valve function using 2D examination and Doppler measurements in a manner identical to cardiology-based echocardiography. The identification and quantification of severe valvular stenosis or regurgitation is especially important, as it may have a major influence on the management of the patient with hemodynamic failure.

References

1. Quinones MA, Douglas PS, Foster E, et al. ACC/AHA Clinical Competence Statement on Echocardiography. *J Am Coll Cardiol*. 2003;41:687–708.
2. Cape EG, Yoganathan AP, Weyman AE, Levine RA. Adjacent solid boundaries alter the size of regurgitant jets on Doppler color flow maps. *J Am Coll Cardiol*. 1991;17:1094–1102.
3. Zoghbi WA, Enriquez-Sarano M, Foster E, et al. American Society of Echocardiography. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. *J Am Soc Echocardiogr*. 2003;16:777–802.
4. Klein AL, Burstow DJ, Tajik AJ, et al. Age-related prevalence of valvular regurgitation in normal subjects: a comprehensive color flow examination of 118 volunteers. *J Am Soc Echocardiogr*. 1990;3:54–63.
5. Cormier B, Iung B, Porte JM, Barbant S, Vahanian A. Value of multiplane transesophageal echocardiography in determining aortic valve area in aortic stenosis. *Am J Cardiol.* 1996;77:882–885.
6. Currie PJ, Seward JB, Reeder GS, et al. Continuous-wave Doppler echocardiographic assessment of severity of calcific aortic stenosis: a simultaneous Doppler-catheter correlative study in 100 patients. *Circulation*. 1985;71:1162–1169.
7. Zoghbi WA, Farmer KL, Soto JG, Nelson JG, Quinones MA. Accurate noninvasive quantification of stenotic aortic valve area by Doppler echocardiography. *Circulation.* 1986;73:452–459.
8. Otto CM. Valvular stenosis. In: Otto CM, ed. *Textbook of Clinical Echocardiography*. 3rd ed. Philadelphia, Pa: WB Saunders Co; 2004:287.
9. Oh JK, Seward JB, Tajik JA. Valvular heart disease. In: Oh JK, Seward JB, Tajik JA, eds. *The Echo Manual*. 3rd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2007:191.
10. deFilippi CR, Willett DL, Brickner ME, et al. Usefulness of dobutamine echocardiography in distinguishing severe from nonsevere valvular aortic stenosis in patients with depressed left ventricular function and low transvalvular gradients. *Am J Cardiol*. 1995;75:191–194.
11. Roberts BJ, Grayburn PA. Color flow imaging of the vena contracta in mitral regurgitation: technical considerations. *J Am Soc Echocardiogr*. 2003;16:1002–1006.
12. Takenaka K, Dabestani A, Gardin JM, et al. A simple Doppler echocardiographic method for estimating severity of aortic regurgitation. *Am J Cardiol.* 1986;57:1340–1343.
13. Tribouilloy CM, Enriquez-Sarano M, Fett SL, Bailey KR, Seward JB, Tajik AJ. Application of the proximal flow convergence method to calculate the effective regurgitant orifice area in aortic regurgitation. *J Am Coll Cardiol.* 1998;32:1032–1039.
14. Oh JK, Seward JB, Tajik JA. Valvular heart disease. In: Oh JK, Seward JB, Tajik JA, eds. *The Echo Manual.* 3rd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2007:204.
15. Li JS, Sexton DJ, Mick N, et al. Proposed modifications to the Duke criteria for the diagnosis of infective endocarditis. *Clin Infect Dis.* 2000;30:633–638.

This page intentionally left blank

CHAPTER 17

Echocardiographic Features of Adult Congenital Heart Disease

Renée J. Roberts and Anthony D. Slonim

INTRODUCTION

Congenital heart disease (CHD) is defined as a gross structural abnormality of the heart, great arteries, or great veins that is present at birth.[1] Of all the congenital anomalies, cardiovascular anomalies are the most common, comprising 30% of the total congenital disease.[2] Although congenital cardiac malformations are relatively uncommon, the total number of adolescent and adult patients surviving into adulthood is growing considerably, with almost one million people >20 years with CHD in the United States (US).[3–6] Approximately 20,000 open-congenital heart operations are performed annually in the US, and >85% of these infants can now expect to reach adulthood due to advances in diagnostics, surgical techniques, intensive care, and interventional devices.[1,6–10] The importance of recognizing heart disease in the critically ill adult lies not so much on the potential for possible surgical or nonsurgical intervention but mostly on management of the extracardiac manifestations of long-standing shunting, ventricular hypertrophy, erythrocytosis, or cyanosis and their potential relationship to the presenting condition in the intensive care unit (ICU) as a cerebrovascular accident, manifestation of pulmonary hypertension, or cardiac decompensation from endocarditis.[7] Furthermore, as the CHD adult ages, the superimposition of other medical conditions (hypertension, coronary artery disease, and diabetes) further complicates the management.

This chapter will focus on the most common undiagnosed lesions in symptomatic adults and adults with diagnosed disease that is corrected.[11–14] Here, the focus will be on the basic anatomy, associated cardiac anomalies, hallmarks of diagnosis, and brief sequelae of each lesion. Consideration is given to the echocardiographic findings in the primary diagnosis of defects commonly presenting in adult life, with a brief discussion of follow-up diagnosis and typical echo findings of simple corrected lesions.

ECHOCARDIOGRAPHIC EVALUATION OF CONGENITAL CARDIAC DISEASE IN THE ADULT PATIENT

Echocardiography is the mainstay of diagnosis and follow-up for most patients with CHD.[15] Echocardiography should be performed using a standardized approach, with an understanding of the underlying anatomy (congenital or postsurgical) and the most likely residual or acquired lesions. This approach prevents the misinterpretation of the findings, poor image formation, or incorrect technique.[9]

Congenital cardiac lesions can be classified in several ways, all of which aid in understanding the physiology.[5,7,10,14] The first classification schema is by anatomic location, which is helpful when performing a screening echo (Table 17.1). The second classification schema is to group the lesions by physiology, which is helpful when doing Doppler studies (Table 17.1). Finally, they can be classified by presentation (Table 17.1).

The echocardiographic approach to CHD lesions is the basic segmental exam, a systematic and sequential approach.[16] The first step in the exam is to determine the basic anatomical relationships of the great arteries and the heart chambers. An L-transposition has occurred if the great arteries arise from the wrong ventricles (ventricular arterial discordance) and if the atria and valves are also switched (atrioventricular discordance). This condition is compatible with normal health into adulthood.[17,18] Although this lesion is rare and will not be discussed further, it is very easy to discern and is the very important first step of the CHD screening exam. The rest of the exam involves evaluating ventricular size and function, atrial size, valvular function, flows in the great arteries, and ventricular peak systolic pressures.[11] The simplified segmental examination for CHD and the lesions discussed in this

TABLE 17.1. Classification of congenital heart disease lesions

Anatomic classification	
Atrial level:	Atrial septal defect, patent foramen ovale
Ventricular level:	Ventricular septal defect
Aortopulmonary level:	Patent ductus arteriosus
Left heart malformation:	Bicuspid aorta, l-transposition of the great arteries
Right heart malformation:	Tetraology of Fallot
Physiologic classification	
Acyanotic with left to right shunt:	Atrial septal defect, ventricular septal defect, patent ductus arteriosus
Acyanotic without a shunt:	Atrioventricular septal defect, l-transposition of the great arteries, bicuspid aorta
Cyanotic:	Tetraology of Fallot, l-transposition of the great arteries, Eisenmenger's complex
Classification by presentation	
Asymptomatic with a murmur:	Atrial septal defect, small ventricular septal defect, bicuspid aorta
Symptomatic with a murmur:	Bicuspid aorta, atrial septal defect, large ventricular septal defect, tetraology of Fallot

TABLE 17.2. Lesions as diagnosed by segmental echocardiographic exam

Systemic circuit		
Atrium	morphology, pulmonary venous connections, atrial septum	ASD
AV connection		AVSD
Ventricle	morphology, size and function, septum	VSD
VA connection	aortic valve	
Bicuspid, ITGA		
Great artery	aorta	TGA, PDA
Pulmonary circuit		
Atrium	atrial morphology, coronary sinus	
AV connection		
Ventricle	morphology, size and function, outflow tract	ITGA, TOF
VA connection	pulmonary valve	TOF
Great artery	pulmonary arteries	TOF

ASD indicates atrial septal defect; AV, atrioventricular (AV connection: the tricuspid and mitral valve); AVSD, atrioventricular septal defect; PDA, patent ductus arteriosus; TGA, transposition of the great arteries; TOF, tetralogy of Fallot; VA, ventricular arterial (VA connection: the aorta and pulmonary artery [PA]); VSD, ventricular septal defect.

chapter are presented in Table 17.2. Transthoracic echocardiography (TTE) is the most widely used primary imaging technique for characterizing simple and complex structural cardiac defects.[9,19] It is noninvasive and universally available, and can provide detailed and quantifiable information on intracardiac morphology and function, valve gradients, pulmonary artery (PA) pressure, chamber hypertrophy, and enlargement at the bedside of the critically ill patient.[9,12] However, in adult patients who often have poorer transthoracic windows because of body size, chest deformity, or previous median sternotomy, image quality may be limited. Transesophageal echocardiography (TEE) is an excellent alternative when any of these conditions exist.[9] The strengths of TEE, in conjunction with Doppler, are improved visualization of posterior structures (especially the atria and atrioventricular (AV) valves, atrial septum, and the mitral valve), improved localization of the pulmonary veins, and improved evaluation of the branch pulmonary arteries for stenosis, septal defects, and the presence of a patent foramen ovale (PFO).[8,12,19] In contrast, information regarding the ventricular chambers, right ventricular outflow tract (RVOT), and pulmonary arteries is impaired with TEE.[9]

PATENT FORAMEN OVALE

GENERAL: The most common atrial communication is a PFO.[7]

INCIDENCE: Anatomic obliteration of the foramen ovale ordinarily follows its functional closure soon after birth, but a residual mobile flap of tissue that is "probe patent" upon autopsy is a normal variant in approximately 25% of people; atrial septal defect (ASD) denotes a true deficiency of the atrial septum and implies functional and anatomic patency.[10]

PRESENTATION: Most people with a PFO are asymptomatic. However, several well-described clinical associations include patients <5 years of age with cryptogenic stroke (prevalence 50%), peripheral systemic emboli, or decompression sickness during a dive.[8,15,20]

ANATOMY: The PFO is a small hole in the atrial septum that is used during fetal circulation. Normally, the foramen ovale closes at birth when increased blood pressure on the left side of the heart forces the opening to close. When persistent, it becomes a PFO, allowing blood to flow from the heart's right atrium (RA) to the left atrium (LA), and vice versa.[21]

PATHOPHYSIOLOGY: The PFO works like a flap valve opening during conditions with increased intrathoracic pressure (Figure 17.1).[21] This increased pressure or shunting occurs when people strain during a bowel movement, cough, or sneeze (transient Valsalva maneuver).[21] A PFO can also be associated with atrial septal aneurysms, which are characterized by excessive mobility of the atrial septum.[21] If the pressure is great enough, clot or particles in the blood can cause paradoxic emboli to the brain, causing a cerebrovascular event, or into a coronary artery, causing myocardial ischemia.[1]

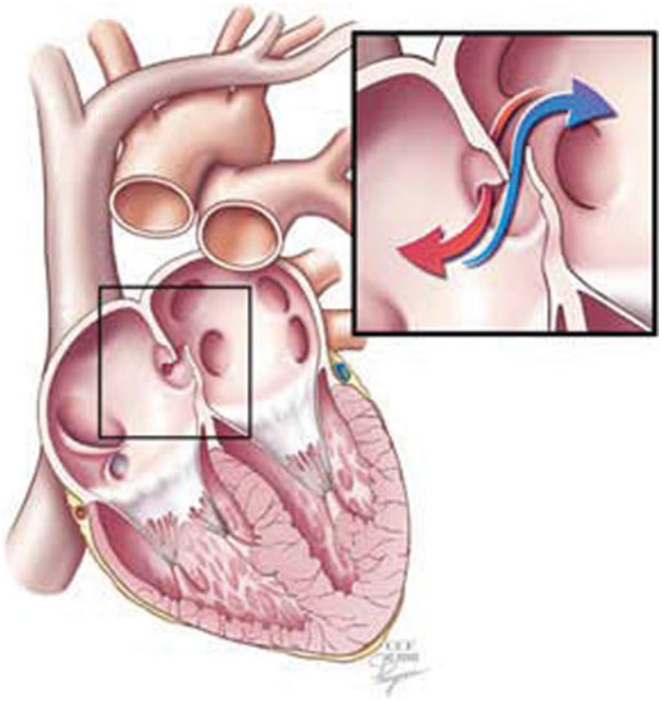


Figure 17.1. Depiction of a PFO showing its flaplike characteristics. (From Ref. 21.)

ECHOCARDIOGRAPHY: The presence of a PFO is best assessed by using an echocardiographic "bubble" contrast medium composed of agitated saline to entrain microbubbles of air into solution.[22] While the sensitivity for TTE is 7%, it can be improved to 50% if contrast is used.[15] TEE with contrast is considered the "gold standard" for the diagnosis of PFOs, even small ones, with a sensitivity of 79%.[8] A Valsalva maneuver can be used during the examination to increase pressure in the RA and the flow of blood from the right to LA, thereby improving visualization of the PFO.[21]

VIEW: The optimal view for a PFO is a longitudinal plane of a TEE (Figure 17.2).

FINDINGS: The optimal method to evaluate an PFO was described very well by Seiler.[20] To get the best results, the patient should be in a left lateral position; the Valsalva maneuver should be practiced a few times, then the evaluation should to be started without prior inspiration; and it has to last about 5–10 seconds. The enhanced volume supply to the right atrium during the release phase of the Valsalva maneuver can be supported by injecting echo contrast from the right cubital vein and by lifting this arm at the end of the maneuver. The Valsalva maneuver should lead to a visual change in the convexity of the fossa ovalis membrane from right to left. Contrast bubbles in the LA occur within three to four heartbeats after release of the Valsalva maneuver; by TEE, it can be recognized that the structure of the bubbles is altered when they cross the lung (smaller, and less echo dense). The washout of contrast bubbles by flow from the inferior vena cava (IVC) streaming alongside a prominent eustachian valve can be so dynamic that they do not reach the PFO. In such cases, a higher contrast volume (5 mL instead of 2 mL) can be administered to overcome the problem, or the contrast injection should be performed from a vein in the foot. Finally, the size of the PFO is estimated using a score of 0–3, with a score of 1 representing the crossover of a few single bubbles, and a score of 3 representing the shunt of an entire cloud of bubbles (score 2 between 1 and 3).

FOLLOW-UP: If asymptomatic, patients with a PFO do not require treatment.[21] Patients who have had a stroke or transient ischemic attack (TIA) may be placed on anticoagulants or

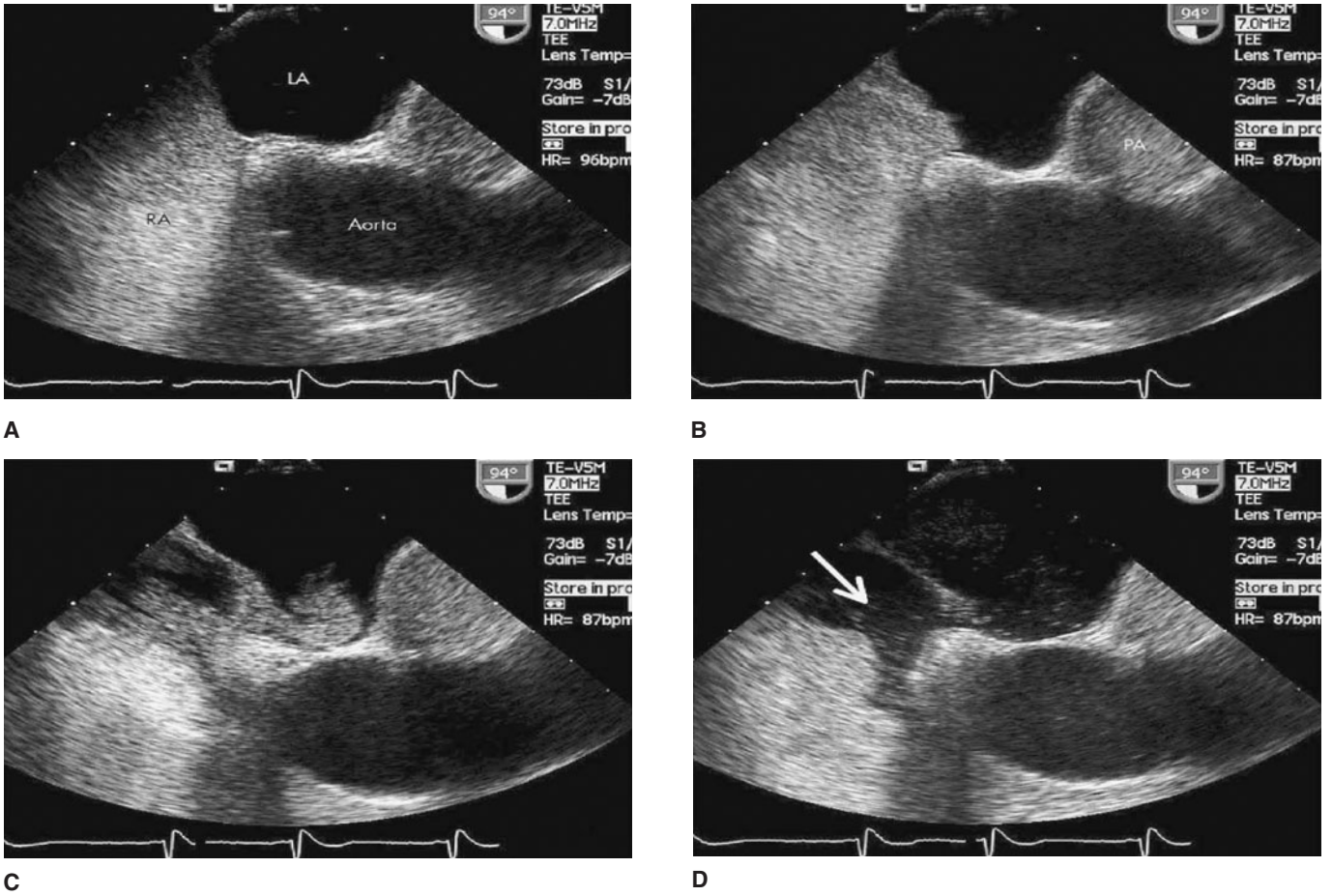


Figure 17.2. Transesophageal contrast echocardiography (TEE) for the detection of patent foramen ovale (PFO). (A) Transesophageal echocardiography long-axis view (right side—cranial; left side—caudal) showing the left atrium (LA) and the aortic root free of ultrasound contrast medium as well as the right atrium (RA) filled with contrast bubbles. The image is taken close to the end of the Valsalva strain phase with the interatrial septum bulged toward the RA. (B) Identical TEE image plane as in all other panels (long-axis view), taken immediately after release of the Valsalva strain phase: the interatrial septum (fossa ovalis region) now bulges toward the LA, thus indicating a pressure rise in the RA above that in the LA (PA, PA). (C) Long-axis view image obtained instantaneously after that in panel B revealing a shunt of contrast medium across a PFO from the right to the left atrium. (D) The shunt is much less pronounced on this next image (PFO grade 3). Washout of contrast medium in the RA is visible (arrow), which is caused by the inflow of contrast-free blood from the inferior vena cava. (From Refs. 7, 14, 20.)

recommended for device closure to prevent recurrence.[21]

ATRIAL SEPTAL DEFECT

GENERAL: An ASD is a direct communication or "hole" between the atrial chambers.[1] The defects vary in size from the smallest fenestrated ASD (a few millimeters) to the largest defect of an absent atrial septum with a common atrium. The variation in location determines the type of ASD (Figure 17.3).

INCIDENCE: Atrial septal defects are among the most common forms of CHD in adults, accounting for approximately one third of CHD cases, with men affected twice as often as women.[1,5,22]

PRESENTATION: Atrial septal defects are detected regularly as incidental findings on echocardiography undertaken for other reasons.[14,15] An associated congenital defect may be seen in up to 30% of cases.[1] The hallmark of an ASD is fixed splitting of the second heart sound with breathing and incomplete right bundle branch block (RBBB) on electrocardiogram (EKG).[14]

ANATOMY: The names and locations of the ASDs are related to events and structures in embryological development.[23] The first atrial septum (septum primum) arises from the endocardial

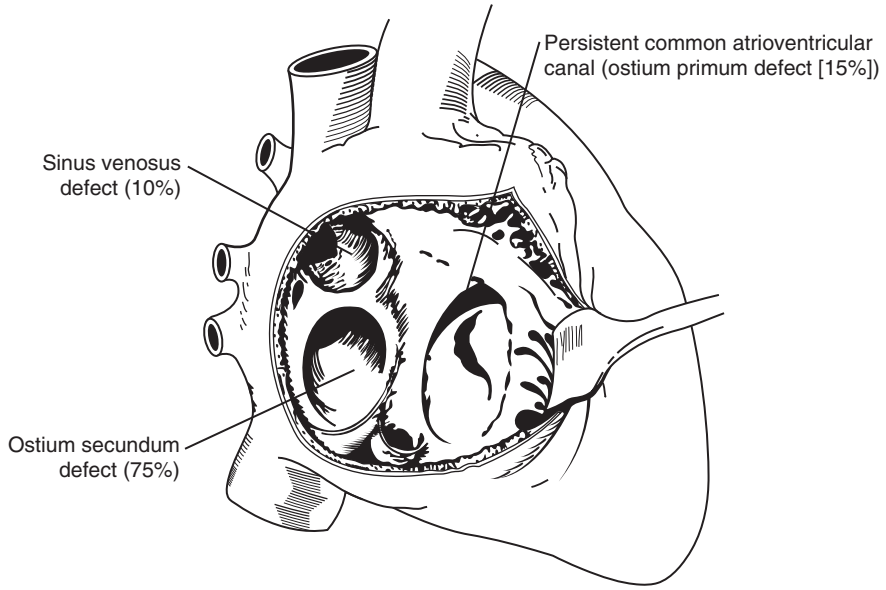


Figure 17.3. Anatomic location of atrial septal defects. (From Ref. 7.)

cushion and migrates inferiorly starting at the atrioventricular valves[8,23] Failure of migration leads to a constellation of ASD-primum defects: atrioventricular septal defect (AVSD), endocardial cushion defect, or atrioventricular canal (AVC).[23] A second septum migrates inferiorly down the right side of the first, and normally completely covers the right side of the atrial septum except for an "ovale," which, if it remains unfused, becomes a PFO. If this growth is aborted, the hole in the middle of the atria is an ASD secundum.[8] A third form of ASD is the sinus venosus defect; a hole arises from failure of the embryonic superior vena cava (SVC) to properly merge with the atria.[8] Rarer forms of ASD exist, including an "inferior" sinus venosus defect associated with failure of the IVC to merge with the atria and a coronary sinus ASD that is basically an unroofed coronary sinus.[7,8] Fifteen to 20% of ASDs are the primum type and are associated with a common AV valve (atrial and ventricular septae do not fuse, leaving a confluent defect with a common valve ring).[15,22] Secundum ASDs account for approximately 75% of all ASDs, and two thirds of these present late in life.[1,8,11] Two percent of secundum ASDs are associated with partial anomalous pulmonary venous (connection of the right upper pulmonary veins to the right atrium) and mitral valve prolapse.[1,22]

PATHOPHYSIOLOGY: Normally, oxygenated blood from the higher-pressure LA passes into the RA, increasing right ventricular (RV) output and pulmonary blood flow.[8] A small defect (<0.5 cm in diameter) is associated with a small shunt and no hemodynamic sequelae.[22] A sizable defect (>2 cm in diameter) may be associated with a large shunt, with substantial hemodynamic consequences.[22] In most adults with ASDs, the RV is more compliant than the left ventricle (LV); as a result, LA blood is shunted to the RA, causing increased pulmonary blood flow and dilation of the atria, RV, and pulmonary arteries.[22] Symptoms usually develop when the pulmonary-to-systemic shunt is >5:1. Atrial septal defects are usually asymptomatic and not accompanied by striking physical examination abnormalities; therefore, they often remain undetected.[22] As the shunt increases, symptoms include fatigue, exertional breathlessness, and palpitations from arrhythmias.[14] With aging, the LV compliance declines and concomitant hypertension and coronary artery disease increase the magnitude of the shunt. This in turn causes RA enlargement, atrial arrhythmias, pulmonary arterial hypertension, and RV failure, which may be the presenting symptoms for the ICU patient with decompensated congestive heart failure (CHF).[7,8,10] Although pulmonary pressures are

modestly elevated, severe pulmonary hypertension resulting in Eisenmenger's physiology occurs in <15% of patients.[7,8] Patients can experience paradoxical right-to-left emboli.[8] Interestingly, paradoxical emboli may be more common in patients with an PFO than an ASD.[8]

ECHOCARDIOGRAPHY: An ASD should be suspected on echocardiography if there is evidence of RA enlargement or RV volume overload demonstrated by a flattened septal motion during systole.[16] On M-mode it will appear as paradoxical (anterior) septal motion.[10,15]

VIEW: Although an apical, right parasternal or subcostal four-chamber view can be used to demonstrate the atrial septum, the subcostal view is best because the intraatrial septum is perpendicular to the ultrasound beam and provides optimal images of the gap in the septum.[10,15] It is the ideal view to distinguish among primum, secundum, and sinus venosus ASDs.[16] If the ultrasound beam is almost parallel to the atrial septum, dropout of the signal can be misinterpreted as a defect, especially in the apical and parasternal views.[11] The transducer can be tilted anteriorly to demonstrate the high septum at its junction with the SVC and to check for a sinus venosus defect if there is no other apparent ASD.[16]

FINDINGS: Echocardiography demonstrates the location and size of the defect and the direction of shunting.[1] Ostium primum (Figure 17.4) or secundum (Figure 17.5) defects are often visualized directly with TTE, but sinus venosus defects (Figure 17.6) are elusive and may be better visualized on TEE.[8,16,22] When identified, the defect's size should be measured and the rim of atrial tissue should be assessed.[15] The sensitivity of echocardiography may be enhanced by bubble contrast, which will move across the defect into the LA.[22] Because the difference in atrial pressures is small, the Doppler sample velocity should be aligned perpendicular to the atrial septum in the subcostal view, so that the low velocity jet can be recorded.[16] Bidirectional flow and smaller defects can be confirmed with color-flow and pulsed Doppler.[8,15,17] Care should be taken not to mistake the signal of flow into the RA from the SVC or tricuspid regurgitation for ASD flow.[15] A TEE is helpful when TTE quality is suboptimal; it improves the sensitivity (up to 100%) for small shunts and provides a more accurate assessment of the size and location of atrial communications.[7,16] In particular, TEE and Doppler color-flow echocardiography are useful in identifying sinus venosus defects and anomalous pulmonary venous drainage.[7,22] Both venae cavae can easily be seen entering the RA with superior and inferior

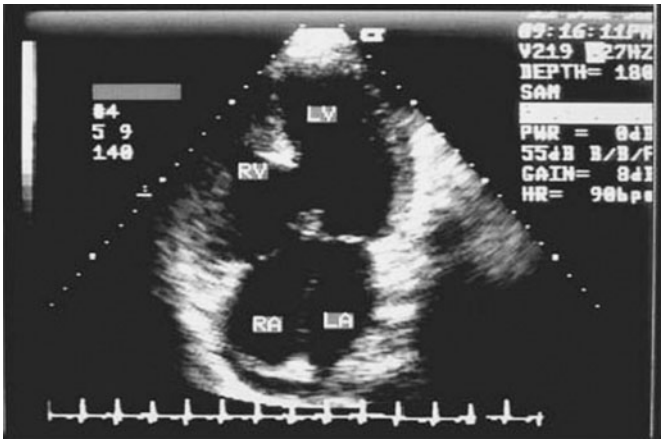
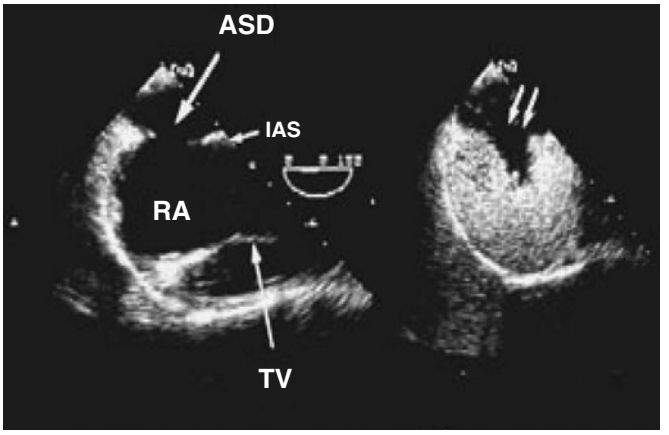
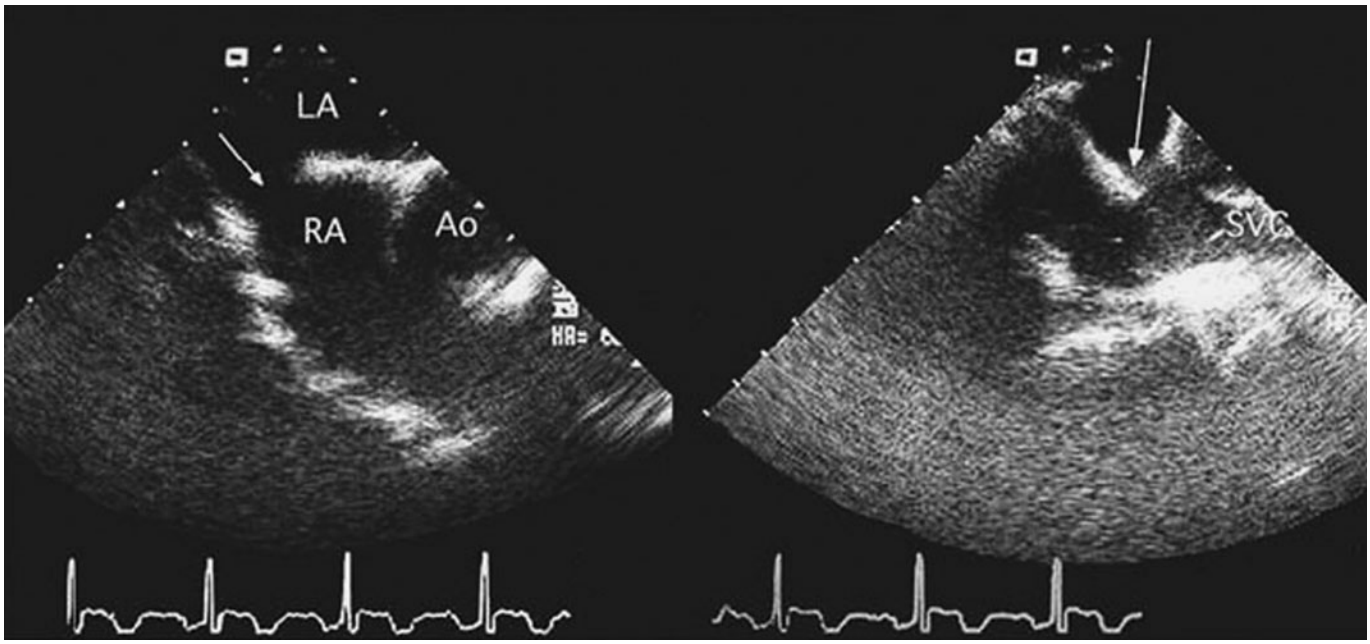


Figure 17.4. Apical four-chamber view from a transthoracic echocardiogram in a patient with atrioventricular septal defect and Down syndrome. The crux of the heart is missing, and a large ostium primum atrial septal defect and large ventricular septal defect are evident. LA indicates left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle. (From Ref. 7.)



A B

Figure 17.5. Transesophageal echocardiogram of a patient with an ostium secundum atrial septal defect (ASD). (A) The image clearly demonstrates the position of the ASD in the midportion of the interatrial septum (IAS). (B) The image is obtained after intravenous injection of agitated saline, which opacifies the right atrium (RA). The negative contrast effect produced by the unopacified left atrial blood entering the RA is clearly demonstrated (double arrow). TV indicates tricuspid valve. (From Ref. 7.)



A B

Figure 17.6. Transesophageal echocardiogram of a 50-year-old man with a sinus venosus atrial septal defect. (A) Horizontal view showing the defect (arrow) in the superior portion of the interatrial septum. (B) The defect (arrow) is clearly demonstrated in this longitudinal plane view. Ao indicates aorta; LA, left atrium; RA, right atrium; SVC, superior vena cava. (From Ref. 7.)

scanning in the vertical plane.[24] In the horizontal plane (0°) the entire septum is interrogated, from the SVC/RA superiorly to the coronary sinus/RA junction inferiorly so as to ensure small defects at the septal margins are identified.[17] Rotation of the image plane to the bicaval (biatrial) view (90°) displays the septum in an orthogonal plane, from IVC to SVC, thereby ensuring that inferior and superior sinus venosus ASDs are not missed.[17] Dimensions should be measured in multiple planes to ensure proper sizing.[16]

The partial AVSD or ASD primum includes abnormal development of the AV septal region and valves, all of which must be assessed in detail.[17] The abnormality can be ascertained from an apical four-chamber view where absence of tissue in the inferior atrial septum is diagnostic.[16] The atrioventricular valves are attached to the edge of the ventricular septum in the same plane rather than the normal more apical position of the tricuspid valve.[16] The left AV valve usually has three leaflets (the anterior leaflet divided into superior and inferior parts by a "cleft," which is best viewed in the parasternal short-axis view.[16,17] Both AV leaflets insert into the crest of the ventricular septum, thereby "sealing off" an interventricular connection, often giving the upper septum an aneurysmal appearance.[17] Doppler will show AV valve regurgitation—the mitral valve often has an eccentric posteriorly directed flow.[16] A complete AVSD, which additionally has an inlet ventricular septal defect (VSD), is rare in adults.

Atrial septal defect primum/partial AVSD defects are best visualized on TEE in the horizontal four-chamber view.[24] In the transgastric horizontal plane, the cleft in the "anterior" leaflet may appear as a fold or cleft between the superior and inferior leaflets.[24] Regurgitation is identified by color-flow mapping.[17] This is best seen at 0°, flexing slightly to interrogate the superior part of the anterior leaflet and by scanning through the valve from 0° to 130°, where it is seen lengthways.[17] The chordal arrangement of both AV valves should also be assessed and can be performed by scanning up and down the transverse plane.[17] Short-axis transgastric views of the AV valves are obtained by careful full flexion of the probe at 0°.[17] This may show the position of each leaflet and the site of regurgitation.[17] Partial ASVD where the AV valve attachments diminish the VSD requires a separate interrogation. In common with other unusual or variable findings, this can be done efficiently by keeping the region of interest in the center of the scan sector and performing a careful 180° sweep, first with two-dimensional echo and then color-flow mapping.[17]

OTHER: The size of the left-to-right shunt through the ASD is reflected in the RV dimension, but the appearance is also found with other causes of volume overload, such as anomalous pulmonary venous drainage or tricuspid or pulmonary regurgitation.[7,15] Just as RV pressure can be measured from signal of tricuspid regurgitation, PA pressure can be estimated from the peak velocity of the tricuspid regurgitant jet.[7] Common associations with an ASD should be sought, in particular mitral valve prolapse or an anomalous pulmonary vein.

Care should be taken not to misdiagnose color-flow mapping jets entering the RA from the cavae. Flow may appear to cross the septum when viewed from the subcostal view, where the eustachian valve appears as a septum, but with Doppler, this flow will show respiratory variation opposite that of a real ASD.[11] Venae cavae flow increases with inspirations and decreases with expiration because of changes in intrathoracic pressure.[11] Atrial septal defect demonstrates the opposite findings.[11] Additional findings heighten the certainty of the diagnosis: RV enlargement, D shaped LV, paradoxical septal motion, RA enlargement, flow across the septum, and elevated pulmonary pressures.[11]

ECHOCARDIOGRAPHY: Repaired ASD: After surgical correction, residual shunts and valve regurgitation necessitates long-term follow-up.[15] Even successful AVSD repair is never perfect; residual or progressive mitral regurgitation may be present in addition to residual shunts.[11] Consequently, in follow-up, in addition to the echocardiographic determination of the presence or absence of residual atrial and ventricular level shunts and pulmonary vascular disease, the degree of MR should also be assessed.[11] In addition, subaortic stenosis/ventricular outflow tract obstruction is another potential late complication.[10,14] Repaired ASDs without residual defects and normal physiology and prognosis may present with arrhythmias.[11] However, if there are normal pulmonary pressures on an early exam, later development of pulmonary hypertension is unlikely.[11] For those repaired late in childhood, RV size does not return to normal in a significant proportion of patients.[11]

OTHER IMAGING: Although echocardiography may provide enough information to guide the management of an ASD, catheterization may be required to determine the magnitude and direction of shunting and the presence and severity of pulmonary hypertension.[22] Cardiac catheterization is also often helpful, especially if there are associated anomalous pulmonary veins, associated malformations, or if coronary artery disease is a possibility.[8,10] Both computed tomography (CT) and magnetic resonance imaging (MRI) can also elucidate the ASD and allow for observation of associated lesions.[8]

FOLLOW-UP: Firm guidelines on indications for defect closure are lacking.[14] However, closure should be considered in patients in whom secondary cardiac symptoms occur, right-sided cardiac chamber enlargement is evident, or cardiac catheterization has confirmed the presence of reversible pulmonary hypertension or a large intracardiac shunt (Qp:Qs >2:1).[14,16] A very small defect with minimal left-to-right shunting (characterized by a ratio of pulmonary to systemic flow of <1.5) usually causes no symptoms or hemodynamic abnormalities and is not usually surgically closed; however, many (20–30%) of the ASDs studied enlarge over time.[5,8,22] Despite poorer surgical results in adults >40 years, a late operation often improves the functional class and eliminates the risk of paradox emboli. The incidence of atrial arrhythmias remains and must be monitored and treated.[12]

BICUSPID AORTIC VALVE

GENERAL: Bicuspid aortic valve is the most common anomaly encountered in adults.[14]

INCIDENCE: It is the most common cause (7%) of CHD, accounting for 2–3% of births.[7]

PRESENTATION: Bicuspid aortic valves are the most often diagnosed pathology in patients <65 years of age with symptomatic aortic stenosis (AS).[1,22] Approximately 20% of patients with bicuspid aortic valves have associated cardiovascular abnormalities, such as patent ductus arteriosus (PDAs), aortic coarctation, VSD, or left-dominant coronary circulation.[1,22] The hallmark of bicuspid aortic valve is an ejection click loudest at the apex occurring after the first heart sound.[14]

ANATOMY: The bicuspid valve occurs when the two commissures fuse, resulting in two rather than three valve leaflets, causing an eccentrically oriented orifice.[1,22] The valve is often

dysplastic, with thickening and rolling of the leaflets.[7]

PHYSIOLOGY: Although the deformed valve is not stenotic at birth, it is subjected to abnormal nonlaminar flow and hemodynamic stress, which leads to thickened, fibrotic, and calcified valve leaflets.[1,7,22] Considered a normal variant by some, a bicuspid aortic valve may function normally throughout life or may develop either stenosis or an LV-to-aorta pressure gradient of variable severity.[1,7] In many patients, there is a coexisting abnormality of the medial layer of the aorta above the valve, which predisposes patients to dilatation, dissection, or aneurysm formation of the aortic root.[1,7,22] Left ventricular outflow tract (LVOT) obstruction may occur at the level of the valve, below the valve, or in the ascending aorta.[1]

ECHOCARDIOGRAPHY

View

Bicuspid aortic valve is best seen in the long-axis view of the LVOT, which also allows measurements to be taken of the aortic valve annulus, sinuses of Valsalva, sinotubular ridge, and ascending aorta (Figure 17.7).

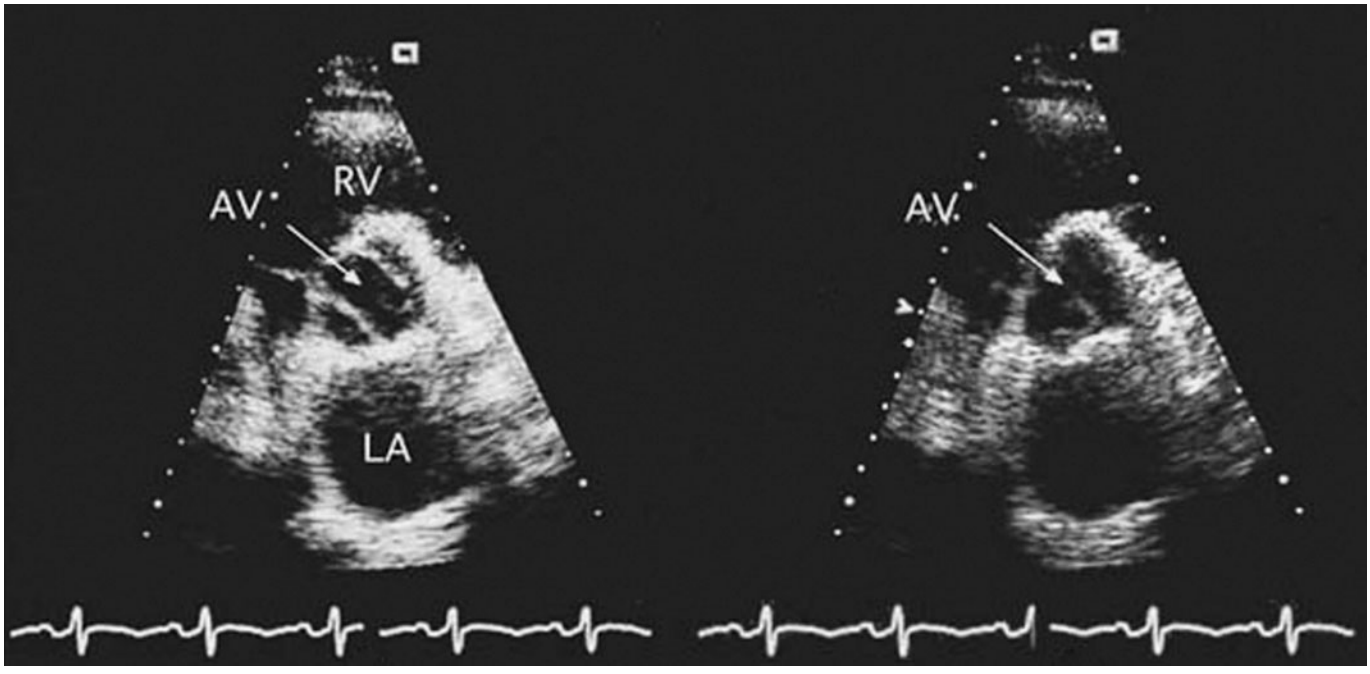
Findings

The abnormally thickened leaflets, doming of the valve, and an oval, not triangular, opening are easily seen during systole.[7,11] Typically, the two unequal-sized aortic cusps and an eccentric closure can be seen on M-mode echocardiography.[1,7] There is often mild poststenotic dilation of the ascending aorta, which can be present without stenosis due to the disturbed flow through the valve.[11]

Other

The presence of associated regurgitation or stenosis should be demonstrated and quantitated, with assessment of the transvalvular gradient.[1] Heavy calcification may obscure the original valve morphology in the older individual with stenosis, thus left ventricular hypertrophy (LVH) and LV function (both systolic and diastolic) should always be assessed.[1,7] Because there is a strong association, coarctation should also be sought.[11] Transesophageal echocardiography has proven value in patients with poor precordial windows.[7]

ECHOCARDIOGRAPHY REPAIRED BICUSPID: For patients with repaired valves, the key parameters to assess are related to valve



A B

Figure 17.7. Transesophageal echocardiographic views of a patient with bicuspid aortic valve. (A) Systolic frame showing two leaflets of the aortic valve (AV) with an ovoid opening (arrow). (B) Diastolic frame showing the single line of coaptation (arrow). LA indicates left atrium; RV, right ventricle. (From Ref. 7.)

stenosis or insufficiency: valve pressure gradients, valve area, qualitative assessment of insufficiency, LV wall thickness, LV dimensions and function.[11]

FOLLOW UP: All patients with bicuspid aortic valves are at risk for endocarditis, which may be the presenting illness to the ICU.[1] Cardiac catheterization is usually reserved for patients in whom either surgical or catheter intervention is contemplated or for additional quantification of the severity of valvular disease.[1] In the adult population, the indications for surgical intervention are the same as for those patients with acquired forms of AS, namely chest pain, CHF, and syncope.[1] Once symptoms of AS develop, the prognosis without valve replacement is poor, with a five-year mortality approaching 90%.[12] Both percutaneous valvuloplasty (if the valve is not calcified) and surgical repair have had limited success due to consequent aortic regurgitation and its progression over time.[1,12]

EISENMENGER'S SYNDROME

GENERAL: Three related, but dissimilar clinical terms bear the name of Eisenmenger. The development of pulmonary hypertension in the presence of increased pulmonary blood flow is called Eisenmenger's reaction.[7] Eisenmenger's syndrome is a general term applied to pulmonary hypertension in the presence of any shunting congenital defect that has reversed flow (right to left).[7] Eisenmenger's complex, as originally described, is the association of a VSD with pulmonary hypertension and shunt reversal. Here, because of its importance to the critically ill adult, the concentration is on Eisenmenger's syndrome: a term applied to any large communication between the systemic and pulmonary circulation that results in irreversible changes in the pulmonary vascular bed, subsequent development of pulmonary hypertension, shunt flow reversal, and cyanosis.[1,12]

INCIDENCE: Eisenmenger's syndrome was seen in 9% of patients with ASD in Wood's initial study, but the prevalence of Eisenmenger's syndrome is declining with improved diagnosis and therapy for CHD.[1] In large adult CHD clinics, approximately 4% of patients have Eisenmenger's syndrome, although there is a higher percentage of Eisenmenger's in patients with cyanotic physiology due to corrected or uncorrected lesions.[1] Those with unrecognized shunts at the atrial level are more likely to be initially diagnosed in adulthood.[1]

PRESENTATION: Commonly, there is a history in infancy of murmur or cyanosis suggesting pulmonary congestion (shunt), symptoms of dyspnea, and exercise intolerance arise in childhood, which then resolve as pulmonary vascular resistance increases and the magnitude of shunting decreases.[4] As the disease progresses (and the shunting reverses), symptoms increase gradually: exertional dyspnea, fatigue, palpitations, chest pain, edema, hemoptysis, and syncope.[1,10] Hallmarks on exam are cyanosis; clubbing; a palpable RV heave and PA impulse; and a pulmonary ejection sound (prominent P2).[7] Laboratory evaluation reveals a compensatory erythrocytosis, iron deficiency, and hyperuricemia.[10]

PATHOPHYSIOLOGY: With substantial left-to-right shunting, the pulmonary vasculature is exposed to increased blood flow and pressure, resulting in the activation of pulmonary vascular elastase, the induction of mediators, smooth muscle migration and hypertrophy, and stimulation of elastin and collagen synthesis.[1] The pathologic changes have a formal grading system. The initial morphologic alterations (medial hypertrophy of the pulmonary arterioles, intimal proliferation and fibrosis, and occlusion of capillaries and small arterioles) are potentially reversible.[4] However, with progression, advanced morphologic changes (plexiform lesions and necrotizing arteritis) are irreversible.[4] The result is obliteration of much of the pulmonary vascular bed, leading to increased pulmonary vascular resistance.[4] As the pulmonary vascular resistance approaches or exceeds systemic resistance, the shunt is reversed.

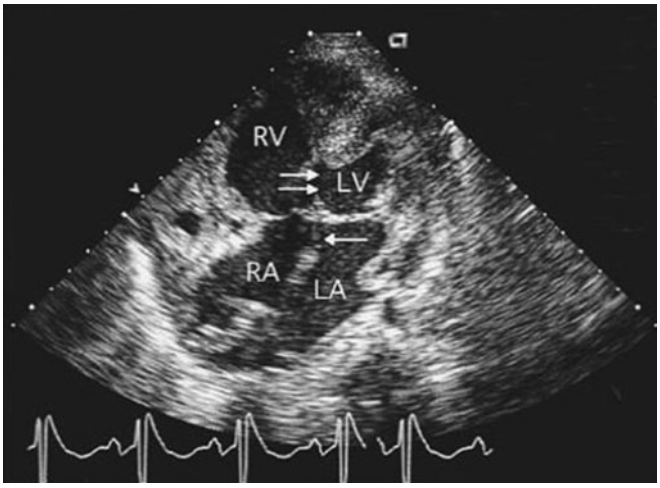
ECHOCARDIOGRAPHY

View

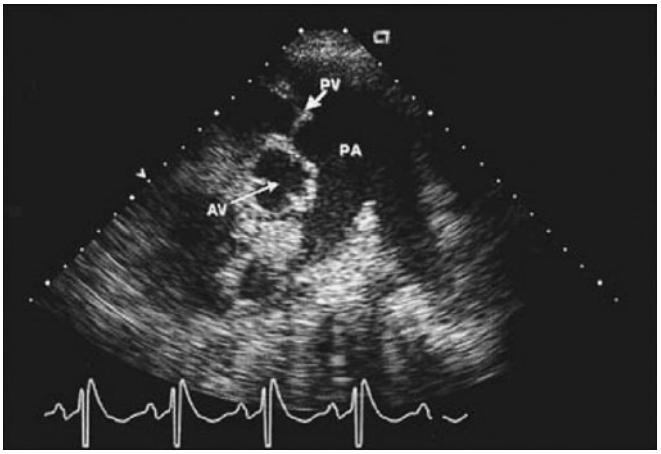
A parasternal long-axis view will demonstrate the RV, LV, and the septum, the four-chamber apical view; the RA and tricuspid valve (Figure 17.8).

Findings

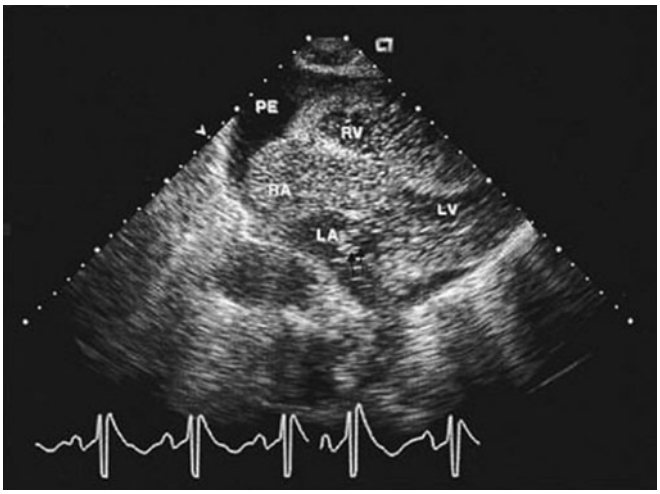
The LV appears small and underfilled; the septum deviates toward the LV.[7] Right ventricular function may be



A



B



C

Figure 17.8. Transthoracic echocardiogram in a 32-year-old woman with Down syndrome. She has Eisenmenger's syndrome due to an atrioventricular septal defect. (A) Four-chamber view shows hypertrophied right ventricular (RV) wall, ventricular septal defect (double white arrow), and ostium primum atrial septal defect (single white arrow). (B) Parasternal short-axis view shows markedly dilated pulmonary artery (PA). (C) Subcostal view after intravenous injection of agitated saline. The right atrium (RA) and right ventricle (RV) are opacified and there is rapid appearance of contrast in the left atrium (LA) and ventricle (LV) (double black arrow). A moderate pericardial effusion (PE) is also noted. AV indicates aortic valve; PV, pulmonary valve. (From Ref. 7.)

normal until the late stages of the disease, when there is severe right ventricular hypertrophy (RVH) and right atrial enlargement.[7] Always evaluate for RV pressure and pulmonary hypertension.[4] Measure the tricuspid regurgitant velocity to estimate the peak RV systolic pressure.[7] Pulmonary insufficiency with a high-velocity regurgitant jet is also a frequent finding.[7] The underlying cardiac defect (usually VSD) can be visualized, although shunting across the defect may be difficult to demonstrate because of low or bidirectional flow due to ventricular pressure equalization (i.e., pulmonary pressure equals systemic pressure).[4,7] Color-flow Doppler and bubble contrast injection may permit the location of the shunt.[4] Other valvular lesions are uncommon, except in ASD primum or AV canal defects, when mitral regurgitation is invariably present. Eisenmenger's syndrome can usually be differentiated from primary pulmonary hypertension noninvasively by TTE, although shunting across a PFO may mimic an ASD with Eisenmenger's syndrome.[7] In these cases, further investigation with TEE or cardiac catheterization is indicated.

Follow up

Despite high pulmonary pressures, patients with Eisenmenger's syndrome have more favorable hemodynamics and a better prognosis than those with primary pulmonary hypertension.[1] The rate of survival after an Eisenmenger's syndrome diagnosis is 80% at 10 years, 77% at 15 years, and 42% at 25 years.[4] Death is usually sudden, presumably caused by supraventricular arrhythmias (seen in 36%), but some patients die of heart failure, hemoptysis, brain abscess, or stroke.[4] There are no prospective studies of long-term outcome in patients with Eisenmenger's syndrome, but a history of syncope, clinically evident RV systolic dysfunction, older age at presentation, poor functional class, complex underlying disease, supraventricular arrhythmias,

low cardiac output, RV dysfunction, severe hypoxemia (O2 saturation <85%), and increased serum uric acid concentration are associated with a poor outcome.[1,4]

Surgical closure of the shunt is an option, but the degree to which pulmonary vascular resistance is elevated before operation is a critical factor determining postsurgical prognosis.[10] If the pulmonary vascular resistance is less than one third of the systemic value, pulmonary vascular disease should not progress.[10] However, if higher, there may be continued progression of disease postoperatively.[10] Improved results in heart–lung transplantation offer an alternative for adolescents and young adults with Eisenmenger's syndrome not amenable to surgical repair of the primary defect, and are promising.[12]

The management of patients with Eisenmenger's syndrome is deceptively simple: avoid destabilizing the "balanced physiology."[1] In general, management includes promptly restoring sinus rhythm; avoiding afterload reduction, which worsens shunting; treating arterial hypertension to reduce the risk of intrapulmonary bleeding and hemoptysis (a potentially fatal complication); and avoiding pregnancy, which is associated with a high maternal mortality and a high risk of fetal complications.[1] Anticoagulation is generally avoided in these patients due to their increased risk of bleeding, but is indicated for atrial fibrillation, atrial flutter, recurrent thromboembolic events, mechanical heart valves, or other high-risk anatomy. Prostacyclin and bosentan have been shown to be of help.[1]

VENTRICULAR SEPTAL DEFECT

GENERAL: A ventricular septal defect (VSD) is a hole in the wall that separates the right and left ventricles of the heart. Only small or moderate-size defects are usually seen initially in adulthood, as most patients with isolated large defects come to medical, and often surgical, attention early in life.[10]

INCIDENCE: A VSD is the most common congenital cardiac defect, occurring in approximately 30% of patients, with an equal male and female frequency.[1] It is much less commonly seen in adults because 75% of VSDs close by the time a child is 10 years of age.[1,5,7,22] Although all types of VSDs may exist in patients reaching adulthood, the most likely to be first diagnosed in adulthood are the perimembranous and outlet VSDs.[11]

PRESENTATION: The presentation in adults is dependent on the size of the shunt and whether there is associated pulmonic or subpulmonic stenosis that has protected the lung from the systemic pressure and volume.[8] Adults may remain asymptomatic, but have a murmur that will be picked up on exam. Or they may present with chest pain, fatigue, tachypnea, and exercise intolerance if the VSD is larger. Unprotected lungs with large shunts invariably lead to symptoms of pulmonary vascular disease and severe pulmonary hypertension.[8] All patients with a VSD may present with endocarditis. Usually an isolated defect, VSDs can also occur in the setting of more complex CHD such as tetralogy of fallot (TOF) and AVSD.[1,10] If patients do not present with symptoms, the exam is notable for a holosystolic murmur at the left sternal border radiating rightward with thrill and an EKG with LVH or RVH.[8]

PATHOPHYSIOLOGY: The ventricular septum consists of the trabecular muscular septum, the inlet septum (mitral and tricuspid valve area), the outlet or infundibular septum (pulmonary and aortic valve area), and the membranous septum, which separates the LVOT from the RV and RA as it courses upward.[1,16] Normal closure of the ventricular septum occurs through three concurrent embryologic mechanisms: (1) downward growth of the outflow tract ridges forming the outlet septum, (2) growth of the endocardial cushions forming the inlet septum, and (3) growth of the muscular septum forming the apical and midmuscular portions of the septum.[25] Failure of growth, alignment, or fusion of these components results in a VSD.[1]

The functional disturbance is dependent primarily on VSD size and on the relative resistances of the systemic and pulmonary vascular beds. Defects can be classified as restrictive and nonrestrictive defects.[7,10,22] In restrictive VSDs, RV pressure is less than one half of the systemic levels. Anatomically, small restrictive defects are less than one third of the aortic root size and moderate restrictive defects are approximately one half the size of the aortic valve.[1] Nonrestrictive defects, by definition, permit equalization of the ventricular pressures.

Ventricular septal defect size has implications for the pathophysiology. A small

restrictive VSD has a high-pressure gradient across it from the LV to the RV, with normal or mildly elevated PA pressure and predominant left-to-right shunting.[7,8] There is little or no functional disturbance because pulmonary blood flow is increased only minimally and pulmonary vascular resistance remains normal.[1,22] Thus the smallest VSD (maladie de Roger) is a hemodynamically insignificant shunt, with a loud murmur, but still carries a risk for endocarditis.[22]

Patients with moderate defects have moderate-to-severe left-to-right shunting, may develop symptoms associated with LV volume overload, and are at risk for developing pulmonary vascular disease.[1] A large, nonrestrictive VSD causes a large, left-to-right shunt initially, and the pulmonary circulation is exposed to systemic pressures early in the course of the disease (in the absence of RVOT obstruction). Over time, the pulmonary vascular resistance usually increases, and the magnitude of left-to-right shunting declines, causing bidirectional shunting.[1,7,22] Eventually, the pulmonary vascular resistance equals or exceeds the systemic resistance (pulmonary hypertension); the shunting of blood from left to right then ceases, and right-to-left shunting begins.[22] Patients with nonrestrictive VSDs usually develop irreversible pulmonary vascular disease within the first decade of life, which can progress to the Eisenmenger physiology.[1]

Unlike patients with an ASD, patients with a VSD present as a volume-loaded left heart, with progressive enlargement of the LA and increased LV vascularity.[5] There is a lesser degree of enlargement of the RV.[5] Because most VSDs close spontaneously and the larger defects are diagnosed and repaired in childhood, it is relatively uncommon to encounter adults with previously undiagnosed VSDs of hemodynamic consequence.[7] The importance of identifying small VSDs is that they pose an ongoing risk for endocarditis and the potential complication of progressive aortic regurgitation.[7]

Classifications, alternate names, and locations of VSDs are presented in Table 17.3. The crista ventricularis is a band of muscle in the posterior RVOT, which is used to divide the "supracristal" or outlet VSDs from the "infracristal" or membranous VSDs.[23] Although they are both subaortic and subpulmonary, the supracristal defect is closer to the pulmonic valve but is associated with aortic insufficiency and many other names. Figure 17.9 shows the anatomic locations of the various types of VSDs.

TABLE 17.3. Types of ventricular septal defects

Location	*Common name*	*Alternate names*
OUTLET		
Below crista	Perimembranous	Membranous
		Infracristal
		Subaortic
		Semimembranous
Above crista	Outlet	Supracristal
		Infundibular
		Conoventricular
		Doubly committed subarterial
		Subpulmonary
With malalignment	Malalignment	Part of tetralogy of Fallot
		Other syndromes
MUSCULAR	Muscular	Trabecular
AV VALVES	Inlet	Endocardial cushion,
		AV Canal Defects
		AV septal defects

AV indicates atrioventricular.

Inlet VSDs comprise 5–10% of VSDs and occur high, near the junction of the mitral and tricuspid valves (so-called atrioventricular canal defects).[22,25] They are always associated with abnormalities of the relationship of the two valves and a cleft in the anterior mitral leaflet.[23] Inlet septal defects rarely close spontaneously.[1]

Muscular defects or defects of the trabecular septum account for 20% of all VSDs.[1] The muscular VSDs, often multiple, may be located in any region of the trebecular septum—inlet trebeculum or outlet trebeculum—but are not to be confused with inlet or outlet VSDs.[7] Common sites for small defects are the apex of the RV and at the anterior junction with the RV free wall.[23] Muscular VSDs ("Swiss cheese VSDs") are the type that most

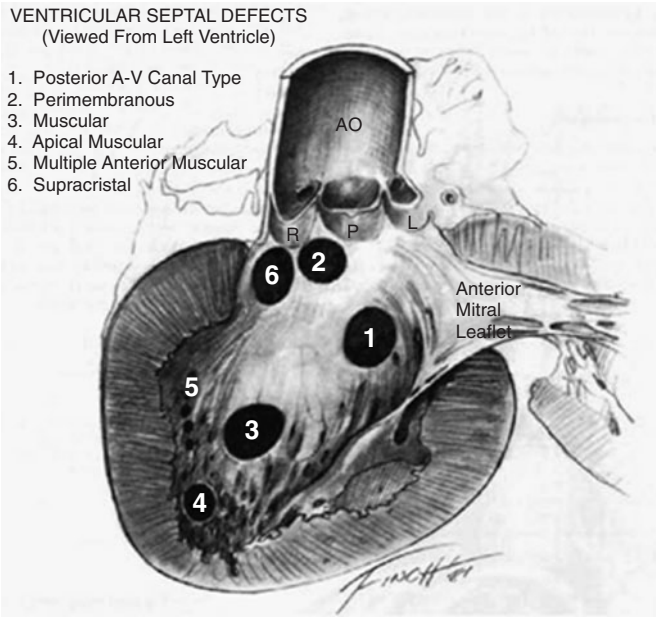


Figure 17.9. Locations of ventricular septal defects 1: Inlet; 2: Perimembranous; 3–5: Muscular; 6: Outlet. Malalignment is not shown. (From Ref. 26.)

often close spontaneously, usually in the first two years of life.[16,25]

The perimembranous VSD is the most common VSD in the adult, and comprises 75% of VSDs overall.[22,23] Because this VSD occurs at the junction of the inlet, outlet, and trabecular septum, it becomes "perimembranous" because it most often extends variably into these regions.[1,16] These VSDs may undermine part of the aortic annulus, resulting in aortic insufficiency in 2–5% of patients.[23] The perimembranous VSD underlies the septal leaflet of the tricuspid valve and may decrease in size or close spontaneously due to adherence of septal leaflet tissue to the defect, resulting in a ventricular septal aneurysm.[1]

An outlet VSD is located in the RV infundibulum beneath the pulmonary valve and comprises 5–7% of VSDs.[25] It can undermine the aortic valve annulus, allowing the right coronary leaflet to prolapse, resulting in regurgitation and a decrease in VSD size.[1,22] Because of the location of this VSD, the disturbed flow may be mistaken for pulmonary stenosis (PS).[23]

Malalignment VSDs are always associated with overriding of one of the great arteries relative to the ventricular septum.[23] It is always associated with a syndrome like TOF.[7]

ECHOCARDIOGRAPHY: Two-dimensional echocardiography with Doppler flow can confirm the presence and location of the VSD, and color-flow mapping provides information about the magnitude and direction of shunting.[8,22] The key elements to evaluate are the velocity of flow across ventricular septum, and size of the overloaded chambers by measuring RV and PA pressures, LV size, and LA size.[8,11] In general, slow velocity across the defect <4 m/sec suggests a large shunt or a high pulmonary resistance or both.[11] The higher the gradient across the septum, the smaller the defect and the left-to-right shunt.[8]

VIEW: Multiple views are required to examine the entire septal region, as the septum is curved and does not lie in a single echocardiographic plane.[16] Figure 17.10 shows the best echocardiographic views for each type of VSD site.[15,16] The VSD can usually be identified with imaging and color Doppler studies of ventricular septum long-axis, short-axis and four-chamber sweeps.[15] For the most common types of VSDs (perimembranous and outlet), viewing is best done from the parasternal long- and short-axis window.[11,16] Muscular and inlet VSD jets are best seen in the apical or subcostal four-chamber views, but the study should include more medial positions to demonstrate some apical muscular or inlet VSDs.[7,15]

FINDINGS: Color-flow Doppler imaging allows VSD localization by demonstrating a high-velocity (aliased) systolic jet across the VSD into the RV.[7] Saline bubble contrast administration can help the degree and direction of shunting. In the parasternal short-axis view, the jet may be seen in the region of the tricuspid valve (perimembranous VSD) (Figure 17.11) or toward the PA (outlet VSD).[7] Muscular VSDs (Figure 17.12) may appear as narrow, irregular channels, where the orifice on one side is displaced for the orifice on the other side. In addition to the absence of septum, inlet VSDs can be confirmed by assessing the relative position of the two AV valves. If the tricuspid valve is not in its normal apical position and is instead on the same planes as the mitral valve, this is pathognomonic for an AVSD.

Whenever a VSD is suspected, Doppler imaging diagnoses location and characterizes flow direction and velocity.[16] The spectral Doppler signal shows a

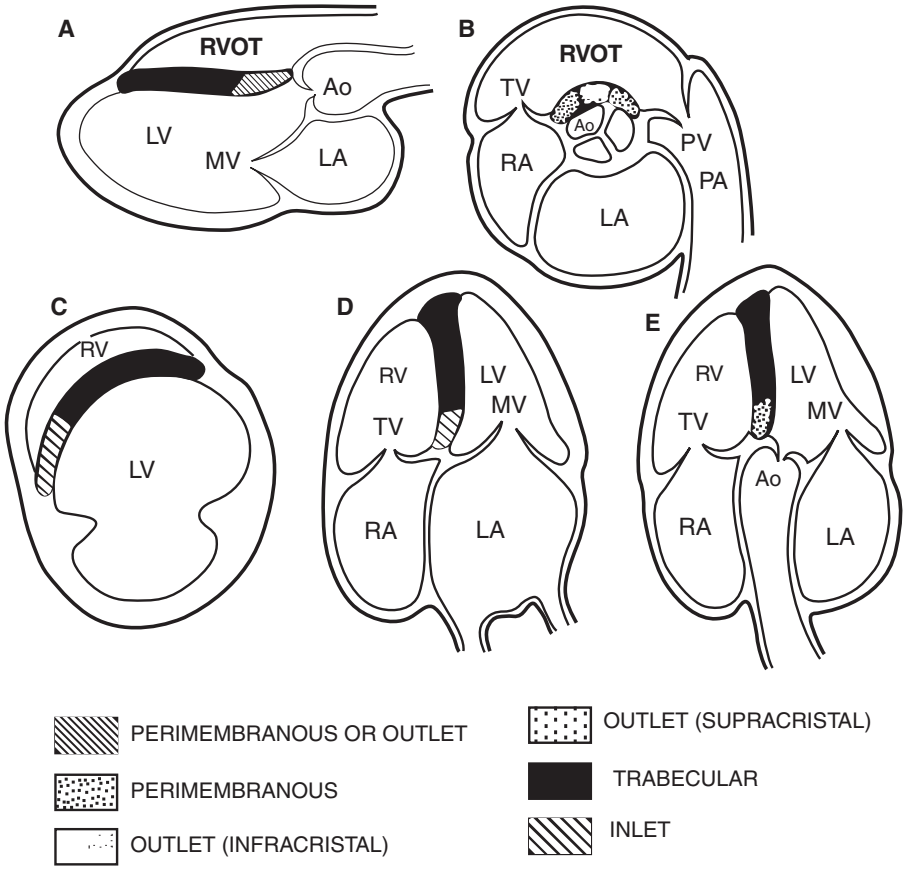


Figure 17.10. Schematic diagram of the location of the various types of ventricular septal defect when viewed using two-dimensional echocardiography. Ao indicates aorta; LA, left atrium; LV, left ventricle; MV, mitral valve; PV, pulmonary valve; RA, right atrium; RV, right ventricle; RVOT, right ventricular outflow tract; TV, tricuspid valve. (From Ref. 16.)

rapid early systolic rise to a midsystolic peak and rapid late systolic fall.[15] The velocity through the septum assesses the magnitude of shunting.[8] Color-flow Doppler through a small restrictive VSD is a thin, turbulent, high-velocity, left-to-right jet, whereas larger, slow-velocity, wider jets indicate larger defects.[16] In continuous-wave Doppler, orienting the transducer parallel to the flow allows accurate peak velocity of the jet and provides a true LV-RV gradient (using the modified Bernoulli equation).[7,16] If there is no LVOT obstruction, subtracting this gradient from the LV systolic pressure measured by cuff theoretically gives the peak RV systolic pressure.[15] In the absence of RVOT obstruction (no pressure gradient), this value is equal to the PA systolic pressure and gives an estimate of the presence and severity of pulmonary hypertension.[7,16] The presence of RV hypertrophy and high RV pressure confirms the diagnosis.

However, there are potential inaccuracies in this calculation, so it is more prudent to take the velocity into account: a high velocity indicates low RV and pulmonary pressure; a low velocity, a high pressure (as in Eisenmenger's syndrome).[15] In this instance there is probably bidirectional flow, which can be shown on spectral Doppler (Figure 17.13).[16] The pressure gradient may also be inaccurate in the setting of tortuous or serpiginous defects often seen in muscular VSDs, where the modified Bernoulli equation is not applicable.[1] If there is tricuspid regurgitation, RV pressure can be indirectly estimated from continuous-wave Doppler interrogation of the tricuspid regurgitation jet.[15] Care must be taken with this approach because the regurgitant jet may be contaminated by the VSD jet (particularly with perimembranous defects), resulting in inaccurate RV pressure estimation.[1]

OTHER: A variety of easily detected associated lesions occur with VSDs. The most common is a ventricular septal aneurysm, a thin membranous tissue at edge of defect (usually

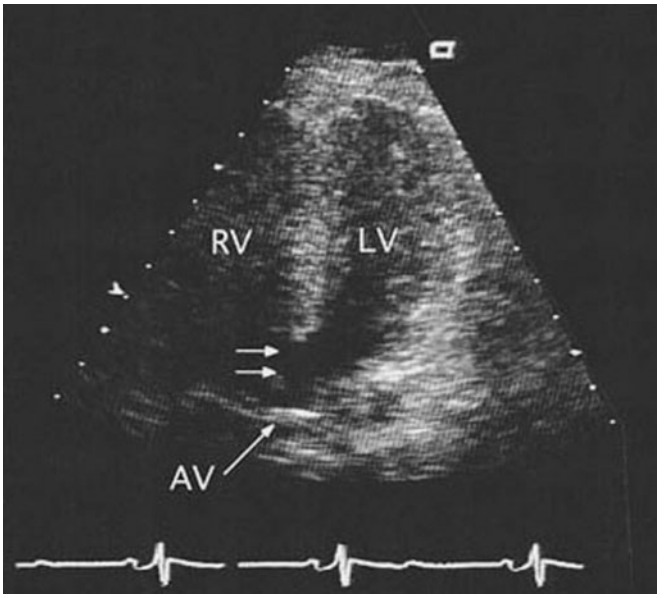


Figure 17.11. Transthoracic echocardiogram of a 40-year-old woman with a large membranous ventricular septal defect (double arrow). The right ventricle (RV) was enlarged because of pulmonary hypertension. AV indicates aortic valve; LV, left ventricle. (From Ref. 7.)

perimembranous) just below the aortic valve that may incorporate the septal leaflet of the tricuspid valve.[7,16] This leaflet then may spontaneously close the VSD. Aneurysms appear best in the parasternal long and short axis and may be highly mobile, protruding through the defect into the RV during systole.[16] Color-flow Doppler determines the aneurysm's patency and evaluates associated tricuspid regurgitation. A second association is aortic insufficiency in outlet VSDs due to absence of the myocardium below the aortic valve destabilizing the annulus.[16] Third, perimembranous (subaortic) VSDs may cause aortic right-coronary cusp prolapse and resultant aortic insufficiency.[1,8] Both lesions can easily be evaluated by color Doppler. Inlet VSDs often are associated with mitral valve clefts and abnormalities of chordal insertion and should be evaluated as part of AVSD, as discussed earlier. If the VSD is one of malalignment, continue evaluation for TOF.[11]

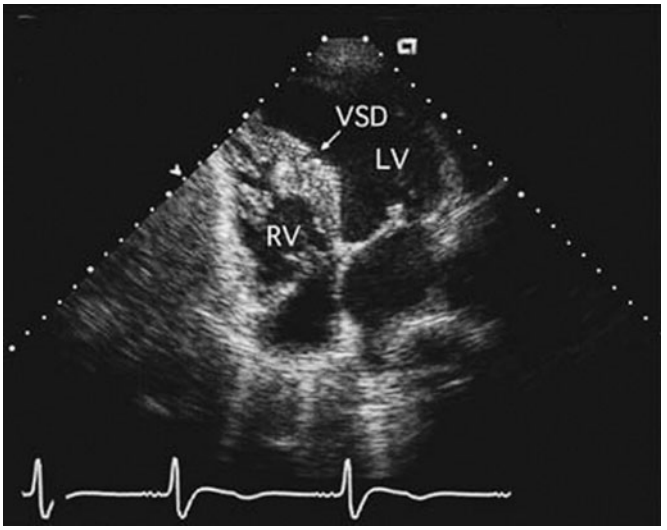


Figure 17.12. Transthoracic echocardiogram of a 45-year-old woman with a small muscular ventricular septal defect (VSD). LV indicates left ventricle; RV, right ventricle. (From Ref. 7.)

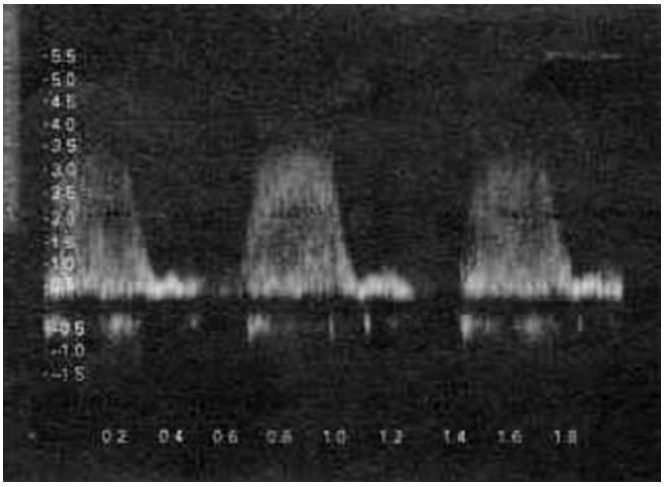


Figure 17.13. Spectral Doppler recording of flow through a ventricular septal defect in a woman with Eisenmenger's syndrome. The velocity is low (maximum 2.3 m/sec) and the pattern demonstrates flow from left to right (above the line) in systole and bidirectional in diastole. (From Ref. 15.)

The outlet VSD may mimic pulmonic stenosis on color Doppler examination because the disturbed flow starts just below the pulmonic valve, and turbulence continues out into the main PA in systole.[11] Careful pulsed-wave and continuous-wave Doppler of the direction, pattern, and timing will distinguish pulmonic stenosis from an outlet VSD.[11]

ECHOCARDIOGRAPHY: Repaired VSD: Evaluation is the same as for a native VSD. In the post-repair patient, the VSD patch may or may not be apparent, depending on the size of the original defect.[7] Once endothelialized, the patch does not cause acoustic shadowing (or distal echo blackout).[7] Color-flow Doppler is the most sensitive technique for detecting residual

shunt due to patch leaks at the peripheral suture lines.[7,16]

Transesophageal echocardiography imaging will provide a clear image of the defect when standard views are unsatisfactory.[15] The ventricular septum is a complex structure that requires careful interrogation in multiple planes.[17] Single-plane (horizontal) TEE is reliable in assessment of the membranous or inlet portions, but has limitations for inlet and muscular VSDs. Biplane TEE is much more ideal: the vertical plane is good for delineating outlet and muscular VSDs.[24] From the midesophageal window (0°), flexion displays the LVOT and thin membranous part of the septum (see perimembranous VSD).[17] Advancing the probe reveals the muscular/trabecular part of the septum (see muscular VSD), but it may be impossible to fully interrogate the apex in this view.[17] Image plane rotation to 30–45° produces a short-axis view of the heart analogous to an inverted parasternal short-axis view.[17] By rotating the probe to image in a plane passing below the level of the aortic valve, perimembraneous and outlet VSDs can be seen and differentiated.[17] Further rotation of the image plane to 130° produces an approximation of the apical long-axis view, providing further imaging of the perimembranous septum, another imaging plane through the muscular septum, and an ideal view to identify an overriding septum (i.e., TOF).[17] Having performed the two-dimensional echo sweep, this is repeated using color-flow mapping.[17] Although it may be difficult to align the Doppler beam parallel to VSD flow, it is important to look out for turbulence within the RV cavity and a flow convergence zone within the LV cavity.[17] In order to interrogate the muscular septum fully, a transgastric transverse (0°) image is obtained and swept from its highest point to its deepest, first by two-dimensional echo.[17] From the deep fundal view, a view analogous to the apical TTE views may be obtained; this provides excellent imaging of the apex.[17] In patients with perimembraneous VSD, TEE is valuable in identifying the mechanism of spontaneous closure and associated findings—for example, suction of the right aortic cusp into the defect, resulting in aortic regurgitation or involvement of tricuspid subvalvar tissue.[17]

OTHER IMAGING: Magnetic resonance imaging and CT can often visualize the defect and describe any other anatomic abnormalities.[8] Magnetic resonance imaging and radionuclide flow studies are sometimes used to quantify the relative size of the left-to-right shunt.[8] Cardiac catheterization can confirm the presence and location of the defect, as well as determine the magnitude of shunting and the pulmonary vascular resistance.[22] A pulmonary vascular resistance to systemic vascular resistance (PVR/SVR) ratio of >0.7 usually implies inoperability.[8]

FOLLOW UP: Adults with small defects and normal pulmonary arterial pressure are generally asymptomatic, and pulmonary vascular disease is unlikely to develop.[22] Such patients do not require surgical closure, but they are at risk for infective endocarditis and should therefore receive antibiotic prophylaxis.[22] Nonrestrictive VSDs can cause LV failure or pulmonary hypertension with associated RV failure, which is important to evaluate to determine suitability for closure.[11,22] Surgical closure of the defect is recommended if the magnitude of pulmonary vascular obstructive disease is not prohibitive.[22]

Severe pulmonary hypertension (Eisenmenger's complex) is more frequent in adults with large uncorrected VSDs than in those with ASDs.[12] In those patients with severe pulmonary vascular disease, a therapeutic approach that has had a midterm benefit is bilateral lung transplantation.[5] Early results with continuous intravenous prostacyclin therapy also are encouraging, but remain largely under research protocols.[5]

Overall, late outcome after early surgical closure of a VSD is excellent.[1] Residual shunts are common, seen in up to 20% of cases after surgery, but are usually small, and second operations are required in only 5% of patients.[1,12] Late complications after surgical repair include endocarditis (if a residual shunt persists after surgery), surgically induced aortic or pulmonary regurgitation, and tricuspid regurgitation.[1] Arrhythmias and conduction disturbances may be seen: RBBB occurs in 30–60%, first-degree AV block in 10%, and complete heart block in 1–3% over long-term follow-up.[1] Patients may have LV dysfunction with late repair of the defect or with significant aortic regurgitation.[1] Patients may have persistent pulmonary hypertension despite successful closure of their shunt.[1] In general, patients undergoing early repair without a residual shunt, evidence of pulmonary hypertension, arrhythmias, or conduction block survive to lead normal adult lives.[12]

TETRALOGY OF FALLOT

GENERAL: Tetralogy of Fallot (TOF) is the most common repaired complex CHD in adults.[14] It is characterized by a large malaligned ventricular septum that results in a VSD, obstruction

of the RVOT, RV hypertrophy, and a dilated anterior and rightward aorta that in about 50% of patients overrides the ventricular septum.[4,8,10] Without surgical intervention, most patients die in childhood; occasionally, an acyanotic patient with only mild PS and minimal right-to-left shunting is encountered as an adult (pink TOF).[7]

INCIDENCE: Tetralogy of Fallot accounts for 10% of all CHD, with a slight male preponderance.[1] Without surgical intervention, the rate of survival is 11% at 20 years, 6% at 30 years, and 3% at 40 years.[4,5,12]

PRESENTATION: Uncorrected adults present with a history of cyanosis from birth and sudden hypoxic "spells," characterized by tachypnea and hyperpnea that resolved in adolescence.[4] They re-present as adults with dyspnea and limited exercise tolerance. As the shunt worsens, progressive cyanosis ensues.[4] Presenting symptoms to the ICU include erythrocytosis, hyperviscosity, abnormalities of hemostasis, cerebral abscesses or stroke, and endocarditis.[4] Physical exam reveals a parasternal RV lift from dilation, and a systolic ejection murmur from RVOT obstruction. The more severe the obstruction, the louder and longer the outflow track murmur is with a diminished P2.[5] The EKG demonstrates right-axis deviation, RV hypertrophy, and RA enlargement with occasional ventricular ectopy if the RVOT is severely obstructed.[5] With repaired tetralogy, there will be a normal S1 and delayed S2 with a low-pitched diastolic murmur from residual pulmonary insufficiency at the left sternal border. Electrocardiogram abnormalities are usually present after repaired TOF.[7]

Several abnormalities may occur in association with TOF, including right aortic arch in 25% (no functional significance), ASD or PFO in 15% (pentalogy of Fallot), coronary arterial anomalies in 10% , and multiple VSDs.[1,4,7]

PATHOPHYSIOLOGY: Although it is called a tetralogy, only the perimembranous nonrestrictive VSD and PS contribute to the pathophysiology of this disorder.[7] The severity of PS determines the RV systolic pressure and thus the degree of right-to-left shunting across the nonrestrictive VSD.[1,7,12] The PS can occur anywhere along the pulmonary outflow tract from the valve to the branch arteries, but most commonly is subvalvular (infundibular) due to an obstructing muscular band in the RVOT.[1,7] Right ventricular hypertrophy occurs as a consequence of both RVOT obstruction and the large, nonrestrictive VSD.

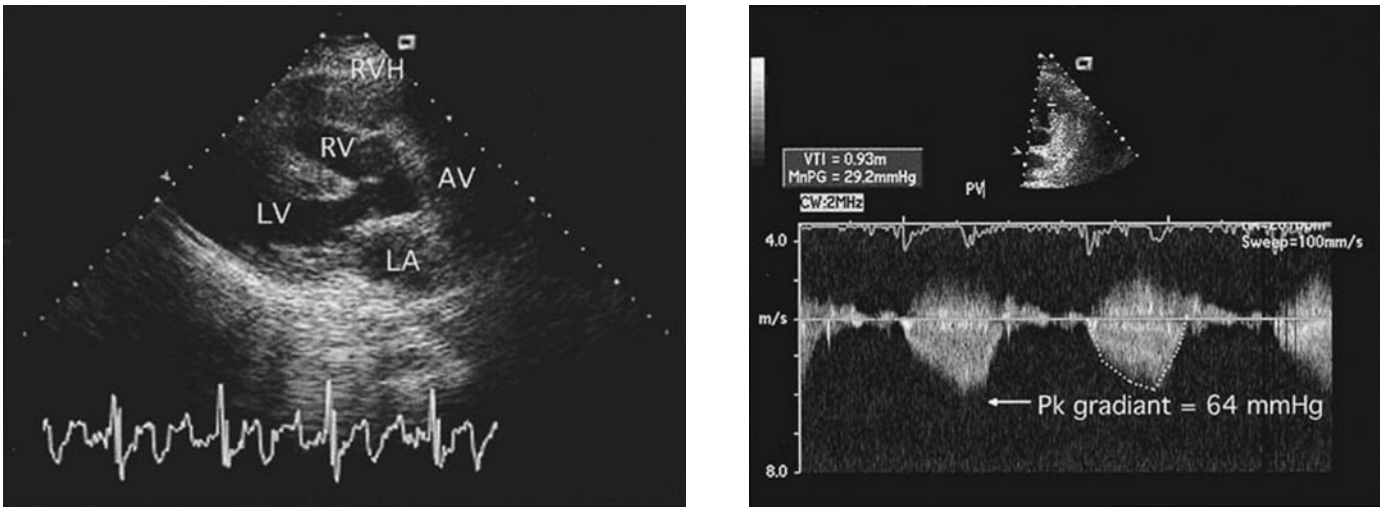
Because the resistance to flow across the RVOT is relatively fixed, changes in systemic vascular resistance affect the magnitude of right-to-left shunting.[4] When the obstruction is severe, the pulmonary blood flow is reduced markedly, and a large volume of desaturated systemic venous blood is shunted from right to left across the VSD, resulting in severe cyanosis. Unlike patients who have isolated, large VSDs, patients with TOF are "protected" by PS from the development of pulmonary hypertension, and they are often surgical candidates as adults.[12] Intracardiac repair with closure of the VSD and correction of the pulmonary or infundibular stenosis is considered one of the most successful operations in congenital heart disease.[12] The sequelae of repaired TOF include residual outflow obstruction, pulmonary valve regurgitation, RV aneurysms, and VSD patch leak and conduction abnormalities.[1,12]

The most common repair is by branch pulmonary dilitation, VSD closure, resection of the stenosis, and use of an RVOT patch extending through the pulmonary valve into the PA (transannular patch). If the conduit is valveless, the pulmonic regurgitation over time increases the RV volume and necessitates a pulmonary valve replacement.[8,17]

ECHOCARDIOGRAPHY: Transthoracic and Doppler echocardiography diagnose the tetrad of features, assess the presence of associated abnormalities, and determine the level and severity of the obstruction of the RVOT and, in the repaired state, the integrity of the patches and any residual lesions (Figure 17.14).[4,7]

VIEW: Two-dimensional echocardiography from the parasternal long axis demonstrates the malalignment of the septum and VSD and the overriding aortic root, and the parasternal short-axis and subcostal coronal views can assess the various potential levels of RVOT stenosis.[16]

FINDINGS: A large perimembranous VSD and right-to-left shunting can be visualized by color Doppler imaging, although the peak velocity of the VSD jet seen by spectral Doppler is usually low.[7] Right ventricular hypertrophy should be evident and evaluated along with estimates of RV size and function.[7,15] The site of obstruction of the RVOT should be determined by imaging



A B

Figure 17.14. Transthoracic echocardiogram of a patient with tetralogy of Fallot. (A) The parasternal long-axis view shows the ventricular septal defect (VSD), overriding aorta. (B) Continuous-wave Doppler signal across the right ventricular outflow tract shows a high-velocity, late-peaking jet. This demonstrates severe outflow tract obstruction. The late peak suggests a component of dynamic obstruction due to the hypertrophied infundibulum. AV indicates aortic valve; LA, left atrium; LV, left ventricle; RV, right ventricle; RVH, right ventricular hypertrophy. (From Ref. 7.)

pulmonary valve and the outflow tract including the main PA and its branches as far as possible.[8] Systolic color-flow aliasing and a continuous-wave Doppler gradient should be detectable across the RVOT or pulmonic valve,[4,7,16] but if the distal obstruction imaging is poor RV pressure may give a better estimate of the gradient than spectral velocity.[15] Short-axis and suprasternal views determine the size of the pulmonary arteries any post-stenotic dilation.[7,16] Last, the aortic root is variably enlarged and overrides the VSD (if >50% overrides the LV, it is TOF; if >50% overrides the RV, it is double outlet RV).[7,16] Measure aortic root size and look for aortic regurgitation.[7,8]

ECHOCARDIOGRAPHY REPAIRED TOF: After repair of TOF, echocardiography plays a key role in assessing surgical results. The long-term sequelae to monitor on echocardiographic exam are RV hypertrophy and dilatation, RVOT aneurysms, PS, and (severe) pulmonary regurgitation[7,11,17] In addition, there may be residual VSD patch leak or residual RVOT obstruction.[7,11,17] The aortic root remains large and dilated.[17] Homografts, over time, are susceptible to calcific degeneration, causing homograft stenosis and/or regurgitation and endocarditis.[17] Transesophageal echocardiography is particularly suited to define the anatomy of the RVOT and the pulmonary valve when precordial imaging is difficult.[7] The horizontal plan can be used to assess the distal main PA and its bifurcations and the right PA.[24] Vertical plane TEE is essential for precise assessment of valvular, subvalvular, and proximal supravavlular RVOT lesions and conduits.[24] Calcifications, however, make imaging difficult even with TEE.[17]

VIEW: Parasternal long-axis view will reveal the VSD patch as a linear structure passing obliquely from the septum to the anterior aortic root.

FINDINGS: Right ventricular outflow tract conduit obstruction is indicated by a high-velocity spectral signal and high RV pressure. However, if there is severe tricuspid regurgitation, the RV may not demonstrate high pressure despite a significant obstruction. Measure maximum velocity at any areas of aliasing.[7] Evaluate for residual VSDs, especially at the edge of the grafts. Color-flow Doppler and the swirling of blood around the upper and lower anastomoses of the grafts may reveal an aneurysm, pseudoaneurysm or abscess.[11,17] Assess pulmonic regurgitation, an expected finding if a valvotomy was performed or a transannular patch was grafted.[8,11] Color Doppler may

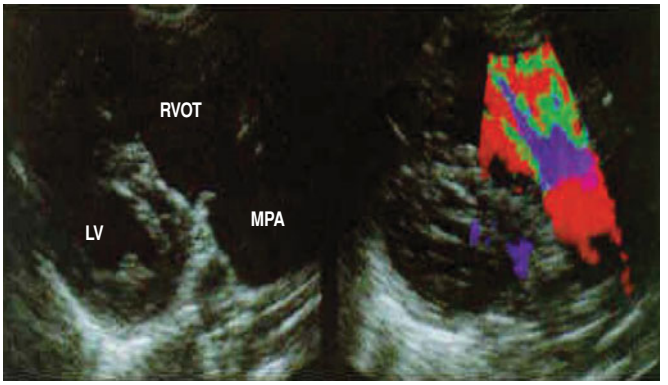


Figure 17.15. Views along the right ventricular outflow tract (RVOT) and main pulmonary artery in a patient who has undergone repair of tetralogy of Fallot. The right ventricle, outflow tract, and pulmonary artery are dilated, and there is jet of pulmonary regurgitation occupying the full width of the pulmonary ring. (From Ref. 15.)

underestimate significant pulmonic regurgitation, thus the severity of regurgitation may be better assessed from the RV volume overload and the Doppler backflow signal in the PA and into the RV (Figure 17.15).[8,15]

Increased pressures in PA and RV indicate a peripheral or postpatch pulmonic stenosis, especially in those who have had a pulmonary homograft (patch enlargement) to augment a hypoplastic pulmonary.[11] A high-velocity spectral signal will indicate an obstructive lesion, but because there can be narrowing at more than one level and a distal lesion may not be visualized, it is not possible to ensure that there is not an additional lesion.[15] The shape of the PA conduit prosthesis means that a spectral Doppler signal is likely to underestimate the transconduit gradient.[15] Most repaired adults have tricuspid regurgitation, thus serial measurement of RV pressure is the best echocardiographic means of assessing the progression of obstruction.[15]

OTHER IMAGING: Because echocardiographic imaging and velocity gradients are poor indicators of RVOT obstruction and because good images are seldom obtained due to calcification of the RVOT valve or conduit, other imaging is necessary.[15] Magnetic resonance imaging/CT can identify any RVOT stenosis and quantitate pulmonary insufficiency and RV volumes.[8,15] With catheterization, it is possible to confirm the diagnosis and obtain additional anatomical and hemodynamic data, including the location and magnitude of right-to-left shunting, the level and severity of RVOT obstruction, anatomic features of the RVOT including the pulmonary valve and arteries, and the origin and course of the coronary arteries.[4,7,10,15]

FOLLOW-UP: The sequelae of TOF repair include residual RVOT obstruction, RV aneurysms, pulmonary valve regurgitation, VSD patch leak, conduction abnormalities, and aortic root dilation.[1,4,12] Residual or recurrent obstruction of the RVOT should be evaluated with serial imaging, and may require repeated surgery.[1,4] Even though substantial regurgitation can be tolerated for long periods, the presence of progressive RV enlargement, worsening tricuspid regurgitation, arrhythmias, and evidence of deteriorating exercise tolerance are all indications for pulmonary valve replacement.[1,4,7,15] Approximately 10–20% of patients with repaired TOF have residual VSDs requiring repeated surgery if the defects are of sufficient size.[4,7,15] Conduction abnormalities (RBBB, premature ventricular contraction [PVC]) occur in a majority of patients. Rarely, AV block and ventricular tachycardia (VT) occur and should be treated.[1] The new onset of an arrhythmia should always prompt investigation of the patient's hemodynamic status, with correction of lesions and cryoablation as indicated.[1] Finally, mild aortic regurgitation from increased root dimension persists.[1,4,15] Tetralogy of Fallot repair has a 90–95% survival at 10 years, and reintervention is needed in approximately 10% of patients older than 20 years.[1]

References

1. Gring C, Griffin BP. Transesophageal echocardiography. In: Topol EJ, ed. *Textbook of Cardiovascular Medicine.* 3rd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2007.
2. Findlow D, Doyle E. Congenital heart disease in adults. *British J Anaesth*. 1997;78:416–430.
3. Wren C, O'Sullivan JJ. Survival with congenital heart disease and need for follow up in adult life. *Heart*. 2001;85:438–443.
4. Brickner ME, Hillis LD, Lange RA. Congenital heart disease in adults. Second of two parts. *New EnglJ Med*. 2000;342:334–342.

5. Moodie DS. Diagnosis and management of congenital heart disease in the adult. *Cardiol Rev*. 2001;9:276–281.
6. Perloff JK, Warnes CA. Challenges posed by adults with repaired congenital heart disease. *Circulation*. 2001;103: 2637–2643.
7. Tede N, Foster, E. Congenital Heart Disease in Adults. In: Crawford MH, ed. *Current Diagnosis & Treatment in Cardiology.* 2nd ed. McGraw-Hill; 2003:427–443.
8. Bashore TM, Granger CB, Hranitzky P. Heart. In: McPhee SJ, Papadakis MA, Tierney LM, eds. *Current Medical Diagnosis & Treatment 2007.* 46th ed. McGraw-Hill; 2007.
9. Stumper, O. Imaging the heart in adult congenital heart disease. *Heart*. 1998;80:535–536.
10. Braunwald E, Fauci AS, Hauser SL, et al. Congenital heart disease. In: Braunwald E, Fauci AS, Kasper DL, Hauser SL, eds. *Harrison's Principles of Internal Medicine.* 15th ed. McGraw-Hill; 2001.
11. Linker DT: The Teenager, the Adult, and the Previously Diagnosed Patient. In: Linker DT, ed. *Practical Echocardiography of Congenital Heart Disease From Fetus to Adult.* Philadelphia, Pa: Churchill Livingstone; 2001:137–139.
12. Foster E. Congenital heart disease in adults. *West J Med*. 1995;163:492–498.
13. Perloff JK. Congenital heart disease in adults: a new cardiovascular subspecialty. *Circulation*. 1991;84:1881–1890.
14. Alvarez N, Prieur T, Connelly M. The ten most commonly asked questions about management of congenital heart disease in adults. *CardiolRev*. 2002;10:77–81.
15. Houston A, Hillis S, Lilley S, et al. Echocardiograhy in adult congenital heart disease. *Heart*. 1998;80:12S–26S.
16. Feigenbaum H, Armstrong WF, Ryan T. Congenital heart diseases. In: Arner FB, ed. *Echocardiography.* 6th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2005:559–636.
17. Masani ND. Transoesophageal echocardiography in adult congenital heart disease. *BMJ Heart*. 2001;86 Supplement II:ii30–ii40.
18. Snider RA, Sewer GA, Ritter SB. Abnormalities of ventricoarterial connection. In: Snider RA, Serwer GA, Ritter SB, eds. *Echocardiography in Pediatric Heart Disease.* St Louis, Mo: Mosby; 1997:297–322.
19. Rice MJ, Sahn DJ. Transesophageal echocardiography for congenital heart disease–who, what, and when. *Mayo Clin Proc*. 1995;70:401–402.
20. Seiler C. How should we assess patent foramen ovale? *Heart*. 2004;90:1245–1247.
21. Cleveland Clinic, Heart and Vascular Institute. Patent foramen ovale. Available at: http://www.clevelandclinic.org/heartcenter/pub/guide/disease/congenital/pfo.htm. Accessed February 7, 2009.
22. Brickner ME, Hillis LD, Lange RA. Congenital heart disease in adults: first of two parts. *New Engl J Med*. 2000;342:256–263.
23. Linker DT. Simplified nomenclature and physiology of congenital heart disease. In: Linker DT, ed. *Practical Echocardiography of Congenital Heart Disease From Fetus to Adult.* Philadelphia, Pa: Churchill Livingstone; 2001:25–28.
24. Marelli AJ, Child JS, Perloff JK. Transoesophageal echocardiography in congenital heart disease in the adult. *Cardiol Clin*. 1993;11:505–520.
25. Emedicine Specialties: Ventricular septal efect, perimembranous. Available at: http://www.emedicine.com/ped/topic2544.htm. Accessed February 7, 2009.
26. UTMB Cardiology: Ventricular Septal Defects. Available at: http://www.cardiology.utmb.edu/slides/sec-3-imaging/sec-3-imaging.shtm. Accessed February 7, 2009.

This page intentionally left blank

CHAPTER 18

Echocardiographic Evaluation of Cardiac Trauma

David A. Vitberg and Dorothea McAreavey

INTRODUCTION

Trauma to the heart occurs in association with either blunt thoracic or penetrating injuries to the chest wall. Tears in the great vessels and many cardiac insults, such as acute cardiac rupture or valvular regurgitation, are often incompatible with life and the patient dies prior to or shortly after arrival at the emergency department (ED).[1] In viable patients with more slowly developing problems, an aggressive plan is essential to detect and manage potentially treatable injuries.

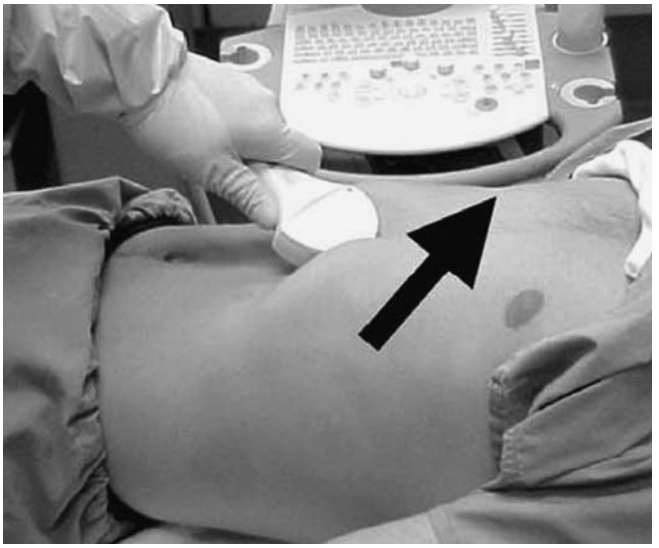
BEDSIDE ULTRASOUND FOR ASSESSMENT OF CARDIAC TRAUMA

The quintessential principle in cardiac ultrasound for all trauma patients, particularly those with chest trauma and potential cardiac injury, is early detection of blood (or blood clot) in the pericardium. The observation of Beck's triad—elevated jugular (central) venous pressure, muffled heart sounds, and hypotension—should always prompt the clinician to further evaluate the heart and pericardial space. However, these textbook physical examination findings are often absent and may occur very late in the course of traumatic cardiac injury.[2] Net circulating volume status will affect the clinician's ability to detect the hemodynamic changes typically associated with cardiac trauma. Patients who have sustained multiple traumatic injuries with hemorrhage may have such a profoundly reduced intravascular volume that central venous pressure remains low despite devastating injuries like cardiac rupture with worsening tamponade. Furthermore, because of advances in prehospital emergency care, some patients may arrive in the ED with potentially life-threatening thoracic wounds and yet be relatively asymptomatic.[3] The integration of bedside cardiac ultrasound into the early portion of the trauma assessment of patients with multiple traumatic injuries, and particularly those with chest trauma, is important.

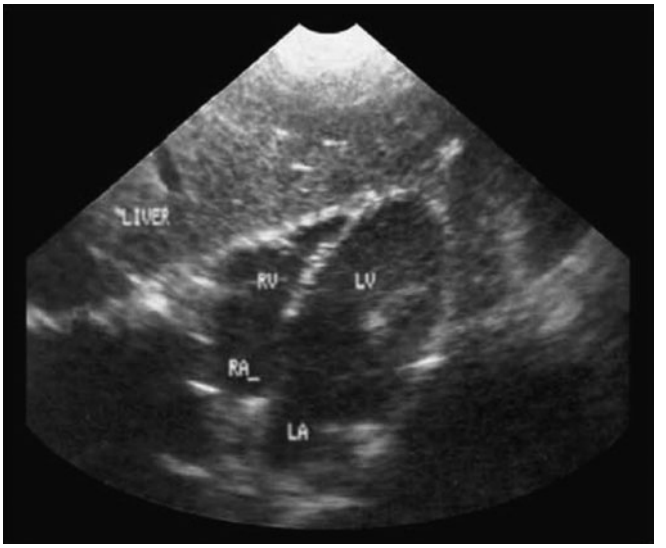
Focused abdominal sonography in trauma (FAST) has become a standard diagnostic component during the early evaluation phase of trauma patients (see Chapter 28). First described by Rozycki et al.,[4] this examination intentionally has limited goals that allow the physician to make rapid decisions, identify life-threatening disorders, and expedite definitive operative interventions. A 3.5-MHz transducer is placed in the pericardial, right upper quadrant, left upper quadrant, and pelvic regions to evaluate for the presence of free fluid. The FAST examination is noninvasive, can be accomplished rapidly, and can be carried out with handheld or highly portable equipment. It is easily incorporated into the primary or secondary survey of trauma patients. Unlike more invasive procedures, serial FAST examinations can be performed to detect evolving injuries (i.e., accumulation of blood in the pericardium) or to reassess a patient when there are significant hemodynamic changes.

The FAST examination is included in both the 1° and 2° patient surveys outlined in Advanced Trauma Life Support (ATLS) guidelines.[5] Under "C" of the "ABCs" of the ATLS 1° survey, the physician is advised to "consider the diagnosis of cardiac tamponade" and urged to rapidly perform the FAST examination (particularly the pericardial view) for patients "with shock that is unresponsive to volume, particularly with penetrating chest trauma" as this "may confirm the diagnosis" (Figure 18.1). Most surgeons would argue that patients with penetrating chest trauma and hemodynamic instability should undergo emergent sternotomy or thoracotomy without delay, even for the brief period of time it would take to perform a FAST examination. Furthermore, ATLS guidelines state that the FAST examination should be considered during the 2° survey of patients with blunt abdominal trauma.

At exactly what point during a trauma assessment the clinician performs the rapid visualization of the



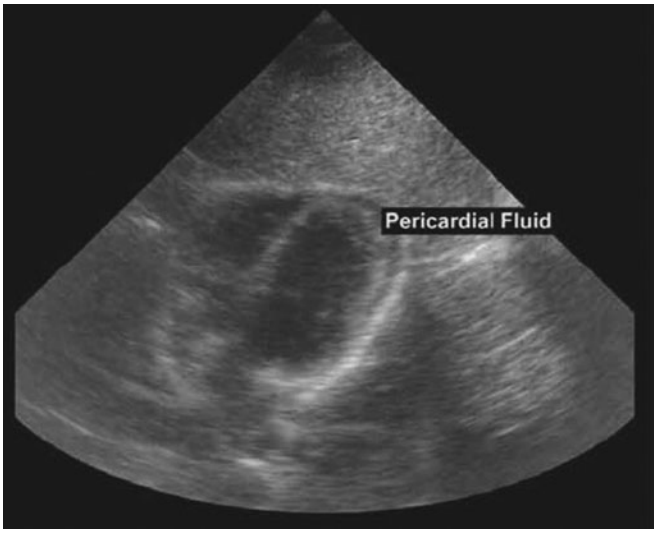
A



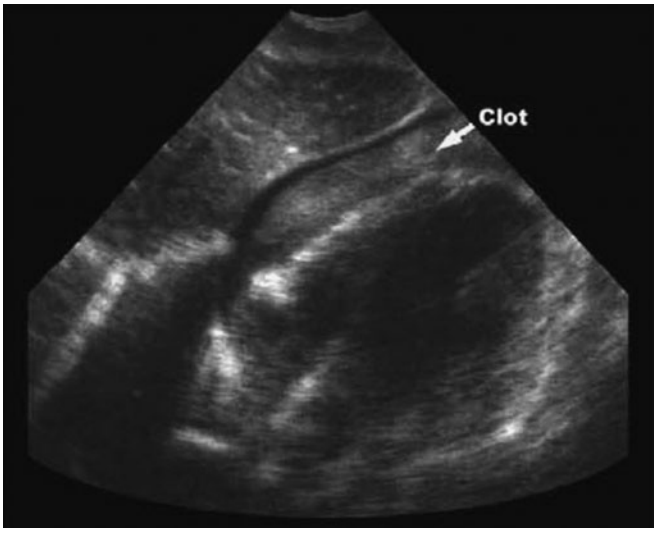
B

Figure 18.1. The FAST examination in the pericardial window view. (A) Location of the transducer to obtain cardiac views. (B) Normal cardiac anatomy and liver. LA indicates left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle. (Reproduced with permission from Dr. RA Jones.)

heart and pericardium, in isolation or as part of the FAST examination, is variable. Unlike computerized tomography (CT), a FAST examination can be performed during resuscitation and concurrent with other assessments and interventions. While the exact timing of this examination is variable, it is critical to look for hemopericardium early in the assessment of patients with chest trauma that are not immediately taken to the operating room for exploration (Figure 18.2). If the FAST examination is not performed by one clinician while another clinician undertakes 1° and 2° surveys, it should be included as part of the 2° survey.[5] Formal echocardiography performed by an ultrasonographer or cardiologist with all of the conventional windows is rarely used in a rapid trauma assessment and resuscitation. Instead, the simple subxiphoid or parasternal cardiac views obtained during the FAST examination are more likely to be used to rapidly detect hemopericardium by



A



B

Figure 18.2. The FAST examination in the pericardial window view. (A) Cardiac window and presence of a pericardial effusion. (B) Cardiac window with clot in the pericardial space. (Reproduced with permission from Dr. RA Jones.)

the surgical or emergency medical staff involved in the acute resuscitation.

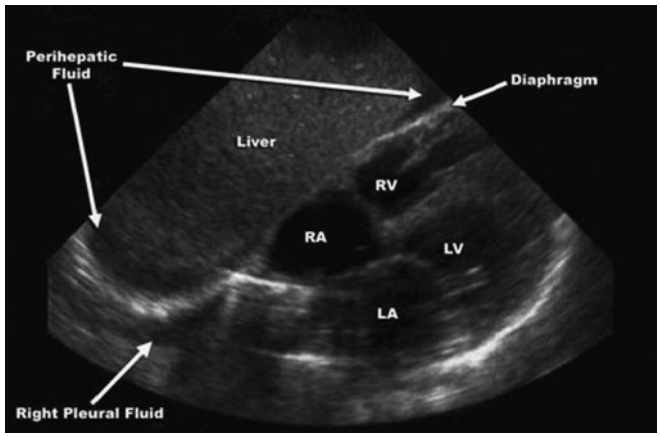
The overriding principle for evaluation of patients with potential cardiac trauma is early diagnosis and rapid surgical repair. Salvage rates increase with the detection of hemopericardium before hypotension ensues.[6] The use of diagnostic pericardiocentesis and subxiphoid pericardial windows has been markedly reduced by the increasing availability of bedside ultrasound. Patients may be spared thoracotomy and the exploration of penetrating chest wounds when hemodynamically stable if the bedside echocardiogram fails to show a pericardial effusion.

Use of beside ultrasound for the detection of hemopericardium in the patient with potential cardiac or chest trauma does have limitations (Table 18.1). Subcutaneous emphysema, pneumopericardium, and lung movement from mechanical ventilation may all make obtaining good ultrasonographic windows difficult. Pain or tenderness with ultrasound probe placement, spinal immobilization, and ongoing procedures may also make image acquisition difficult. Narrow intercostal spaces may make a parasternal or transthoracic view impossible. The examination is also challenging in the presence of obesity, muscular chest physique, hyperinflated lung changes associated with obstructive lung disease, calcified rib cartilage, and abdominal distention.

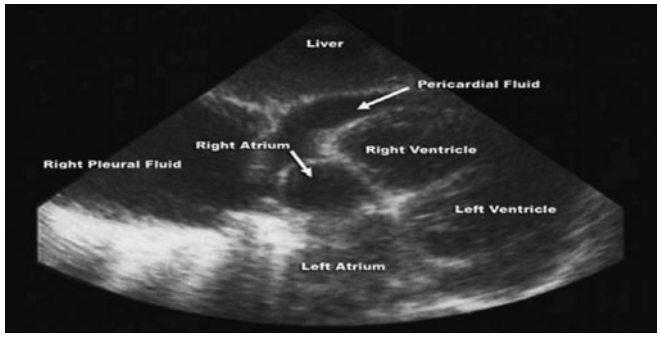
TABLE 18.1. Limitations of bedside ultrasound for the detection of hemopericardium in trauma patients

Subcutaneous emphysema
Presence of chest tubes
Pneumopericardium
Lung movement from mechanical ventilation
Pain or tenderness at site of probe placement
Restraining devices/straps associated with spinal immobilization
Procedures occupying the chest wall or subcostal space
Obesity
Muscular chest
Hyperinflated lungs (COPD)
Calcified rib cartilage
Abdominal distention

COPD indicates chronic obstructive pulmonary disease.



A



B

Figure 18.3. The FAST examination in the pericardial window view. (A) Cardiac window and presence of a right pleural effusion as well as perihepatic fluid. (B) Cardiac window with both pericardial and pleural effusions. (Reproduced with permission from Dr. RA Jones.)

Care must be taken to differentiate pleural fluid from pericardial fluid (Figure 18.3). In the parasternal long-axis view, a pericardial effusion tends to taper as it approaches the left atrium. The best way to differentiate between pericardial and pleural fluid is to note that the descending aorta can only be separated from the left atrium by a pericardial effusion but not by a pleural effusion. Absence of an echo-free space between the descending aorta and the left atrium makes any retrocardiac fluid collection more likely pleural than pericardial in origin.

Emphasis should be placed on the detection of pericardial fluid posterior to the left ventricle because anterior fluid may be negligible with small (or early) effusions. Of importance, anterior epicardial fat can cause an echo-free space that may be mistaken for hemopericardium. In general, with a large pericardial effusion, the heart tends to settle posteriorly, resulting in greater accumulation of fluid anteriorly. Conversely,

TABLE 18.2. Estimated right-sided volume/pressure status of trauma patient at time of cardiac ultrasound

	Euvolemia	*Hypovolemia*	*Chronic RV/RA pressure elevation (and/or RV hypertrophy)*	
Clinical examples	• Hemorrhage with prior volume resuscitation • Isolated injury	• Significant hemorrhage • Ongoing hemorrhage despite volume resuscitation • Multiple traumatic injuries	• Pulmonary hypertension	
Elevated CVP or detectable JVD?	YES (acute increase)	NO ("low pressure tamponade")	YES (chronically elevated)	
IVC status	Plethoric; Blunted respiratory variation	Collapsed	Plethoric; Variable respiratory variation	
RV diastolic collapse likely with large pericardial effusion?	YES	YES	Euvolemia Hypovolemia	NO Hypertrophy or elevated RA/RV pressures may prevent right-sided collapse
Response to IV fluids?	May prevent right-sided collapse	May prevent right-sided collapse	Euvolemia	RV was likely not collapsing prior to volume infusion May prevent right-sided collapse

CVP indicates central venous pressure; JVD, jugular venous distension; IV, intravenous; IVC, inferior vena cava; RA, right atrium; RV, right ventricle.

with smaller pericardial effusions, fluid tends to accumulate posteriorly.

The instance in which circulating blood volume is so low that clinical evidence of tamponade is lacking is referred to as "low-pressure tamponade." While jugular venous distention may not be present in low-pressure tamponade, one should still see echocardiographic evidence of right ventricular diastolic collapse and atrial collapse. Exponential volume resuscitation, as is typical in hypotensive patients with traumatic injuries, may increase right atrial and ventricular pressures to the point that the right-sided collapse disappears.

Right-sided collapse may also be absent in patients with a hypertrophied right ventricle or elevated right-sided chamber pressures. Under normal physiologic circumstances, the low pressure chambers and thin walls of the right atrium and ventricle are easily compressible with elevated pericardial pressure. Table 18.2 demonstrates how intravascular volume status at the time of ultrasound examination and cardiac function prior to a traumatic injury affects the ability to detect classical findings associated with cardiac tamponade.

Patients with preexisting, nontraumatic pericardial effusions are at high risk for developing cardiac tamponade. Hypovolemia induced by noncardiac trauma can cause a reduction in preload and right-sided pressures to the point that pressure in the pericardial compartment exceeds that in the right-sided cardiac chambers. While emergent thoracotomy and pericardiotomy is typically reserved for patients who have sustained penetrating chest trauma, this procedure can be lifesaving in patients with blunt trauma who develop cardiac tamponade as a result of acute hypovolemia and a preexisting pericardial effusion.[7]

BLUNT (NONPENETRATING) CARDIAC TRAUMA

The use of echocardiography in patients with blunt cardiac trauma is directed at the diagnosis of aortic

TABLE 18.3. Potential cardiac injuries and abnormalities associated with blunt chest wall trauma

Aortic transection
Blunt cardiac injury (formerly myocardial contusion; especially right ventricle dyskinesia)
Septal rupture
Free wall rupture
Valvular disruption (especially tricuspid valve)
Coronary artery thrombosis (especially left anterior descending)
Cardiac failure
Minor ECG or enzyme abnormality
Complex arrhythmia
Pericardial effusion/tamponade

ECG indicates electrocardiographic.

TABLE 18.4. Echocardiographic findings associated with aortic rupture after blunt chest trauma

1. Abrupt and discrete change in aortic diameter (normal diameter proximal and distal to the site of rupture; lumen is widened at site of rupture)
2. Presence of one or more consistent linear echoes indicative of transaction flaps dividing the aortic lumen into two or more lumina
3. Focal but complete intimal and medial disruption with formation of a pseudoaneurysmatic cavity
4. Color doppler identification of a pseudocoarctation pattern (flow acceleration through the injured aortic segment)
5. Periaortic hematoma

transection, blunt cardiac injury (formerly referred to as cardiac contusion), cardiac rupture, and valvular disruption (Table 18.3).

Aortic Transection

Aortic transection should be suspected in any patient who has sustained a high-speed deceleration injury to the chest and has an abnormal chest x-ray, specifically, a widened mediastinum. Aortography is considered the "gold standard" diagnostic test for evaluation of the blunt trauma patient with a widened mediastinum. Aortography, however, is time consuming and involves transfer of a potentially very unstable patient to the radiology suite. The portable chest x-ray obtained as part of a rapid trauma assessment is a suboptimal screening study for aortic injury due to limited exposure capability, expiratory views, patient motion, difficult patient positioning, and magnification and distortion of the mediastinum in the supine position. Additionally, the positive yield for patients with a widened mediastinum is rather low; <20% of patients with a widened mediastinum have an aortic injury, while up to 28% of those with aortic rupture have a normal chest x-ray.[8]

Table 18.4 lists the echocardiographic findings associated with aortic rupture after blunt chest trauma. Many different criteria for the diagnosis of traumatic aortic rupture exist.[9–11] Periaortic hematomas may arise from multiple other injuries that produce bleeding in the mediastinum, including tearing of mediastinal veins, rib fractures, sternal fractures, injury to the brachiocephalic vessels, and caudal dissection of blood from cervical injuries.

Most patients with aortic transection will die unless the bleeding is trapped by soft tissue and a false aneurysm is produced.[12] Only 20% of patients with aortic rupture survive for >1 hour after injury, and 80% of patients die at the scene.[13] In initial survivors, if the injury is not surgically repaired, 40% die within 24 hours and 90% are dead by 10 weeks. Echocardiography, particularly transesophageal echocardiography (TEE), is very accurate in the identification of aortic rupture. Studies comparing transthoracic (TTE) with TEE for evaluation of aortic injury after blunt chest trauma support the utilization of TEE whenever possible. Image quality is frequently suboptimal with TTE due to associated chest wall injuries. Ninety percent of aortic ruptures occur at the aortic isthmus, a region that is impossible to visualize with a TTE. A major disadvantage of TEE is that it often requires the presence of a cardiologist or trained specialist. Other advantages and disadvantages of TEE are outlined in Table 18.5.

Blunt Cardiac Injury (Cardiac Contusion)

Some experts use the term "blunt cardiac injury" to refer only to wall motion abnormalities following chest trauma (the classical "cardiac contusion"), while others include all injuries to cardiac structures caused by blunt chest wall trauma under the generic term of "blunt cardiac injury." For our purpose, "myocardial" or "cardiac contusion" is now called "blunt cardiac

TABLE 18.5. Transesophageal echocardiography for detection of aortic injury after blunt cardiac trauma

Advantages	*Disadvantages*
1. Markedly improved image quality compared with TTE	1. Semiinvasive procedure
2. Can be performed concurrent to other diagnostic, therapeutic, and resuscitative measures	2. Potential complications in patients with esophageal or gastric trauma
3. Reduces need for aortography and decreases time to definitive surgical repair	3. May cause excessive head and neck movement in patients with cervical spine injuries
4. Greater accuracy for detection of aortic rupture	4. Poor visualization of the distal ascending aorta and proximal aortic arch (due to the interposition of the air-filled trachea and left main bronchus)
5. Better visualization of the aortic isthmus	

TTE indicates transthoracic echocardiography.

injury" across the trauma literature. The precise definition and criteria required to make the diagnosis of a blunt cardiac injury remains controversial. However, blunt cardiac injury has been defined as the presence of wall motion abnormalities detected by echocardiography, including either, or both, ventricles, following nonpenetrating chest trauma in the absence of transmural myocardial infarction detected on electrocardiogram (ECG). Although prior wall motion abnormalities may exist, the diagnosis is made on clinical grounds when there is a high index of suspicion for traumatic cardiac injury. The left anterior descending coronary artery, tricuspid valve, and right ventricle are the most commonly injured structures due to their proximity to the chest wall.

The reported incidence of blunt cardiac injury ranges from 8% to 71% in patients sustaining blunt chest trauma. In one case series of victims with fatal nonpenetrating chest trauma, 15% had blunt cardiac injury demonstrated at autopsy.[14] Patients with significant chest trauma (i.e., multiple rib fractures, pulmonary contusions, hemothorax, major intrathoracic vascular injury) have an estimated 13% incidence of significant blunt cardiac injury.

Patients with blunt cardiac injury may present with a wide spectrum of signs and symptoms ranging from asymptomatic to severe cardiac compromise. Arrhythmias and conduction defects are the most common complications of blunt cardiac injury. There are many reasons why a patient with blunt chest trauma may be hypotensive, hypoxic, tachycardic, or in heart failure. These include pulmonary contusion, pneumothorax, hemorrhagic shock, and cardiac tamponade. A thorough investigation to rule out these potentially reversible causes of pump failure is essential before focusing on blunt cardiac injury as the etiology.

Current trauma guidelines recommend an admission ECG for all patients with suspected blunt cardiac injury.[15] Hemodynamically stable patients with a normal ECG require no further workup for blunt cardiac injury, as the risk of serious complications is minimal. If the admission ECG is abnormal, the patient should be admitted for continuous ECG monitoring for 24 to 48 hours. Echocardiography is recommended only in the patient with suspected blunt cardiac injury that causes hemodynamic compromise. If adequate windows on the chest wall are unobtainable, a TEE should be performed. Presence of a sternal fracture does not predict the presence of blunt cardiac injury and, thus, is not an indication for cardiac monitoring or ECG evaluation.[15]

Of importance, however, echocardiography should not be used as a primary screening modality but should be reserved for patients with hemodynamic instability of unclear etiology, an abnormal ECG, or cardiac arrhythmias with documented risk of blunt cardiac injury.[10,16–19] Creatine kinase levels and cardiac specific markers (CK-MB, cTnI) may all be elevated in trauma patients for a variety of reasons and should not be used as a screening tool for blunt cardiac injury.[20] Repeat echocardiography in patients with blunt cardiac injury will often show normalization of wall motion abnormalities.

The same physical examination findings that alert the clinician to the potential presence of blunt cardiac injury may limit the ability to perform a comprehensive TTE. Chest wall tenderness, a flail chest, or crepitus may all limit placement and manipulation of the ultrasound probe. Near-field artifact during TTE limits

evaluation of the right ventricular outflow tract, a site which, in blunt chest trauma patients, can be focally injured. Thus, the provider should consider performing a TEE in patients with a very high clinical suspicion for blunt cardiac injury but a suboptimal or unrevealing transthoracic examination.

Patients with blunt abdominal trauma should always be screened for pericardial effusion and tamponade. This is a routine practice at most trauma centers where the FAST examination is performed in patients not immediately taken to the operating room. A quick screen for pericardial effusion and tamponade is particularly important in patients with blunt abdominal trauma who develop increasingly unstable hemodynamic parameters. Patients with a preexisting pericardial effusion can develop cardiac tamponade from acute hypovolemia related to blunt abdominal trauma.[7]

Cardiac Rupture

Cardiac rupture after blunt chest trauma is usually a rapidly fatal event. The precise incidence is unknown because many of these patients will die at the scene of the trauma. Rapid prehospital transportation, early diagnosis, and surgical repair offer the only chance for long-term survival. There are several case reports that describe the use of echocardiography in the ED to diagnose pericardial effusion in patients found later to have cardiac tears.[21,22]

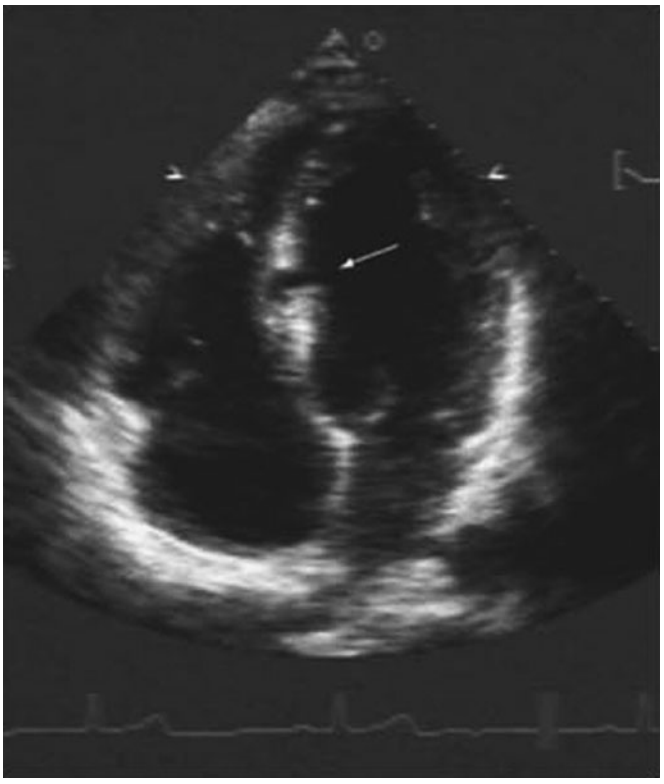


Figure 18.4. Transthoracic echocardiogram apical four-chamber view demonstrating incomplete transverse tear (arrow) of the interventricular septum. This image was obtained from a 50-year-old man involved in a 40 MPH motor vehicle crash into a tree. (Image courtesy of Lisa Motavalli, M.D. retrieved from http://cme.mcgill.ca/php/pre.php?id=597 and hosted by the McGill Faculty of Medicine.)

Other Intracardiac Injuries

Intracardiac injuries, such as septal defects or valvular lesions, are rare complications of blunt chest trauma. Echocardiography with Doppler capability can quickly and noninvasively identify and quantify these lesions.

Traumatic rupture of the interventricular septum is an uncommon entity but has been reported after blunt chest trauma. It rarely occurs as an isolated lesion. In an autopsy series by Parmley and colleagues, only 5 out of 546 cases of nonpenetrating injuries to the chest had isolated ventricular septal rupture.[13] Incomplete ventricular septal tears occur even less frequently. Rupture may be the consequence of a laceration of the septum from traumatic injury, or may be from a contused septum that subsequently becomes necrotic, and later ruptures.[23] A tear in the interventricular septum is more likely to arise when severe chest compression and abrupt deceleration occurs during late diastole or early systole when the ventricles are full and the valves are closed (Figures 18.4 and 18.5). This time period in which the heart is vulnerable to the proposed mechanism of injury is about 6.25% of the cardiac cycle.[24] The most common site of septal tear is in the muscular portion of the interventricular septum near the apex.[25]

Severe hypoxemia is not uncommon after blunt chest trauma and is usually the result of pulmonary injuries, intrapulmonary shunting, and altered chest wall mechanics. Valvular injury and dysfunction may explain severe hypoxemia when pulmonary and chest wall injuries are less severe. Traumatic tricuspid regurgitation with intracardiac right-to-left shunting through a patent foramen ovale has been reported after blunt chest trauma.[26] Injury to right heart structures is often well tolerated and the onset of distressing symptoms may be delayed.

There are case reports of rupture of both atrioventricular valves after blunt chest trauma.[27] For patients undergoing operative repair of a single atrioventricular valve injury, strong consideration should be given to performing an intraoperative TEE to exclude injury to the other atrioventricular valve.

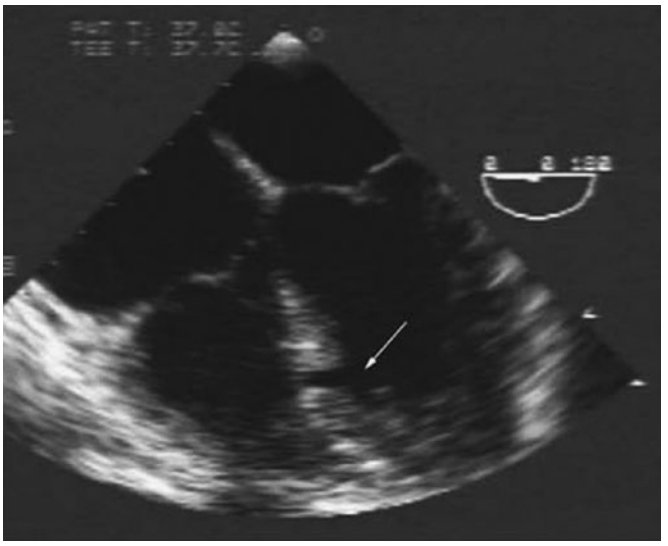


Figure 18.5. Transesophageal echocardiogram four-chamber view from a 50-year-old man involved in a 40 MPH motor vehicle crash into a tree (same patient as in Figure 18.4), demonstrating incomplete septal tear (arrow). Only a thin membrane in the area of the rupture separates the two ventricles. (Image courtesy of Lisa Motavalli, M.D. retrieved from http://cme.mcgill.ca/php/pre.php?id=597 and hosted by the McGill Faculty of Medicine.)

PENETRATING CARDIAC TRAUMA

Most patients with penetrating cardiac trauma die before reaching medical attention or present with rapidly progressive hemorrhagic shock culminating in cardiac arrest. Despite advances in prehospital and hospital-based emergency care over the last few decades, reported survival rates from penetrating cardiac trauma have only minimally improved. Reports of hospital survival after penetrating cardiac trauma may decline or not appear to be improving as prehospital emergency medical systems become better at resuscitating and rapidly delivering unstable patients to the hospital. The incidence of stab wounds and gunshot wounds among the subset of patients who actually make it to the ED with signs of life is roughly equal.[28]

Physiologic status of the patient at ED presentation has consistently been shown to be the best predictor of survival. When a patient arrives in the ED with no vital signs, even in the instance where prehospital care providers report signs of life just prior to hospital arrival, the mortality rate with vigorous resuscitation including thoracotomy approaches 100%. Patients that arrive with profound hypotension (systolic blood pressure <60 mm Hg) and agonal respiratory effort have a mortality rate near of 60%. Gunshot wounds of the heart are generally associated with 2–4 times the mortality of stab wounds to the heart. This is thought to be related to the surrounding tissue injury of the high-velocity projectile versus the low velocity of the stab instrument.[29,30]

The anatomic site of cardiac injury and the number of cardiac chambers injured affects prognosis. The right ventricle is affected more often than the left ventricle due to its anterior anatomic location, but left ventricular wounds carry a worse prognosis. The left or right atrium is affected in 20% of cases.[31] One third of penetrating cardiac wounds affect multiple chambers, and the relative risk of death is 2.5 times greater than single-chamber injuries.[32] In 5% of cases, a coronary artery is lacerated. These injuries usually involve a distal segment of the artery and rarely produce significant acute myocardial infarction when they are ligated, whereas more proximal coronary artery lacerations require coronary bypass.[33] Rarely, the interventricular septum, a valve, papillary muscle, or chordae tendineae is injured, producing an acute shunt or valvular insufficiency. These lesions are poorly tolerated and can quickly cause massive pulmonary edema and cardiogenic shock. Intrapericardial aortic injuries are almost universally fatal.

Penetrating cardiac injury may result in exsanguinating hemorrhage if the cardiac lesion communicates freely with the pleural cavity or cardiac tamponade if the hemorrhage is contained within the pericardium. The reported incidence of acute pericardial tamponade is approximately 2% in patients with penetrating trauma to the chest and upper abdomen. Cardiac tamponade is itself a life-threatening condition, but appears to offer some degree of protection and increased survival in patients with penetrating cardiac wounds.[32,34] It occurs more commonly with stab wounds than with gunshot wounds, and 60–80% of patients with stab wounds involving the heart develop tamponade. Intermittent hemorrhage from the intrapericardial space may cause an intermittently decompressing tamponade. This latter condition may be tolerated for a longer period.

Hypotension, cardiopulmonary resuscitation, nonthoracic injuries (other significant, noncardiac injuries), and emergency thoracotomy are all risk factors that have been associated with death after penetrating cardiac trauma. Emergency department thoracotomy is associated with a higher mortality rate than operating-room thoracotomy. Blood pressure >90 mm Hg and pericardial tamponade are prognostic signs often associated with improved survival in patients with penetrating cardiac trauma.[32,34] Clinical

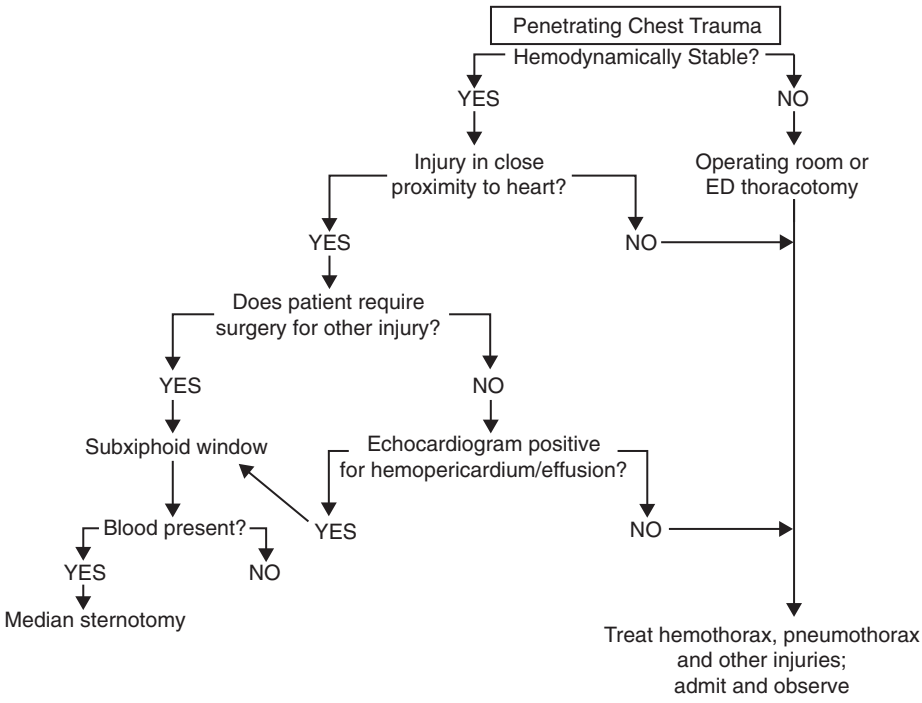


Figure 18.6. Algorithm incorporating bedside echocardiogram into the evaluation of penetrating chest trauma.

signs of pericardial tamponade in penetrating cardiac injuries are the exception rather than the rule. Given the paradoxical protective effect of pericardial tamponade on patients with cardiac injuries, patients who survive the acute stage after penetrating cardiac trauma (i.e., those who make it to the hospital alive) are more likely to have tamponade. It is critical to identify patients with cardiac injury (and tamponade) early, before the "protective" effect is lost. Figure 18.6 integrates a rapid bedside 2D echocardiogram (usually performed within the context of the FAST examination) into the assessment of a patient with penetrating chest trauma.

Few patients with significant penetrating cardiac trauma present without symptoms. In rare instances, a patient may present with much delayed symptoms of penetrating cardiac trauma. Echocardiography is invaluable in evaluating this subset of patients.

Two-dimensional echocardiography has the capacity to visualize cardiac defects very well. It also has the advantage of being able to evaluate left ventricular function and to determine whether there is pericardial effusion. Doppler echocardiography can be used to further visualize flow from one compartment to another. Two-dimensional echocardiography is very sensitive and specific in patients without hemothorax (sensitivity 100% and specificity 89%) but is less useful when the patient has a hemothorax (sensitivity 56% and specificity 93%).[2,35] An explanation for the poor sensitivity of this test in the subset of patients with hemothoraces lies in the fact that many patients with penetrating injury to cardiac structures lacerate the pericardium. Pericardial blood escapes into the pleural cavity, resulting in an empty pericardial space and the presence of a hemothorax.

One multicenter study of surgeon-performed ultrasound demonstrated a sensitivity of 100% and specificity of 97% for the detection of hemopericardium in penetrating precordial injuries, resulting in minimal delay to operation.[36] Another demonstrated 100% sensitivity and 99.3% specificity in patients with penetrating precordial or transthoracic trauma.[37] Emergency physicians using bedside ultrasound demonstrated that they could reliably examine the pericardium for effusion with an accuracy of 97.5%.[38] Time to diagnosis and disposition to the operating room is significantly reduced and survival increased for patients with penetrating cardiac injuries when 2D echocardiography is included in the initial trauma assessment.[39]

Pericardiocentesis is unreliable for the detection of hemopericardium in the setting of penetrating chest trauma with an approximate 20% false-positive and false-negative rate. The most sensitive test for detection of occult cardiac injury and posttraumatic

tamponade is a (subxiphoid) pericardial window,[40] but this invasive intervention requires general anesthesia in the operating room. There has been a significant drop in the performance of pericardiocentesis and subxiphoid exploration with the advent of bedside ultrasound. Similarly, utilization of pericardiocentesis as a temporary therapeutic maneuver has been abandoned and emergency thoracotomy is being used with increasing frequency. For patients undergoing laparotomy for abdominal injuries, subxiphoid exploration and pericardiotomy can be performed in the operating room if there is a suspicion of cardiac injury.

Penetrating cardiac injuries are highly lethal injuries that can present with normal hemodynamic parameters or cardiac arrest. Pericardial tamponade is the most reversible etiology that produces cardiac arrest in patients with penetrating injuries. A hemodynamically unstable patient with a penetrating thoracic injury should proceed to emergent thoracotomy without any ultrasound or other imaging. During the initial evaluation of the hemodynamically stable patient, a focused examination for the detection of hemopericardium can be performed in the context of the FAST examination pericardial views. Because it is rapid and easily repeatable, bedside cardiac ultrasound is especially useful for the assessment of patients with thoracic wounds, not only to establish the presence or absence of hemopericardium, but also to reevaluate a patient who suddenly becomes hypotensive.

MISCELLANEOUS TRAUMA

Endocardial biopsies from patients who have sustained significant electrical shocks have shown patchy endomyocardial necrosis.[41] Echocardiographic changes following alternating current (AC) electrocution have shown left ventricular dysfunction with reduced fractional shortening as well as global hypokinesis. These changes may resolve over time. Similar findings occur with higher voltage injuries, but the changes are more likely to be permanent.[42]

IATROGENIC CARDIAC TRAUMA

Cardiac perforation with resultant pericardial effusion is a complication that may occur in the cardiac catheterization laboratory. An intravascular ultrasonic catheter placed in the right atrium can detect pericardial fluid. Pacemaker and implantable cardiac defibrillator leads may penetrate into the myocardium and perforate the right ventricular free wall. The risk of perforation is minimized when the lead is screwed into the thicker ventricular septum at the right ventricular apex rather than the ventricular free wall. Patients with prior myocardial infarction or dilated cardiomyopathy with thinner ventricular walls may be at greater risk for this complication. Lead repositioning and/or explantation should be performed in the operating room with surgical backup under TEE guidance.[43]

TABLE 18.6. Goal-directed echocardiography for cardiac trauma

Nonpenetrating cardiac trauma	*Penetrating cardiac trauma*
1. Obtain subcostal, parasternal long-axis and apical four-chamber views	1. Obtain subcostal and parasternal long-axis views
2. Assess for the presence of a pericardial effusion, which suggests mediastinal trauma or possible cardiac rupture	2. Assess for the presence of a pericardial effusion, which suggests myocardial laceration or perforation
3. Assess wall motion for dyskinesis associated with blunt myocardial injury	3. Evaluate the size, location, and hemodynamic effects of the effusion, focusing particularly on the presence of RA and RV diastolic collapse
4. Evaluate for valvular dysfunction or ventricular septal defect	4. Identify a "safe" track for therapeutic pericardiocentesis, if indicated
5. Assess the thoracic aorta for the presence of hematoma, intimal flap, and altered contour, suggesting aortic transection	5. Perform serial examinations for patients under observation or with sudden clinical or hemodynamic changes
6. Consider TEE for improved visualization of the aorta and to improve diagnostic accuracy	

RA indicates right atrium; RV, right ventricle; TEE, transesophageal echocardiography.

CONCLUSIONS

Intensivists must recognize the tension between the need for accurate noninvasive diagnosis of injury and the need for immediate surgical intervention in patients presenting with cardiac and thoracic trauma. The goal of the intensivist should be to perform limited, goal-directed ultrasonography, a philosophy that has been promoted by emergency medicine physicians (Table 18.6). The noncardiologist critical care provider should not be expected to identify subtle cardiac abnormalities. The goal of cardiac imaging in trauma should be to rapidly answer specific binary (yes or no) questions to help determine the presence or absence of disease, specifically a hemopericardium or other life-threatening cardiac injury, and whether or not emergency surgical intervention is indicated.

References

1. Shorr RM et al. Blunt thoracic trauma. Analysis of 515 patients. *Ann Surg.* 1987;206:200–205.
2. Nagy KK et al. Role of echocardiography in the diagnosis of occult penetrating cardiac injury. *J Trauma.* 1995;38: 859–862.
3. Ivatury RR et al. Penetrating cardiac injuries: twenty-year experience. *Am Surg.* 1987;53:310–317.
4. Rozycki GS et al. A prospective study of surgeon-performed ultrasound as the primary adjuvant modality for injured patient assessment. *J Trauma.* 1995;39:492–498; discussion 498–500.
5. American College of Surgeons. *ATLS Advanced Trauma Life Support Program for Doctors.* 7th ed. Chicago, IL; 2004.
6. Rohman M et al. Emergency room thoracotomy for penetrating cardiac injuries. *J Trauma.* 1983;23:570–576.
7. Menaker J et al. Ultrasound-diagnosed cardiac tamponade after blunt abdominal trauma-treated with emergent thoracotomy. *J Emerg Med.* 2007;32:99–103.
8. Goarin JP et al. Early diagnosis of traumatic thoracic aortic rupture by transesophageal echocardiography. *Chest.* 1993;103:618–620.
9. Smith DC, Bansal RC. Transesophageal echocardiography in the diagnosis of traumatic rupture of the aorta. *N Engl J Med.* 1995;333:457–458.
10. Brooks SW et al. The use of transesophageal echocardiography in the evaluation of chest trauma. *J Trauma.* 1992;32:761–765; discussion 765–768.
11. Shapiro MJ et al. Cardiovascular evaluation in blunt thoracic trauma using transesophageal echocardiography (TEE). *J Trauma.* 1991;31:835–839; discussion 839–840.
12. Snow CC et al. Diagnosis of aortic transection by transesophageal echocardiography. *J Am Soc Echocardiogr.* 1992;5:100–102.
13. Parmley LF, Manion WC, Mattingly TW. Nonpenetrating traumatic injury of the heart. *Circulation.* 1958;18:371–396.
14. Kissane RW. Traumatic heart disease; nonpenetrating injuries. *Circulation.* 1952;6:421–425.
15. EAST Practice Parameter Workgroup for Screening of Blunt Cardiac Injury: Pasquale MD, Nagy K, Clarke J. *Practice Management Guidelines for Screening of Blunt Cardiac Injury.* Eastern Association for the Surgery of Trauma; 1998. Available at http://www.east.org/tpg/chap2.pdf. Accessed on March 25, 2009.
16. Cachecho R, Grindlinger GA, Lee VW. The clinical significance of myocardial contusion. *J Trauma.* 1992;33:68–71; discussion 71–73.
17. Christensen MA, Sutton KR. Myocardial contusion: new concepts in diagnosis and management. *Am J Crit Care.* 1993;2:28–34.
18. Karalis DG et al. The role of echocardiography in blunt chest trauma: a transthoracic and transesophageal echocardiographic study. *J Trauma.* 1994;36:53–58.
19. Malangoni MA, McHenry CR, Jacobs DG. Outcome of serious blunt cardiac injury. *Surgery.* 1994;116:628–632; discussion 632–633.
20. Ferjani M et al. Circulating cardiac troponin T in myocardial contusion. *Chest.* 1997;111:427–433.
21. Baxa MD. Cardiac rupture secondary to blunt trauma: a rapidly diagnosable entity with two-dimensional echocardiography. *Ann Emerg Med.* 1991;20:902–904.
22. Schiavone WA et al. The use of echocardiography in the emergency management of nonpenetrating traumatic cardiac rupture. *Ann Emerg Med.* 1991;20:1248–1250.
23. Ilia R et al. Spontaneous closure of a traumatic ventricular septal defect after blunt trauma documented by serial echocardiography. *J Am Soc Echocardiogr.* 1992;5:203–205.
24. Rutherford EJ et al. Immediate isolated interventricular septal defect from nonpenetrating thoracic trauma. *Am Surg.* 1993;59:353–354.
25. Rollins MD et al. Traumatic ventricular septal defect: case report and review of the English literature since 1970. *J Trauma.* 2005;58:175–180.
26. Ribichini F et al. Subacute tricuspid regurgitation with severe hypoxemia complicating blunt chest trauma. *Chest.* 1996;109:289–291.
27. Bailey PL et al. Mitral and tricuspid valve rupture after moderate blunt chest trauma. *Ann Thorac Surg.* 2000; 69:616–618.
28. Mittal V et al. Penetrating cardiac injuries. *Am Surg.* 1999; 65:444–448.

29. DeGennaro VA et al. Aggressive management of potential penetrating cardiac injuries. *J Thorac Cardiovasc Surg.* 1980;79:833–837.
30. Marshall WG Jr, Bell JL, Kouchoukos NT. Penetrating cardiac trauma. *J Trauma.* 1984;24:147–149.
31. Brown J, Grover FL. Trauma to the heart. *Chest Surg Clin N Am.* 1997;7:325–341.
32. Rhee PM et al. Penetrating cardiac injuries: a population-based study. *J Trauma.* 1998;45:366–370.
33. Wall MJ Jr et al. Acute management of complex cardiac injuries. *J Trauma.* 1997;42:905–912.
34. Tyburski JG et al. Factors affecting prognosis with penetrating wounds of the heart. *J Trauma.* 2000;48:587–590; discussion 590–591.
35. Meyer DM, Jessen ME, Grayburn PA. Use of echocardiography to detect occult cardiac injury after penetrating thoracic trauma: a prospective study. *J Trauma.* 1995;39:902–907; discussion 907–909.
36. Rozycki GS et al. The role of ultrasound in patients with possible penetrating cardiac wounds: a prospective multicenter study. *J Trauma.* 1999;46:543–551; discussion 551–552.
37. Rozycki GS et al. Surgeon-performed ultrasound for the assessment of truncal injuries: lessons learned from 1540 patients. *Ann Surg.* 1998;228:557–567.
38. Mandavia DP et al. Bedside echocardiography by emergency physicians. *Ann Emerg Med.* 2001;38:377–382.
39. Plummer D et al. Emergency department echocardiography improves outcome in penetrating cardiac injury. *Ann Emerg Med.* 1992;21:709–712.
40. Trinkle JK et al. Affairs of the wounded heart: penetrating cardiac wounds. *J Trauma.* 1979;19:467–472.
41. Jensen PJ et al. Electrical injury causing ventricular arrhythmias. *Br Heart J.* 1987;57:279–283.
42. Homma S, Gillam LD, Weyman AE. Echocardiographic observations in survivors of acute electrical injury. *Chest.* 1990;97:103–105.
43. Merla R et al. Late right ventricular perforation and hemothorax after transvenous defibrillator lead implantation. *Am J Med Sci.* 2007;334:209–211.

CHAPTER 19

Echocardiographic Evaluation of Cardiopulmonary Interactions

Antoine Vieillard-Baron

INTRODUCTION

Cardiopulmonary interactions are not a new issue. A PubMed search (www.ncbi.nlm.nih.gov/PubMed) using the keywords "heart–lung interactions" yielded more than 100 references published since 1962. But heart–lung interactions are not just of interest in terms of discussions among physiologists. They also have a strong impact on our ability to accurately interpret hemodynamic measurements and blood gas analysis and to manage patients in the intensive care unit (ICU). In many situations, such as acute respiratory distress syndrome (ARDS), severe asthma, chronic obstructive pulmonary disease (COPD), and mechanical ventilation, heart–lung interactions may account for considerable hemodynamic and blood gas changes. For example, Kumar and colleagues first suggested that positive end-expiratory pressure (PEEP)-induced oxygenation improvement could actually be due to a marked decrease in cardiac index rather than to an improvement in lung recruitment.[1]

Echocardiography is currently the most accurate tool to evaluate cardiac function beat by beat during the respiratory cycle. It does not require highly sophisticated equipment, but only the opportunity to correctly visualize the heart in either spontaneously breathing or mechanically ventilated patients. The study of cardiopulmonary interactions requires two-dimensional, time-motion, and Doppler modes. Visualization of the airway pressure tracing on the screen of the machine is mandatory.[2] We have exhaustively illustrated the different mechanisms promoting heart–lung interactions[3] and have also developed a Web site devoted to echocardiography in the ICU, a large part of which covers heart–lung interactions.[4]

The objectives of this chapter are to briefly report the mechanisms that promote heart–lung interactions, to illustrate how they can be easily described using echocardiography, and to discuss their clinical implications in the ICU. At the end, readers will better understand how to use echocardiography and how fundamental heart–lung interactions are to effective patient management in the ICU.

PHYSIOLOGICAL REMINDERS

Cardiopulmonary interactions are mainly related to the fact that a circulation, the pulmonary circulation, is interposed between the right heart and the left heart. This circulation contains close to 500 milliliters (mL) of blood. So, whereas the preload reserve for the right heart is the systemic venous circulation,[5] the preload reserve for the left heart is actually pulmonary capillaries and veins.[6] For a given cardiac function, the more blood there is in the pulmonary circulation, the higher the left ventricular stroke volume.[6]

Heart–lung interactions can be separated according to their direct and indirect consequences for cardiac function. The main consequences are direct and are promoted by changes in intrathoracic and transpulmonary pressures and have been extensively reported by Scharf and collegues since 1977.[7] They are usually described as the respiratory variations in systolic and pulse pressures, reflecting respiratory variations in left ventricular stroke volume. In spontaneously breathing patients, these variations are called the "pulsus paradoxus" and are observed in diseases such as severe asthma, where left ventricular stroke volume, and thereby pulse pressure, decrease during inspiration and increase during expiration.[8] In mechanically ventilated patients, they are called "reverse pulsus paradoxus"[9,10] because, conversely, pulse pressure increases during inspiration and decreases at expiration. (Figure 19.1).[11] Briefly and simply, increased intrathoracic pressure will induce a decrease in systemic venous return by reducing the pressure gradient between the periphery and the right atrium (Figure 19.2),[5] and also by increasing the resistance to flow.[12] Many clinical situations may be responsible for this: PEEP

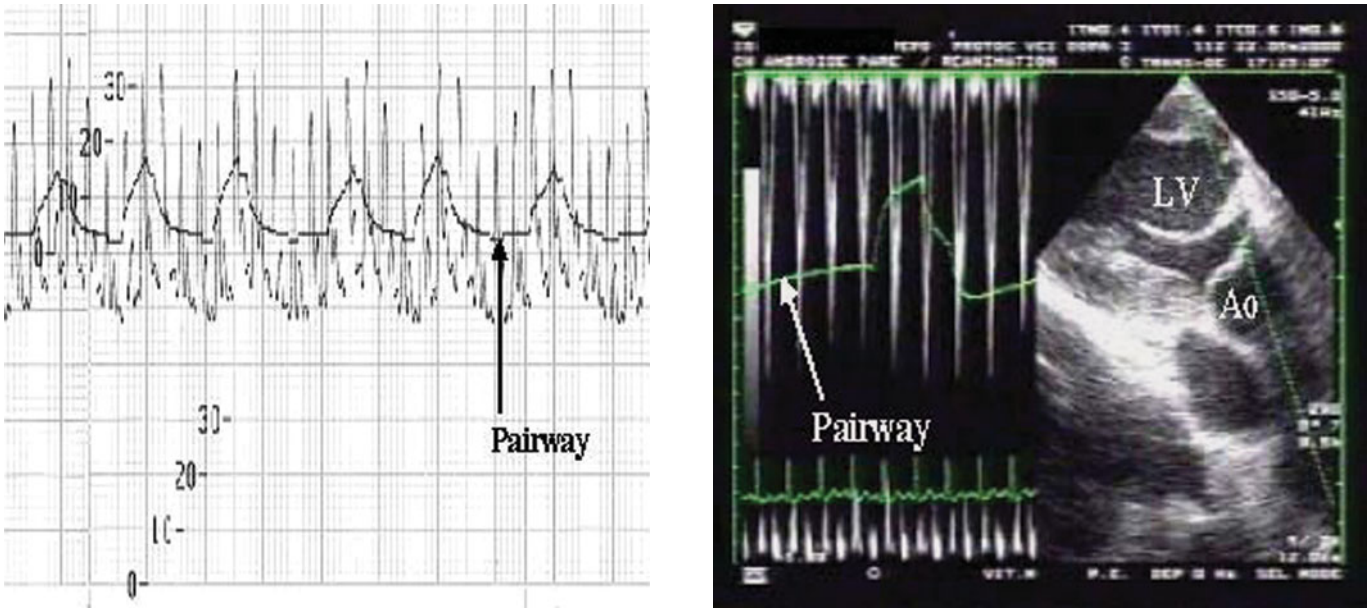


Figure 19.1. Respiratory variations in blood pressure (panel A) and left ventricular stroke volume (panel B) in a mechanically ventilated patient. Left ventricular stroke volume was evaluated by recording the flow into the left outflow track (pulsed Doppler mode). Pulse pressure and left ventricular stroke volume increased during inspiration, and decreased during expiration. Ao indicates aorta; LV, left ventricle; pairway, pressure in the trachea.

(either intrinsic, as in exacerbation of COPD and in severe asthma, or therapeutic), controlled ventilation in pressure or volume, decreased chest wall compliance. Increased transpulmonary pressure will augment right ventricular afterload by its ability to act on the pulmonary capillaries and so to increase pulmonary vascular resistance (Figure 19.3).[13,14] But it is seen when the distending pressure of the lung is high, and so especially occurs in ARDS,[15] where lung compliance is severely depressed, or in severe asthma,[16] where lung overdistension is present.

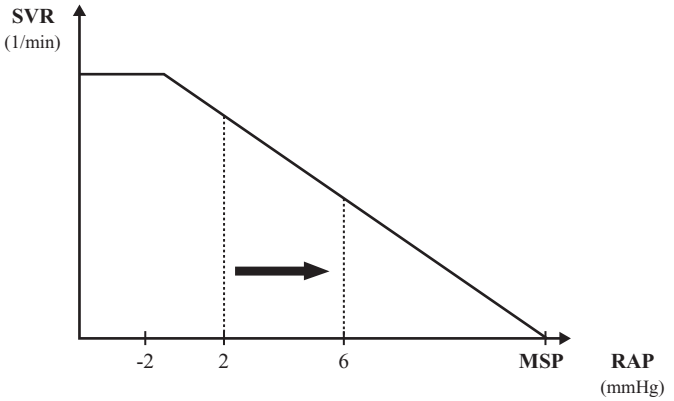


Figure 19.2. Guyton's representation of the systemic venous return (SVR), which depends on the pressure gradient between the mean systemic pressure (MSP) and the right atrial pressure (RAP). Any increase in RAP induces a decrease in SVR.

Indirect consequences of heart–lung interactions are promoted by changes in PaO_2, $PaCO_2$, and pH, according to change in ventilation or ventilatory settings, and their effects on the pulmonary circulation. For example, by their strong vasoconstrictor effect on the pulmonary circulation, decrease in PaO_2 and increase in $PaCO_2$ are able to impair right ventricular function.[17,18,19] Once again, such effects may occur in many clinical situations in the ICU related either to the patient's status (Figure 19.4) or to the intensivist's ventilatory strategy.

Finally, acute lung injury and ARDS are great models for heart–lung interactions. Because most patients are septic, initially mechanical ventilation and application of PEEP significantly alter the hemodynamics by decreasing systemic venous return.[20] After initial resuscitation and control of the septic process, heart–lung interactions may explain development of right ventricular dysfunction, called acute cor pulmonale.[21] Occurrence of acute cor pulmonale is strongly related to the plateau pressure (Figure 19.5),[22] and also to the PEEP[15] and hypercapnia.[21] The plateau pressure (which mainly reflects the transpulmonary pressure in

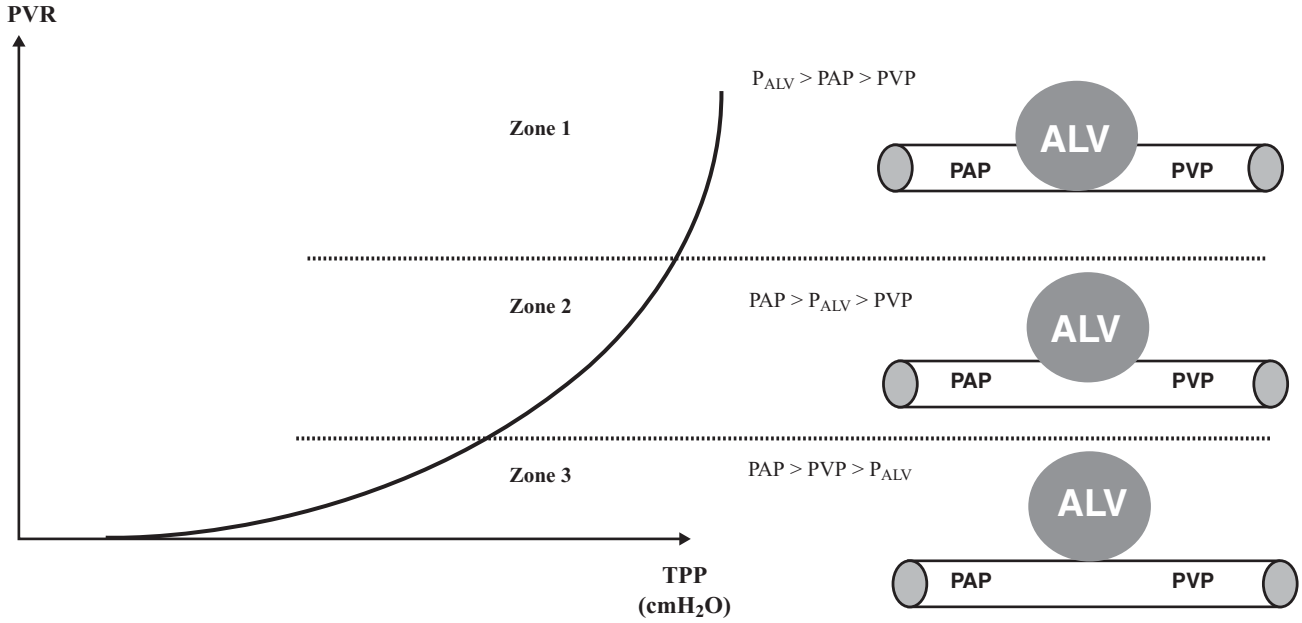


Figure 19.3. Relationship between pulmonary vascular resistance (PVR, y axis) and transpulmonary pressure (TPP, x axis). This relationship is nonlinear and can be separated into three zones, according to West's zones. In zone 3, pulmonary artery pressure (PAP) and pulmonary venous pressure (PVP) always remain higher than the distending pressure of the alveoli (P_{ALV}): the PVR is then only slightly increased. In zone 2 and in zone 1, the distending pressure becomes higher than the PVP and/or the PAP: the capillary is collapsed, the flow is restricted, and PVR is markedly increased.

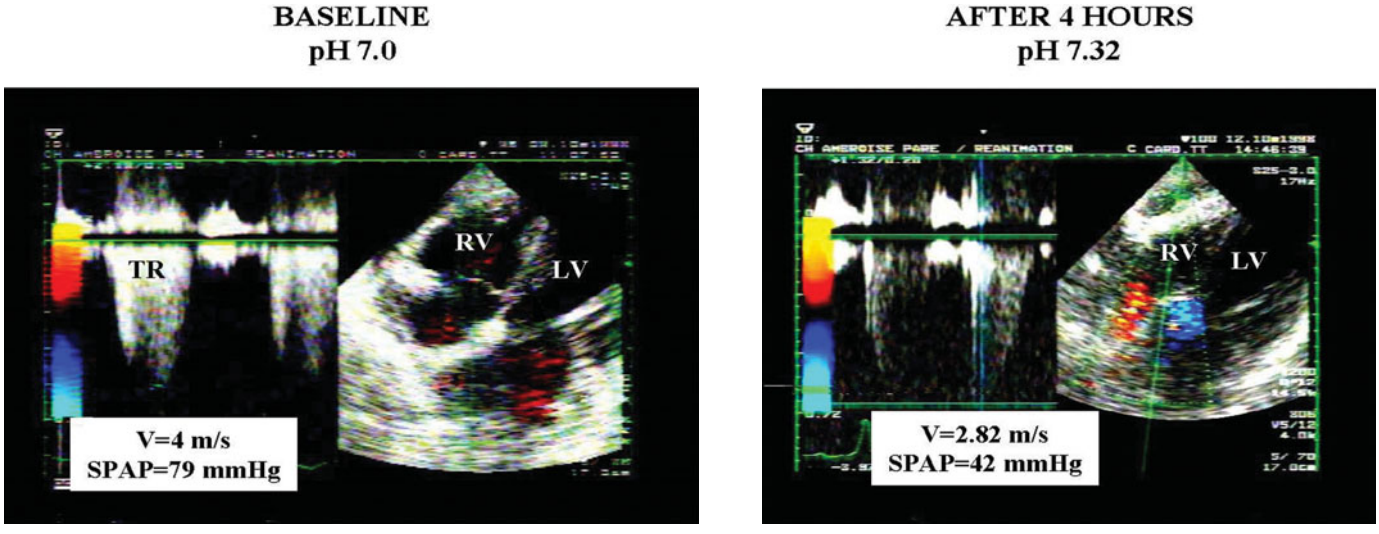


Figure 19.4. Echocardiographic evaluation of pulmonary artery pressure using tricuspid regurgitant flow in a patient hospitalized for acute exacerbation of chronic obstructive pulmonary disease. At admission, the patient had severe respiratory acidosis with a pH of 7.0. Echocardiography showed enlargement of the right ventricle with a high maximal velocity of the tricuspid regurgitation. Systolic pulmonary artery pressure was calculated as 79 mm Hg. After four hours of noninvasive ventilation, pH was 7.32, right ventricular size was normalized, and systolic blood pressure was calculated as 42 mm Hg. LV indicates left ventricle; RV, right ventricle; SPAP, systolic pulmonary artery pressure; TR, tricuspid regurgitation; V, velocity.

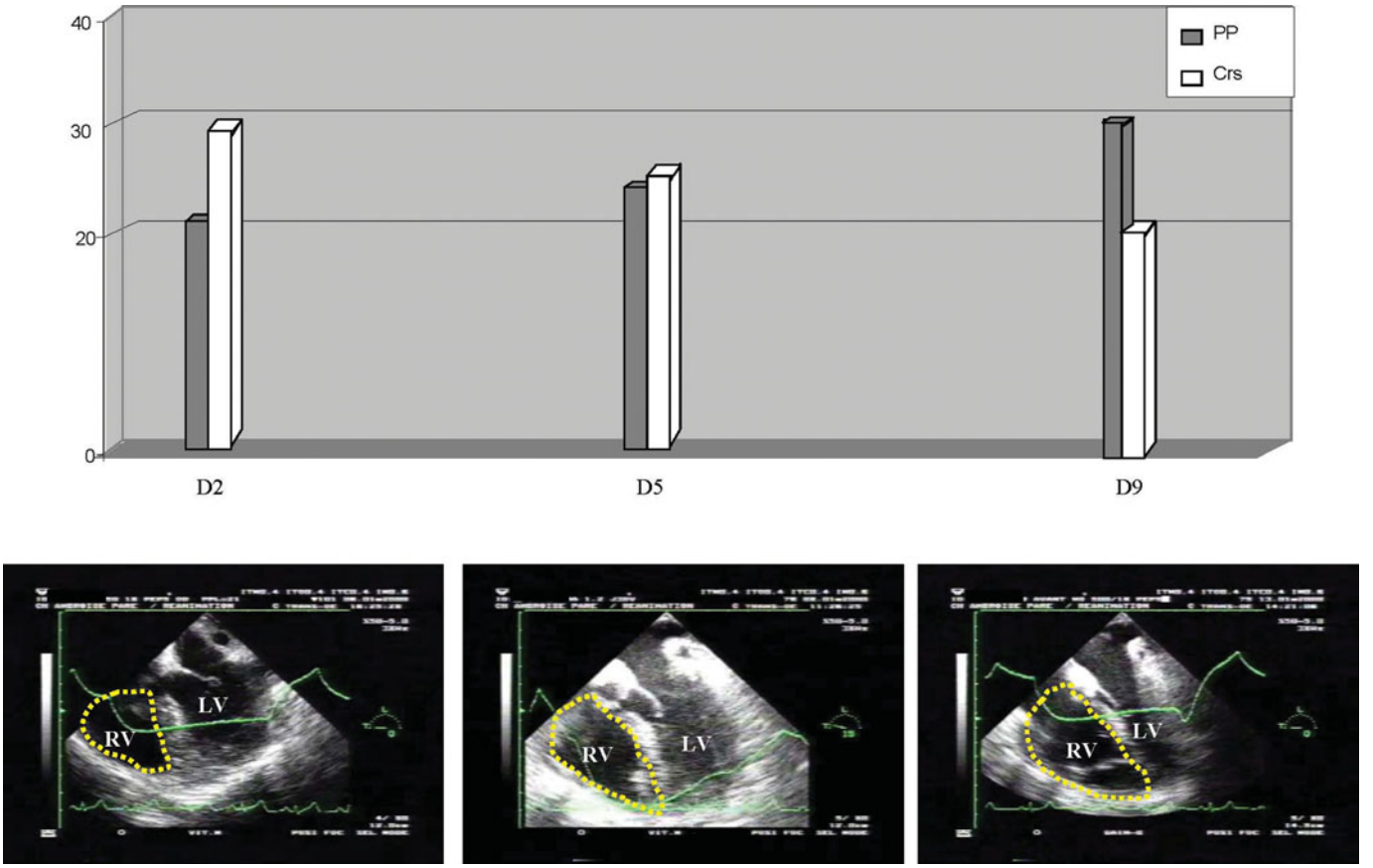
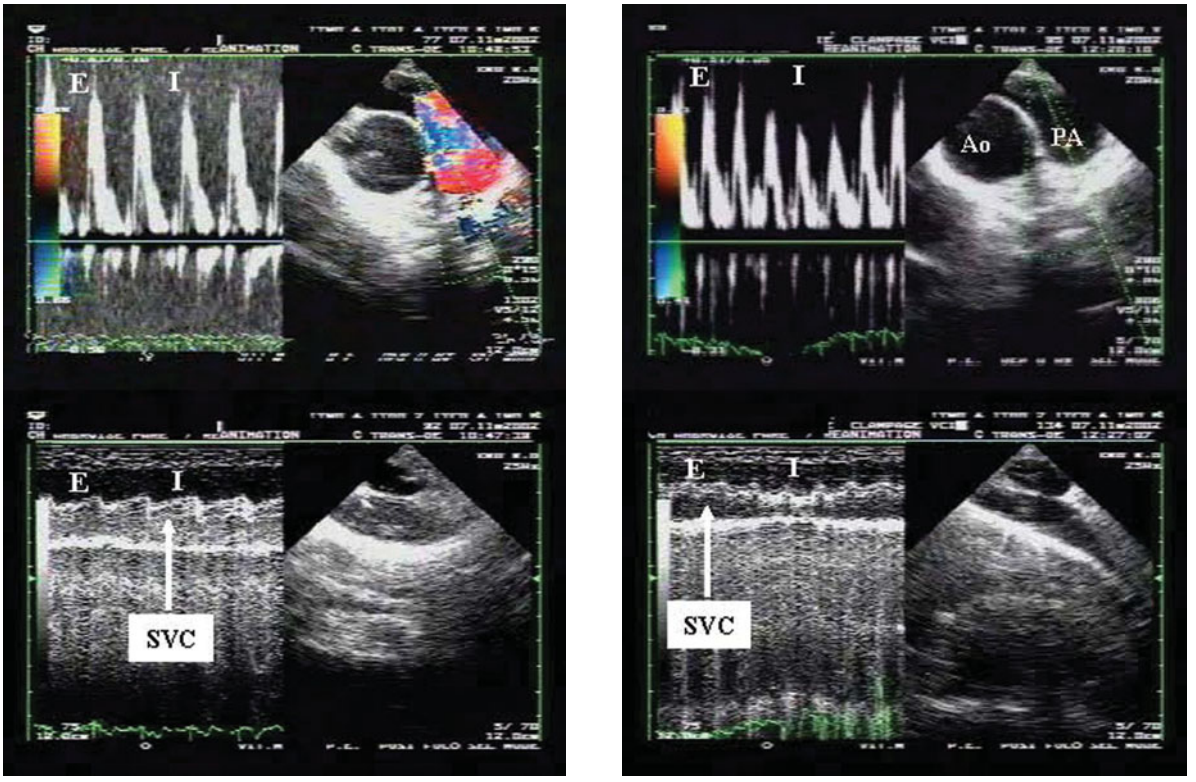


Figure 19.5. Patient hospitalized in the ICU for ARDS related to severe pneumonia. Top: plateau pressure (PP) and compliance of the respiratory system (Crs) after two, five, and nine days of mechanical ventilation. Bottom: long-axis view of the left ventricle by a transesophageal approach at days 2, 5, and 9, permitting evaluation of the size of the right ventricle. Progressive impairment in respiratory mechanics (decreased Crs and increased PP) was associated with progressive dilatation of the right ventricle. Whereas at day 2 the right ventricle was slightly dilated without any hemodynamic impairment, subsequently there was pronounced right ventricular dilatation, especially at day 9, leading to severe restriction of the left ventricle. The patient required increased doses of norepinephrine on day 5 and day 9. ARDS indicates acute respiratory distress syndrome; ICU, intensive care unit; LV, left ventricle; RV, right ventricle.

ARDS) deemed "safe" for the right ventricle has been reported as <27 cmH_2O in a population of more than 300 patients.[22]

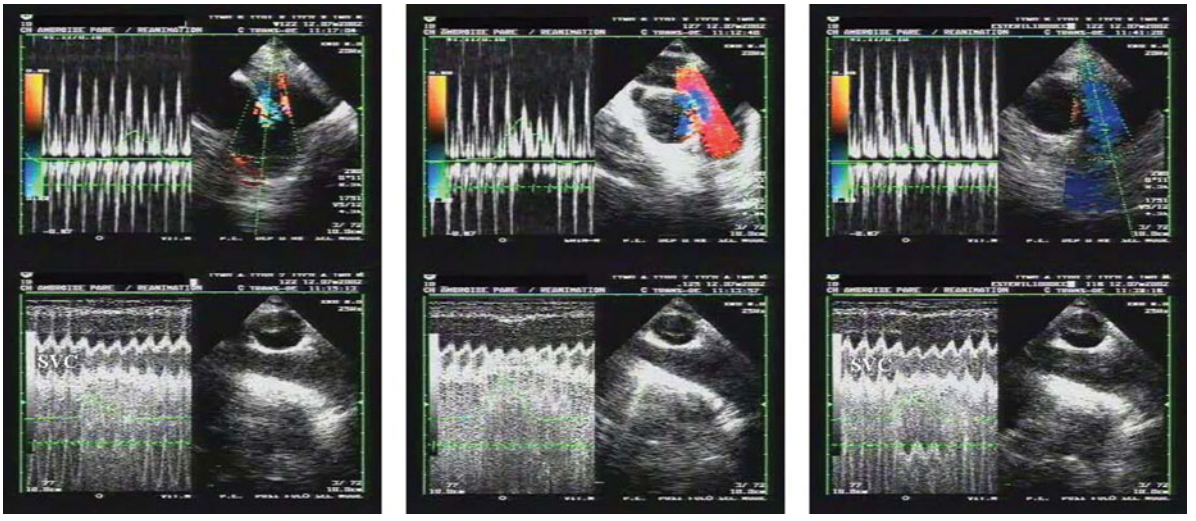
CARDIOPULMONARY INTERACTIONS, ECHOCARDIOGRAPHY AND CLINICAL IMPLICATIONS

Study of cardiopulmonary interactions led intensivists to develop new echocardiographic indices to manage the need for fluids in mechanically ventilated patients. Reductions in systemic venous return during tidal ventilation not only depend on changes in intrathoracic pressure, as emphasized above, but also on the filling status of patients. In other words, the lower the patient's filling status, the greater the consequences of changes in intrathoracic pressure.[23] As first hypothesized by Fessler et al.,[12] Vieillard-Baron and colleagues have demonstrated that reductions in systemic venous return related to tidal ventilation in humans is in part related to collapse of a great vessel, the superior vena cava (SVC), interposed between the periphery and the right atrium.[24] The surrounding pressure of the SVC is the intrathoracic pressure, which significantly increases during inspiration. In certain conditions, this increase may induce total or partial collapse of the vessel (Figure 19.6). The same authors have shown that these respiratory variations in the SVC, as defined by the "SVC collapsibility index," are a strong predictor of



A B

Figure 19.6. Transesophageal echocardiography in a patient during surgery for hepatic metastasis. Echocardiography recorded the right ventricular ejection flow, using pulsed Doppler in the pulmonary artery, and respiratory variations in the superior vena cava, using the time-motion mode. At baseline (panel A), no significant change was observed during inspiration. During cross-clamping of the inferior vena cava by the surgeon (panel B), the vessel collapsed during inspiration, resulting in a significant decrease in right ventricular ejection flow. Ao indicates aorta; E, expiration; I, inspiration; PA, pulmonary artery; SVC, superior vena cava.



A B C

Figure 19.7. Patient mechanically ventilated for an acute lung injury related to aspiration. Echocardiography by a transesophageal route recorded the right ventricular ejection flow and the respiratory variations in the superior vena cava in three conditions: at baseline without PEEP (panel A), after application of a PEEP of 5 cmH_2O (panel B), and with a PEEP of 5 cmH_2O after infusion of 500 mL of fluid (panel C). Application of a PEEP led to inspiratory collapse of the superior vena cava, resulting in a fall in right ventricular ejection flow. After fluid infusion, this was corrected and cardiac index increased significantly. PEEP indicates positive end-expiratory pressure; SVC, superior vena cava.

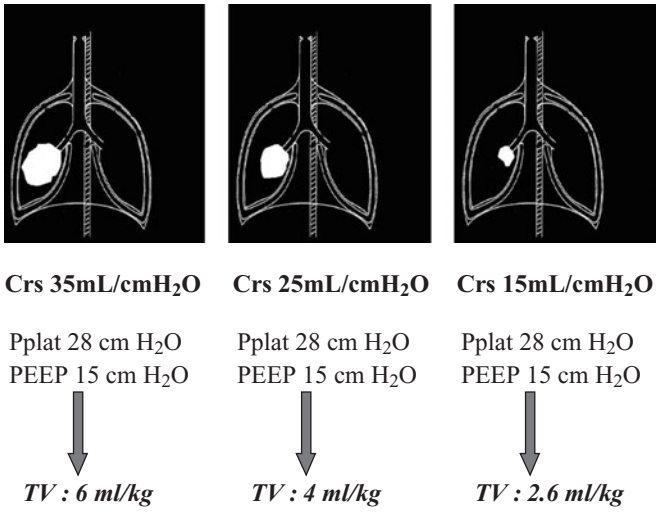


Figure 19.8. Schematic representation of the baby lung concept in ARDS. For a compliance (Crs) of 35 mL/cmH_2O, application of a PEEP of 15 cmH_2O requires a tidal volume (TV) of 6 mL/kg to maintain a plateau pressure (Pplat) at 28 cmH_2O. However, a compliance at 25 or 15 mL/cmH_2O, for the same Pplat and the same PEEP, requires a decrease in tidal volume to 4 or even 2.6 mL/kg, leading to severe hypercapnia. ARDS indicates acute respiratory distress syndrome; PEEP, positive end-expiratory pressure.

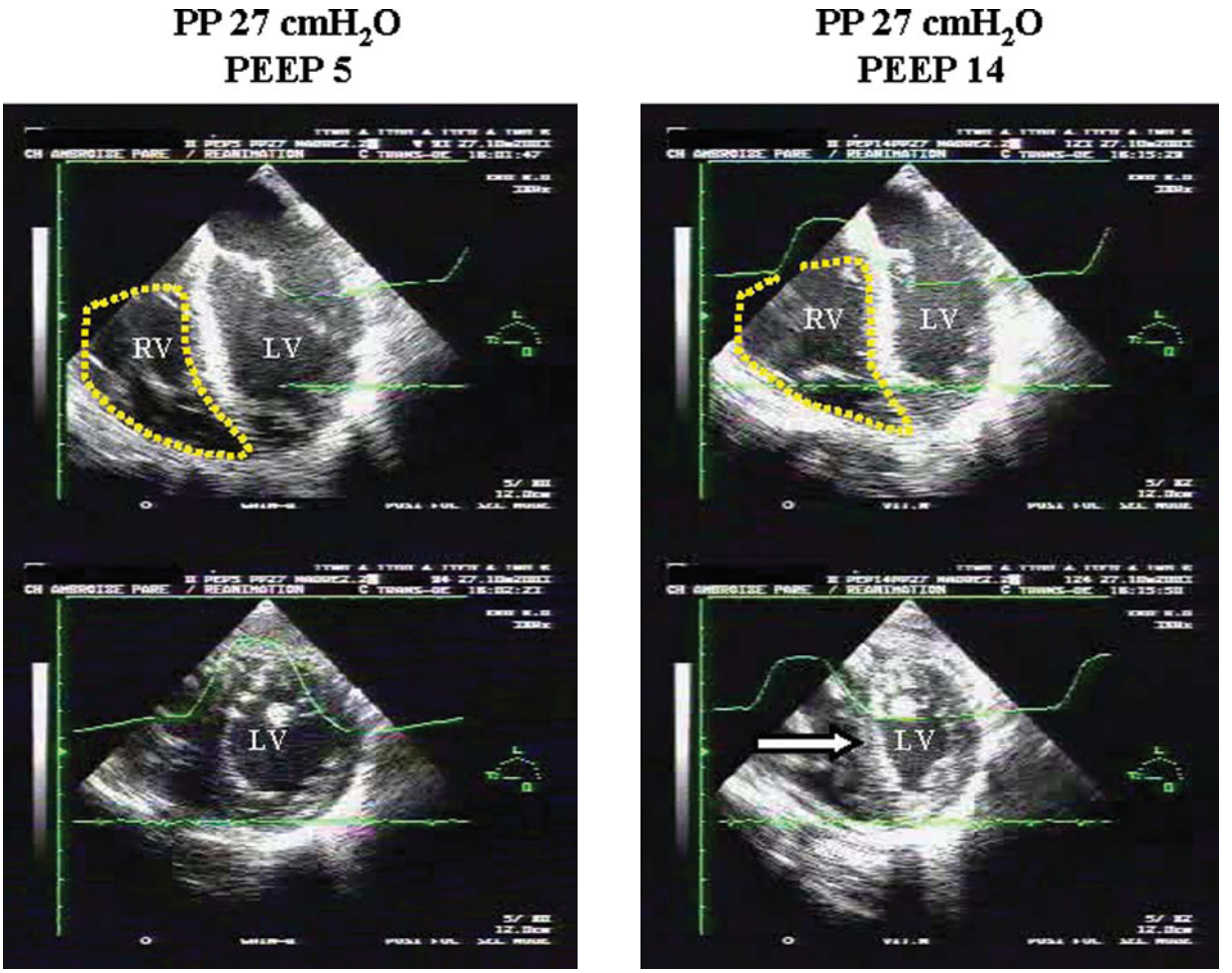


Figure 19.9. Transesophageal echocardiography in a patient mechanically ventilated for ARDS with two different PEEPs but the same plateau pressure. With a PEEP of 5 cmH_2O, the long-axis view of the left ventricle showed only a slight increase in right ventricular size and the short-axis view showed the absence of paradoxical septal movement. Application of a PEEP of 14 cmH_2O induced RV dilatation associated with paradoxical septal motion (arrow), resulting in hemodynamic impairment. ARDS indicates acute respiratory distress syndrome; LV, left ventricle; PEEP, positive end-expiratory pressure; PP, plateau pressure; RV, right ventricle.

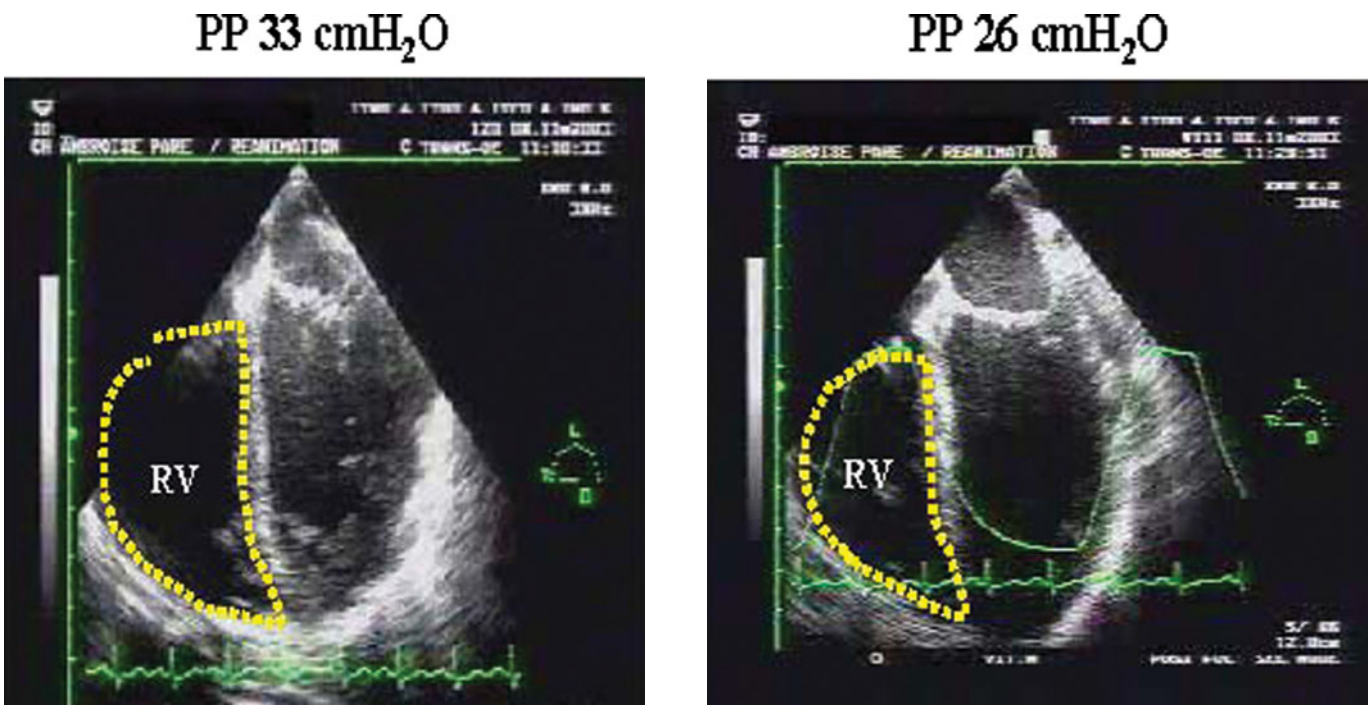


Figure 19.10. Another patient ventilated for ARDS. Transesophageal echocardiography was performed at two different plateau pressures, first 33 cmH_2O, then 26 cmH_2O, obtained by decreasing the tidal volume. At 26 cmH_2O, echocardiography visualized a decrease in right ventricular size and in left ventricular restriction. This was associated with an improvement in hemodynamics. ARDS indicates acute respiratory distress syndrome; PP, plateau pressure; RV, right ventricle.

hypovolemia and of fluid responsiveness.[25] Such respiratory variations in the SVC are dependent on volume status, and also on ventilatory settings, and well illustrate the concept of heart–lung interactions (Figure 19.7).

Another very practical application of the study of heart–lung interactions regards adaptation of ventilatory settings in ARDS patients to hemodynamics and especially to right ventricular function. For instance, echocardiography can be used to check how well patients with severe ARDS tolerate increasing PEEP.[26] High PEEP may impair right ventricular function by two mechanisms: first, by overloading the right ventricle during expiration when it is still overloaded by tidal ventilation; second, by inducing severe hypercapnia related to a tidal volume decreased to avoid any increase in plateau pressure above 30 cmH_2O (Figure 19.8). Examples of adjustment of PEEP and plateau pressure, in the light of the echocardiographic examination, are demonstrated in Figures 19.9 and 19.10.

To conclude, cardiopulmonary interactions are crucial to the understanding and management of many diseases in the ICU and, especially, mechanically ventilated patients and patients with severe asthma or ARDS. Echocardiography, by its ability to analyze cardiac function beat by beat during respiration, is the most valuable tool to study these interactions. More than an intriguing topic of conversation between physiologists, the study of heart–lung interactions strongly influences management: it yields new parameters of fluid responsiveness, enhances understanding of the impact of ventilatory settings on right ventricular function, and so enables adaptation of respiratory settings to this function.

References

1. Kumar A, Falke KJ, Geffin B, et al. Continuous positive-pressure ventilation in acute respiratory failure. *N Engl J Med.* 1970;283:1430–1436.
2. Vieillard-Baron A, Loubieres Y, Schmitt JM, Page B, Dubourg O, Jardin F. Cyclic changes in right ventricular output impedance during mechanical ventilation. *J Appl Physiol.* 1999;87:1644–1650.
3. Vieillard-Baron A, Chergui K, Augarde R, et al. Cyclic

changes in arterial pulse during respiratory support revisited by Doppler echocardiography. *Am J Respir Crit Care Med.* 2003;168:671–676.

4. Echocardiography in intensive care. Available at: http://www.pifo.uvsq.fr/hebergement/webrea/index.php. Accessed February 7, 2009.
5. Guyton AC, Lindsey AW, Abernathy B, Richardson T. Venous return at various right atrial pressures and the normal venous return curve. *Am J Physiol.* 1957;189:609–615.
6. Milnor WR, Jose AD, McGaff CJ. Pulmonary vascular volume, resistance, and compliance in man. *Circulation.* 1960;22:130–137.
7. Scharf SM, Caldini P, Ingram RH Jr. Cardiovascular effects of increasing airway pressure in the dog. *Am J Physiol.* 1977;232:H35–H43.
8. Knowles GK, Clark TJ. Pulsus paradoxus as a valuable sign indicating severity of asthma. *Lancet.* 1973;2:1356–1359.
9. Werko L. *The Influence of Positive Pressure Breathing on the Circulation in Man*. Stockholm, Sweden: Alb. Bonniers Boktrycken; 1947:50.
10. Massumi R, Mason D, Zakauddin V, Zelis R, Otero J, Amsterdam E. Reversed pulsus paradoxus. *N Engl J Med.* 1976; 289:1272–1275.
11. Jardin F, Farcot JC, Gueret P, Prost JF, Ozier Y, Bourdarias JP. Cyclic changes in arterial pulse during respiratory support. *Circulation.* 1983;68:266–274.
12. Fessler HE, Brower RG, Wise RA, Permutt S. Effects of positive end-expiratory pressure on the gradient for venous return. *Am Rev Respir Dis.* 1991;143:19–24.
13. Whittenberger JL, McGregor M, Berglund E, Borst HG. Influence of state of inflation of the lung on pulmonary vascular resistance. *J Appl Physiol.* 1960;15:878–882.
14. West JB, Dollery CT, Naimark A. Distribution of blood flow in isolated lung; relation to vascular and alveolar pressures. *J Appl Physiol.* 1964;19:713–724.
15. Jardin F, Brun-Ney D, Cazaux P, Dubourg O, Hardy A, Bourdarias JP. Relation between transpulmonary pressure and right ventricular isovolumetric pressure change during respiratory support. *Cathet Cardiovasc Diagn.* 1989;16:215–220.
16. Jardin F, Farcot JC, Boisante L, Prost JF, Gueret P, Bourdarias JP. Mechanism of paradoxic pulse in bronchial asthma. *Circulation.* 1982;66:887–894.
17. Rabinovitch M, Gamble W, Nadas AS, Miettinen OS, Reid L. Rat pulmonary circulation after chronic hypoxia: hemodynamic and structural features. *Am J Physiol.* 1979; 236:H818–H827.
18. Balanos GM, Talbot NP, Dorrington KL, Robbins PA. Human pulmonary vascular response to 4 h of hypercapnia and hypocapnia measured using Doppler echocardiography. *J Appl Physiol.* 2003;94:1543–1551.
19. Viitanen A, Salmenpera M, Heinonen J, Hynynen M. Pulmonary vascular resistance before and after cardiopulmonary bypass. The effect of $PaCO_2$. *Chest.* 1989;95:773–778.
20. Michard F, Chemla D, Richard C, et al. Clinical use of respiratory changes in arterial pulse pressure to monitor the hemodynamic effects of PEEP. *Am J respir Crit Care Med.* 1999;159:935–939.
21. Vieillard-Baron A, Schmitt JM, Augarde R, et al. Acute cor pulmonale in acute respiratory distress syndrome submitted to protective ventilation: incidence, clinical implications, and prognosis. *Crit Care Med.* 2001;29:1551–1555.
22. Jardin F, Vieillard-Baron A. Is there a safe plateau pressure in ARDS? The right heart only knows. *Intensive Care Med.* 2007;33:444–447.
23. Perel A, Pizov R, Cotev S. Systolic blood pressure variation is a sensitive indicator of hypovolemia in ventilated dogs subjected to graded hemorrhage. *Anesthesiology.* 1987;67:498–502.
24. Vieillard-Baron A, Augarde R, Prin S, Page B, Beauchet A, Jardin F. Influence of superior vena caval zone condition on cyclic changes in right ventricular outflow during respiratory support. *Anesthesiology.* 2001;95:1083–1088.
25. Vieillard-Baron A, Chergui K, Rabiller A, et al. Superior vena caval collapsibility as a gauge of volume status in ventilated septic patients. *Intensive Care Med.* 2004;30: 1734–1739.
26. Vieillard-Baron A, Prin S, Chergui K, Dubourg O, Jardin F. Echo-Doppler demonstration of acute cor pulmonale at the bedside in the medical intensive care unit. *Am J Respir Crit Care Med.* 2002;166:1310–1319.

SECTION III

ULTRASOUND EVALUATION OF THE NECK, TRUNK, AND EXTREMITIES

This page intentionally left blank

CHAPTER 20

Ultrasound Evaluation of the Neck and Upper Respiratory System

Christian Butcher

INTRODUCTION

Ultrasound of the neck and upper respiratory system has many potentially useful clinical applications.[1] Aside from vascular access (see Chapter 30), some of these indications include confirmation of satisfactory endotracheal tube placement, evaluation of the larynx, guidance for percutaneous tracheostomy, and evaluation of the paranasal sinuses. The data demonstrating improved outcomes by using ultrasound for imaging the upper airway remain scarce. However, there are important opportunities to improve care for the intensive care unit (ICU) patient that can be derived from its use.

PARANASAL SINUSES

Ultrasound use for the evaluation of the paranasal sinuses was recognized in Europe as technically feasible to confirm the presence of sinus disease as early as the 1960s.[2] However, widespread clinical application emerged only recently with the development of low-cost, high-quality bedside ultrasound imaging technology. Earlier studies established ultrasound as an alternative to computed tomography (CT) for the diagnosis of maxillary sinus disease and described the typical findings associated with sinusitis.[3,4] With improvements in imaging, more recent reports focused on improving the diagnostic accuracy of ultrasound by performing postural maneuvers.[5] In 2006, Vargas and colleagues investigated the role of ultrasound for performing transnasal puncture of the maxillary sinus in intubated ICU patients. In patients suspected of having sinusitis, they found ultrasonographic evidence of maxillary sinusitis in 70% of patients, and of these, 93% had positive results from transnasal puncture, demonstrating the comparability of ultrasound to CT for the diagnosis and transnasal puncture of sinusitis.[6]

There are no studies that describe an improvement in ICU outcomes by using ultrasound instead of standard CT, even though a CT scan has more radiation, is more expensive, and requires the transportation of critically ill patients to and from the radiology department and the use of valuable critical care nursing time. There are important roles, however, for CT imaging of the sinuses that cannot be duplicated with ultrasonography. These include any planned surgical procedure involving the sinuses, suspected sinus trauma, and suspected malignant disease. This discussion focuses on the use of ultrasound for the evaluation of paranasal sinusitis.

Sinus disease is important to recognize in critically ill patients because it is a source of fever, which leads to costly diagnostic workups and empiric therapeutic regimens.[7,8] Additionally, maxillary sinus disease is an independent risk factor for the development of nosocomial lung infections.[9] Although not studied in any systematic fashion, it is also conceivable that undiagnosed sinusitis may lead to significant pain and agitation, resulting in the increased use of sedatives and analgesics, which could then delay extubation.

Technique

The anatomy of the paranasal sinuses is shown in Figure 20.1. The sinuses most amenable to ultrasonographic examination are the maxillary and frontal sinuses; however, most studies have been performed on the maxillary sinus. The maxillary sinus is contained within the maxilla, and is bordered by the orbital floor superiorly, the hard palate inferiorly, the nasal wall medially, and the zygoma laterally. In the normal state, the sinus is air filled, thus impairing the transmission of ultrasound energy. In this case, what is seen is the anterior wall only, with some artifact known as "acoustic shadowing" (Figure 20.2), which obscures all underlying structures; this is considered a negative study. When filled with fluid, ultrasound penetrates the anterior wall, "travels" through the fluid, and strikes the posterior or lateral walls and "reflects" back to the transducer, resulting in an image of the sinus cavity

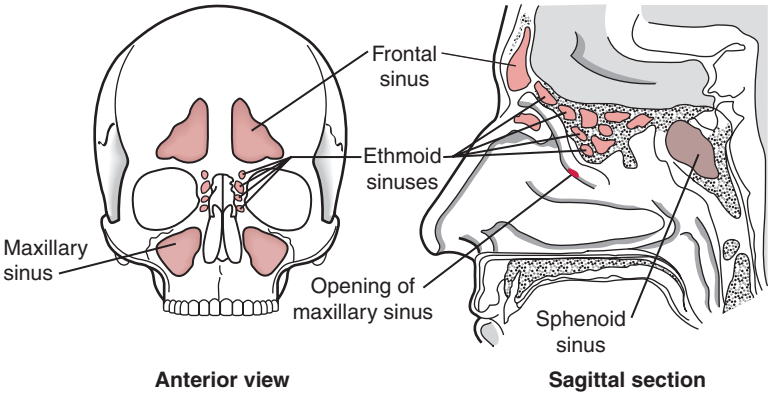


Figure 20.1. Anatomy of the paranasal sinuses. (*Source:* http://www.merck.com/mmpe/sec08/ch089/ch089a.html (in public domain).)

in its entirety (Figure 20.3). This is known as a "sinusogram," which is a positive study. A partial sinusogram, where only the posterior wall or a side wall is seen, can occur due to the presence of an air-fluid level in the sinus or mucosal thickening. The patient's position influences the appearance of the fluid in a partial sinusogram. In the supine position, fluid can "layer out" away from the anterior wall, resulting in either acoustic shadowing or a partial sinusogram. However, when placed in a semirecumbent or upright position, the fluid (if present) will follow gravity and cover the floor of the sinus, coming in contact with the anterior wall. When imaged, this results in either a partial or complete sinusogram, depending on how much fluid is present and on the transducer orientation or angulation (Figure 20.4).

For a sinus ultrasound, patients can be placed in a semirecumbent position. A 3–5 MHz cardiac probe with a small footprint is used. Proper transducer position is demonstrated in Figure 20.5. The horizontal plane is scanned first, angulating the probe cephalad (toward the orbital floor) and caudally (toward the floor of the sinus); this is followed by turning the transducer 90° and scanning from the medial to the lateral wall. The technique is then repeated on the alternate side. A complete ultrasound maxillary sinus scan, in contrast to CT scanning, can be performed in <60 seconds (sec). If a complete sinusogram is seen, no further evaluation is

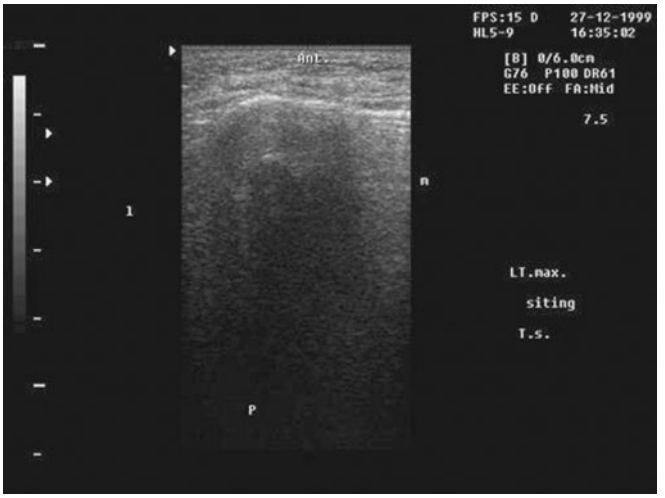


Figure 20.2. Normal maxillary sinus showing anterior wall only.

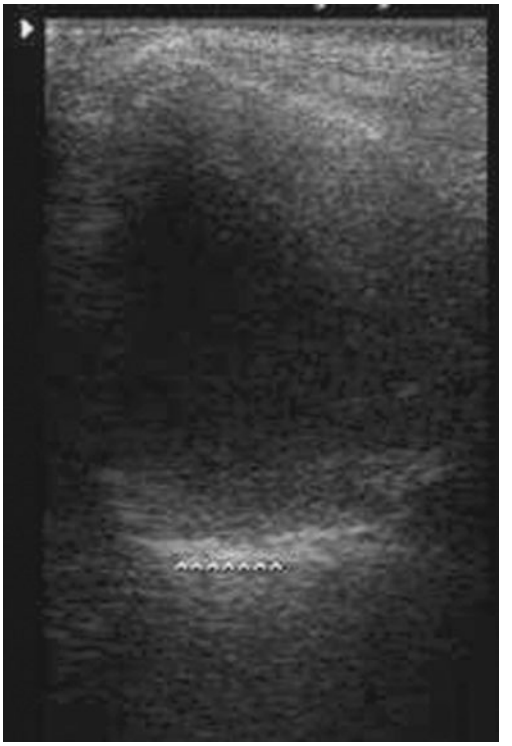


Figure 20.3. Abnormal, fluid-filled maxillary sinus showing anterior and posterior walls.

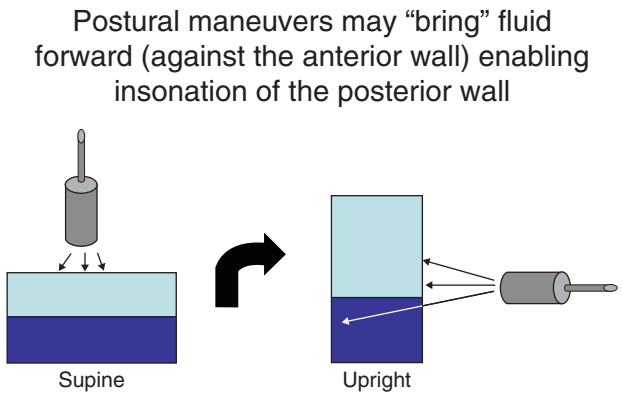


Figure 20.4. Diagram showing the effect of changing patient position from supine to upright. Note that in the supine position, fluid (dark blue) does not contact the anterior wall, thus the ultrasound beam cannot "penetrate." However, in the upright position, the fluid layers out inferiorly with gravity, and comes into contact with the anterior wall. The ultrasound then penetrates to the posterior wall, resulting in either a partial sinusogram (if imaged vertically) or a complete sinusogram (if imaged horizontally).

necessary and the patient should be treated for sinusitis. If a partial sinusogram is obtained, postural maneuvers may help elucidate the cause: sinusitis vs. mucosal thickening. Until proficiency with the ultrasound technique is mastered, bedside ultrasound can be correlated with CT scan.

LARNYX/ ENDOTRACHEAL INTUBATION

In 1987, Raphael and Conard used B-mode 2D transtracheal ultrasound to assess the capability of ultrasound to visualize and confirm ETT placement (REF).[10] In this study, the investigators were primarily interested in verifying the intratracheal placement in patients already known to have successful tracheal intubations. The study was not attempting to identify esophageal intubation or any other malposition. The authors suggested that ETT cuffs be filled with saline to reduce the acoustic impedance of the air-filled ETT balloon, which would improve ultrasound transmission (similar to having a full bladder during abdominal ultrasonography). The contrast between the air-filled trachea and saline-filled balloon allows the position of the ETT to be identified more easily. It was also suggested that a longitudinal view, combined with a slight to-and-fro motion, could improve visualization. They concluded that this technique was beneficial for certain patient

Figure 20.5. Proper transducer position. Top: horizontal. Bottom: vertical.

populations like pregnant women or patients receiving frequent chest radiographs to monitor ETT position.

A blinded, prospective study of 40 patients undergoing elective surgery was performed to identify esophageal intubation using 2D ultrasound.[11] The authors identified esophageal intubations with a sensitivity of 100% and correctly identified all five esophageal

intubations, and 34 out of 35 tracheal intubations. Two additional studies, using both live patients and cadavers, confirmed the high sensitivity and specificity of 2D ultrasound to evaluate ETT position.[12,13] In addition, improved sensitivity and specificity could be achieved by using a dynamic approach (visualization of the tube during placement) as compared with a static approach (confirmation of placement after the fact).[13]

Endotracheal tube malposition in the right mainstem bronchus can also be identified by using bilateral pleural ultrasound.[14] The parietal–visceral pleural interface can be easily identified with a high-frequency probe (Figure 20.6). This interface, known as the visceral-parietal pleural interface (VPPI), has a characteristic "shimmering" appearance during lung ventilation. The two pleural surfaces can be seen to slide past one another, which is responsible for producing the shimmering effect. If the ETT is positioned in a mainstem bronchus, the sliding or shimmering will either be greatly reduced or absent on the contralateral side. This assumes that there is no anatomic airway obstruction, such as an obstructing tumor, causing the reduced or absent pleural shimmer, which could lead to a false-positive test and inappropriate repositioning of the ETT. This approach was confirmed by Weaver et al.,[15] using cadavers. The sensitivity for identifying esophageal intubation was 95–100% and the sensitivity for a right mainstem intubation versus a tracheal intubation was lower, at 70–75%, using the sliding lung sign.

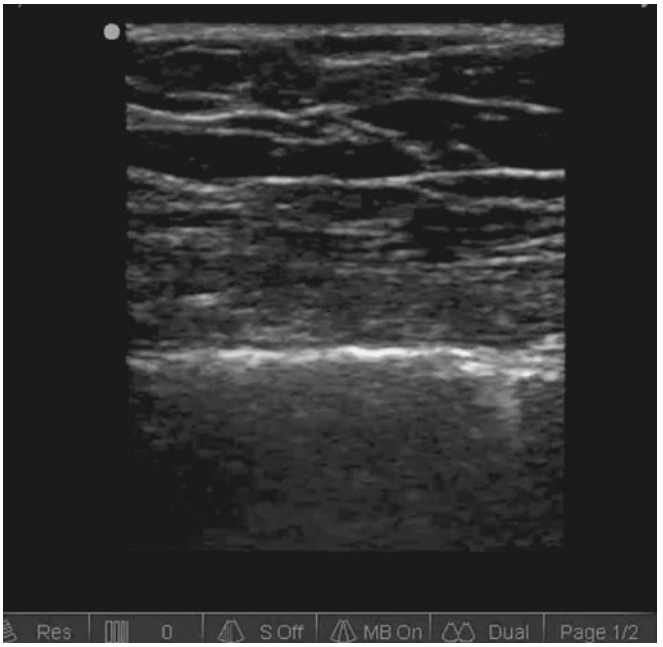


Figure 20.6. Visceral-parietal pleural interface (VPPI). Normal appearance of the pleural surfaces. The white line "shimmers" during respiration.

Other applications for upper airway ultrasound, aside from verification of endotracheal tube position, include appropriate ETT size selection, the prediction of a difficult airway, and the prediction of postextubation stridor. Lakhal et al. studied whether or not appropriate selection of ETT size could be informed by using translaryngeal ultrasound. After measuring the diameter of the airway in the subglottic region by translaryngeal ultrasound, the investigators then compared their measurement to those obtained by using magnetic resonance imaging (MRI) and found a strong correlation between the two measurements. The authors suggested that ultrasound could accurately gauge the diameter of the subglottic airway and could guide selection of an appropriately sized ETT.[16]

Pretracheal soft tissue (PST) swelling is identified as a risk factor for difficult laryngoscopy and can be identified using ultrasound.[17] Patients were clinically classified as either "difficult" or not, followed by ultrasonographic quantification of PST. Difficult patients had PST measurements of 28 ± 2.7 millimeters (mm), compared with 17.5 ± 1.8 mm for nondifficult laryngoscopy. These findings were refuted by Komatsu, who concluded that ultrasound quantification was unable to predict subsequent difficult laryngoscopy in a cohort of obese patients.[18] While having a noninvasive tool to assist with predicting difficult laryngoscopy and difficult-to-intubate patients, the data available are equivocal for this indication.

Ding and colleagues studied whether translaryngeal ultrasound measurements of airway column width around the endotracheal tube could predict postextubation stridor. A preextubation air leak test and ultrasonographic measurements of air column widths were obtained on intubated patients. The air column width was measured with the balloon inflated and deflated. The patients who developed stridor had an air leak at 25 cc compared with 300 cc in the nonstridor group. The air column width in the normal group was 6.4 mm, compared with 4.5 mm in the stridor group. The authors concluded that preextubation translaryngeal quantification of the air column width could help identify patients at risk for postextubation stridor.[19] There are several limitations to this study, including the effect of endotracheal tube size on air column width and the presence of secretions, which would decrease air column width measurements. Although inconclusive, this represents an exciting opportunity for further investigation.

Technique

Endotracheal tube malposition is a common problem in the ICU. Accordingly, most of the data on the use of ultrasound for airway management focuses on the verification of ETT position. Two basic techniques are available for this purpose: translaryngeal ultrasound to evaluate the proximal trachea and evaluate for proximal malposition, and pleural ultrasound to evaluate for distal malposition of the ETT.

Translaryngeal 2D ultrasound for visualization of the ETT is best performed longitudinally during ETT placement, so called "dynamic guidance." This requires that the ultrasound equipment be readily available during intubation. A midline longitudinal view, using a 5–7 MHz linear array (vascular) probe, through the larynx at the level of the cricoid cartilage is optimal. The "depth" of the image should be set deep enough to visualize the tracheal lumen and not just the anterior tracheal wall. Remember that air produces significant artifact due to its high acoustic impedance and structures lying deeper to air will generally not be seen. However, with careful examination and practice, the presence of the ETT can be seen with this view as an echogenic stripe lying deep to the anterior tracheal wall. While this may initially be considered a reverberation artifact from the tracheal wall itself, with further practice, the anterior surface of the ETT can be appreciated, particularly in the setting of a "high," or subglottic intubation. In this case, the echogenic stripe does not continue the entire length of the image (Figure 20.7). Angulation of the probe under the manubrium is sometimes necessary to visualize the tip of the ETT (Figure 20.8). In cases of a relatively low intubation, the tip will not be seen. To enhance this technique, a transverse ultrasound examination looking for the curved, echogenic contour of the anterior endotracheal tube may be helpful (Figure 20.9). Again, angulation under the manubrium may be required. In general, if the tip of the tube is seen without significant angulation under the manubrium in either the longitudinal or transverse view, it is likely to be within the proximal trachea and may require advancement.

Pleural ultrasound (see Chapter 22), is an extremely useful tool to evaluate for esophageal intubation, confirm tracheal intubation, and determine mainstem intubation in most patients. Conveniently, the same probe used for translaryngeal ultrasound (linear array, 5–7 MHz) can also be used to evaluate the VPPI. As discussed above, in cases of satisfactory tracheal intubation, and in the absence of unilateral airway obstruction from a tumor or foreign body, there will be bilateral and equal pleural sliding during respiration. In cases of esophageal intubation, there will be minimal or no pleural sliding (caused by the cardiac cycle, known as the lung pulse). This would be associated with desaturation, reduced breath sounds, epigastric gurgling with bagging, and an undetectable end-tidal CO_2 prompting immediate tube removal and tracheal reintubation. In cases of distal malposition, such as a right mainstem intubation, there would be vigorous pleural sliding on the right, with diminished or absent sliding on the left. There is importance in comparing one side of the chest to the other, similar to techniques

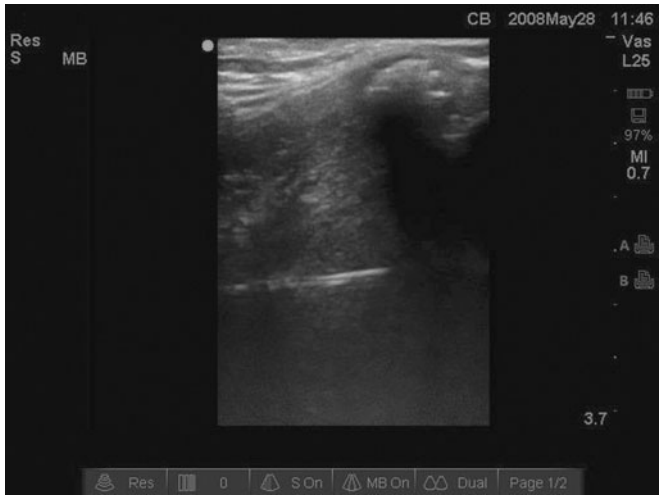


Figure 20.7. Longitudinal view through the trachea showing the distal tip of the endotracheal tube (bright white line).

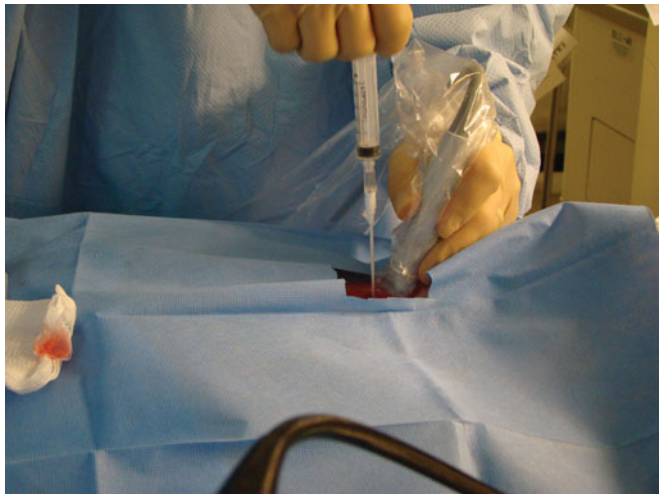


Figure 20.8. Angulation of the probe to image inferiorly to the manubrium.

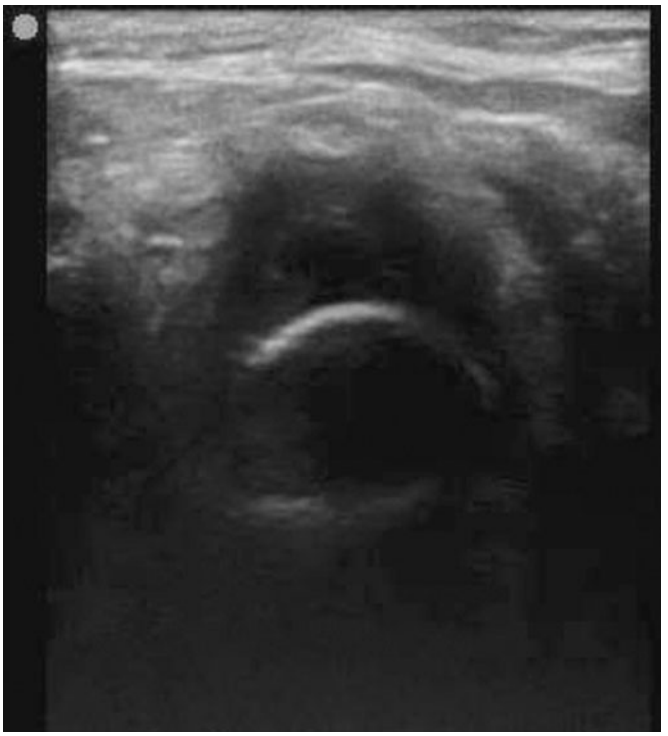


Figure 20.9. Transverse view through the trachea showing the anterior surface of the endotracheal tube (bright white, curvilinear structure).

of lung auscultation learned in medical school. A combined approach of translaryngeal ultrasound to identify proximal malposition of the ETT, paired with pleural ultrasound to identify distal malposition, may be a viable alternative to chest radiography to verify tube position. Figure 20.10 demonstrates an algorithm that we use for this purpose. This same basic technique can be applied to other situations, such as confirming proper double lumen ETT placement.[1] The limitations of using lung sliding to verify endotracheal tube position include the presence of any process that causes loss of lung sliding such as pneumothorax, pleural scarring, or severe parenchymal lung disease that prevents lung inflation (e.g. severe ARDS or pneumonia).

PERCUTANEOUS TRACHEOSTOMY

Percutaneous dilatational tracheostomy (PDT) has become the procedure of choice for providing long-term airway access in many institutions. Once considered only for "ideal" candidates, the procedure is now being performed safely in the morbidly obese and in those with prior neck surgery or coagulopathy. Recently, there has been significant attention to studying the application of ultrasound to PDT to reduce complications, especially in patients belonging to high-risk groups.

Examination of the anterior neck prior to surgical tracheostomy was first described by Bertram et al. in 1995,[20] in a study in which 50 patients underwent an ultrasound evaluation of the neck prior to the operation. This study established the feasibility of performing a targeted ultrasound examination to identify anatomic variations that may complicate the procedure. Sustic et al. first reported on the use of ultrasound guidance for percutaneous tracheostomy,[21] and the

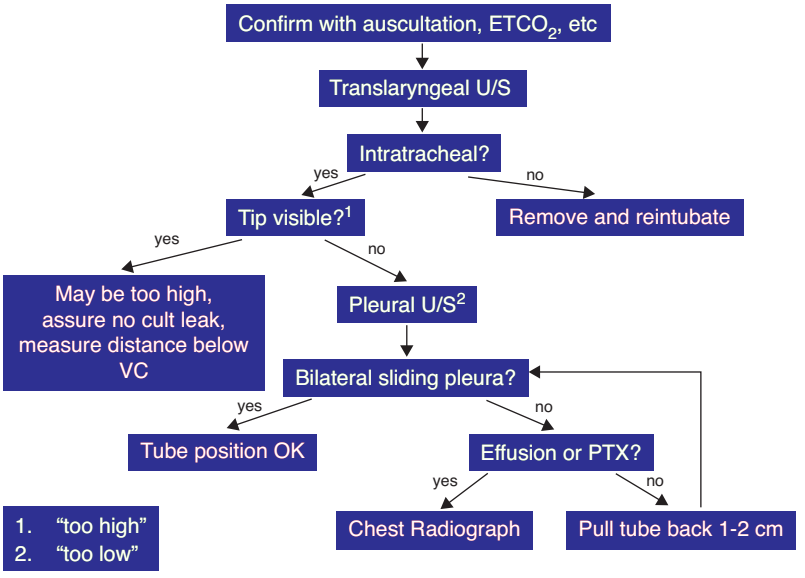


Figure 20.10. Algorithm for determining satisfactory endotracheal tube placement.

clinical utility of this technique has been confirmed in several other reports.[22–25] These studies demonstrate the benefits of ultrasound for both the selection of an insertion site and an appropriately sized tracheostomy tube,[26] in addition to allowing visualization of aberrant anatomy.

Although safe, PDT can be associated with life-threatening complications, such as severe bleeding and malposition. Most bleeding events are delayed two to three weeks after placement, and are due to erosion of the brachiocephalic artery caused by the high-pressure tracheostomy tube cuff. However, bleeding can also occur at the time of tracheostomy placement. A recent case of fatal hemorrhage following PDT was reported.[27] In this case, the authors cited the cause of hemorrhage as an aberrant right subclavian artery, following thyroid surgery that coursed high in the neck at the level of the cricoid cartilage before turning toward the extremity. In a follow-up letter, Gwilym and Cooney[28] reported their experience with hemorrhage caused by injury to the lowest thyroid artery during PDT. Ultrasound mapping of the neck was not performed in either case, although the latter group now uses ultrasound regularly. Additionally, Muhammad and colleagues published their series of 497 cases in which bleeding occurred in 5%, usually due to an inferior thyroid vein, brachiocephalic vein, or high communicating anterior jugular vein.[29] Clearly, aberrant vascular anatomy exists and should be screened for.

Malposition is another complication of PDT. However, the selection of an appropriate insertion site minimizes risk. Proximal malposition could cause damage to the vocal cords at the time of insertion (immediate damage), or could cause the tracheostomy tube to lie directly underneath the vocal cords and damage them over time, resulting in permanent vocal cord dysfunction and hoarseness. Distal malposition could result in the tip of the tracheostomy tube abutting the carina during neck flexion or lying in a mainstem bronchus. Contact with the carina results in cough, patient-ventilator dysynchrony, difficult suctioning, suction trauma, and bleeding. Using ultrasound, the tracheal cartilages can be counted and the appropriate site marked on the skin, usually between the second and third tracheal cartilage, which can easily eliminate proximal malposition. Careful ultrasound examination to identify the insertion site reduces the incidence of cranial malposition from 33% to 0%.[30] In addition, the distance from the carina to the subglottic area can be measured by bronchoscopy, providing a rough estimate of tracheal length, which minimizes the risk of distal malposition. Therefore, using these two techniques, malposition of any kind can be virtually eliminated.

Another potential complication of PDT, as commonly performed, is hypercarbia. Most procedures are endoscopically guided, which is associated with hypercarbia when compared to surgical tracheostomy or tracheostomy with ultrasound guidance alone (without bronchoscopy). In one study comparing pCO_2 levels in patients undergoing endoscopically guided PDT, ultrasound-guided PDT, or surgical tracheostomy, the pCO_2 increased up to 24 mm Hg in the bronchoscopy group, compared with 8 mm Hg in the ultrasound group and 3 mm Hg in the operative group.[31] This has implications for patients in whom hypercarbia is undesirable, such as neurotrauma patients or patients with elevated intracranial pressure.

Technique

Selection of an appropriately sized tracheostomy tube can be aided by ultrasound in several ways. A B-mode scan of the pretracheal soft tissues, utilizing a 7 MHz linear array probe, allows an estimation of the distance from the skin to the anterior tracheal wall. Based on this information, the operator can choose either a standard tube for small distances or one with a proximal long segment for longer distances. This measurement can be performed in either a longitudinal or transverse orientation (Figure 20.11) and is best performed at the proposed insertion site. Also, the diameter of the trachea can be easily estimated by a transverse measurement of the tracheal lumen, again at the level of the proposed insertion site. This measurement guides the selection of a tube with a compatible diameter (Figure 20.12).

Selection of an insertion site is easily accomplished with a midline, longitudinal view of the trachea from cricoid cartilage to sternal notch (Figure 20.13). The tracheal cartilages can be counted, and the appropriate site can be marked on the skin, usually between the second and third tracheal cartilage. This, of course, requires that the patient be sedated enough that there is no movement of the head or neck between skin marking and tracheostomy insertion. If movement occurs during this window, the area needs to be rescanned to confirm that the skin mark is still appropriate.

After selection of an insertion site, a scan of the overlying tissues with color-flow Doppler should be performed to identify any vascular structures at risk for damage during the procedure. This is performed in both a longitudinal and transverse orientation to look for "bridging" jugular veins, high brachiocephalic vessels, or thyroid veins or arteries. When examining the

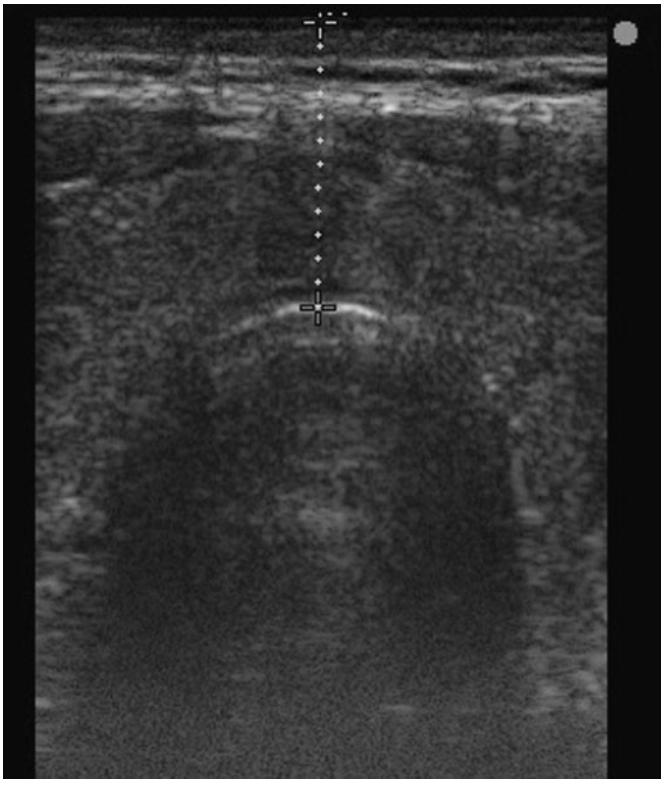


Figure 20.11. Measuring the pretracheal soft tissue to help with proper tracheostomy tube selection. Distances much greater than 2.5 cm may require a proximal long tracheostomy type.

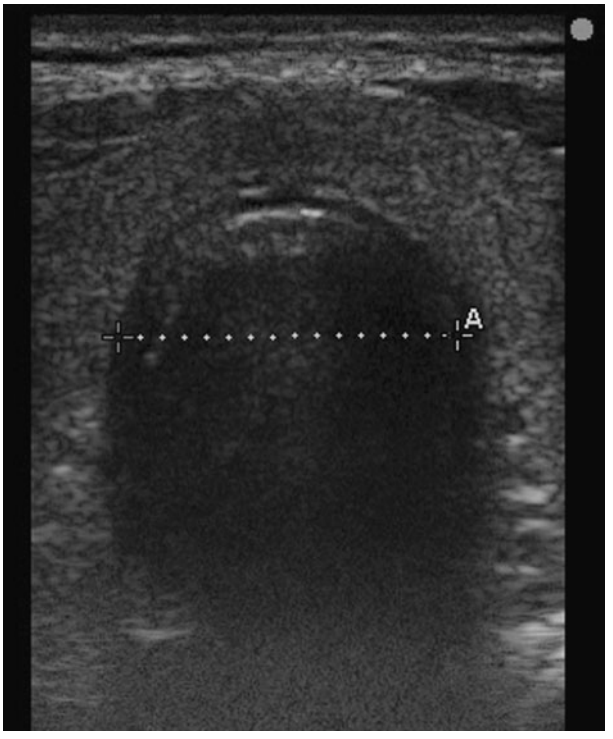


Figure 20.12. Measuring the tracheal diameter to aid proper tracheostomy tube selection.

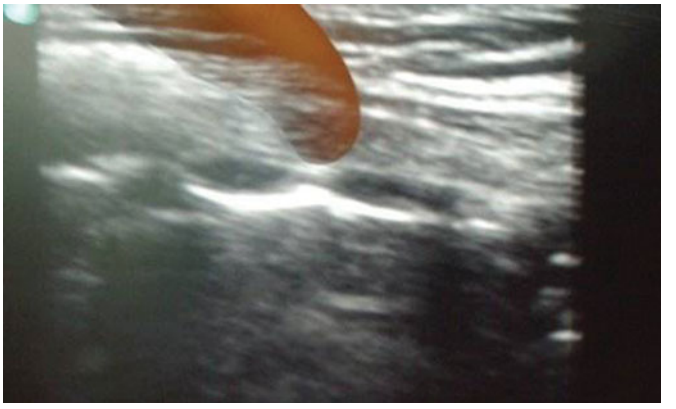


Figure 20.13. Midline longitudinal view of trachea, showing the tracheal rings. Superior is to the left, inferior to the right. The ring furthest to the left is the fist tracheal ring. The thumb indicates the desired insertion site.

trachea transversely, the thyroid gland, including both lobes, the isthmus, and the location of the carotid arteries and jugular veins, especially in patients with prior neck surgery, should be examined (Figure 20.14).

Cannulation of the trachea can be visualized under dynamic guidance. To achieve this, a longitudinal view just lateral to the midline is obtained. The cannulation needle is then applied to the skin in the midline; the needle causes an indentation of the subcutaneous tissue (Figure 20.15), which approximates the needle tract.

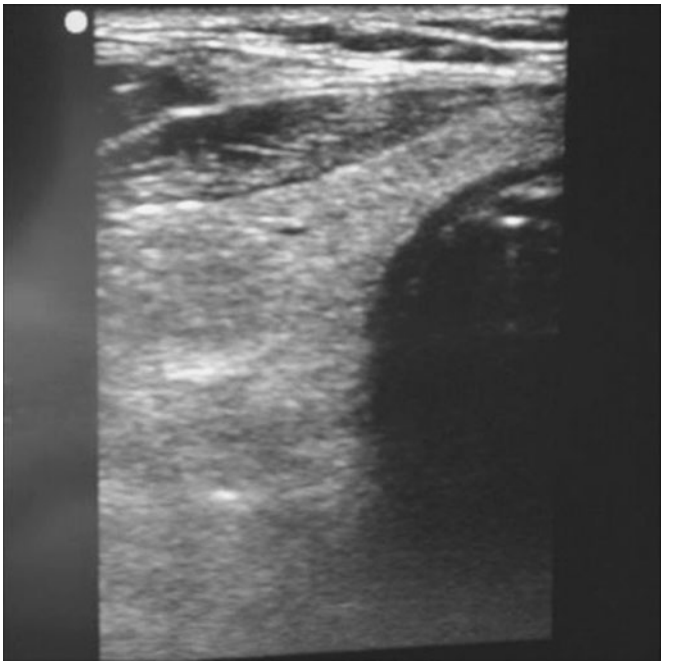


Figure 20.14. Transverse view through the trachea, showing the right lobe of the thyroid gland as well as the thyroid isthmus.

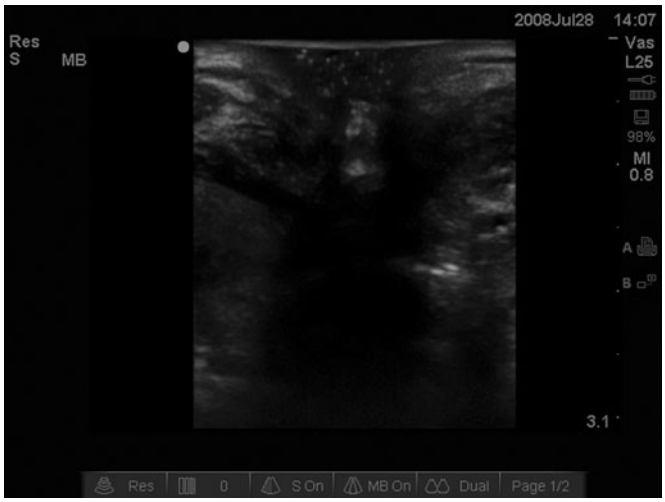


Figure 20.15. Cannulating the trachea under dynamic guidance utilizing a longitudinal ultrasound view.

The needle position can be adjusted either caudally or cranially, until this indentation overlies the desired puncture site. This is called longitudinal positioning. Once the desired longitudinal position is achieved, the probe can be rotated 90° to guide the transverse position of the needle (Figure 20.8). Transverse position is less important than longitudinal position, as the puncture site is somewhat anterior. The ultrasonographic view is seen in Figure 20.16. When the needle is in the desired position, cannulation can occur in the conventional manner. After tube placement, especially if bronchoscopy is not used to verify tube position, remember to scan the anterior chest bilaterally for sliding pleura; this confirms an intratracheal placement that is not too low.

In our practice, ultrasonography has not replaced bronchoscopy in the guidance of PDT. Bronchoscopy retains certain advantages, including the direct visualization of the position of the ETT during withdrawal and visualization of the posterior tracheal wall during needle cannulation and dilation. Additionally, in cases of overzealous ETT tube withdrawal, one can easily reintubate the trachea over the bronchoscope.

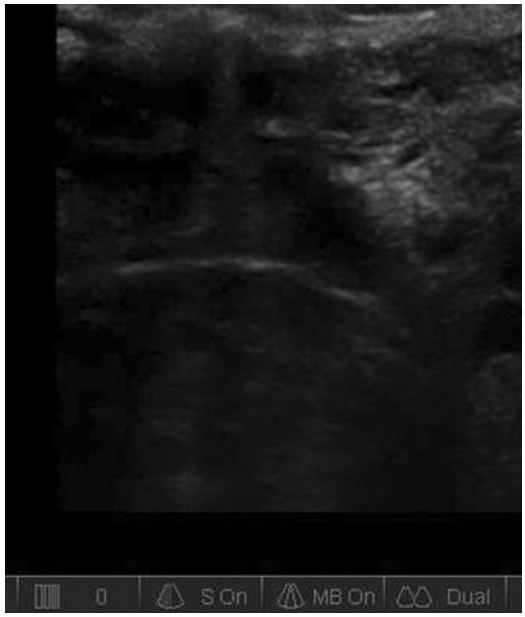


Figure 20.16. Transverse view through the trachea showing cannulation in the midline. The needle position can be adjusted under ultrasound guidance to assure a midline puncture. The acoustic shadow can be seen as two angled vertical lines overlying the mid portion of the tracheal ring.

SUMMARY

Ultrasound examination of the neck and upper airway has many useful clinical applications in the ICU. Maxillary sinusitis is commonly overlooked, yet easily identifiable with ultrasound, sometimes obviating the need for CT. Confirmation of ETT placement, probably the most common indication for "STAT" portable chest radiographs in the ICU, can be achieved with a high degree of confidence by using ultrasound. Percutaneous tracheostomy can be made even safer by performing a few simple scans to identify aberrant anatomy and to locate an appropriate insertion site. Utilization of this technology can help provide timely, cost-effective, and safe care for patients.

References

1. Sustic A. Role of ultrasound in the airway management of critically ill patients. *Crit Care Med.* 2007;35:S173–S177.
2. Abdurasulov DM, Amilova AA, Fazylov AA, et al. On the use of ultrasonics in the diagnosis of diseases of the maxillary sinuses. *Nov Med Priborostr.* 1964;24:30–33.
3. Lichtenstein D, Biderman P, Meziere G, et al. The sinusogram, a real-time ultrasound sign of maxillary sinusitis. *Intensive Care Med.* 1998;24:1057–1061.
4. Hilbert G, Vargas F, Valentino R, et al. Comparison of B-mode ultrasound and CT in the diagnosis of maxillary sinusitis in mechanically ventilated patients. *Crit Care Med.* 2001;29:1337–1342.
5. Vargas F, Boyer A, Bui HN, et al. A postural change test improves the prediction of a radiological maxillary sinusitis by ultrasonography in mechanically ventilated patients. *Intensive Care Med.* 2007;33:1474–1478.

6. Vargas F, Bui HN, Boyer A, et al. Transnasal puncture based on echographic sinusitis evidence in mechanically ventilated patients with suspicion of nosocomial maxillary sinusitis. *Intensive Care Med.* 2006;32:858–866.
7. Holzapfel L, Chastang C, Demingeon G, et al. A randomized study assessing the systematic search for maxillary sinusitis in nasotracheally mechanically ventilated patients. *Am J Respir Crit Care Med.* 1999;159:695–701.
8. Marik PE. Fever in the ICU. *Chest.* 2000;117:855–869.
9. Rouby JJ, Laurent P, Gosnach M, et al. Risk factors and clinical relevance of nosocomial maxillary sinusitis in the critically ill. *Am J Respir Crit Care Med.* 1994;150:776–783.
10. Raphael DT, Conard FU 3rd. Ultrasound confirmation of endotracheal tube placement. *J Clin Ultrasound.* 1987;15:459–462.
11. Milling TJ, Jones M, Khan T, et al. Transtracheal 2-D ultrasound for identification of esophageal intubation. *J Emerg Med.* 2007;32:409–414.
12. Werner SL, Smith CE, Goldstein JR, et al. Pilot study to evaluate the accuracy of ultrasonography in confirming endotracheal tube placement. *Ann Emerg Med.* 2007;49:75–80.
13. Ma G, Davis DP, Schmitt J, et al. The sensitivity and specificity of transcricothyroid ultrasonography to confirm endotracheal tube placement in a cadaver model. *J Emerg Med.* 2007;32:405–407.
14. Chun R, Kirkpatrick AW, Sirois M, et al. Where's the tube? Evaluation of hand-held ultrasound in confirming endotracheal tube placement. *Prehosp Disaster Med.* 2004;19:366–369.
15. Weaver B, Lyon M, Blaivas M. Confirmation of endotracheal tube placement after intubation using the sliding lung sign. *Acad Emerg Med.* 2006;13:239–244.
16. Lakhal K, Delplace X, Cottier JP, et al. The feasibility of ultrasound to assess subglottic diameter. *Anaesth Analg.* 2007;104:611–614.
17. Ezri T, Gewurtz G, Sessler DI, et al. Prediction of difficult laryngoscopy in obese patients by ultrasound quantification of anterior neck soft tissue. *Anaesthesia.* 2003;58:1111–1114.
18. Komatsu R, Sengupta P, Wadhwa A, et al. Ultrasound quantification of anterior soft tissue thickness fails to predict difficult laryngoscopy in obese patients. *Anaesth Intensive Care.* 2007;35:32–37.
19. Ding LW, Wang HC, Wu HD, et al. Laryngeal ultrasound: a useful method in predicting post-extubation stridor. A pilot study. *Eur Resp J.* 2006;27:384–389.
20. Bertram S, Emshoff R, Norer B. Ultrasonographic anatomy of the anterior neck: implications for tracheostomy. *J Oral Maxillofac Surg.* 1995;53:1420–1424.
21. Sustic A, Zupan Z. Ultrasound guided tracheal puncture for non-surgical tracheostomy. *Intensive Care Med.* 1998;24:92.
22. Bonde J, Norgaard N, Antonsen K, et al. Implementation of percutaneous dilation tracheotomy-value of preincisional ultrasonic examination? *Acta Anaesthesiol Scand.* 1999;43:163–166.
23. Hatfield A, Bodenham A. Portable ultrasonic scanning of the anterior neck before percutaneous dilational tracheostomy. *Anaesthesia.* 1999;54:660–663.
24. Muhammad JK, Patton DW, Evans RM, et al. Percutaneous dilational tracheostomy under ultrasound guidance. *Br J Oral Maxillofac Surg.* 1999;37:309–311.
25. Sustic A, Zupan Z, Eskinja N, et al. Ultrasonographically guided percutaneous dilational tracheostomy after anterior cervical spine fixation. *Act Anaesthesiol Scand.* 1999;43:1078–1080.
26. Muhammad JK, Major E, Patton DW. Evaluating the neck for percutaneous dilational tracheostomy. *J Craniomaxillofac Surg.* 2000;28:336–342.
27. Shlugman D, Satya-Krishna R, Loh L. Acute fatal haemorrhage during percutaneous tracheostomy. *Br J Anaesth.* 2003;90:517–520.
28. Gwilym S, Cooney A. Acute fatal haemorrhage during percutaneous trachestomy. *Br J Anaesth.* 2004;92:298.
29. Muhammad JK, Major E, Wood A, et al. Percutaneous tracheostomy: hemorrhagic complications and the vascular anatomy of the anterior neck. A review based on 497 cases. *Int J Oral Maxillofac Surg.* 2000;29:217–222.
30. Sustic A, Kovac D, Zgaljardic Z, et al. Ultrasound guided percutaneous dilational tracheostomy: a safe method to avoid cranial misplacement of the tracheostomy tube. *Intensive Care Med.* 2000;26:1379–1381.
31. Reilly PM, Sing RF, Giberson FA, et al. Hypercarbia during tracheostomy: a comparison of percutaneous endoscopic, percutaneous Doppler, and standard surgical tracheostomy. *Intensive Care Med.* 1997;23:859–864.

CHAPTER 21

Ultrasound Evaluation of the Pleura

Lewis Eisen and Peter Doelken

INTRODUCTION

Pleural ultrasonography has great utility for the intensivist. As early as 1967, it was apparent that ultrasound was ideally suited for the detection of pleural effusions.[1] In addition to permitting imaging of pleural effusions, thoracic ultrasonography can also detect less common pleural pathology and is ideally suited to guide thoracentesis and other pleural procedures.

GENERAL CONSIDERATIONS IN PLEURAL ULTRASOUND

Ultrasound examination of the pleura is influenced by the surrounding structures. The ribs completely absorb ultrasound waves and prevent deeper structures from being visualized. In contrast, air reflects ultrasound. The surface of aerated lung will reflect most of the ultrasound waves. The point of reflection is immediately below the pleura. However, if the lung is consolidated or atelectatic, it can be readily visualized.

In addition to the artifacts seen in other aspects of medical ultrasonography, there are specific artifacts, such as rib shadowing, that are found commonly in pleural ultrasound. Air reverberation artifacts, which originate below the pleura, are another artifact type that can be commonly observed by clinicians (see Chapter 22). Translational artifacts, due to patient breathing or mechanical ventilation, may also confuse the examiner.[2]

Obesity and subcutaneous edema can degrade image quality. Significant edema may also present problems in judging the depth for procedures. The presence of subcutaneous air will make visualization of deeper structures problematic. The use of firm pressure on the skin and the use of a coupling medium will reduce some artifacts. Artifacts are often visible in only one scanning plane, so changing the probe angle may cause artifacts to disappear. Furthermore, artifacts usually will not move with the respiratory cycle. The observation of an image throughout several respiratory cycles often clarifies the issue.

ULTRASOUND MACHINE REQUIREMENTS AND MACHINE CONTROL

Pleural ultrasonography can be performed with many different types of two-dimensional ultrasound machines. Doppler capability is not needed. A probe with a small enough "footprint" to easily fit between rib spaces should be used. The preferred ultrasound probe is a sector or curved-array transducer with a frequency of 2–5 MHz (typically 3.5 MHz). Probes with higher frequencies can visualize the pleural surface but lack adequate penetration for most clinical applications.

In order to enhance one's knowledge of pleural ultrasonography, the use of a uniform probe orientation and screen marker is suggested. This allows easy comparison of images with those found in the literature. Furthermore, the recording and reporting of images will be standardized. The machine should be prepared so that the image marker on the screen is in the upper left corner of the screen. In longitudinal scanning, the probe should be oriented with the marker positioned cephalad. If this orientation is maintained, the cephalad projection will always be on the left of the screen.

Before making any clinical judgments, the clinician should set the gain and depth on the machine. The gain should be set so that images of the pleural surface and chest wall are optimized. Additionally, gain should be adjusted so that deeper structures such as the liver or spleen are clearly visualized. It is recommended that the depth setting first be set to near-maximum depth, which allows for a general overview of the entire area and prevents oversight of deeper structures. When better visualization of the pleural surface and superficial structures is required, the depth setting can be adjusted to allow for better magnification of near-field structures.

NORMAL PLEURAL EXAMINATION

The pleural ultrasound examination should be performed in a systematic fashion. With the probe applied in an interspace in a longitudinal orientation, the

pleural surface appears as a bright line between the chest wall and the air artifact of the lung. By sliding the probe longitudinally along the chest wall, adjacent interspaces can be examined. After completing an entire scan line, the probe can be moved medially or laterally and another scan line can be obtained. In this way, a near-complete mapping of the pleura can be obtained. The diaphragmatic pleura can be viewed through a transhepatic approach. On the patient's left side, in the absence of pleural fluid, the full length of the diaphragmatic pleura may not be visible due to air artifact. However, in most cases, clinically relevant information can still be obtained. The mediastinal pleura generally cannot be visualized with a transthoracic probe. The visceral subcostal pleura may be obscured by rib shadowing. As the intensivist becomes more skilled in image acquisition, changing of probe angle or alteration of patient position can overcome this problem.

The normal pleura is 0.2–0.4 millimeters (mm) thick.[3] Although the frequency of the probe used for general ultrasonography cannot individually resolve the parietal and visceral pleura, this does not have any clinical relevance for the intensivist. A complete ultrasound examination of the pleura in the ambulatory patient is usually performed with the patient in an upright position. This poses particular problems for the intensivist because patients are in the supine position while mechanically ventilated and sedated. Fortunately, many abnormalities can be detected via an anterior and lateral thorax examination of a supine patient. If the posterior chest must be examined, the supine patient may be placed in a lateral decubitus position. If a major change in a patient's position is required to perform pleural ultrasonogaphy, the intensivist must pay careful attention to support lines and tubes to avoid unplanned device removal.

PLEURAL EFFUSION

Pleural effusion is a common problem in the intensive care unit (ICU). Mattison et al. reported a prevalence of 62% in medical ICU patients.[4] The most common causes were heart failure, atelectasis, parapneumonic effusion, and hepatic hydrothorax. Malignancy accounted for 3.2% and empyema accounted for 1.6%. Compared with patients without effusions, patients with effusions are sicker and have longer ICU stays and longer durations of mechanical ventilation.

Ultrasonography is well suited for the identification and evaluation of fluid because fluid is less echogenic than soft tissue. Many studies have demonstrated the usefulness of ultrasound for this indication. Pleural effusions as small as 3–5 milliliters (mls) can be detected ultrasonographically.[5] Clinical examination is neither sensitive nor specific for the detection of pleural effusion.[6] Pleural ultrasonography is superior to standard chest radiography in detecting the presence of pleural effusions and in distinguishing pleural effusions from atelectasis or pleural thickening.[5,7] Compared with the "gold standard" of chest computerized tomography (CT) scan, pleural ultrasound has 93% sensitivity and specificity for pleural effusions.[8] When a patient has complete opacification of a hemithorax, ultrasound has 95% sensitivity for pleural effusion.[9]

The supine chest radiograph in patients in the ICU has poor performance characteristics for the detection of pleural fluid. Intensive care unit radiographs suffer from problems with penetration, rotation, and magnification. In the supine patient, pleural effusions accumulate in dependent areas. Thoracic opacities in a supine chest radiograph may be caused by pleural effusions, atelectasis, consolidation, or any combination of these processes. For example, a patient with acute respiratory distress syndrome may have all three of these processes. Pleural ultrasound outperforms chest radiography when compared with chest CT for this indication.[8] ICU radiographs often cannot distinguish between pleural and parenchymal abnormalities.[10–12] In a series of ICU patients, supine radiographs detected only 61.4% of the incidents of pleural fluid as compared with those detected by ultrasound.[13] The gold standard in this case was withdrawal of pleural fluid during thoracentesis.

Free-flowing pleural effusions will layer posteriorly in the thorax of the supine patient. Patients with multiple lines or compromised hemodynamic and oxygenation status will be difficult to position sitting upright in bed. If the patient is supine, the bed may prevent the easy visualization of small pleural effusions. One option is for the examiner to place the transducer in the posterior axillary line while angling the probe up toward the center of the body to visualize smaller effusions. In unstable patients who have effusions that are difficult to visualize, positioning the patient in a lateral decubitus position may be helpful. The examiner should always identify three findings (Table 21.1) indicating the presence of a pleural effusion (Figure 21.1, and Video 21.1 in enclosed DVD).

1. Anatomic boundaries: This requires clear identification of the diaphragm and subdiaphragmatic organs (liver or spleen, depending on the side), the chest wall, and the lung, which should be clearly differentiated from the pleural effusion.

TABLE 21.1. Ultrasonographically detectable characteristics helpful in pleural effusion evaluation

1. Pleural effusion boundaries
   Chest wall, diaphragm with underlying liver or spleen, lung, heart, spinal column
2. Dynamic signs
   Floating lung, sinusoid sign, curtain sign, undulating fronds or strands, swirling particles
3. Echogenicity
   Anechoic, homogeneously echogenic, heterogeneously echogenic (complex), strands, fronds, septations, particles
4. Semiquantitative assessment of effusion volume

2. Echo-free space: The relatively echo-free space surrounded by typical anatomic boundaries is the pleural effusion.
3. Dynamic changes: Characteristic changes for a pleural effusion should be identified.

In the supine position, the examiner should start scanning in the posterior axillary line. The diaphragm should be visualized as a curved structure that moves with respiration. Its location should be confirmed by clearly identifying the subdiaphragmatic structures. Generally, pleural fluid will be less echogenic than the adjacent liver or spleen. Rarely, a complex pleural effusion will have echogenicity similar to these organs. The misidentification of pleural fluid in this instance could have deleterious effects if thoracentesis is planned.

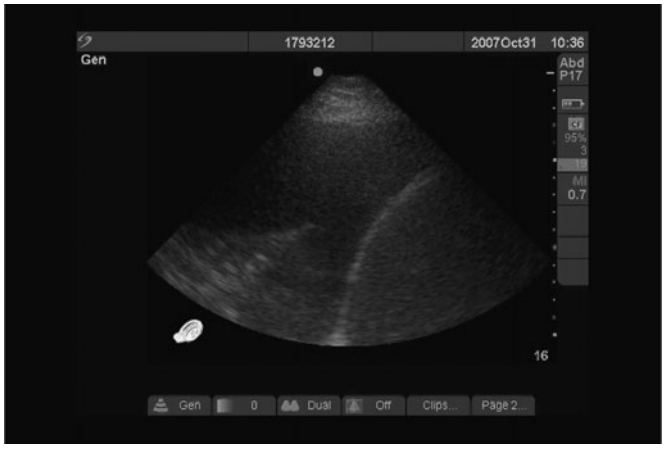


Figure 21.1. Large pleural effusion showing characteristic anatomic boundaries: diaphragm, chest wall, and atelectatic lung surrounding a hypoechoic pleural effusion.

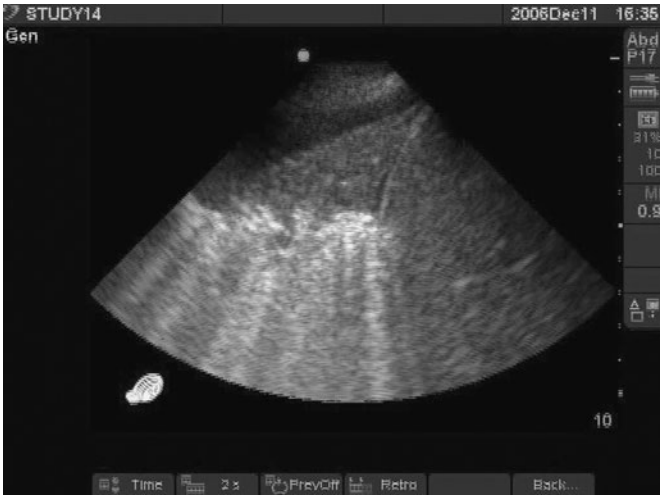


Figure 21.2. Small pleural effusion showing characteristic anatomic boundaries: diaphragm, chest wall, and atelectatic lung with adjacent hypoechoic pleural effusion. The atelectatic lung has the same echogenicity as the adjacent liver.

The inside of the chest wall, because it is a stationary structure, should not undergo dynamic changes with respiration. Local lung tissue will generally have tissue density, but becomes atelectatic when it is compressed by the neighboring pleural effusion and appears as a tissue density structure on ultrasonographic examination (Figure 21.2, and Video 21.2 in enclosed DVD).

This is termed sonographic hepatization of lung, as sonographically, atelectatic lung has the same echodensity as liver. The compressed lung can be visualized floating in the pleural fluid. This has been termed lung flapping or the jellyfish sign (Video 21.3 in enclosed DVD). Aerated lung may move into the scanning field during the respiratory cycle. Interposition of aerated lung into the scanning plane will block visualization of deeper structures. This has been termed the curtain sign (Video 21.4 in enclosed DVD). If the operator is familiar with M-mode ultrasound, examination of the pleural surface generally will show a sinusoid sign, indicating dynamic movement of the pleural surface within a fluid-filled space. (Figure 21.3).[14]

Other findings characteristic of pleural effusions include the plankton sign (Video 21.5 in enclosed DVD), which is caused by swirling debris agitated by cardiac or respiratory motion in a pleural effusion. Fibrin strands may be seen moving in synchronization with cardiac pulsations or respiratory motion (Video 21.6 in enclosed DVD). These strands may be visibly connected to one or more of the pleural surfaces. The hematocrit sign may be observed in cases of cellular pleural effusions of different etiologies. The effusion is

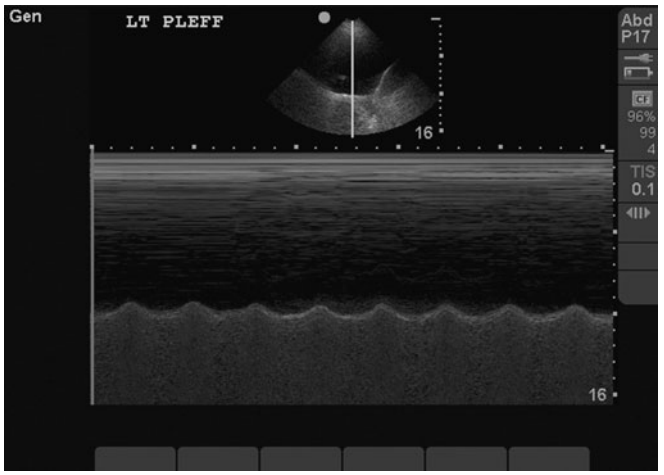


Figure 21.3. Sinusoid sign. M-mode ultrasonography of a pleural effusion shows the hypoechic pleural effusion and movement of the lung during respiratory cycling. This is a convenient method of documenting the presence of a pleural effusion without storage of a video clip.

layered into two phases of different echogenicity by its gravitational effect (Video 21.7 in enclosed DVD).

In addition to the identification of a pleural effusion, the examiner should seek to characterize the fluid and quantify its amount (Table 21.2). Several authors have developed rules to quantify the amount of fluid in the pleural space with reasonable accuracy.[15–18] In most circumstances, it is sufficient to qualitatively judge a pleural effusion as small, moderate, or large. The echogenecity of the fluid should be characterized. Transudates are almost always anechoic.[19] However, they may also have a complex nonseptated pattern.[20] They should not have a homogenous exudative or septated pattern. Highly cellular exudates may be homogenously echogenic. Echogenic swirling patterns are suggestive of exudates (particularly malignant effusion); however, this pattern can also be seen occasionally in transudative effusions.[21] Exudative effusions may have an echogenic pattern that is not homogenous. Debris, strands, or septations may be visible. In the case of parapneumonic effusions, these structures indicate either a complicated parapneumonic effusion or an empyema.[22] Patients with septated effusions visible on ultrasonography require longer hospital stays, longer chest tube drainage, and more often require fibrinolytic therapy or surgery for adequate drainage (Figure 21.4, and Videos 21.8–11 in enclosed DVD).[20] Pleural ultrasound is superior to CT scan in visualizing septations within a pleural effusion.[23]

TABLE 21.2. Key concepts and findings in pleural ultrasonography

1. Anatomic boundaries: diaphragm, chest wall, ribs, visceral pleura, lung (normal, consolidated, or atelectatic)
2. Other structures: liver, spleen, kidney, heart, spinal column, aorta, IVC
3. Dynamic findings: diaphragmatic motion, floating lung, lung point, respirophasic shape changes (sinusoid sign)
4. Characterization of pleural fluid: echogenicity, presence of strands/debris/septation
5. Miscellaneous findings: pleural-based lesions, masses, or thickening
6. Semiquantitative assessment of fluid volume
7. Limits of pleural ultrasonography: inadequate image quality due to technical limitations, hemothorax, echo dense purulent fluid, mimics of effusion
8. Significance of lung sliding, B-lines, seashore sign, lung point, stratosphere sign for evaluation of pneumothorax

The most complicated effusions are loculated. Loculated effusions characteristically occur in a nondependent position and do not move with changes in body position. Loculated effusions consist of thick-walled circular fluid collections. A complete pleural ultrasound is required to identify the presence of a loculated effusion because its position may be nondependent.

Once pleural fluid has been characterized by ultrasound, the intensivist is left with the question of whether thoracentesis should be performed. Thoracentesis is a safe procedure in the ICU, even in mechanically ventilated patients.[13,24–28] For a particular patient, the intensivist should assess the risk and

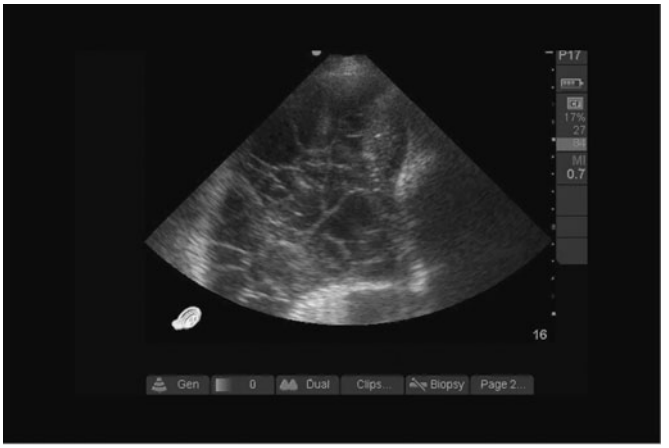


Figure 21.4. Complex septated pleural effusion.

benefits of thoracentesis. A distance of >15 mm from the chest wall to underlying structures should be visualized. The intensivist should observe multiple respiratory cycles to avoid interposition of any vital structure.[29]

Once the feasibility is assured, thoracentesis may be performed for diagnostic or therapeutic reasons. Tu and colleagues studied 94 febrile ICU patients with pleural effusions. A total of 61.7% had infectious exudates of which 15/95 (16.0%) were empyemas. Subgroup analysis of those patients with empyema showed that all of the patients with empyema had either a complex hyperechoic pattern, a complex septated pattern, or a homogenously echogenic pattern. They concluded that in the febrile patient, nonechoic and hypoechoic effusions do not require immediate thoracentesis because they are unlikely to represent empyemas.[22] Fartoukh et al. studied routine thoracentesis of pleural effusions in the ICU. They concluded that of the 82 patients without contraindication to thoracentesis, the presumptive diagnosis was changed in 45.1% of the patients and treatment was changed in 35.4%.[24] This evidence indicates that, in an ICU patient meeting standard indications, thoracentesis should be performed.

The benefits of therapeutic thoracentesis have been harder to prove. Ahmed et al. performed thoracentesis on 22 ventilated patients in the surgical ICU. The mean fluid removed was 1262 mL. Drainage of pleural effusions resulted in increased oxygen delivery and consumption. There was a reduction in pulmonary capillary wedge pressure, and the pulmonary arteriovenous shunt decreased.[30] Doelken et al. demonstrated a reduction in the work of breathing after thoracentesis in eight patients receiving mechanical ventilation.[31] However, no change in respiratory system resistance, compliance, intrinsic positive end-expiratory pressure (PEEP), or gas exchange was evident. Because all of these individual measurements offered only a limited assessment of a complex system, the clinician should weigh the risks and benefits of thoracentesis in each patient. In our opinion, thoracentesis of large pleural effusions helps liberate patients from mechanical ventilation; however, more research in this area is required.

SOLID PLEURAL ABNORMALITIES

A variety of solid pleural abnormalities may be visualized as echogenic structures within the pleural space. Benign pleural tumors such as benign mesothelioma, lipomas, or chondromas may be seen. They will be surrounded by a distinct capsule and will not invade tissue planes. A complementary imaging modality may be required for confirmation. Malignant mesothelioma will have hypoechoic thickening of the pleural surface with irregular or indistinct borders. Malignant mesothelioma may invade the diaphragm or chest wall. It may also be nodular in character. Pleural metastatic disease will often be accompanied by a pleural effusion. Metastatic lesions are frequently multiple. Their echogenecity is variable. Chest wall and diaphragmatic invasion will be apparent. Two case series demonstrated the value and accuracy of ultrasound for detecting chest wall invasion by lung cancer.[32–33] Pleural fibrosis is the end result of multiple types of pleural injury. Pleural thickening will be evident. Respiratory movement will be diminished or absent at this location.[34] In addition to helping to characterize lesions, pleural ultrasonography is ideally suited for guiding biopsy.

References

1. Joyner CR Jr, Herman RJ, Reid JM. Reflected ultrasound in the detection and localization of pleural effusion. *JAMA.* 1967;200:399–402.
2. Schuler A. Image artifacts and pitfalls. In: Mathis G, Lessnau KD, eds. *Atlas of Chest Sonography.* Berlin, Germany: Springer-Verlag; 2003:137–145.
3. Reuss J. The pleura. In: Mathis G, Lessnau KD, eds. *Atlas of Chest Sonography.* Berlin, Germany: Springer-Verlag; 2003:17–35.
4. Mattison LE, Coppage L, Alderman DF, et al. Pleural effusions in the medical ICU: prevalence causes and clinical implications. *Chest.* 1997;111:1018–1023.
5. Gryminski J, Krakowka P, Lypaceqicq G. The diagnosis of pleural effusion by ultrasonic and radiologic techniques. *Chest.* 1976;70:33–7.
6. Diacon AH, Brutsche MH, Soler M. Accuracy of pleural puncture site: a prospective comparison of clinical examination with ultrasound. *Chest.* 2003;123:436–441.
7. Kelbel C, Borner N, Schadmand S, et al. Diagnosis of pleural effusions and atelectasis: sonography and radiology compared. *Rofo.* 1991;154:159–163.
8. Lichtenstein D, Goldstein I, Mourgeon E, et al. Comparative diagnostic performances of auscultation, chest radiography, and lung ultrasonography in acute respiratory distress syndrome. *Anesthesiology.* 2004;100:9–15.
9. Yu C, Yang P, Chang D, et al. Diagnostic and therapeutic use of chest sonography: Value in critically ill patients. *AJR.* 1992;159:695–701.

10. Mirvis SE, Tobin KD, Kostrubiak I, et al. Thoracic CT in detecting occult disease in critically ill patients. *AJR*. 1987;148:685–689.
11. Overfors C, Hedgecock MW. Intensive care unit radiology: problems of interpretation. *Radiol Clin North Am.* 1978; 16:407–409.
12. Yu CJ, Yang PC, Wu HD, et al. Ultrasound study in unilateral hemithorax opacification: image comparison with computed tomography. *Am Rev Respir Dis.* 1993;147:430–434.
13. Lichtenstein DA, Hulot JS, Rabiller A, et al. Feasibility and safety of ultrasound-aided thoracentesis in mechanically ventilated patients. *Intensive Care Med.* 1999;25:955–958.
14. Lichtenstein DA. Pleural effusion and introduction to lung ultrasound. In: Lichtenstein DA, ed. *General Ultrasound in the Critically Ill.* Berlin, Germany: Springer-Verlag; 2005;96–104.
15. Balik M, Plasil P, Waldauf P, et al. Ultrasound estimation of volume of pleural fluid in mechanically ventilated patients. *Intensive Care Med.* 2006;32:318–321.
16. Eibenberger KL, Dock WI, Ammann ME, et al. Quantification of pleural effusions: sonography versus radiography. *Radiology.* 1994;191:681–684.
17. Roch AMD, Bojan M, Michelet P, et al. Usefulness of ultrasonography in predicting pleural effusions >500 ml in patients receiving mechanical ventilation. *Chest.* 2005;127:224–232.
18. Vignon P, Castagner C, Berkane V, et al. Quantitative assessment of pleural effusion in critically ill patients by means of ultrasonography. *Crit Care Med.* 2005;33:1757–1763.
19. Yang PC, Luh KT, Chang DB, et al. Value of sonography in determining the nature of pleural effusion: analysis of 320 cases. *AJR Am J Roentgenol.* 1992;159:29–33.
20. Chen HJ, Tu CY, Ling Sj. Sonographic appearances in transudative pleural effusions: not always an anechoic pattern. *Ultrasound Med Biol.* 2008;34:362–369.
21. Chian CF, Su WL, Soh LH, et al. Echogenic swirling pattern as a predictor of malignant pleural effusions in patients with malignancies. *Chest.* 2004;126:129–134.
22. Tu CY, Hsu WH, Hsia TC, et al. Pleural effusions in febrile medical ICU patients chest ultrasound study. *Chest.* 2004;126:1274–1280.
23. McLoud TC, Flower CD. Imaging the pleura: sonography, CT, and MR imaging. *Am J Roentgenol.* 1991;156:1145–1153.
24. Fartoukh M, Azoulay E, Galliot R et al. Clinically documented pleural effusions in medical ICU patients: how useful is routine thoracentesis? *Chest.* 2002;121:178–184.
25. Gervais DA, Petersein A, Lee MJ, et al. US-guided thoracentesis: requirement for postprocedure chest radiography in patients who receive mechanical ventilation versus patients who breathe spontaneously. *Radiology.* 1997;204:503–506.
26. Godwin JE, Sahn SA. Thoracentesis: a safe procedure in mechanically ventilated patients. *Ann Intern Med.* 1990; 113:800–802.
27. Mayo PH, Goltz HR, Tafreshi M, et al. Safety of ultrasound-guided thoracentesis in patients receiving mechanical ventilation. *Chest.* 2004;125:1059–1062.
28. McCartney JP, Adams JW, Hazard PB. Safety of thoracentesis in mechanically ventilated patients. *Chest.* 1993; 103:1920–1921.
29. Lichtenstein DA. Ultrasound in the management of thoracic disease. *Crit Care Med.* 2007;735:S250–S261.
30. Ahmed SH, Ouzounian SP, Dirusso S, et al. Hemodynamic and pulmonary changes after drainage of significant pleural effusions in critically ill, mechanically ventilated surgical patients. *J Trauma.* 2004;57:1184–1188.
31. Doelken P, Abreu R, Sahn S, et al. Effect of thoracentesis on respiratory mechanics and gas exchange in the patient receiving mechanical ventilation. *Chest.* 2006;130:1354–1361.
32. Suzuki N, Siatoh T, Kitamura S. Tumor invasion of the chest wall in lung cancer: diagnosis with US. *Radiology.* 1993;187:39–42.
33. Sugama Y, Tamaki S, Kitamura S, et al. Ultrasonographic evaluation of pleural and chest wall invasion of lung cancer. *Chest.* 1988;93:275–279.
34. Mayo PH, Doelken P. Pleural Ultrasonography. *Clin Chest Med.* 2006;27:215–227.

CHAPTER 22

Ultrasound Evaluation of the Lung

Paul H. Mayo

INTRODUCTION

Lung ultrasonography is easy to learn, simple to perform, and has strong clinical utility for the critical care clinician. Interestingly, radiologists have not been instrumental in developing critical care applications of lung ultrasonography. Perhaps because lung ultrasonography in the intensive care unit (ICU) is a purely beside technique, it required a frontline ICU clinician to develop the field. Dr. Daniel Lichtenstein is responsible for developing critical care lung ultrasonography. In the 1990s, he published a series of landmark articles that defined the important features of the field. He also developed the semiology of lung ultrasonography that is in current use. Based on his original work, in the past few years there have been numerous published studies from other groups, which have served to validate and expand the field. This section will review critical care applications of lung ultrasonography.

BASIC PRINCIPLES OF LUNG ULTRASONOGRAPHY

Air is the enemy of the ultrasonographer. There is a large difference in the acoustic impedance and velocity of ultrasound between tissue and air. This leads to a reflection of the ultrasound wave at any air–tissue interface. When combined with the unfavorable attenuation coefficient of air, this leads to the homogeneous amorphic grayness that occupies the ultrasound screen deep to a tissue–air interface. This frustrates any attempt to scan through air to deeper body structures.

The normal alveolar lung parenchyma is filled with air, so that lung is not visible as a discreet structural entity with ultrasonography. Air blocks the visualization of normally aerated lung. When a disease process reduces the amount of air within the lung, ultrasound findings change in a predictable fashion. Atelectatic lung is airless, and appears as a discrete structure with tissue density. Likewise, lung that is consolidated from pneumonia appears as a well-defined hyperechoic structure. Lung that is edematous, though still aerated, has ultrasonographic findings that are different from normally aerated lung. One of the limitations of lung ultrasonography is that abnormalities that are surrounded by aerated lung cannot be visualized. Fortunately, most lung processes that are of interest to the intensivist (e.g., pneumonia, hydrostatic pulmonary edema, lesional edema) extend to the periphery of the lung. The effect that fluid accumulation has on ultrasonographic findings is summarized in Figure 22.1. Lung ultrasonography demonstrates a spectrum of patterns that depends on the ratio of air to fluid: from a normal aeration pattern to alveolar/interstitial edema and finally to a tissue density pattern. Each of these findings may have major implications for the management of the critically ill patient.

MACHINE REQUIREMENTS

Lung ultrasonography may be performed with a wide variety of ultrasound machines with two-dimensional (2D) scanning capability. It was fully described using a machine manufactured in 1990. A 3.5–5.0 MHz transducer of convex sector design works well. Vascular transducers of higher frequency may also yield serviceable images, although the examination may be limited by a lack of penetration in the larger patient. A microconvex transducer has the advantage that it fits well between rib interspaces. As lung ultrasonography will generally be performed in the context of a whole body approach, many groups use a cardiac transducer for general critical care ultrasonography (lung, pleura, abdominal) to reduce cost. Transducers of linear design may be used, but these are difficult to use in a longitudinal scanning orientation in the thin individual. Paradoxically, high-end, recent generation ultrasound machines may yield inferior lung ultrasound images compared with machines from the 1990's. Complex image smoothing technology that is appropriate for advanced cardiac imaging may provide suboptimal results for lung ultrasonography. When using this type of machine, the operator may have to bypass machine settings in order to obtain a basic or fundamental

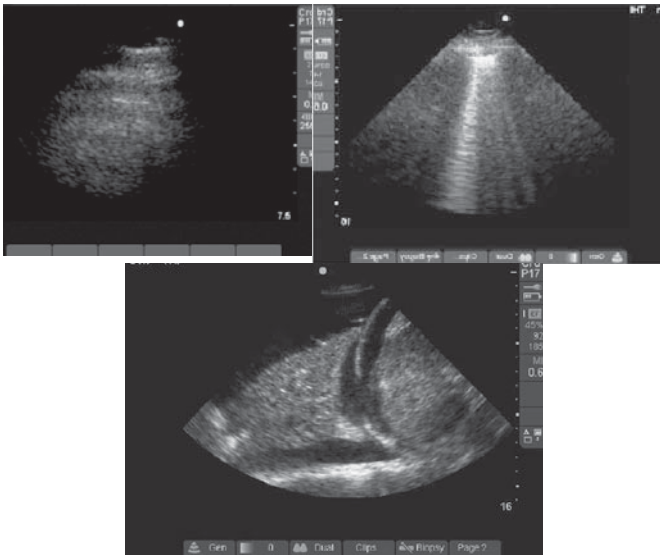


Figure 22.1. Pneumothorax and normal aeration pattern yields A line pattern (image 1) with a high air/fluid ratio. Lung edema cause B lines with a moderate air/fluid ratio (image 2). Pleural fluid and alveolar consolidation (seen together in image 3) with a very low air/fluid ratio. The amount of fluid relative to air results in the characteristic ultrasound patterns of thoracic ultrasonography.

ultrasound image with the clear near-field resolution necessary for lung ultrasonography.

PERFORMANCE OF LUNG ULTRASONOGRAPHY

By convention, lung ultrasonography is performed in a longitudinal scanning plane with the transducer held perpendicular to the skin surface. Multiple sites on the chest are scanned in sequence. It is advisable to scan the thorax using a standard section approach, as results can then be reported in reference to a particular area. For this purpose, the chest may be divided into an anterior and lateral area. The anterior area is bordered by the sternum and the anterior axillary line, while the anterior and posterior axillary lines border the lateral area. Many patients who are critically ill are in a supine position. This represents a challenge to the ultrasonographer because the posterior thorax may be difficult to image by virtue of being blocked by the surface of the bed. This is frequently an important area to image because pleural effusions and posterior consolidation are found in the dependent thorax. To image these areas, the transducer may be pressed into the mattress and angled anteriorly. Alternatively, the patient may be rolled to a lateral decubitus position to fully expose the posterior thorax. Lung ultrasonography is then performed, as with the lateral and anterior exam, by applying the transducer at multiple interspaces on the back. An efficient manner of thoracic scanning is to move the transducer up the chest wall in a series of scan lines examining each interspace and underlying lung in sequence. This allows the examiner to construct a 3D image of the thorax from multiple 2D images gathered in organized scan-line sequences. Thoracic ultrasonography of the patient who is able to sit up with arms abducted allows easy scanning of the entire thorax with the multiple scan-line technique.

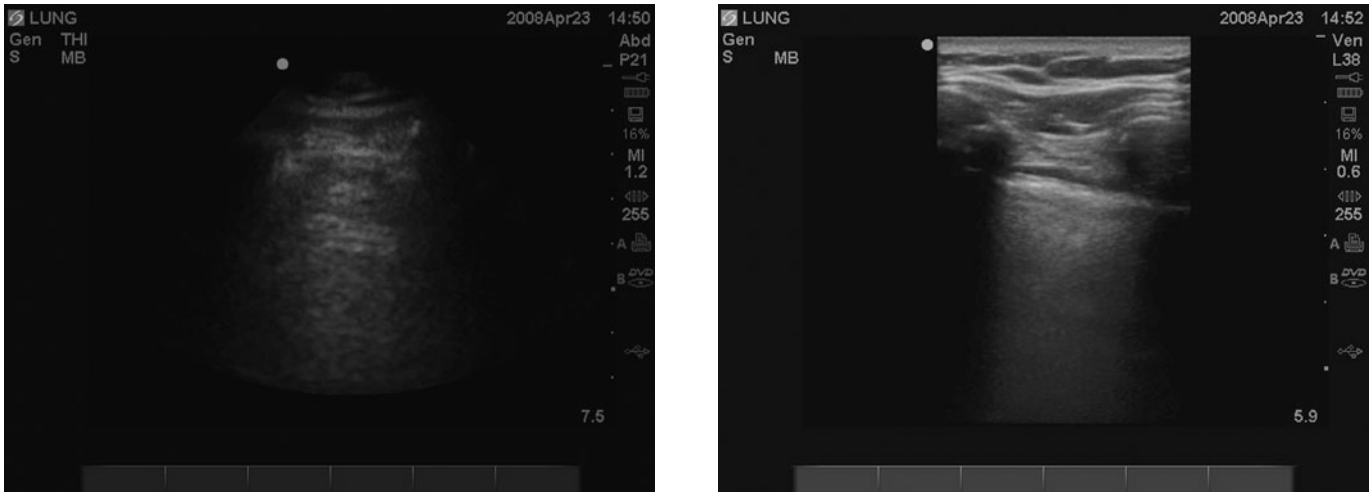
KEY FINDINGS OF LUNG ULTRASONOGRAPHY

Lung ultrasonography is able to detect pneumothorax, normal aeration patterns, alveolar–interstitial fluid accumulation, lung consolidation, and pleural fluid. It is superior to standard supine radiography and similar to chest computerized tomography (CT) in detecting these key findings.[1] The key findings of lung ultrasonography for critical care applications are as follows.

Lung Sliding

With the transducer in a longitudinal orientation perpendicular to the skin surface and centered between two adjacent ribs, a typical lung ultrasound image with the depth adjusted to examine the pleural interface can be displayed (Figure 22.2). Two adjacent rib shadows are noted on either side of the image, with the hyperechoic horizontally orientated pleural line appearing approximately 0.5 centimeters (cm) deep to the origin of the rib shadows. The pleural line represents the apposition of the visceral and parietal pleural surfaces. In the normal examination, the pleural surfaces move against each other during the respiratory cycle. This causes the finding of lung sliding, which is a shimmering mobile pleural line that moves in synchrony with the respiratory cycle (Video 22.1 in enclosed DVD). A related finding is lung pulse. With lung pulse, the pleural line moves synchronously with cardiac pulsation, as the force of cardiac pulsation is sufficient to cause movement of the lung and overlying visceral pleura (Videos 22.2 and 22.3 in enclosed DVD). Sliding lung and lung pulse are dynamic findings that require for their detection real time, 2D scanning. For convenience, they may be recorded with M-mode for purposes of easy documentation (Figure 22.3).

The findings of lung sliding and lung pulse have major significance because they exclude the presence of a pneumothorax at the site of transducer application with a high level of certainty.[2] As air within the pneumothorax space will distribute to the anterior thorax

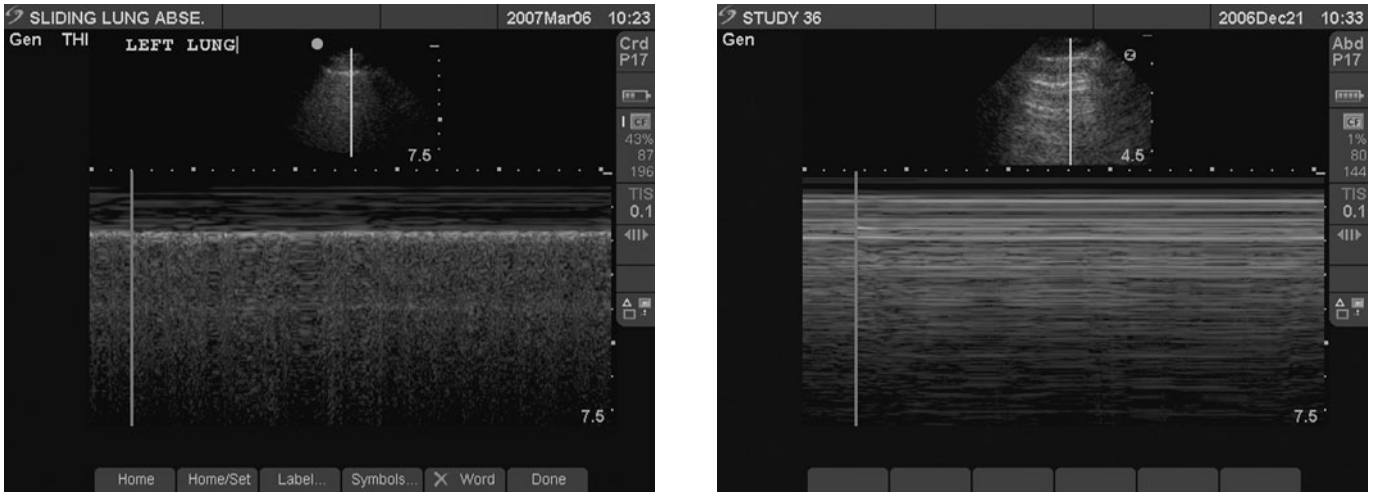


A B

Figure 22.2. (A) The image is obtained using a 3.5 MHz cardiac transducer. The transducer is in longitudinal orientation and placed perpendicular to the chest wall to scan through the second intercostal space. The two adjacent ribs yield shadow artifact. The pleural line is present about 0.5 cm deep to the ribs. (B) The image is obtained using a 7.5 MHz vascular transducer held in an identical fashion to that in (A).

in the supine patient, the critically ill patient is ideally positioned for the examination. Multiple sites may be easily examined for sliding lung over both hemithoraces, so that the intensivist can promptly and confidently rule out pneumothorax. Several groups have reported on the superiority of ultrasonography to rule out pneumothorax when compared with supine chest radiography.[3–6]

Unfortunately, the absence of lung sliding is not as useful (Video 22.4 in enclosed DVD). Loss of lung sliding may occur with pneumothorax, but it also occurs in many other circumstances. Any process that greatly reduces the movement of air into the lung will reduce or abolish lung sliding. Right mainstem bronchial intubation and other causes of mainstem bronchial occlusion (e.g., mucous plug, blood clot, foreign body, tumor) will ablate lung sliding of the left lung. Similarly, any process that impairs lung inflation, such as severe pneumonia, apnea, or severe adult respiratory distress syndrome (ARDS) will result in an absence of lung sliding. Pleural symphysis (inflammatory, neoplastic, cicatricial) will cause a loss of lung sliding. Apnea causes loss of lung sliding, though necessarily of short duration. The *presence* of lung sliding is therefore a powerful sign because it rules out pneumothorax. The *absence* of lung sliding is less useful.[7]



A B

Figure 22.3. M-mode ultrasound image demonstrating "seashore sign": (A) consistent with sliding lung and "stratosphere" sign and (B) consistent with the absence of sliding lung.

In certain situations, lung pulse may be observed in the absence of lung sliding. For example, with a unilateral mainstem bronchial block, lung sliding is lost ipsilateral to the block due to the lack of air entry into the affected lung.[8] Transmission of the cardiac pulsation will remain, providing strong evidence of lung inflation.

Lung Point

Using standard scanning techniques as described above, the presence of a pneumothorax may yield a diagnostic finding on lung ultrasonography called lung point. The lung point is found at the site where partially collapsed lung is apposed to the inside of the chest wall. Some pneumothoraces are total, i.e., the lung is completely collapsed, but most are partial with some remaining apposition of the visceral and parietal pleural. With careful technique, the examiner searches for the site at which the two pleural surfaces touch. It appears as intermittent lung sliding, because the collapsed lung still inflates synchronously, to some extent, with the respiratory cycle. Designated as the lung point, this finding is diagnostic of pneumothorax (Video 22.5 in enclosed DVD).[9] Unfortunately, while 100% specific for pneumothorax, it is relatively insensitive. The sensitivity is, in part, related to operator experience. The detection of lung sliding is an entry-level skill, while lung point requires more experience.

A Lines

Using standard scanning technique with a scanning depth set to examine deeper structures, normally aerated lung yields a characteristic pattern of air artifact called "A lines." A lines are one or more horizontally orientated lines seen deep to the pleural line (Figure 22.4, and Video 22.6 in enclosed DVD). Their depth is a multiplicative of the distance between the skin surface and the pleural line because they are reverberation artifacts from ultrasound reflection between these two surfaces. In the presence of sliding lung, A lines indicate normally aerated lung. They are strongly correlated with a normal aeration pattern on CT scan.[1] In the absence of sliding lung, A lines are less useful. They are found in pneumothorax. They are also found in the absence of pneumothorax without lung sliding (see above).

B Lines

Using standard scanning techniques with a scanning depth set to examine deeper structures, edematous lung yields a characteristic pattern of air artifact termed B lines. B lines have several characteristics (Figure 22.5, and Video 22.7 in enclosed DVD):

1. They are horizontal in orientation and may occur as one or more per field (sometimes termed comet tails or lung rockets)
2. They originate at the pleural interface
3. They extend raylike to the bottom of the screen
4. They efface A lines where the two intersect
5. They generally move in synchrony with lung sliding. However, they are not necessarily mobile, as in the case of B lines in the absence of lung sliding
6. Their presence excludes pneumothorax[10]

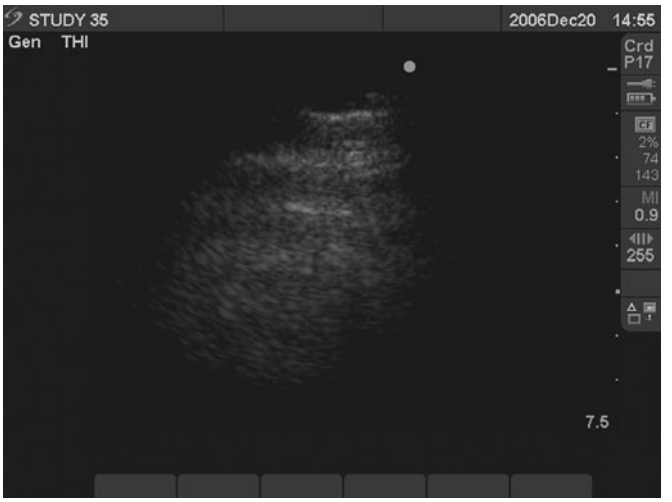


Figure 22.4. A lines are demonstrated using a 3.5 MHz transducer in longitudinal orientation scanning through an intercostal space.

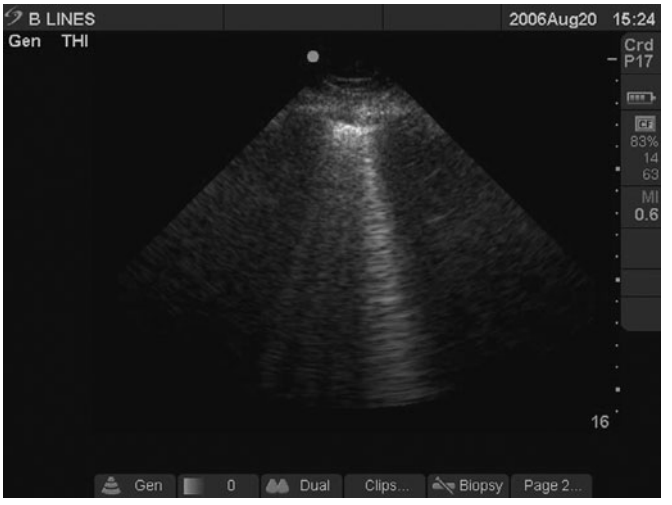


Figure 22.5. B lines are demonstrated using a 3.5 MHz transducer in longitudinal orientation scanning through an intercostal space.

B lines are characteristic of lung edema or any process that infiltrates the interstitium of the lung, such as inflammation, neoplasm, or scarring.[11–15] They are thought to be caused by ring-down artifact from small subpleural fluid collections or tissue densities. The presence of B lines is strongly correlated with alveolar or interstitial pattern abnormalities on CT scan (ground glass or reticular pattern abnormality).[1] Normal individuals may have a few B lines in the lateral lower lung. Both A lines and B lines may be found diffusely or focally on lung ultrasonography, depending on the underlying pathophysiology of the lung process in question.

Consolidation

Using standard scanning techniques with a scanning depth set to examine deeper structures, consolidated lung yields a characteristic ultrasound pattern (Figure 22.6, and Videos 22.8–10 in enclosed DVD). Consolidated lung is tissue density.[16] It has similar echogenicity as the liver; hence, it is sometimes referred to as sonographic hepatization of lung. If the bronchial structures that supply the affected consolidated lung are patent, the consolidated lung may have sonographic air bronchograms within it. These appear as hyperechoic foci that represent small amounts of air in the bronchi. They may be mobile, reflecting movement of air within the bronchus due to respiratory activity. The examiner may easily localize consolidation to a specific lobe; and, with experience, to specific segments of the lung. The finding of consolidation with lung ultrasonography is strongly correlated with results of CT scanning.[1] It is important to understand that the finding of consolidation on lung ultrasonography is purely descriptive, similar to the finding of consolidation on chest radiography or CT scanning. Any process that renders the alveolar compartment airless will demonstrate consolidation on lung ultrasonography or in other radiographic techniques. All causes of airless lung, such atelectasis (compressive, resorptive, or cicatricial), infiltrative processes (tumor, purulent material as in pneumonia), or severe pulmonary edema with complete filling of the alveolar compartment, will yield the ultrasonographic finding of lung consolidation. Lung ultrasonography identifies the consolidation; the clinician determines its cause.

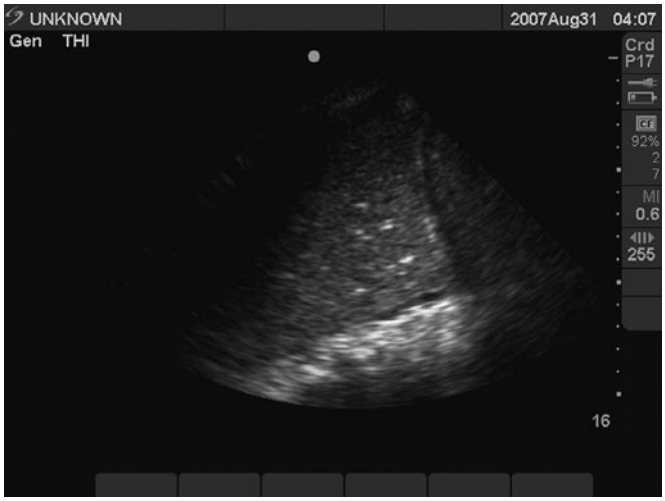


Figure 22.6. Alveolar consolidation pattern of right lower lobe. Sonographic air bronchgrams (white dots in the tissue density lung) are present in the lung. The diaphragm is visible as a curvilinear structure between the liver and lung. The image is obtained using a 3.5 MHz transducer in longitudinal orientation scanning through the fifth intercostal space midaxillary line on the left.

Pleural Effusion

Pleural effusion may be observed when performing lung ultrasonography. Knowledge of lung ultrasonography is essential for proficiency in pleural ultrasonography. A complete discussion of pleural ultrasonography is found in Chapter 21.

CLINICAL APPLICATIONS OF LUNG ULTRASONOGRAPHY

Clarification of the Ambiguous Chest Radiograph

Chest radiography in the supine critically ill patient on ventilator support is frequently difficult to interpret. A complex 3D structure is viewed in two dimensions compounded by the common occurrence of rotation, penetration, and projection artifact. The resultant radiograph shows nonspecific radiodensity; the clinician cannot discern between normal, lung, alveolar or interstitial abnormalities, consolidation, or pleural effusion. Lung ultrasonography is an excellent means of clarifying the results of an ambiguous chest radiograph in the ICU.

Evaluation for Pneumothorax

Lung ultrasonography is very effective for rapidly ruling out pneumothorax. In patients on mechanical ventilatory support, a pneumothorax is particularly dangerous because it may be under the threat of tension.

The supine chest radiography is unreliable in ruling out pneumothorax.[3] Lung ultrasonography may be used to rule out pneumothorax with minimal time delay and a high degree of accuracy in the ICU. In addition, it is useful in the emergency evaluation of pneumothorax in cases of thoracic trauma.[5,6] Lung ultrasonography may be used to evaluate for postprocedure pneumothorax (following thoracentesis or subclavian/internal venous access). It is a straightforward exercise to identify sliding lung *before* the planned procedure. Following the procedure, the continued presence of sliding lung rules out procedure-related pneumothorax, whereas the new absence of sliding lung where it was previously present is strong evidence for a procedure-related pneumothorax.

It is a simple matter to rule out a pneumothorax. Definitive diagnosis of a pneumothorax is more difficult. Absence of sliding lung is suggestive but not diagnostic. Unequivocal diagnosis of pneumothorax requires identification of lung point, which requires an experienced examiner.

Rapid Evaluation of Acute Respiratory Failure

The critical care clinician frequently evaluates the patient with acute respiratory failure. The limitations of standard chest radiography have already been discussed and include, in the acute situation, an element of time delay. Chest CT has time delay, risk of transport of the unstable patient, and major radiation exposure.[17] The intensivist who is equipped with a portable ultrasound unit can bring to the bedside a device that yields more accurate imaging than standard chest radiography. Lung ultrasonography allows prompt identification of the cause for acute respiratory failure in rapid-response-team events, the emergency department, and at the bedside of the acutely decompensating patient in the ICU. In a recent study, Lichtenstein et al. have confirmed the excellent operational utility of lung ultrasonography for the assessment of acute respiratory failure.[7] Based on their data, they have proposed a simple algorithm for evaluating the patient with acute dyspnea derived from performing lung ultrasonography in a large group of patients (Figure 22.7).

In similar fashion, lung ultrasonography is useful in evaluating the patient on mechanical ventilator support with acute severe desaturation, where the intensivist needs immediate diagnosis of a potentially life-threatening event. Barring obvious causes (e.g., mechanical failure of the ventilator/endotracheal tube system, unplanned extubation, severe patient/ventilator dysynchrony etc.), the intensivist may use ultrasonography to rule out pneumothorax and mainstem

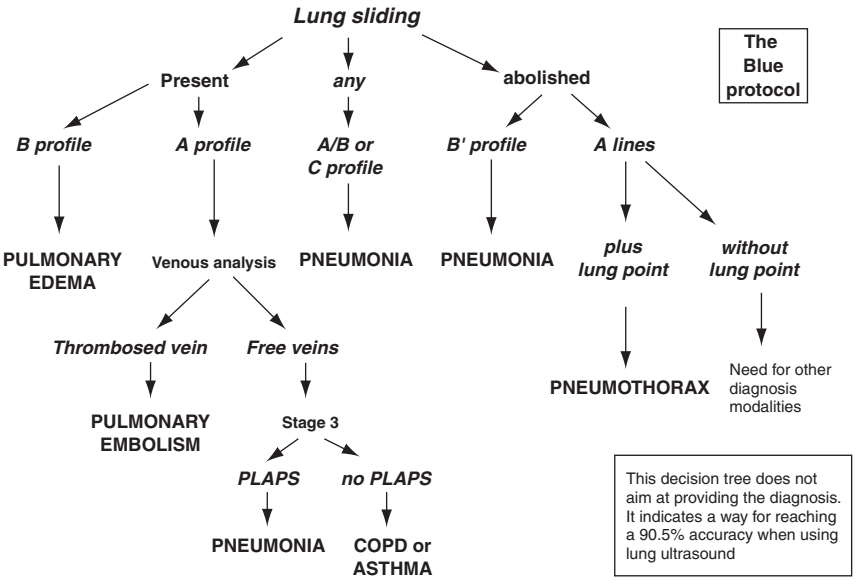


Figure 22.7. A decision tree utilizing lung ultrasonography to guide diagnosis of severe dyspnea.

bronchial block (endotracheal tube movement or mucous plugging) and evaluate for acute pulmonary edema or embolism.[1] Lung ultrasonography is easily combined with basic echocardiography and leg study for deep venous thrombosis to yield maximal diagnostic information in the emergency evaluation of severe cardiopulmonary failure.

Advanced Applications of Lung Ultrasonography

Lichtenstein has described a number of lung ultrasound findings that are of interest to the intensivist, but which are beyond the scope of this text.[18] An interesting potential application of lung ultrasonography is to observe for lung recruitment during ventilator manipulation.[19] Another potential application is in determining whether lung edema is related to a hydrostatic mechanism (heart failure) or lung injury (lesional, as in ARDS). Lung ultrasonography may be used to guide transthoracic needle or device insertion for purposes of biopsy of a lesion that is adjacent to the pleural surface.[20] Intraparenchymal lesions are not visible because the surrounding lung is aerated and blocks the view of the ultrasound. An exception to this principle is lung abcess, where the surrounding lung is generally consolidated thus permitting visualization of the abscess.[21] Lung ultrasonography can be used for the targeted insertion of a drainage catheter into a lung abscess, if clinically indicated. Lung ultrasonography may be used to confirm successful endotracheal tube placement[22] and to rule out a right mainstem bronchial intubation. Lung ultrasonography has utility in the diagnosis of high-altitude pulmonary edema in remote locations[23] and for the identification of lung contusion.

LIMITATIONS OF LUNG ULTRASONOGRAPHY

Operator-Related

Lung ultrasonography requires that the intensivist have specific training in image acquisition, image interpretation, and integration of the results into an effective management strategy. Lung ultrasonography is performed by the frontline intensivist without input from the radiologist or ultrasound technician. A bedside technique, its clinical utility is completely dependent on the skill of the intensivist–ultrasonographer. Adequate training in the field is obviously a requirement.

Machine-Related

Not all ultrasound machines can produce adequate image quality for lung ultrasonography. In particular, extensive image processing may degrade the image quality required for lung ultrasonography. Near-field clutter and lack of resolution of the pleural interface are problems with some recent-generation machines.

Documentation-Related

Critical care lung ultrasonography is frequently performed on an emergency basis in the ICU. The intensivist may be handling multiple tasks, and there may be insufficient time to label and save images that are being quickly obtained from multiple sites on the thorax or to issue a complete written report of all findings. Chest radiography and chest CT scans result in durable reviewable hard copies that are read and documented by well-organized radiology services. The same may not be said for lung ultrasonography.

Difficulty with documentation can lead to problems. First, lack of documentation is a medical–legal concern. Second, the lack of documentation also impacts on the need to know what previous lung ultrasound results showed in a particular patient. In a busy ICU, the ICU team might perform numerous rapid lung scans each day and use the results for important immediate management decisions. Deficient documentation and a limited ability to retrieve ephemeral images for review prevent the clinician from comparing today's result with those of yesterday.

Miscellaneous

Lung ultrasonography cannot resolve lesions that are surrounded by aerated lung. Chest wall dressings and massive edema or obesity may preclude adequate imaging of the lung. Subcutaneous air prohibits lung ultrasonography at the site of the air collection.

CONCLUSION

Lung ultrasonography has strong utility for the frontline intensivist. It can identify pneumothorax, alveolar/interstitial changes, consolidation, and pleural effusion. It is superior in many respects to supine radiography in the ICU. Proficiency in lung ultrasonography will help the intensivist clarify the ambiguous chest radiograph, promptly recognize *postprocedure* pneumothorax, and rapidly evaluate the patient with acute respiratory failure.

References

1. Lichtenstein D, Goldstein I, Mourgeon E, Cluzel P, Grenier P, Rouby JJ. Comparative diagnostic performances of auscultation, chest radiography, and lung ultrasonography in acute respiratory distress syndrome. *Anesthesiology.* 2004;100:9–15.
2. Lichtenstein DA, Menu Y. A bedside ultrasound sign ruling out pneumothorax in the critically ill. Lung sliding. *Chest.* 1995;108:1345–1348.
3. Lichtenstein DA, Mezière G, Lascols N, et al. Ultrasound diagnosis of occult pneumothorax. *Crit Care Med.* 2005;33:1231–1238.
4. Soldati G, Testa A, Sher S, Pignataro G, La Sala M, Silveri NG. Occult traumatic pneumothorax: diagnostic accuracy of lung ultrasonography in the emergency department. *Chest.* 2008;133:204–211.
5. Blaivas M, Lyon M, Duggal SA. Prospective comparison of supine chest radiography and bedside ultrasound for the diagnosis of traumatic pneumothorax. *Acad Emerg Med.* 2005;12:844–849.
6. Kirkpatrick AW, Sirois M, Laupland KB, et al. Hand-held thoracic sonography for detecting post-traumatic pneumothoraces: the Extended Focused Assessment with Sonography for Trauma (EFAST). *J Trauma.* 2004;57:288–295.
7. Lichtenstein DA, Meziere GA. Relevance of lung ultrasound in the diagnosis of acute respiratory failure: the BLUE protocol. *Chest.* 2008;134:117–125.
8. Lichtenstein DA, Lascols N, Prin S, Mezière G. The "lung pulse": an early ultrasound sign of complete atelectasis. *Intensive Care Med.* 2003;29:2187–2192.
9. Lichtenstein D, Mezière G, Biderman P, Gepner A. The "lung point": an ultrasound sign specific to pneumothorax. *Intensive Care Med.* 2000;26:1434–1440.
10. Lichtenstein D, Mezière G, Biderman P, Gepner A. The comet-tail artifact: an ultrasound sign ruling out pneumothorax. *Intensive Care Med.* 1999;25:383–388.
11. Lichtenstein D, Mezière G. A lung ultrasound sign allowing bedside distinction between pulmonary edema and COPD: the comet-tail artifact. *Intensive Care Med.* 1998;24:1331–1334.
12. Lichtenstein D, Mézière G, Biderman P, Gepner A, Barré O. The comet-tail artifact. An ultrasound sign of alveolar-interstitial syndrome. *Am J Respir Crit Care Med.* 1997;156:1640–1646.
13. Agricola E, Bove T, Oppizzi M, et al. "Ultrasound comet-tail images": a marker of pulmonary edema: a comparative study with wedge pressure and extravascular lung water. *Chest.* 2005;127:1690–1695.
14. Agricola E, Picano E, Oppizzi M, et al. Assessment of stress-induced pulmonary interstitial edema by chest ultrasound during exercise echocardiography and its correlation with left ventricular function. *J Am Soc Echocardiogr.* 2006;19:457–463.
15. Jambrik Z, Monti S, Coppola V, et al. Usefulness of ultrasound lung comets as a nonradiologic sign of extravascular lungwater. *Am J Cardiol.* 2004;93:1265–1270.
16. Lichtenstein DA, Lascols N, Mezière G, Gepner A. Ultrasound diagnosis of alveolar consolidation in the critically ill. *Intensive Care Med.* 2004;30:276–281.
17. Brenner DJ, Hall EJ. Computed Tomography—an increasing source of radiation exposure. *N Engl J Med.* 357:2277–2284.
18. Lichtenstein DA. Lung. In: Lichtenstein DA, ed. *General Ultrasound in the Critically Ill.* Berlin, Germany: Springer-Verlag; 2005:116–128.
19. Tsubo T, Yatsu Y, Tanabe T, et al. Evaluation of density area in dorsal lung region during prone position using transesophageal echocardiography. *Crit Care Med.* 2004;32:83–87.
20. Sheth S, Hamper UM, Stanley DB, Wheeler JH, Smith PA. US guidance for thoracic biopsy: a valuable alternative to CT. *Radiology.* 1999;210:721–726.
21. Lichtenstein D, Peyrouset O. Is lung ultrasound superior to CT? The example of a CT occult necrotizing pneumonia. *Intensive Care Med.* 2006;32:334–335.
22. Weaver B, Lyon M, Blaivas M. Confirmation of endotracheal tube placement after intubation using the ultrasound sliding lung sign. *Acad Emerg Med.* 2006;13:239–244.
23. Fagenholz PJ, Gutman JA, Murray AF, Noble E, Thomas SH, Harris NS. Chest ultrasonography for the diagnosis and monitoring of high-altitude pulmonary edema. *Chest.* 2007;131:1013–1018.
24. Soldati G, Testa AR, Silva F, Carbone L, Grazia P, Nicolò GS. Ultrasonography in Lung Contusion. *Chest.* 2006;130:533–538.

CHAPTER 23

Ultrasound Evaluation of the Abdomen

Alan Cook and Heidi L. Frankel

INTRODUCTION

The intensivist must often consider the abdomen of the critically ill patient as a source for concern. Whether in the medical, surgical, cardiac, neurological, or transplant intensive care unit (ICU) patient, the source of bleeding, fever, pain, jaundice, or elevated white blood cell (WBC) count is often found in the peritoneal cavity or retroperitoneum. Focused ultrasonographic assessment can often help to identify the problem and assist with therapy.

The American Institute of Ultrasound in Medicine (AIUM) delineates eleven indications for an abdominal ultrasound examination (Table 23.1).[1] All but indications number "6" and "7" may be applicable to the ICU patient. Acute appendicitis, cholecystitis, mesenteric ischemia or an abscess may all cause abdominal pain and are potentially detectable by ultrasound. Jaundice may be due to acalculous cholecystitis whose sonographic features are discussed below. A mass caused by a hernia with bowel intrusion into a sac may be appreciated by ultrasound. Further, the cause of an elevated white blood cell count or the presence of fascial dehiscence may also be readily apparent by ultrasound. Trauma patients with solid visceral organ damage (liver, spleen, or kidney) can be followed for the continued wisdom (or lack thereof) of nonoperative management. Renal and hepatic transplant patients can undergo ultrasound screening for evaluation of the perfusion of their transplanted organs, as can those with new onset failure of their native ones. Finally, the presence and nature of peritoneal fluid can be assessed by ultrasound to guide drainage.

Of course, the concept of a focused ultrasound exam performed by nonradiologists to address a specific question originated with a limited abdominal examination performed by trauma surgeons, the so-called focused assessment with sonography for trauma (FAST) protocol.[2–4] Although the acronym is now known as the "focused *assessment* with sonography for trauma," in recognition of the components of the exam that assess the thorax, the "A" in FAST originally stood for "abdomen."[5] We believe that all ICU patients undergoing abdominal ultrasonographic examination should be studied with a variant of the FAST, that is, a search for fluid. The amount, location, and loculation of the fluid should be assayed and compared to prior studies, if any. Additional focused components of the exam should be added as needed (Table 23.2). Indeed, the initial description of the ultrasound evaluation of trauma patients relayed detailed examinations of intraperitoneal and retroperitoneal structures, not merely a search for free fluid.[6–8]

Schacherer and colleagues reported on their experience of abdominal ultrasonography of 400 ICU patients over three years.[9] They described a median examination time of 18 minutes and an accuracy of 83.7%; however, they noted that the test was of limited quality or unhelpful in 50% of cases, suggesting that ultrasound is best used as a screening tool in patients who cannot undergo more definitive diagnostic procedures. For detection of fluid and biliary tract disease, however, ultrasonography is the diagnostic study of choice.

EQUIPMENT AND TRANSDUCER SELECTION

Base Unit. The ideal ultrasound unit for use in the ICU should be portable, easy to use, highly reliable, relatively indestructible, and inexpensive. Ultrasmall units, although intuitively attractive, particularly in locations with limited space, may be taken from the ICU and not be readily available when needed. We find that even larger ultrasound units, particularly if easy to use and with good resolution, often get removed from our ICU. In our ICU, we resolved this problem by assigning the less expensive equipment to simpler tasks (i.e., central venous catheter insertion) and leaving the more sophisticated machines for torso imaging. In the future, we look forward to ultrasound technology that can be plugged into the overhead column in the ICU with disposable, inexpensive, single-patient use transducers. Until then, the choice of equipment remains a highly personal decision.

Most focused ICU abdominal ultrasound examinations do not rely on the use of Doppler imaging. More

TABLE 23.1. AIUM indications for abdominal ultrasonography

1. Abdominal, flank, and/or back pain
2. Signs/symptoms referred from abdominal organs, such as jaundice
3. Palpable abnormalities such as a mass
4. Abnormal lab value suggestive of abdominal pathology
5. Follow-up of known or suspected abdominal pathology
6. Search for metastatic disease or occult primary neoplasm
7. Evaluation of suspected congential abnormalities
8. Abdominal trauma
9. Pre- and posttransplantation evaluation
10. Planning and guidance for an invasive procedure
11. Search for the presence of free or loculated peritoneal and/or retroperitoneal fluid

AIUM indicates American Institute of Ultrasound in Medicine. (From Ref. 1)

advanced renal ultrasound may require this modality to assist in diagnosis. However, the addition of reliable Doppler capability to the ultrasound unit may complicate the issues of size, expense, and training.

Transducer Selection. The examinations described below all require imaging with good penetration and less resolution; hence a low-frequency transducer is appropriate (e.g., 2–5 MHz). Furthermore, a curvilinear transducer with a small footprint (i.e., microconvex probe) is more appropriate in ICU patients to navigate intercostal spaces, the subcostal region, and painful areas. By using another base unit to perform central venous catheter insertions, we reduce the difficulties associated with multiple transducers and base unit disinfection.

Data Capture. Portable printers can be added to ICU ultrasound units to capture images for documentation purposes. However, digital capture through video cards that allow for archiving of motion pictures tend to be more useful.

Ultrasound Coupling Agent. Proprietary ultrasound gel need not be used to acquire quality ultrasound images. Packets of lubricant found at the bedside of an ICU patient may be more convenient. Liechtenstein and colleagues describe using only water as a coupling agent, with adequate image acquisition and no apparent harm to their ultrasound units.[10]

Cleaning Transabdominal Transducers. The AIUM recommends that after each use probes be cleaned with soap and water or quaternary ammonium sprays or wipes as directed by the manufacturer in the

TABLE 23.2. Components of a focused abdominal ultrasound

Goal	*Task*	*Views*
Identify fluid	Amount, Location, Loculation	RUQ, LUQ, Bladder, Gutters
Characterize mass	Locate fascia	Local
Elevated WBC, pain	View gallbladder, appendix Bowels, aorta	Complete
Jaundice	Presence of stones, fluid	GB
	Wall thickness	Long and transverse
Decreased urine output	Is bladder full?	Long and transverse
	Is there hydronephrosis?	
	Are kidneys perfused?	
Transplant organ evaluation	Arterial/venous flow?	Over organ
	Organ size and tenderness	

GB indicates gallbladder; LUQ, left upper quadrant; RUQ, right upper quadrant; WBC, white blood cell (count).

operating manual. Heavy contamination with blood or enteric contents may warrant additional cleaning.[11]

IMAGE ORIENTATION AND ANATOMIC CORRELATION

The majority of scans obtained in a focused ICU abdominal ultrasonographic assessment are performed in the longitudinal or transverse plane. Exceptions are noted in the original FAST, where the right and left upper quadrant views were more sagittal than longitudinal (i.e., lined up in a plane with the sagittal sinus of the brain) because the views started very posterior to avoid intestinal and stomach air. The FAST bladder view starts transverse but moves coronal (and matches the images seen in computerized tomography [CT] scanning). In the longitudinal orientation of general abdominal ultrasound imaging (in contrast to the convention used in cardiac imaging), the patient's head is at the left of the screen and toes are oriented toward the right. The transverse images of general abdominal ultrasonography match the convention used with CT scanning (i.e., the left side of the screen corresponds to the patient's right side and the right side of the screen to the patient's left side). Thus, the indicator on the transducer is oriented to the patient's right (and the screen's left) in a conventional abdominal imaging setup on the software package (Figure 23.1). Of course, with additional experience, the transition between purely longitudinal or purely transverse images is blurred and the sonographer answers a focused clinical question "on the move."

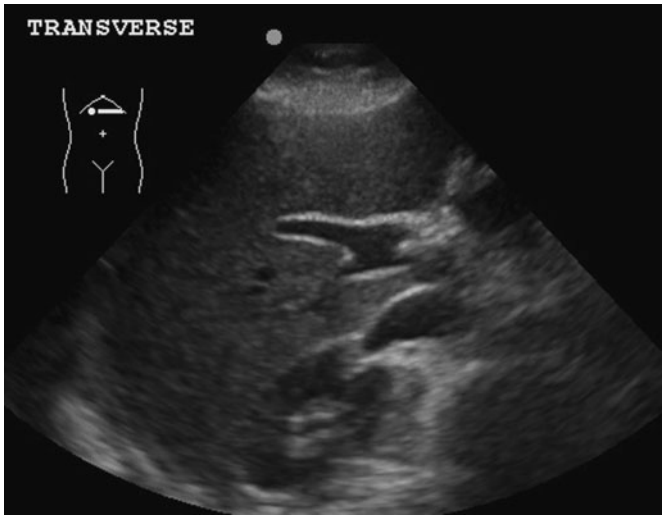


Figure 23.1. In an abdominal transverse ultrasound view, the transducer indicator is located on the patient's right and on the left of the screen.

As in all ultrasound imaging, structures seen in general abdominal ultrasonography are either anechoic (or hypoechoic), echoic, or hyperechoic. Jet black or anechoic structures may be either artifactual (e.g., a rib shadow) or free fluid (meaning that it contains no clot, sludge, or loculations as it might in an abscess, infected biliary fluid, or resolving hemoperitoneum including organizing hematomas after injury). Hyperechoic structures are artifactual (caused by gas), real (such as bone, other calcifications or omentum), or pathological (e.g., gas in tissues, chronic renal failure) conditions. Finally, echoic structures include normal parenchyma or complex fluid, as described above.

There are multiple limitations to performing a complete abdominal ultrasound in the ICU patient, even one designed to answer a specific question. Rib shadows and bowel gas may obscure the ability to obtain quality images. Obese patients and those who have received large volume fluid resuscitation often provide similar challenges, as do injured patients with extensive dressings, particularly those covering an "open abdomen." Of course, deeper structure visualization (e.g., aorta and pancreas) is the most difficult task here, whereas right and left upper quadrant views may be adequate. For most patients, it is possible to complete a focused ICU abdominal ultrasound examination in the supine position (unlike a focused cardiac echocardiographic examination, where we find it more helpful to have patients placed in the left lateral decubitus position).

FAST AND OVERALL ABDOMINAL EVALUATION

The classic FAST protocol consists of a subxiphoid or cardiac view in addition to three abdominal views (right upper quadrant or Morrison's pouch, left upper quadrant or splenorenal, and suprapubic or retrovesical) (Figure 23.2). Traditionally, the cardiac view is obtained first to allow the operator to establish the overall gain based on the fluid inside of the cardiac chambers. Further discussion of the performance of this and other cardiac and thoracic views can be found in Chapters 7 and 22. Attention here will be placed on the purpose of the FAST in the abdomen for the identification of free fluid. For the injured patient in the emergency department, the assumption is that free fluid in an unstable patient equates to blood. This is not necessarily the case, of course, and it can be quite embarrassing to perform a laparotomy on a patient with ascites due to liver failure and a baseline low blood pressure. It is equally disconcerting to triage the hypotensive patient

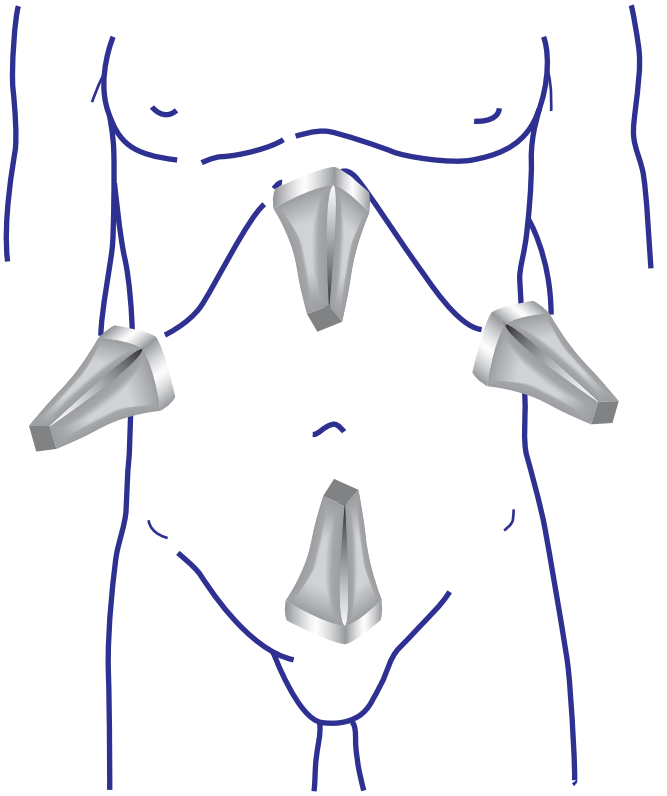


Figure 23.2. Views of the focused assessment with sonography for trauma (FAST).

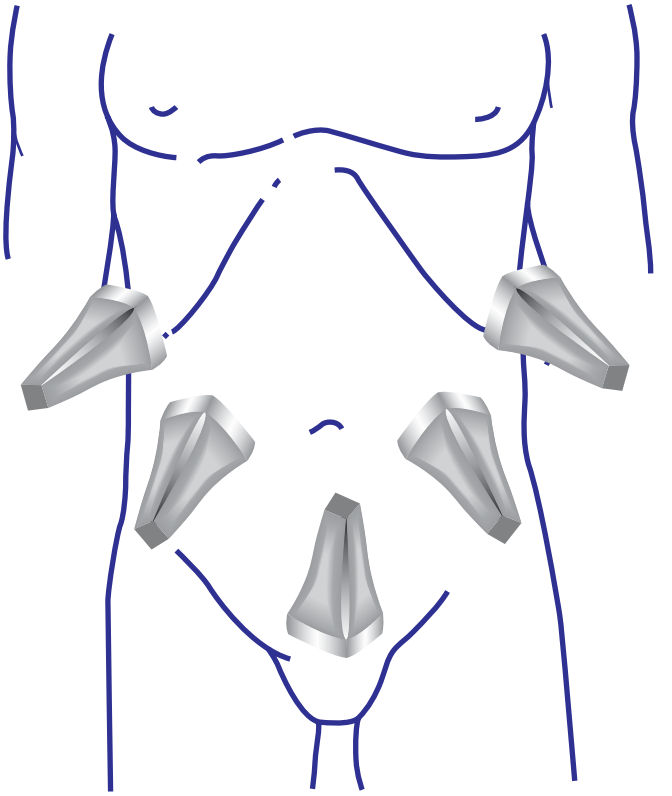


Figure 23.3. Views to identify fluid in the peritoneal space.

with a pelvic fracture and positive FAST to laparotomy before pelvic arteriography to control bleeding, when the cause of the positive FAST is a ruptured bladder and the cause of the hypotension is pelvic fracture hemorrhage. Cases such as these have resurrected the use of diagnostic peritoneal aspiration or lavage in the emergency department. Generally, hemodynamic instability with a positive FAST mandates expeditious laparotomy that will invariably be therapeutic.

However, the inability of FAST to distinguish fresh blood from ascites makes the use of focused abdominal ultrasound not very helpful in the ICU patient. In addition to the three abdominal FAST views discussed above, the gutters should also be assessed for fluid (Figure 23.3).

The right upper quadrant, hepatorenal or Morrison's pouch, view is most helpful to detect the presence of free fluid in a supine patient (most sensitive). The retropubic view is the most sensitive view to detect fluid in an upright patient. The right upper quadrant view is a sagittal one at intercostal space 10/11 at the posterior axillary line that generates the image seen in Figure 23.4. Fluid will first collect in the space between the liver and the right kidney, first anteriorly and then posteriorly, usually occurring first in intercostal space 7/9 at the midclavicular line. If there is an extremely large amount of fluid or blood in the abdomen, the liver will appear to be floating (Figure 23.5).

The left upper quadrant view requires the operator to move the transducer even more posteriorly and cephalad than the analogous view on the right to avoid gastric air. Fluid may collect either superior or inferior

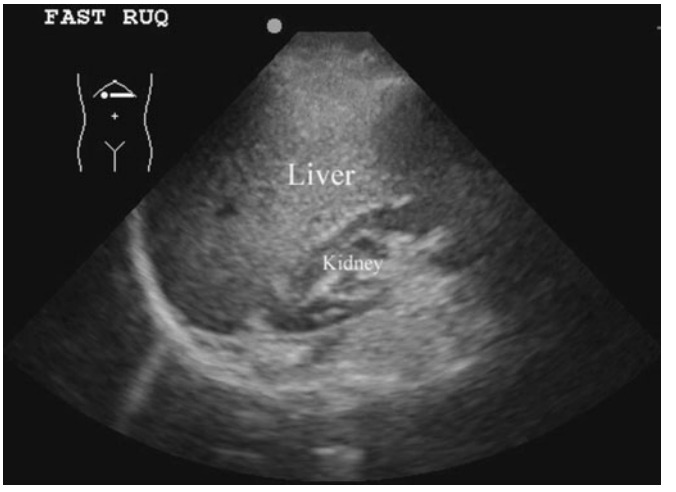


Figure 23.4. FAST right upper quadrant view.

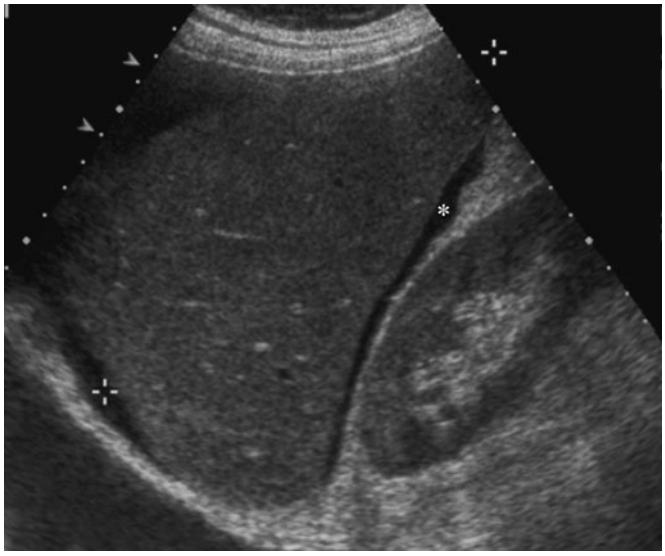


Figure 23.5. FAST right upper quadrant view showing moderate fluid collection in Morrison's pouch. *Fluid. (Image courtesy of Jeffrey Pruitt, MD, Associate Professor of Radiology, UT Southwestern.)

to the spleen (Figure 23.6). Finally, the suprapubic or retrovesical view is a transverse view of the full bladder that looks for fluid in the pouch of Douglas (Figure 23.7). Some also obtain a longitudinal view to confirm the same findings. Bowels seem to be floating in very large amounts of fluid (Figure 23.8).

The flank views are most helpful in the ICU patient because they may assist in therapeutic maneuvers. A paracentesis should be performed where the maximum amount of fluid is located, with the safest path by the needle to the target (Chapter 28). The right upper quadrant view is rarely helpful is reaching this decision.

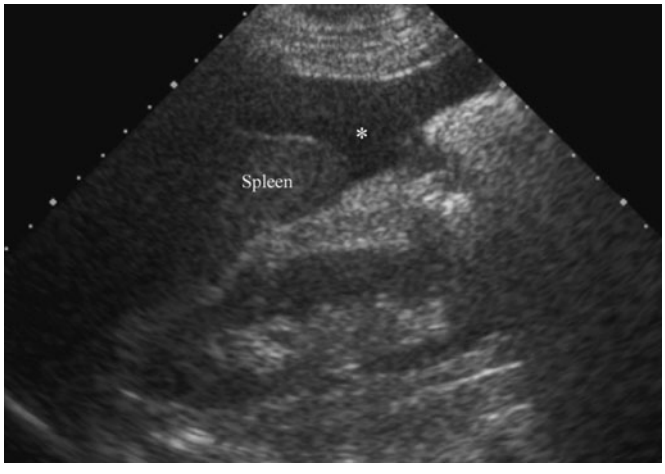


Figure 23.6. FAST left upper quadrant view showing free fluid. (Image courtesy of Jeffrey Pruitt, MD, Associate Professor of Radiology, UT Southwestern.)

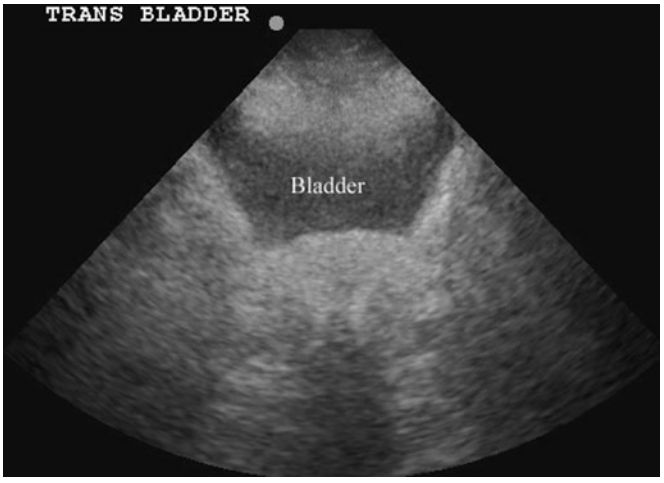


Figure 23.7. FAST suprapubic transverse view.

Ultrasonography practiced in this fashion is highly sensitive in identifying free fluid; as little as 100 cc of fluid (and likely less with machines of higher quality) may be detected.[12,13] In fact, the accuracy of FAST in identifying free fluid that is blood approaches 80%.[14–19] Of course, there are several potential abdominal catastrophes present in ICU patients that may not be associated with large amounts of free fluid. Hollow visceral injury, even with perforation, may be missed, as may a pancreatic injury, even one with transection, if one's abdominal evaluation is limited to the FAST protocol.

The amount of fluid present in the peritoneal cavity has been described by several scoring systems.[20,21] These are valuable in the emergency department to judge the eventual need for therapeutic laparotomy

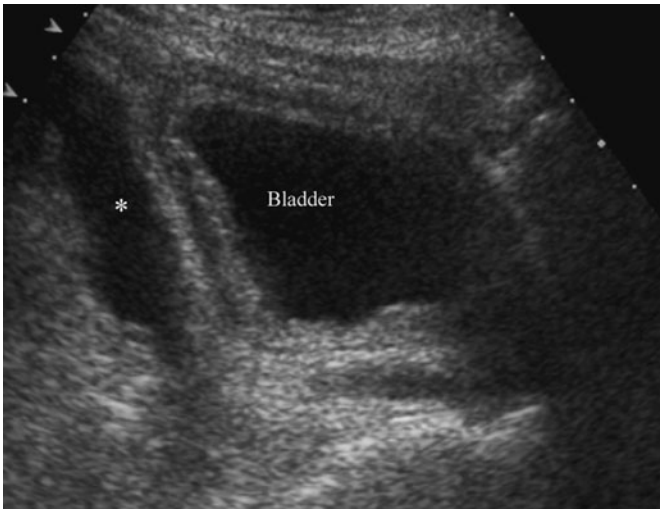


Figure 23.8. Positive FAST suprapubic view showing fluid in the pouch of Douglas (asterisk). (Image courtesy Jeffrey Pruitt, MD, Associate Professor of Radiology, UT Southwestern.)

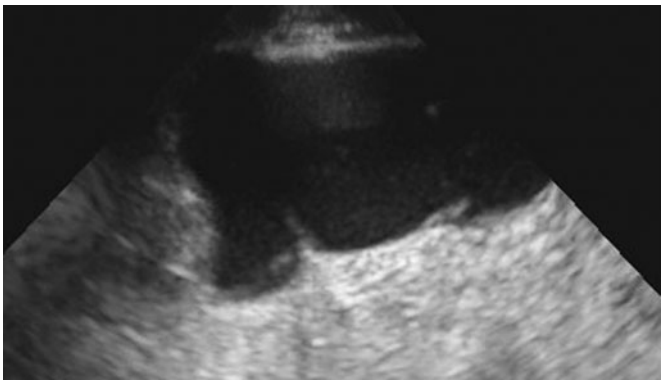


Figure 23.9. Peritoneal fluid with loculations.

even in the face of hemodynamic normality. These scoring systems are less useful in the ICU setting.

The nature of the peritoneal fluid collection should also be assayed with a focused ultrasound. Anechoic free fluid may be fresh blood or ascites related to primary liver failure, portal hypertension, right-sided heart failure, or aggressive volume resuscitation with a capillary leak. The presence of any echogenicity suggests an exudate. This may be due to hemoperitoneum with organizing clots or peritonitis with an infectious etiology. The latter is often associated with many internal septations or loculations that may not be appreciated on CT imaging (Figure 23.9). Nonetheless, even with occasional internal septations, sparsely loculated peritoneal fluid collections can be successfully sampled under ultrasound guidance to further characterize their nature.

Follow-up of the hemodynamically stable, injured patient with solid visceral injury and concomitant orthopedic injuries is also assisted by serial abdominal ultrasonographic assessments. Is the falling hematocrit due to the lacerated liver or the bilateral femur fractures? A rapid repeat FAST may answer this question more quickly and inexpensively than a CT scan without the need for patient transport, which is often difficult in the acutely ill or traumatized patient.

Peritonitis with a perforated hollow viscus should be suspected with the presence of a new peritoneal collection with loculations and echogenicity in a patient who has matched fluid balance. Additionally, evaluation of the adjacent intestines, although a more advanced skill as described below, can further suggest an infectious etiology to the peritoneal fluid collection.

Pneumoperitoneum can also be identified by focused abdominal sonography. Analagous to the lack of pleural sliding with a pneumothorax, there is a lack of peritoneal sliding with pneumoperitoneum. This can be appreciated in a longitudinal view of the abdomen

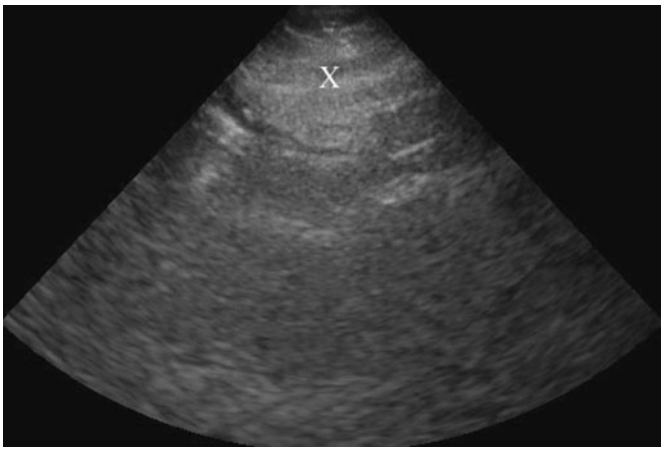


Figure 23.10. Pneumoperitoneum with absence of "gut sliding." (peritoneal edge at "X").

with a low frequency transducer due to the depth of the peritoneum versus the pleura (Figure 23.10). Massive pneumoperitoneum may even be associated with an inability to obtain the traditional FAST views.

Additionally, the presence of pneumoperitoneum can be assayed in M-mode ultrasound, much as one can do for pneumothorax when looking for the presence of the seashore sign. A seashore sign (beach under sea) is normal, as opposed to "all sea or barcode," which suggests a large pneumoperitoneum (Figure 23.11).

Finally, attention to the peritoneum can help to exclude the presence of a dehiscence (Figure 23.12) or identify a hernia sac with bowel incursion (Figure 23.13).

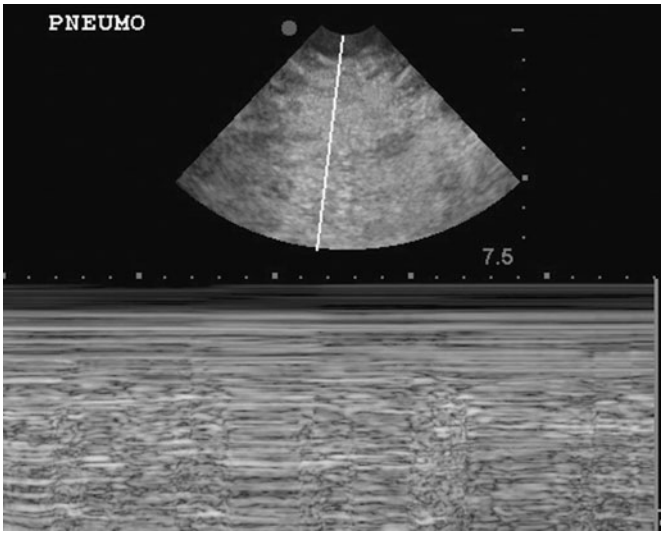


Figure 23.11. Pneumoperitoneum as witness in M-mode with absence of seashore sign (see text). "All sea or barcode sign" is seen intermittently and is partially obscured by patient movements.

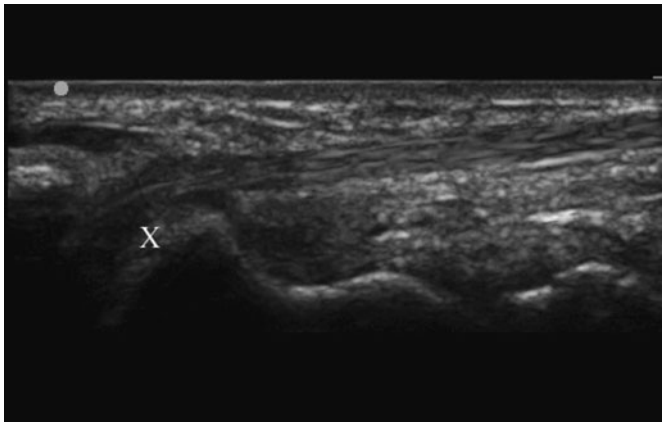


Figure 23.12. Ultrasound image depicting fascial dehiscence ("X" marks fascial edges).

LIVER ULTRASOUND

Defined questions regarding liver anatomy that are helpful to the intensivist are frankly few in number. Focused liver ultrasonography can evaluate for the presence of many lesions (particularly abscesses), intraductal dilatation, portal gas, perfusion, and relative function in a new transplant recipient. Comprehensive analysis of the segments of the liver is beyond the scope of most intensivists. Even appreciation of the features of intrinsic liver diseases such as cirrhosis (surface irregularity known as the "hump sign," smaller size, and a coarse echo texture, in addition to ascites) will rarely impact therapy in the ICU.

The liver is imaged in longitudinal and nearly transverse planes (more truly oblique, or following along the course of the subcostal space [Figure 23.14]). Several anatomic features are constant. Three hepatic veins join the inferior vena cava in the oblique scan and the portal vein branches into right and left divisions (Figure 23.15). In the longitudinal scan, the portal vein is seen posterior to the hepatic artery (medial) and common bile duct (lateral) (Figure 23.16). The common bile duct should be less than 4–7 millimeters (mm) in diameter. Abscesses are generally hypoechoic and may be drained under ultrasound guidance (Chapter 28). Intrahepatic ductal dilatation and the association with stones can be assessed. Portal gas may be seen as hyperechoic imaging in the parenchyma of the liver (Figure 23.17) and generally portends a poor prognosis. Finally, arterial and venous flow into and from a transplanted liver may be evaluated with Doppler technology, although not a routine feature of the intensivist's

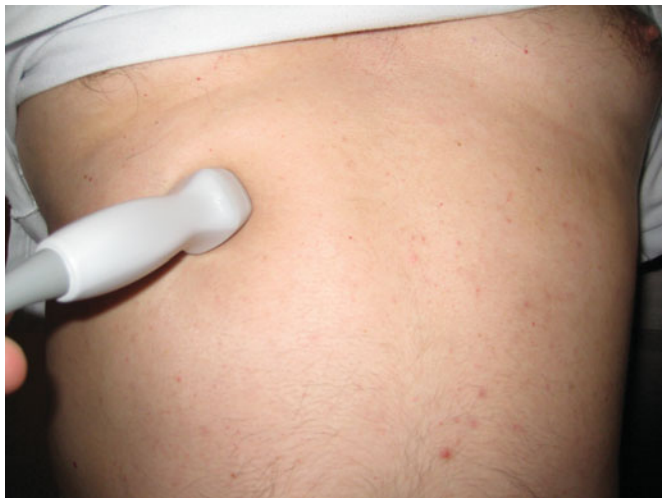


Figure 23.14. Subcostal placement of the ultrasound probe rendering an oblique view of the liver.

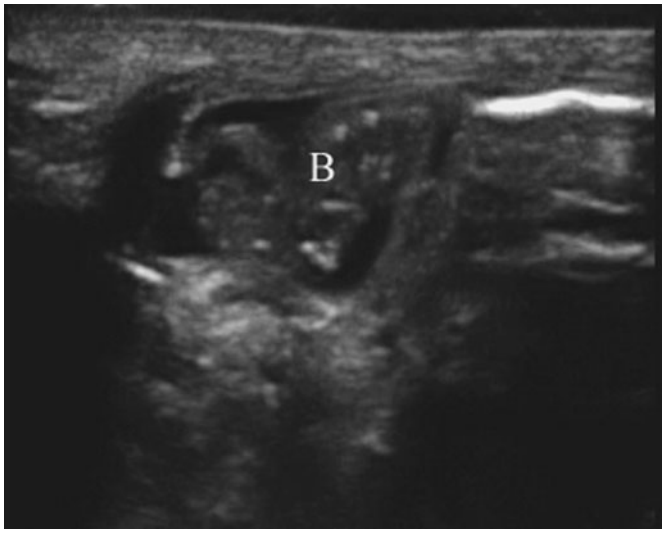


Figure 23.13. Ultrasound image showing hernia with bowel (B) present in the hernia sac.

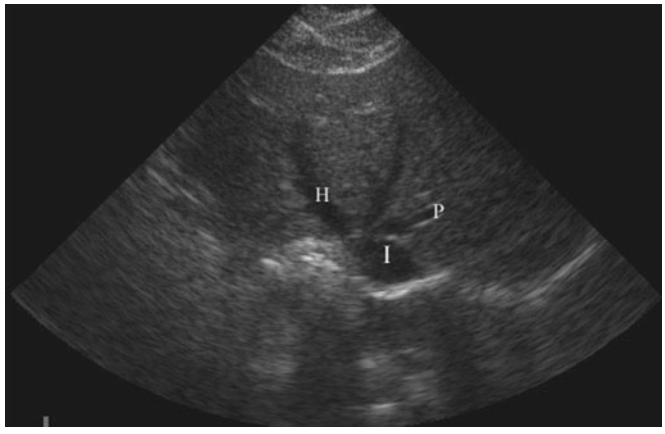


Figure 23.15. Hepatic veins (H) (3) entering the inferior vena cava (I) and portal vein (P) dividing into left and right sides.

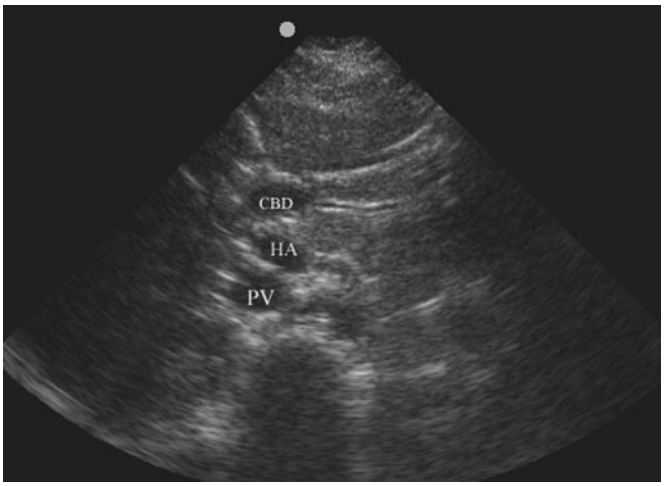


Figure 23.16. Longitudinal view of the portal triad showing relationship between the portal vein (PV), hepatic artery (HA) and common bile duct (CBD).

focused examination. Posttransplant biliary complications may also be assessed. In general, flow in the hepatic veins and inferior vena cava (IVC) is bidirectional and impacted by the cardiac cycle and respiratory phase. Portal flow is normally low-flow and continuous toward the liver, and hepatic artery flow is generally biphasic, not triphasic, as in larger vessels.

SPLENIC ULTRASOUND

As with the liver, focused questions regarding splenic anatomy are rarely helpful to the intensivist. This is particularly true given the normally homogenous nature of splenic parenchyma. Splenic abscesses may appear as hypoechoic structures; however, just as commonly, they may be isoechoic to the spleen and inconsistent to the intensivist. Of course, if visualized by ultrasound and unilocular, splenic abscesses may

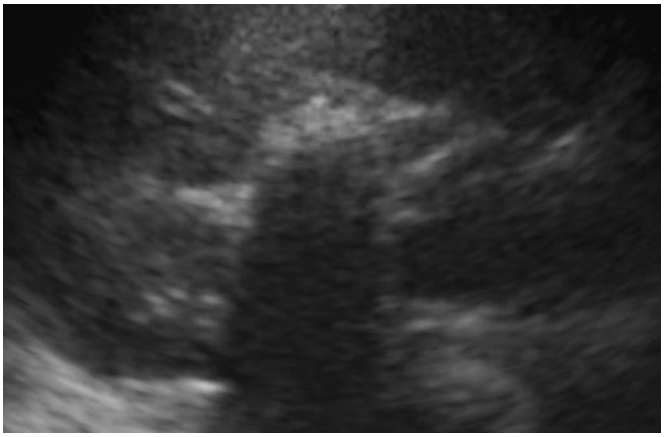


Figure 23.17. Gas in the portal system obscuring anatomy.

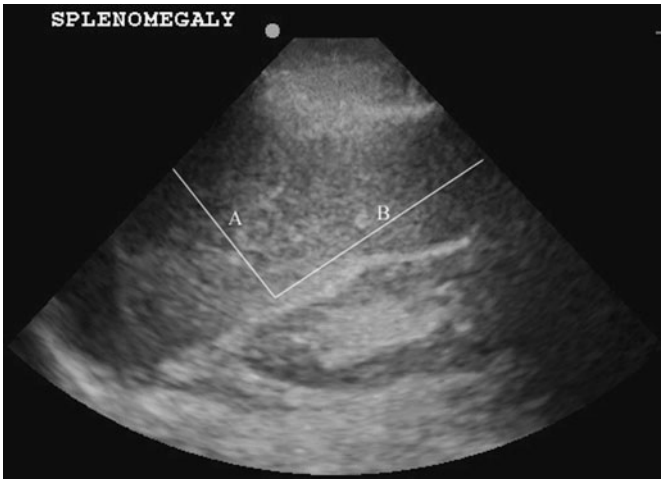


Figure 23.18. Splenomegaly where the product of lines A and B exceeds 20 cm^2.

be percutaneously drained under guidance. Computerized tomography scanning is clearly the better imaging modality in stable patients who can be transported from the ICU. More important is the need to identify the spleen prior to any interventional procedures (e.g., thoracentesis or paracentesis in the presence of splenomegaly). Wang and Chen describe splenomegaly as an ultrasound product of >20 cm^2 of the perpendicular hilar lines seen in Figure 23.18.[22]

BILIARY ULTRASOUND

Far more useful to the intensivist is ultrasound imaging of the biliary tree and, to a lesser extent, the pancreas. Typically, ICU patients are imaged in the supine position and enteral feeds are continued. A longitudinal-axis view of the gallbladder with the common bile duct present anterior to the portal vein is seen in Figure 23.19. This approach provides the intensivist with

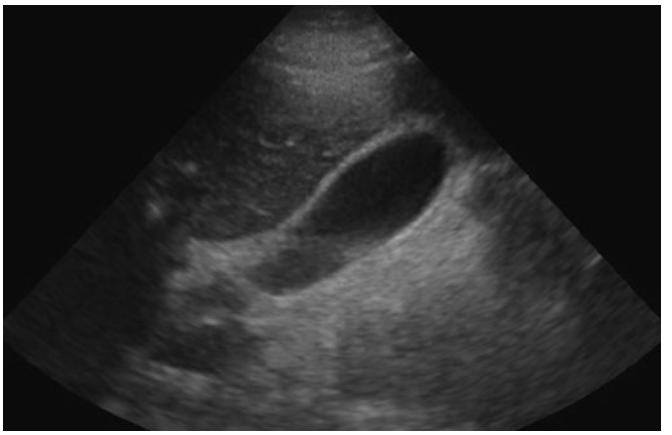


Figure 23.19. Gallbladder visualized in the longitudinal access.

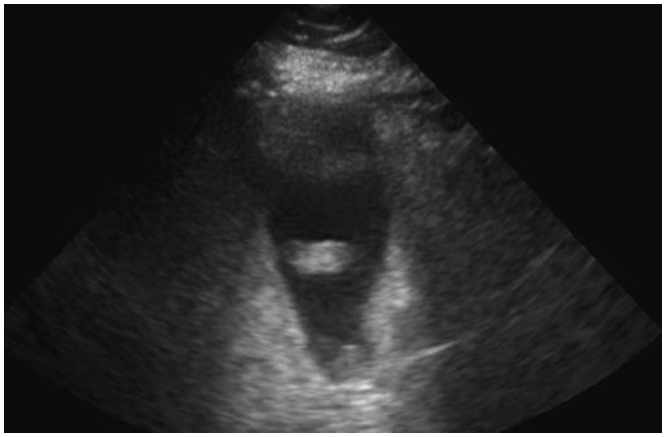


Figure 23.20. Ultrasound showing gallbladder wall thickening and a gallstone.

important answers to the following questions: Does the jaundiced patient have gallstones or sludge, or is acalculous cholecystitis present? Is there evidence of ductal dilatation and choledocolithiasis? Does the septic patient have features of acute cholecystitis (either calculous or not) such as a sonographic Murphy's sign, gallbladder wall thickening, and pericholecystic fluid? Is there a mechanical reason for jaundice?

The diagnosis of acute acalculous cholecystitis, although an infrequent cause of all cases of cholecystitis, is the most common cause of postoperative cholecystitis. It is likely due to an ischemic insult to the gallbladder, although this is far from certain. Furthermore, the sensitivity of ultrasound to establish the diagnosis has been questioned and it is often one of exclusion that is made after the gallbladder has been percutaneously drained. Of course, a negative aspiration in a patient receiving antibiotics also may not exclude the diagnosis. Nonetheless, there are features that are typically seen in patients with acalculous cholecystitis. The first is an enlarged gallbladder, exceeding 90 mm on longitudinal axis and 50 mm on transverse axis.[23] Second, the gallbladder wall is thicker than 3 mm (Figure 23.20), although Slaer suggests that this finding in isolation, particularly in a cardiac patient, is rarely significant.[24] Next, sludge is invariably present in the lumen of the gallbladder (Figure 23.21). Alternatively, a sonographic Murphy's sign (pain on subcostal imaging) is rarely present (likely more a testament to ICU pain control regimens than to the sensitivity of the test), although described as classic, as is pericholecystic fluid (Figure 23.22).

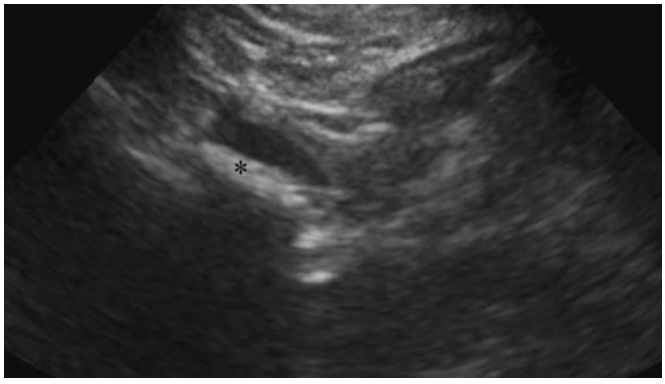


Figure 23.21. Ultrasound showing gallbladder sludge (*).

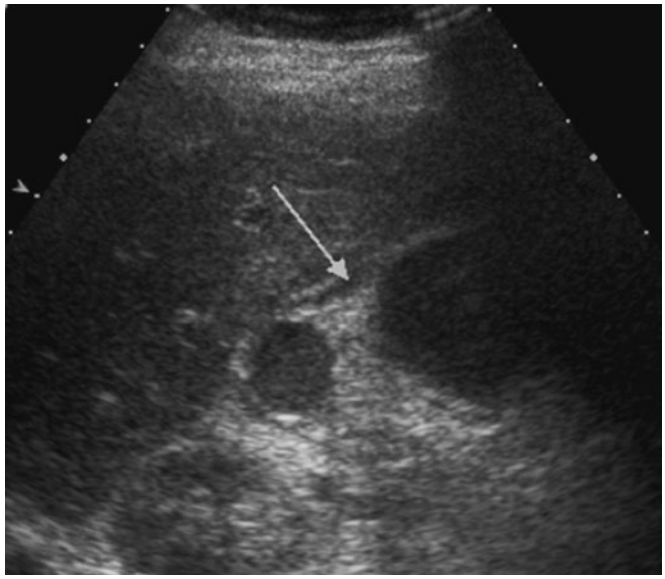


Figure 23.22. Ultrasound showing pericholecystic fluid (arrow). (Image courtesy of Jeffrey Pruitt, MD, Associate Professor of Radiology, UT Southwestern.)

Acute calculous cholecystitis is rarely an ICU disease, unless the patient is admitted for sepsis of unknown etiology. In addition to the features above,

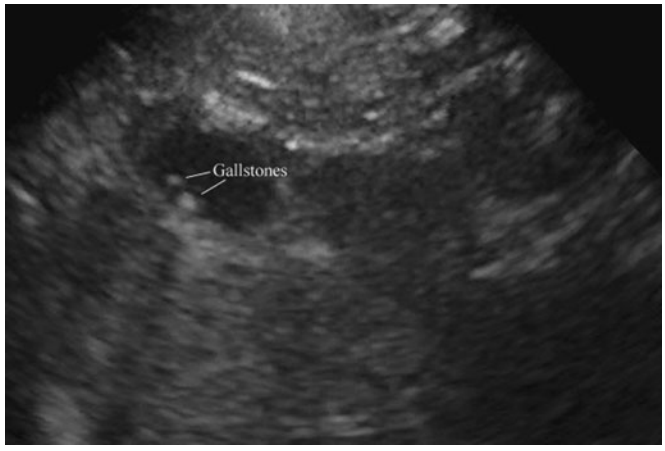


Figure 23.23. Gallstones present with acoustic shadowing.

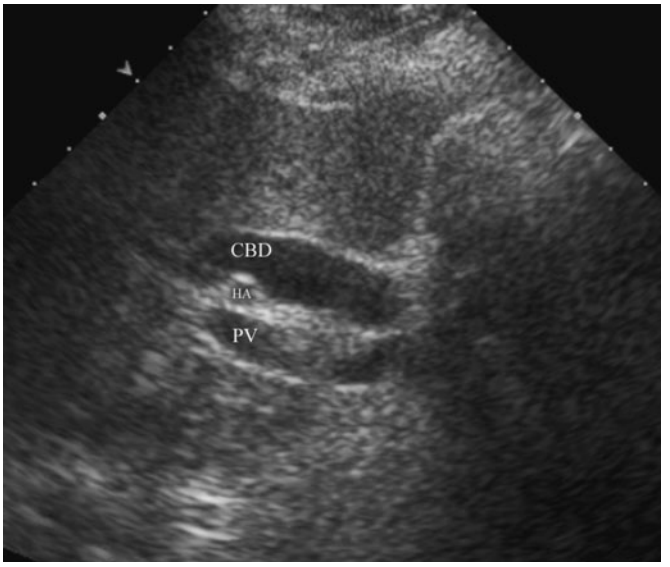


Figure 23.24. Dilation of the common bile duct (CBD) relative to the portal vein (PV). (Image courtesy of Jeffrey Pruitt, MD, Associate Professor of Radiology, UT Southwestern.)

gallstones with acoustic shadowing, as noted in Figure 23.23, are seen.

Intra- or extrahepatic ductal dilation beyond 7 mm is the sonographic hallmark or biliary obstruction. Bile ducts are considered dilated if the same size or larger than the adjacent portal vein (Figure 23.24). The cause for the biliary obstruction (i.e., stone, stricture, or other) should be sought and the level of the obstruction, although transabdominal ultrasound is notoriously inaccurate (as are most other modalities) in identifying common bile duct stones.

The pancreas may be difficult to identify, particularly in large patients with an ileus. Normal pancreatic

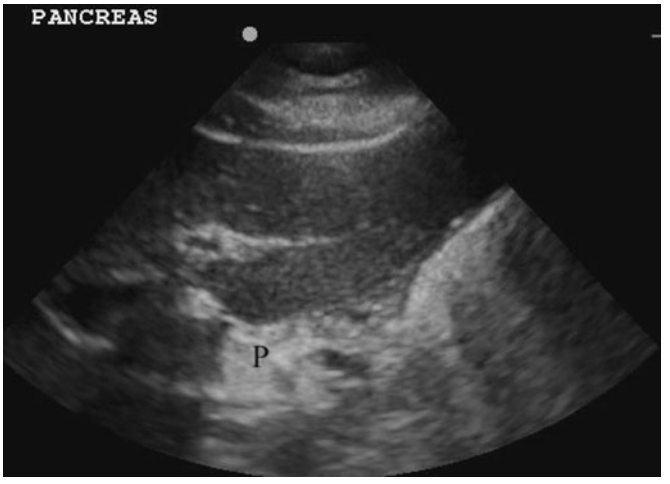


Figure 23.25. Normal pancreas (P).

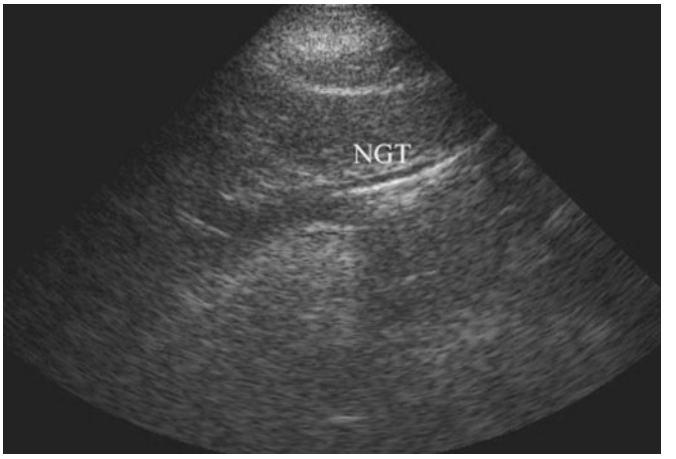


Figure 23.26. Nasogastric tube (NGT) in good position in the stomach collapsed around it.

parenchyma is homogenous with a well-visualized pancreatic duct (Figure 23.25). Pancreatitis may result in an enlarged gland with areas of hypoechoic characteristics, but this is not universal. By far, the better imaging modality in the stable patient is CT scanning. Intensivists should not aspire to become masters of imaging this organ.

ULTRASOUND OF THE GASTROINTESTINAL TRACT

Sonographic imaging of gastrointestinal structures also falls into the realm of an advanced technique that is rarely helpful for the intensivist in ruling out a particular condition, although a positive finding may assist in care of the patient. The location of a nasogastric tube (by the presence of an acoustic shadow and the decompression of the stomach) may be assessed as in Figure 23.26. Bowel peristalsis, wall thickness <4 mm, echoic bowel contents and cross-sectional area <12 mm

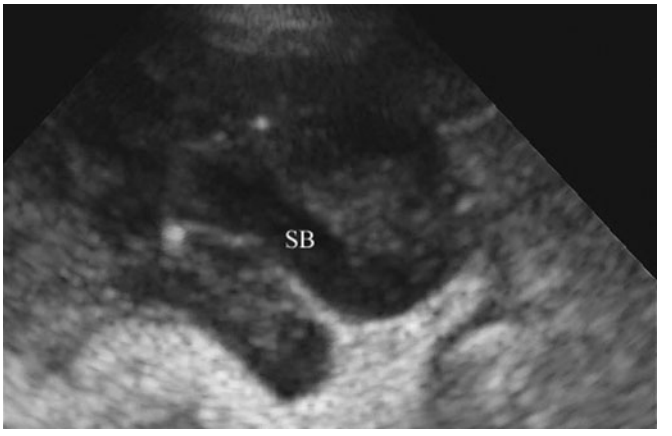


Figure 23.27. Dilated small bowel (SB).

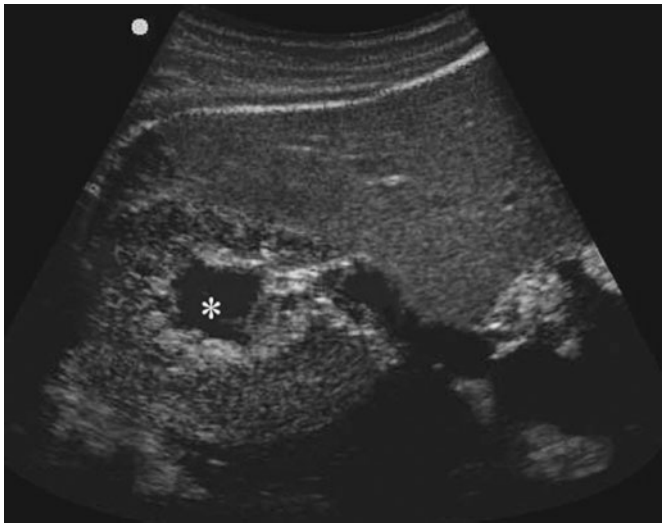


Figure 23.28. Acute obstruction of the renal pelvis (*). (Image courtesy of Kristen Hawkes, SonoSite, Inc.)

indicates a normal small intestine. Pathologic intestines are motionless, with thick walls and large diameter and anechoic bowel contents (Figure 23.27). Differential diagnosis includes ileus with a nonabdominal cause, mechanical bowel obstruction, mesenteric ischemia, or peritonitis. Doppler sonography can assist in determining whether or not perfusion is adequate.

URINARY TRACT ULTRASOUND (ALSO SEE CHAPTERS 24 AND 25)

The diagnosis of acute renal failure, whether due to prerenal, renal (acute tubular necrosis), or postrenal

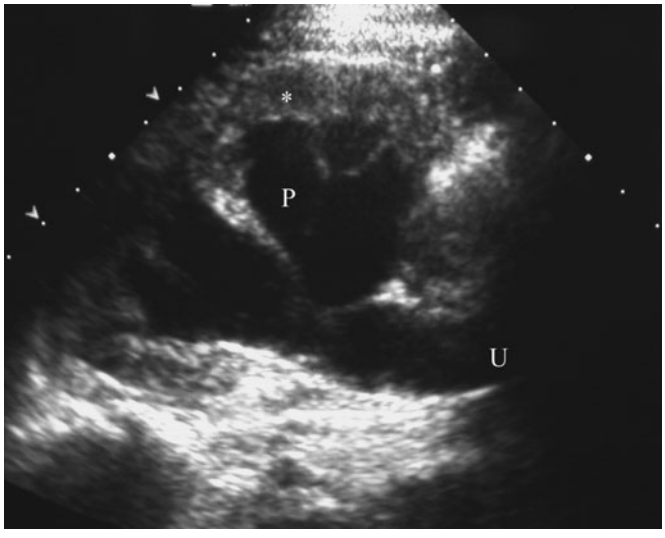


Figure 23.29. Chronic obstruction of the renal pelvis (P) with thin cortex (*) and ureter (U) noted. (Image courtesy of Jeffrey Pruitt, MD, Associate Professor of Radiology, UT Southwestern.)

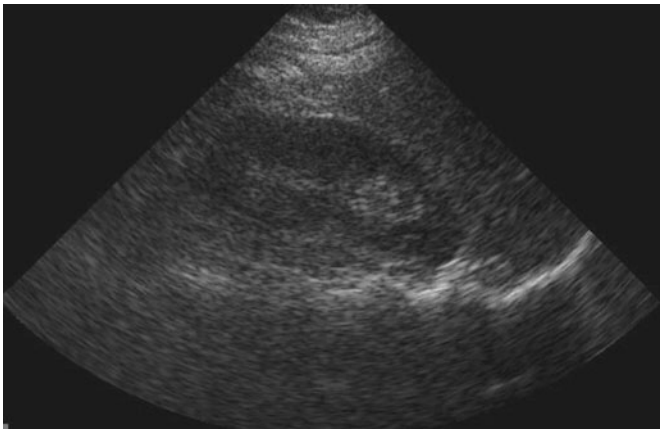


Figure 23.30. Normal renal architecture in a young patient showing relatively hypoechoic parenchyma and hyperechoic renal sinus.

causes, is generally made by the interpretation of laboratory values; however, focused ultrasound may rule out an obstruction. In addition, ultrasound evaluation of the renal parenchyma and perfusion status can provide additional clues as to the etiology of renal failure. Finally, assessment of the bladder is often helpful in the critically ill and injured patient.

Evaluation of the renal pelvis and bladder can be used to exclude postrenal causes of renal failure. A full bladder may indicate obstruction due to a malfunctioning catheter or bladder hematoma. Hydronephrosis with dilation of the calices and renal pelvis may be chronic or due to an acute infection. Acute obstruction of the renal pelvis, such as with infection or a stone, results in less dilation and a concave morphology (Figure 23.28, as opposed to major dilation and a more rounded morphology (Figure 23.29) seen in chronic

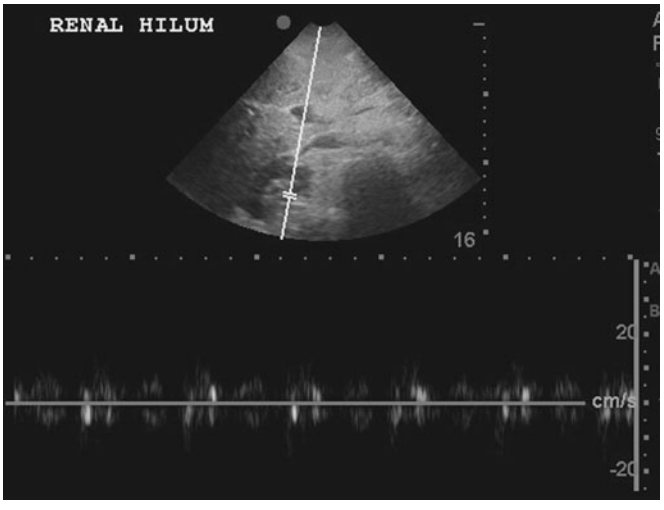


Figure 23.31. Spectral analysis by color Doppler evaluating (normal) renal arterial inflow.

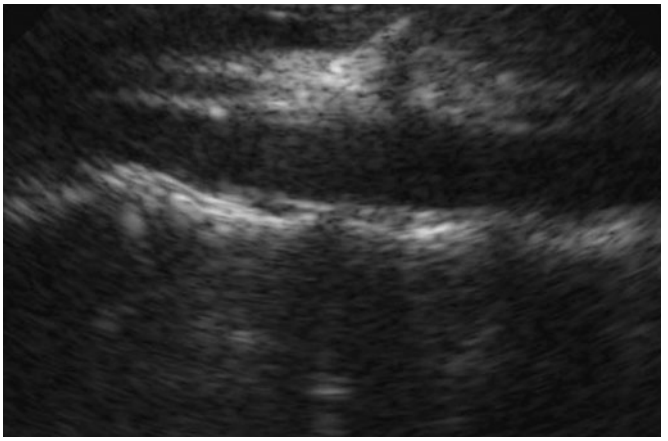


Figure 23.32. Longitudinal view of the aorta.

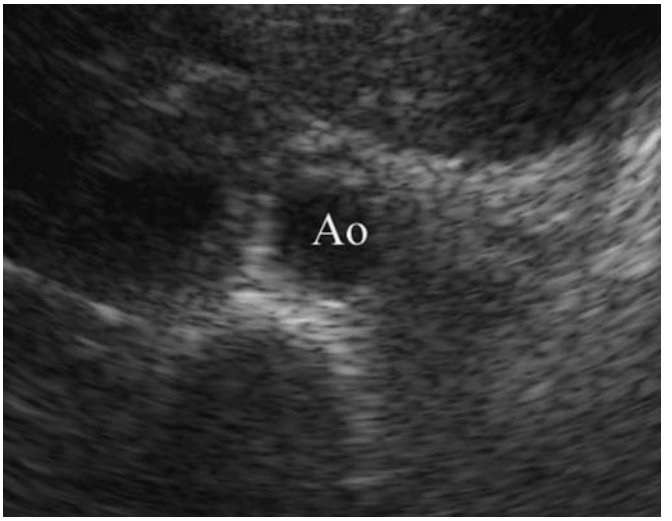


Figure 23.33. Transverse view of the aorta (Ao).

obstruction. Stones are more easily visualized if >5 mm in diameter.[25] Visualization of the ureter in a flank view by ultrasonography is nearly always pathologic and indicates obstruction.

Renal parenchymal evaluation considers the following variables: longitudinal diameter, thickness, margins, echogenicity, and perfusion status. Kidneys are normally 9–12 centimeters (cm) in longitudinal diameter. Those with chronic renal failure have smaller kidneys; those with acute infection may have much larger dimensions. Normal renal thickness varies between 1.5 cm and 1.8 cm, and thins with chronic renal failure. Renal lobulation and irregularity may indicate chronic inflammation or ischemia. Echogenicity increases with age and pathology. Further, the distinction between the more hypoechoic parenchyma and the more hyerpechoic renal sinus is more apparent in younger patients (Figure 23.30). Assessment of renal perfusion by Doppler scanning is an advanced technology that is likely beyond the scope of most intensivists. The right kidney is more easily visualized than the left, and a transverse scanning plane is utilized. A so-called resistive index can be calculated using spectral analysis in color Doppler imaging (systolic peak-diastolic peak/systolic peak × 100), with values over 0.70 pathological for many ICU disease states ranging from sepsis to prerenal causes (Figure 23.31). Venous thrombosis after transplantation can also be assessed, with a tender, enlarged graft, and arterial thrombosis, with resultant anuria. Acute rejection may also result in an elevated resistive index, although this is far from diagnostic.[26]

Bladder scanning using inexpensive technology permits ICU practitioners to remove bladder catheters, allowing patients to spontaneously void, without resulting in distension. Routine bladder scanning in those recovering from acute renal failure can be utilized

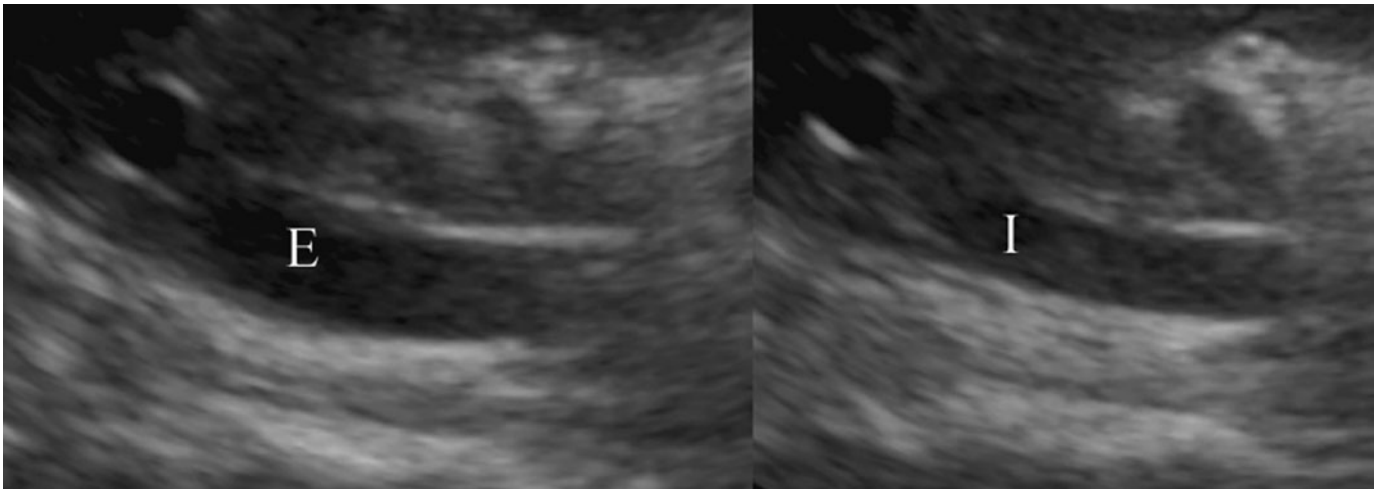


Figure 23.34. Inferior vena cava (IVC) Diameter in inspiration (I) and expiration (E).

instead of daily "in and out" catheterization for monitoring of recovery. Doppler ultrasound showing ureteral jets argues against obstruction.

ABDOMINAL VASCULATURE ULTRASOUND

Ultrasound assessment of the retroperitoneum can assess aortic size and contour, presence of flow in major visceral branches, and the size of the IVC as a measure of volume status (see Chapter 10). Longitudinal (Figure 23.32) and transverse views (Figure 23.33) are obtained; successful imaging can be impaired by the presence of bowel gas. In longitudinal imaging, the first abdominal branch of the aorta, the celiac artery, is visualized, dividing into the common hepatic and splenic arteries (the left gastric artery is not easily seen). The superior mesenteric artery is more easily visualized than in the inferior mesenteric artery. The left renal vein courses anteriorly to the aorta and posteriorly to the superior mesenteric artery, whereas the right renal artery passes posterior to the inferior vena cava. In the transverse view, the posterior hyperechoic nature of the spine is referred to as the horseshoe sign. The compressible and irregularly shaped inferior vena cava is seen to the left of the screen (to the patient's right of the aorta).

An enlarged aorta (measuring from outside wall to outside wall) >3 cm with luminal thrombus and irregularity suggests an aortic aneurysm. Occasionally, an intimal flap can be appreciated. Rarely is "leaking" from an abdominal aortic aneurysm appreciated by sonography. Rather, the presence of an aneurysm in a patient in shock establishes the diagnosis and warrants urgent surgical repair.

Mesenteric ischemia is likely only if two of the three mesenteric vessels (celiac, superior, and inferior mesenteric arteries) are occluded. Atherosclerotic vessels will have an increased flow velocity and turbulence. These are all difficult diagnoses to make because of the interference of adjacent bowel gas, making other diagnostic modalities far more attractive.

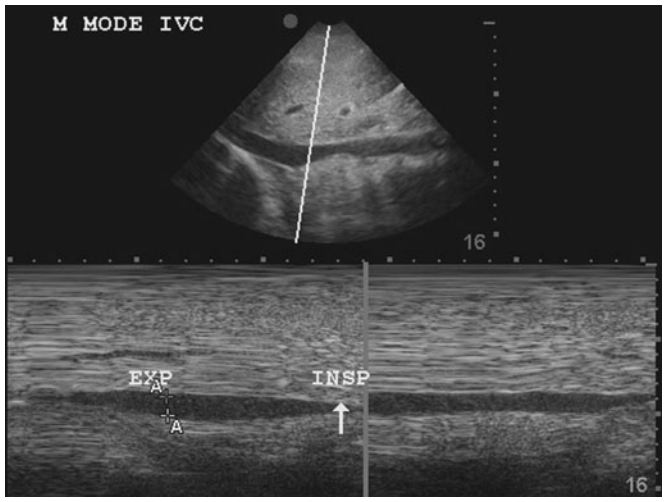


Figure 23.35. M-mode view of the inferior vena cava (IVC) in inspiration (Insp) and expiration (Exp).

Compressibility of the inferior vena cava with inspiration in long axis as it enters the right atrium provides a rapid assessment of a patient's volume in the ICU (Figure 23.34). In addition to assaying this on B-mode ultrasound, this can also be measured in M-mode (Figure 23.35). The presence of positive pressure ventilation can alter the reliability of these assessments, but we have not found this to be the case in surgical intensive care unit patients.

SUMMARY

The intensivist can readily perform a goal-directed abdominal ultrasound on critically ill and injured patients. The presence and nature of peritoneal fluid can be assayed as well as the etiology of pain, a palpable mass, and an elevated white cell count or jaundice. The presence of an ileus, volume overload, and subcutaneous emphysema may render the examination difficult; however, in most cases, the novice sonographer can accomplish an accurate screening test. As equipment continues to become more user-friendly, the indications for abdominal sonography in the intensive care unit will continue to expand.

References

1. Cohen HL, McGahan JP. Practice guidelines for the performance of an ultrasound examination of the abdomen and retroperitoneum. American Institute for Ultrasound in Medicine, 2007. Available at: http://www.aium.org. Accessed February 9, 2009.
2. Rozycki GS, Ochsner MG, Jaffin JH, et al. Prospective evaluation of surgeons' use of ultrasound in the evaluation of trauma patients. *J Trauma*. 1993;34:516–527.
3. Rozycki GS, Ochsner MG, Schmidt JA, et al. A prospective study of surgeon-performed ultrasound as the

primary adjuvant modality for injured patient assessment. *J Trauma*. 1995;39:492–500.

4. Tso P, Rodriguez A, Cooper C, et al. Sonography in blunt abdominal trauma: a preliminary progress report. *J Trauma*. 1992;33:39–44.
5. Scalea TM, Rodriguez A, Chiu WC, et al. Focused assessment with sonography for trauma (FAST): results from an international consensus conference. *J Trauma.* 1999;46:466–472.
6. Amman A, Brewer WH, Maull KI, et al. Traumatic rupture of the diaphragm: real-time sonographic diagnosis. *Am J Radiol*. 1988;140:915–918.
7. Kristensen JK, Buermann B, Kuehl E. Ultrasonic scanning in the diagnosis of splenic hematomas. *Acta Chem Scand*. 1971;137:653–657.
8. Asher WM, Parvin S, Virgilia RW, et al. Echographic evaluation of splenic injury after blunt trauma. *Radiology*. 1976;118:411–417.
9. Schacherer D, Klebl F, Goetz D, et al. Abdominal ultrasound in the intensive care unit: a 3-year survey on 400 patients. *Intensive Care Med*. 2007;33:841–844.
10. Liechtenstein DA. *General Ultrasound in the Critically Ill*. Berlin, Germany: Springer-Verlag; 2005.
11. American Institute of Ultrasound in Medicine. Official Statement. June 22, 2005. Available at: http://www.aium.org. Accessed February 9, 2009.
12. Goldberg BB, Goodman GA, Clearfield HR. Evaluation of ascites by ultrasound. *Radiology*. 1970;96:15–22.
13. Goldberg BB, Clearfield HR, Goodman GA. Ultrasonic detection of ascites. *Arch Intern Med.* 1973;131:217–221.
14. McKenney MG, Martin L, Lentz K, et al. 1000 consecutive ultrasounds for blunt abdominal trauma. *J Trauma*. 1996;40:607–611.
15. Dolich MO, McKenney MG, Varella JE, et al. 2576 ultrasounds for blunt abdominal trauma. *J Trauma*. 2001;50:108–112.
16. Ma OJ, Mateer JR, Ogata M, et al. Prospective analysis of a rapid trauma ultrasound examination performed by emergency physicians. *J Trauma*. 1995;38:879–885.
17. Bode PJ, Niezen RA, van Vugt AB, et al. Abdominal ultrasound as a reliable indicator for conclusive laparotomy in blunt abdominal trauma. *J Trauma*. 1993;34:27–31.
18. Glaser K, Tschmelitsch J, Klinger B, et al. Ultrasonography in the management of blunt abdominal and thoracic trauma. *Arch Surg*. 1994;129:743–748.
19. Golletti O, Shiselli G, Lippolis PV, et al. The role of ultrasonography in blunt abdominal trauma: results in 250 consecutive cases. *J Trauma*. 1994;36:178–182.
20. Huang MS, Liu M, Wu JK, et al. Ultrasonography for the evaluation of hemoperitoneum during resuscitation: a simple scoring system. *J Trauma*. 1994;36:173–177.
21. McKenney KL, McKenney MG, Cohn SM, et al. Hemoperitoneum score helps determine need for therapeutic laparotomy. *J Trauma*. 2001;50:650–656.
22. Wang HP, Chen SC. Upper abdominal ultrasound in the critically ill. *Crit Care Med*. 2007;35(suppl 5)S208–S215.
23. Mirvis SE, Vainright JR, Nelson AW, et al. The diagnosis of acute acalculous cholecystitis: a comparison of sonography, scintigraphy and CT. *Am J Roentgenol*. 1986;147:1171–1179.
24. Slaer WJ, Leopold GR, Scheible FW. Sonography of the thickened gallbladder wall: a non-specific finding. *Am J. Roentgenol*. 1981;136:337–339.
25. Barozzi L, Valentino M, Santoro A, et al. Renal ultrasonography in critically ill patients. *Critical Care Med*. 2007;35(suppl 5)S198–S205.
26. Jakobsen JA, Branrand K, Egge TS, et al. Doppler examination of the allografted kidney. *Acta Radiologica*. 2003;44:3–13.

CHAPTER 24

Ultrasound Evaluation of the Renal System and the Bladder

Yefim R. Sheynkin

INTRODUCTION

Ultrasound is a powerful and inexpensive tool particularly well suited for the diagnosis and monitoring of critically ill patients. While portable bedside sonography may not be the preferred tool for a detailed examination, the development of versatile portable ultrasound machines significantly improves its utility and clinical accuracy.[1]

Easy accessibility for major organs of the urinary system makes ultrasound a commonly performed test in critically ill patients. Sonography of the kidneys and bladder in critical care has multiple applications including evaluation of patients with reduced or absent urinary output, complicated urinary tract infections, and fever of unknown origin, renal trauma, and idiopathic hematuria. It is the most useful initial investigation in the early or late period after kidney transplantation. Sonographic study often provides the clinician with a diagnosis or guidance for rapid decision-making necessary for the treatment of critically ill patients. The most important goal of ultrasound evaluation of the urinary system is to identify or rule out a problem that requires prompt, goal-directed surgical or medical intervention to improve the patient's condition. While not intended as a comprehensive formal examination, ultrasound is a convenient bedside monitoring tool for use in the intensive care unit (ICU).

In addition, many incidental abnormalities may be found during sonographic evaluation of kidneys and bladder. Whereas they may not have an impact on the immediate treatment decision, physicians should be able to recognize them and provide appropriate care if necessary.

SONOGRAPHIC ANATOMY OF URINARY TRACT

The normal adult kidney is a bean-shaped structure surrounded by a well-defined, smooth echogenic capsule representing Gerota's fascia and perinephric fat. The kidneys have a convex lateral edge and concave medial edge called the hilum. The lower pole is located more laterally and anteriorly than the upper pole. The sonographically measured normal adult kidney is between 9 and 12 centimeters (cm) in length and about 4–5 cm wide.

The kidney parenchyma surrounds centrally located hyperechoic fatty renal sinus, which contains renal pelvis, calyces, major branches of renal artery and vein, and lymphatic vessels. Parenchyma corresponds to the area between renal sinus and outer renal surface and has two main components: the more echogenic peripherally located cortex and centrally located hypoechoic medulla, which contains renal pyramids (Figure 24.1). The normal renal parenchyma is 1.0–1.8 cm thick. The visible distinction between the cortex and medulla is a sign of a normal kidney. While easily recognized in children and younger patients, it may not always be detectable in the elderly.

Parenchymal homogeneity is determined in comparison with that of adjacent liver and spleen. Normally, the renal cortex is hypoechoic or isoechoic to the liver (right kidney) and hypoechoic to spleen (left kidney). The collecting system of the kidney is not usually visible with ultrasound because calyces and pelvis are collapsed within renal sinus. The normal ureters measure approximately 8 millimeters (mm) wide and are difficult to evaluate sonographically. However, proximal or distal ends of significantly dilated ureter (hydroureter) can be seen.

The shape and appearance of the normal bladder depends on the degree of distention. When empty, the bladder lies behind the symphysis pubis. On longitudinal transabdominal view, the full bladder has a teardrop-shaped anechoic appearance, with distinct wall, while on the transverse view it appears rectangular. The thickness of the bladder wall varies with the degree of bladder filling. When mildly distended or empty, the bladder wall is thick and irregular. With full

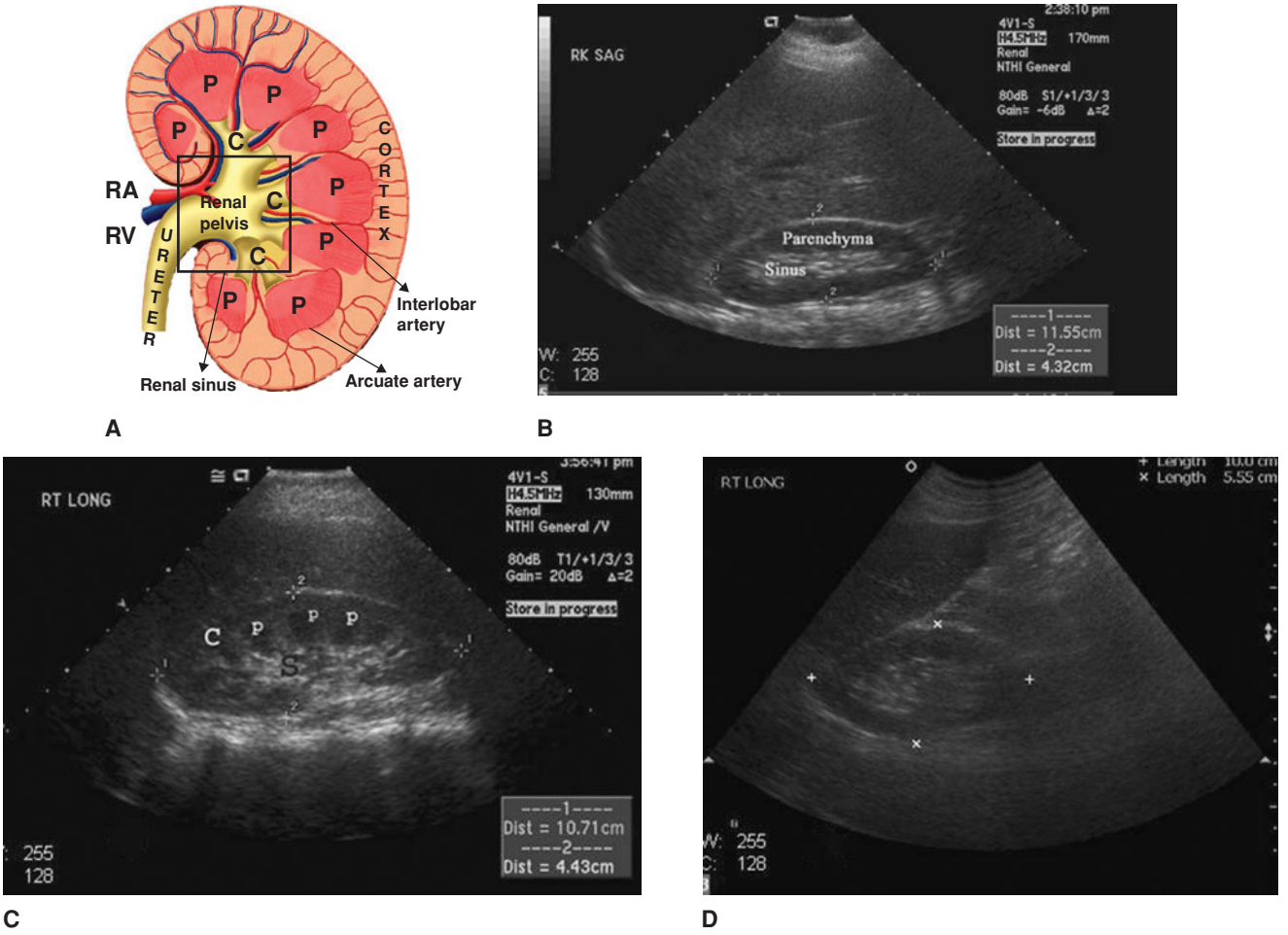


Figure 24.1. (A) Normal renal anatomy. C indicates calyx; P, pyramid; RA, main renal artery; RV, main renal vein. (B) Normal kidney. Longitudinal view of the kidney demonstrates peripheral hypoechoic universally thick parenchyma and central hyperechoic renal sinus. Note the echogenic white Gerota's fascia. Parenchyma is less echogenic then liver. (C) The cortical echogenecity is equal of that of the liver. Several slightly hypoechoic renal pyramids are seen. C indicates cortical echogenecity; L, liver. (D) Portable ultrasound of the normal right kidney. Note less contrast appearance but renal contour, parenchyma and renal sinus are clearly identified.

distension, the normal bladder wall is thin and smooth and does not exceed 4–5 mm in thickness (Figure 24.2).[2]

IMAGING TECHNIQUE

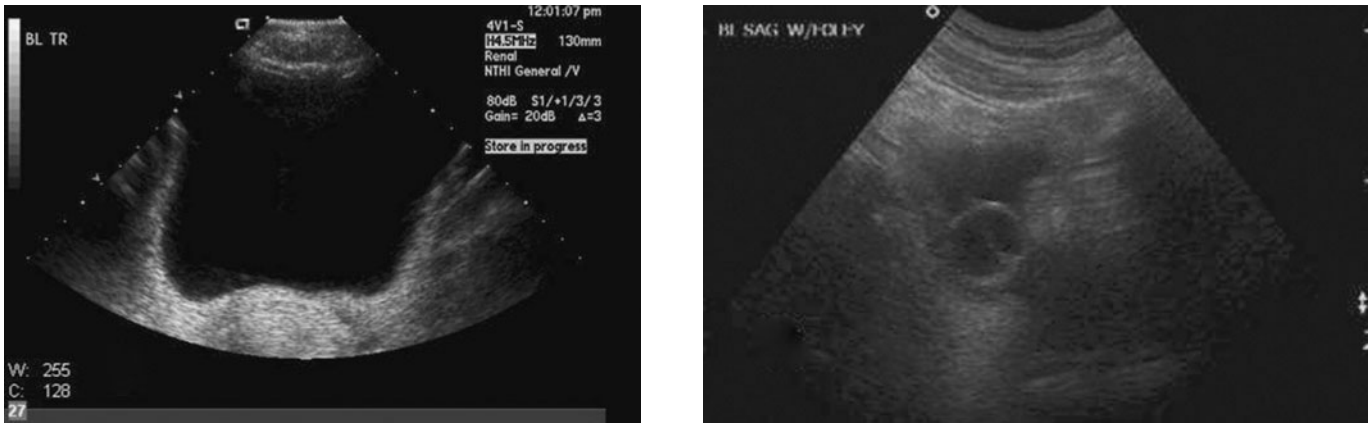
The spectrum of urologic ultrasound includes gray-scale and Doppler evaluation of the kidneys and the bladder. In accordance with American Institute of Ultrasound in Medicine (AIUM) practice guideline, the examination of kidneys should include longitudinal and transverse views and assessment of the cortex and renal pelvis. Renal echogenecity may be compared with echogenecity of the adjacent liver or spleen. Kidneys and perirenal regions should be assessed for abnormalities (Table 24.1).[3]

Sonographic evaluation of a critically ill patient is typically limited by a supine position, lack of patient cooperation, presence of monitoring devices, tissue changes (e.g., bowel gas, edema, ascitis), postsurgical incisions, and dressings.

Kidneys demonstrate a significant mobility with respiration (about 2–3 cm), which complicates the evaluation of patients on a ventilator.[4]

Commonly, a sector or curved-array transducer (3–5 MHz) is used, while higher-frequency probes (5–7 MHz) with higher space resolution may be necessary to evaluate children, thin patients, and transplanted kidneys. Imaging of the urinary tract must always include evaluation of both kidneys and the bladder.

The right kidney is best examined in the supine or left lateral decubitus position through the liver, which



A B

Figure 24.2. (A) Transabdominal ultrasound (transverse scan) of a normally distended bladder. (B) Foley catheter in the collapsed bladder (portable ultrasound).

serves as an acoustic window. The probe should be placed along the right lateral subcostal margin in the anterior axillary line, scanning through the liver to locate the right kidney. After visualization of the whole kidney, the optimal longitudinal view is obtained by slowly adjusting the probe's position up and down or side to side. The kidney is traditionally measured in the longest axis (length and width) because the longitudinal diameter has minor inter- and intraobserver variations. If needed, a transverse plane (short-axis view) can be obtained by rotating the probe 90° and evaluating upper, mid, and lower portions of the kidney separately.

The left kidney is typically less visible due to its location in a more superior position, the lack of the sonographic window generated by the liver and the overlying small bowel, and gastric gas. If possible, placing the patient in the right lateral decubitus position with the probe positioned in the posterior axillary line or left costovertebral angle may improve visualization. If

TABLE 24.1. Renal ultrasound in critically ill patients

Parameter	*Description*
Longitudinal diameter	Easy to obtain and reproduce with less intra and inter-observe variation. Average length is between 9 cm and 12 cm
Parenchymal thickness	Measurements between renal surface and hyperechoic sinus, normally >1 cm
Kidney margins	Sharp and regular. V-shaped indentation may represent persistent fetal lobulation. The outline depression with rounded angles indicates inflammatory or ischemic scars
Parenchymal echogenecity	Cortex hypo/isoechogenecity compared with that of liver or spleen is usually normal, while hyperechogenecity indicates a diffuse parenchymal pathology. Medulla is slightly less echogenic than cortex
Collecting system	Visible only when dilated (hydronephrosis), mostly secondary to mechanical obstruction
Calcifications	Small hyperechogenic lesions are nonspecific and may represent small stones, vascular or intraparenchymal calcifications. Larger stones are easily diagnosed by the characteristic posterior acoustic shadowing
Renal/extrarenal masses	Solid masses are usually neoplastic and require further evaluation with CT or MRI. Simple renal cyst is anechoic thin-walled space occupying lesion with good through-transmission and no internal echoes
Resistive index	Color Doppler study of renal perfusion. Normal RI <0.70. High resistance pattern indicates decrease perfusion of various causes

CT indicates computerized tomography; MRI, magnetic resonance imaging; RI, resistive index.

bowel gas obscures the kidney (especially left) and reflects the ultrasound waves, the transducer can be positioned in the mid or posterior axillary line.[5]

The bladder can be examined only when it is distended. Sonographic evaluation is usually performed from a transabdominal location, with the patient in a supine position. A probe is placed 1 cm above the symphysis and angled laterally, inferiorly, and superiorly. Most commonly, the transverse scan is obtained first. A normal bladder is located in the midline without deviation, and appears symmetric, smooth, and without irregularities of inner surface. On the longitudinal scan, the bladder is oriented toward the umbilicus and tapered anteriorly. Transverse and longitudinal scans provide a fairly accurate calculation of the urine volume within the bladder. If needed, postvoid residual urine volume can be automatically calculated.

Detailed bladder ultrasound may have a limited application in critically ill patients with draining indwelling Foley catheters. However, it can provide immediate bedside diagnosis of urinary retention in patients with decreased or absent urinary output.

COLOR DOPPLER ULTRASOUND

Complex Doppler studies have not been used routinely as a bedside test in critically ill patients. However, the technical improvement of portable ultrasound machines with color and power Doppler equipment enables the ability to combine gray-scale ultrasound with limited Doppler study in the critical care setting. Changes in the perfusion of renal parenchyma are commonly associated with different renal pathology. Color-flow and spectral Doppler studies are able to provide noninvasive, indirect global assessment of renal blood flow and identify the vessels at the level of the renal hilum and in the renal parenchyma. Because the spatial resolution of gray-scale sonography is much lower than frequency resolution, the Doppler study is able to detect the arteries on the basis of the flow and not the anatomic size.[2] The Doppler spectral tracing reflects a low vascular resistance and classically has a ski slope appearance. From the many different indices introduced to quantify blood flow, the most commonly used single parameter is the resistive index (RI), a ratio between end-diastolic velocity and peak systolic velocities. Restrictive index is a physiological parameter reflecting the degree of renal vascular resistance. Normal renal blood flow has a low resistance pattern, with a flow maintained throughout diastole. The normal RI values are 0.58 ± 0.10.[2] Values >0.70 are considered abnormal and may be due to lower arterial patency, although major clinical significance is observed for values >0.80. Doppler signals are commonly obtained from renal artery or interlobar arcuate arteries at the corticomedullary junction and border of medullary pyramids. However, the identification of these areas requires more training and experience in performing Doppler ultrasound. The test is routinely performed to evaluate the transplanted kidney. The RI has been proposed to assist with the differential diagnosis between obstructive and nonobstructive hydronephrosis, or diagnosis of acute obstruction when dilatation has not yet developed. A minority of patients with obstructive renal failure may not show hydronephrosis due to dehydration or decompression caused by rupture of calyceal fornix. High intrarenal pressure and changing renal hemodynamics due to the release of vasoactive substances and vasoconstriction secondary to obstruction cause an increase in intrarenal arterial resistance measured by a higher RI. While the diagnostic accuracy of RI still remains controversial due to a wide range of results, a normal RI may still be helpful in arguing against the presence of obstruction.[6–8] Color-flow Doppler ultrasound is frequently performed for the evaluation of the patency of the ureter. Jet phenomenon should be seen in the bladder when the urine bolus from the ureter is being propelled into the bladder cavity due to periodic peristalsis (1–12 jets per minute). Ureteral jets are usually identified during transverse bladder scanning as a color projecting into the bladder lumen from lateral posterior border and coursing superior and medial (Figure 24.3). While most critically ill patients have indwelling Foley catheters, bedside evaluation of ureteral jets may be limited due to the empty bladder.

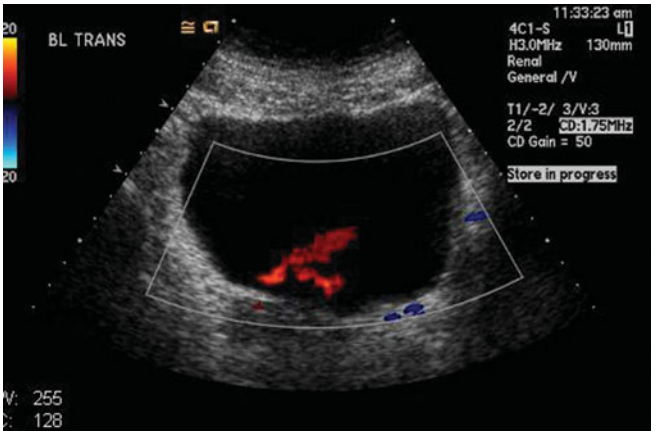


Figure 24.3. Color Doppler ultrasound of the urinary bladder shows crossing bilateral ureteral jets.

CLINICAL APPLICATIONS

Renal Failure

Acute and acute-on-chronic renal failure (ARF) is relatively common in critically ill patients with a reported prevalence ranging between 16% and 23%.[9] While physical examination and laboratory tests are invaluable in making a correct diagnosis, sonography rapidly provides useful information about the kidneys independent of renal function. The American College of Radiology Appropriateness Criteria suggests ultrasound as a primary imaging technique in acute renal failure.[10]

Traditionally, renal failure is categorized as prerenal, intrinsic to the kidney, and postrenal. While prerenal kidney failure will not be associated with specific sonographic abnormalities, intrinsic and especially postrenal (obstructive) causes usually will have visible ultrasound features. Ultrasound evaluation can establish the presence of kidneys, their size, shape, and echogenecity. The absence of kidney in the normal anatomical position (pelvic kidney is usually located close to midline, just above the bladder) requires further investigation.

Renal parenchymal damage is a major cause of intrinsic renal failure. Sonographic evaluation is not helpful in providing a precise diagnosis of renal disease. However, it may provide some information regarding the nature of renal insufficiency. Normal or enlarged kidneys are likely associated with acute renal failure. Parenchymal echogenecity is equal or greater compared to the liver. It is important to remember that liver echogenecity may also be altered in a critically ill patient. In more severe cases the echogenecity of the renal parenchyma is equal to the renal sinus echoes.

The most common cause of ARF in critical care patients is acute tubular necrosis (ATN).[11] While sonography is not a diagnostic method used for ATN, recent research provides support for the possible use of color Doppler for the monitoring of improvement of renal hemodynamics in the critically ill patient. The recovery of renal function has been characterized by improvement in RI when there are still no significant changes in the diuresis.[2]

Chronic renal failure is associated with small (5–8 cm in length) contracted kidneys with increased echogenecity. Renal sinus echoes are still visible, but the parenchyma may show evidence of focal losses (Figure 24.4).

Postrenal ARF can be efficiently corrected if promptly diagnosed. About 5% of patients with ARF suffer from obstructive uropathy (hydronephrosis). It is more

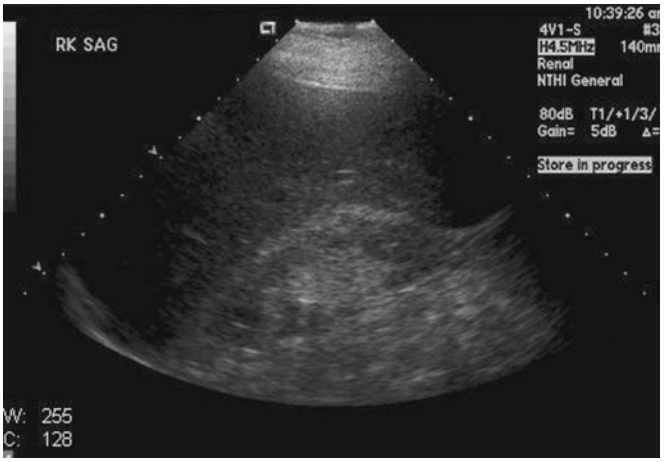


Figure 24.4. Chronic renal failure. Small contracted right kidney. Parenchymal echogenecity is equal to that of the liver and slightly less than of renal sinus.

common in patients with certain predisposing factors including urolithiasis, retroperitoneal cancer, or a solitary kidney. In patients with no risk factors for urinary obstruction, only approximately 1% will have sonographically detected hydronephrosis.[9] Nevertheless, obstructive uropathy remains the most important finding that requires urgent treatment because it is likely to be reversible. Alternatively, knowing that obstruction is absent is as important a finding as treating obstruction.

Sonography can usually diagnose obstruction quickly and simply with a sensitivity of approximately 95%. The dilatation of the renal collecting system (hydronephrosis) is the most important sonographic feature of obstructive uropathy. Renal pelvis and calyceal dilation are characterized by effacement of the renal sinus fat by an anechoic-branched structure with through-transmission. Hydronephrosis is most commonly categorized as mild, moderate, or severe. The degree of renal damage can be quantified on the basis of a reduction in parenchymal thickness. Mild hydronephrosis (grade I) refers to minimal dilatation of the collecting system known as splaying. Moderate hydronephrosis (grade II) shows rounding of the calices with obliteration of the papillae. Cortical thinning is minimal in moderate hydronephrosis. Severe hydronephrosis (grade III) refers to massive dilatation of renal pelvis and calyces associated with cortical thinning (Figure 24.5).

However, the degree of dilatation does not necessarily correlate with the presence or severity of obstruction. Acute, high-grade obstruction may produce only minimal hydronephrosis on early ultrasound before significant dilatation of the collecting system

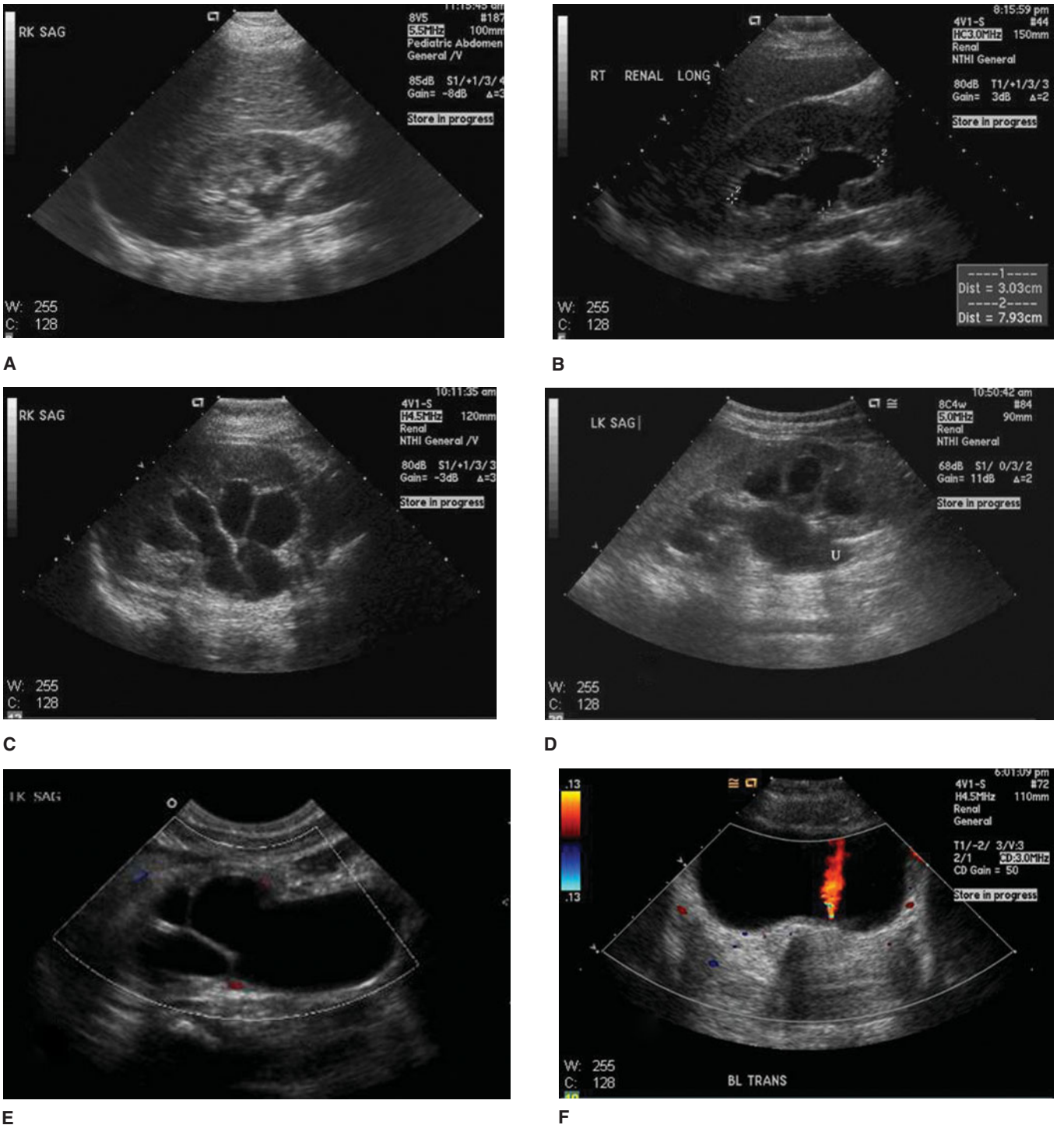


Figure 24.5. (A) Mild hydronephrosis with slight widening of the renal collecting system. (B) Moderate hydronephrosis. (C) Moderate hydronephrosis without loss of renal parenchymal thickness. (D) Hydronephrosis and proximal hydroureter (U). (E) Severe hydronephrosis with thinning of renal parenchyma. (F) Loss of the right ureteral jet in patient with right obstructive hydronephrosis.

develops. This problem may be common in critically ill patients with reduced renal function.

Hydronephrosis does not necessarily equate with obstruction because other factors (e.g., infection, persistent diuresis, and reflux) can cause dilatation of the pelvico-calyceal system. Doppler evaluation has been proposed for the suspected renal obstruction. Normal RIs suggest the absence of obstruction, while RIs greater than 0.70 suggest an obstructive etiology of hydronephrosis. However, this method remains

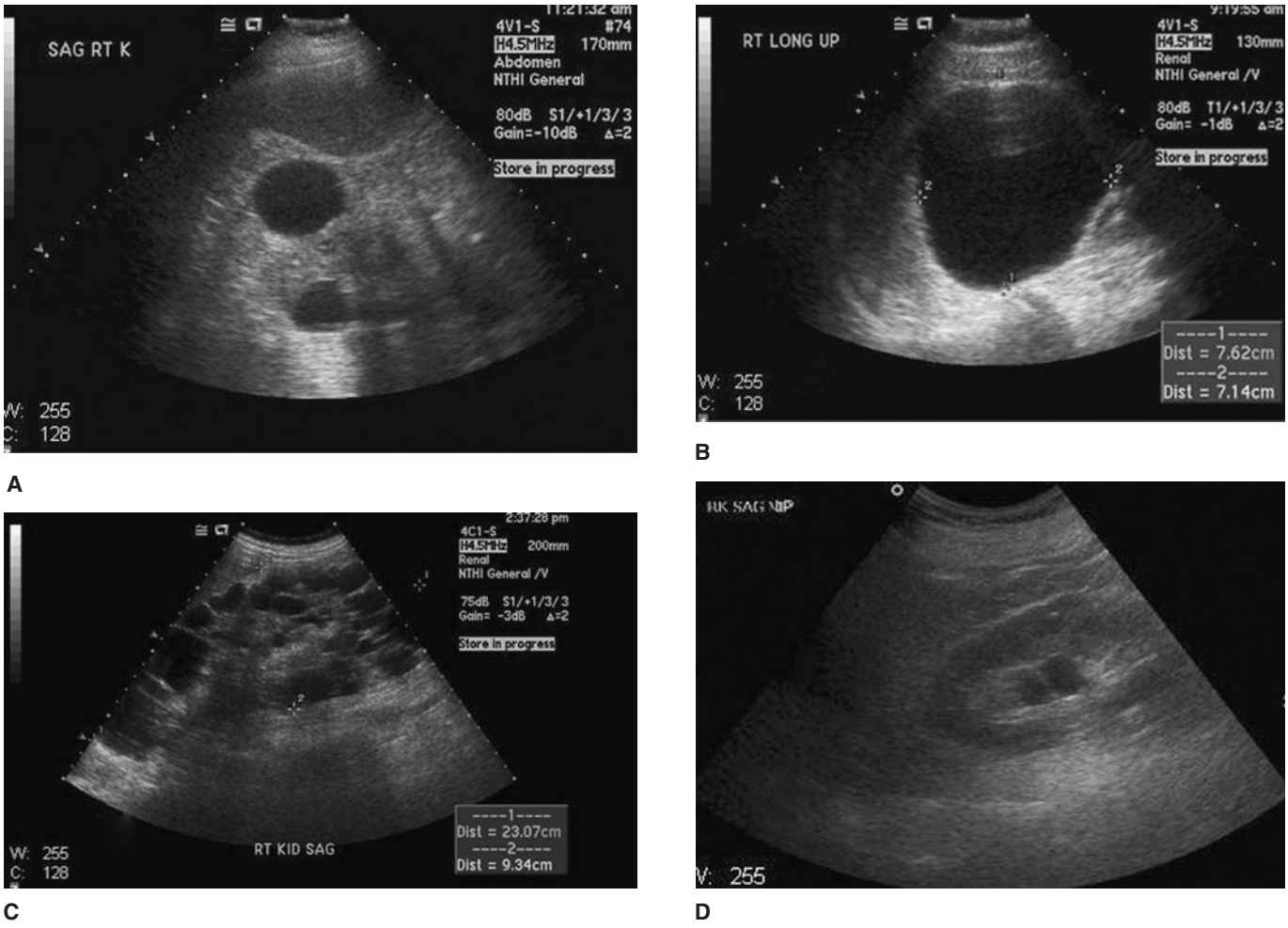


Figure 24.6. (A) Renal cyst may be single or multiple. Borders are well defined. No internal echoes are seen. (B) Large centrally located renal cyst. (C) Polycystic kidneys are usually bilateral. Normal renal parenchyma of enlarged kidney is replaced with multiple cysts of different sizes. (D) Two parapelvic cysts do not communicate with the renal collecting system (portable ultrasound).

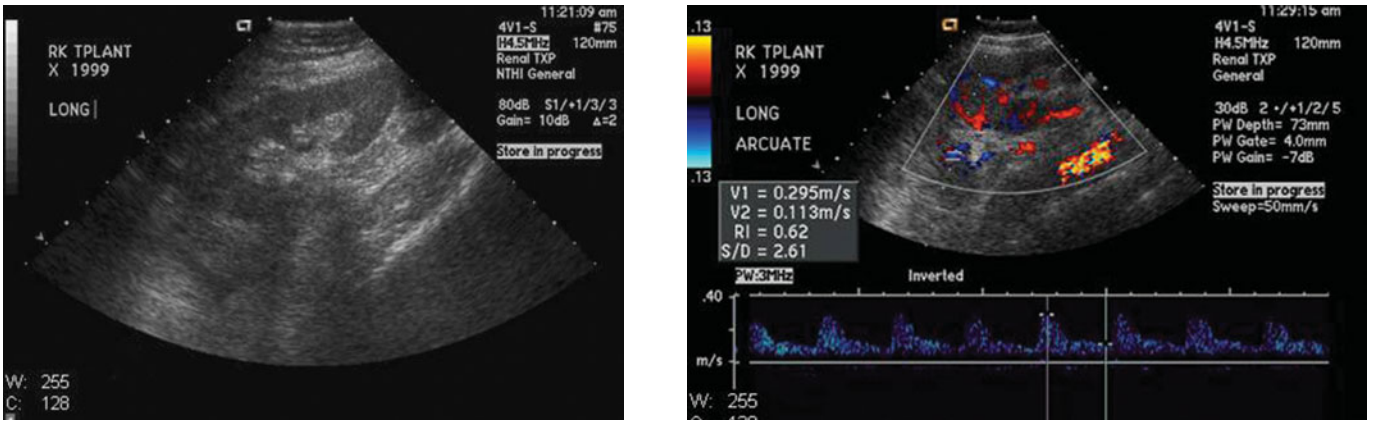
controversial because of equivocal and conflicting results in detection of either acute or partial obstruction.[8]

Analysis of ureteral jets by Doppler interrogation may be another way to diagnose ureteral obstruction. The detection of intermittent flashes of Doppler color (jets) indicates patency of upper urinary tract. The absence of a unilateral jet is highly significant as an indication of obstruction. The presence or absence of ureteral jets does not correspond to the degree of hydronephrosis. The bilateral absence of jets is less specific and may indicate a lack of difference in specific gravity between urine entering the bladder and urine in the bladder. The combined Doppler study (RI and ureteral jets) improves the accuracy of renal ultrasound in the diagnosis of obstruction.[6]

Identification of the obstructing lesion remains the best way to confirm the significance of hydronephrosis. However, it is not always possible with the limited ultrasound evaluation of critically ill patient. Bilateral hydronephrosis in patients with ARF, regardless of its cause, requires emergency decompression of the kidneys to restore urinary output.

Certain sonographic findings mimicking hydronephrosis include renal cysts, an extrarenal pelvis, and polycystic renal disease (Figure 24.6). Questionable findings in patients with anuria may require an extended evaluation beyond ultrasound to confirm a diagnosis of obstruction.

Renal cysts are the most commonly found renal mass. Sonographic features of a simple cyst include a spherical appearance, an anechoic lumen without internal echoes, a well-defined back wall, clear wall demarcations, no measurable wall thickness, and an acoustic enhancement posterior to the cyst. Single or multiple cysts may be located anywhere in the



A B

Figure 24.7. (A) Longitudinal view of the right transplanted kidney. The renal parenchyma is well visualized with bright renal sinus echoes centrally. (B) Color Doppler ultrasound image of the transplanted kidney. Spectral gate is placed over arcuate vessels. A number of indices can be measured simultaneously. Resistive Index <0.7 is considered normal.

kidney. A renal sinus cyst is called parapelvic and accounts for 6% of renal cysts. A parapelvic cyst does not communicate with renal pelvis and calyces. Unlike the cauliflower appearance of a dilated pelvis, a parapelvic cyst is rounder, with good through-transmission. Sonographically, the differential diagnosis between hydronephrosis and parapelvic cyst may be difficult, especially if the cysts are bilateral. Complex cysts do not meet the sonographic criteria of simple cysts. They may be septated and multilocular. While the ultrasound diagnosis of a simple cyst is very accurate, complex cysts may require additional imaging studies (e.g., computerized tomography [CT] or magnetic resonance imaging [MRI]) to rule out malignancy.

Autosomal-dominant polycystic kidney disease is one of the etiologies of end-stage renal failure. Multiple variably sized cysts located in the cortex and medulla are characteristic for this bilateral disease. The kidneys are enlarged and the parenchyma can be identified or completely replaced by numerous cysts. The extrarenal pelvis lies largely outside the kidney rather than in its usual central location. Usually, dilatation of the calyces is not associated with nonobstructed, dilated extrarenal pelvis.

KIDNEY TRANSPLANT

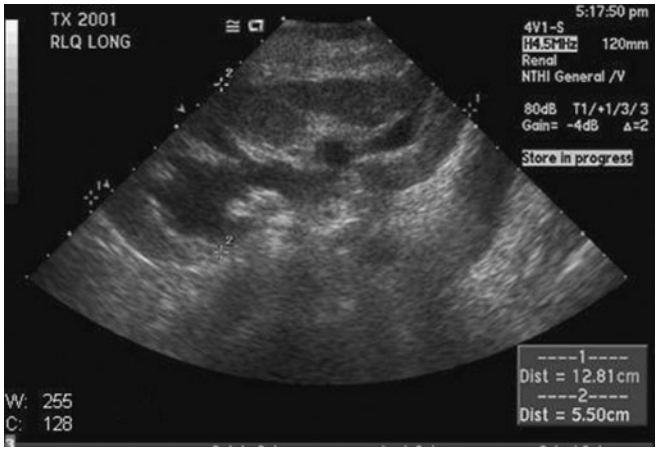
Decreased renal function after transplantation is the most important indication for ultrasound evaluation. The more superficial location of the transplanted kidney requires a 5–7 MHz transducer. Longitudinal and transverse scans over the kidney provide accurate measurements of kidney size, echogenecity, shape and evidence of hydronephrosis, bladder sonogram, and the color Doppler flow studies.

The normal transplanted kidney is similar to the native kidney morphologically. It has a smooth contour and homogenous parenchyma. The urinary bladder should be visualized where possible. Normally, it must be empty because full bladder may cause hydronephrosis (Figure 24.7).[12,13]

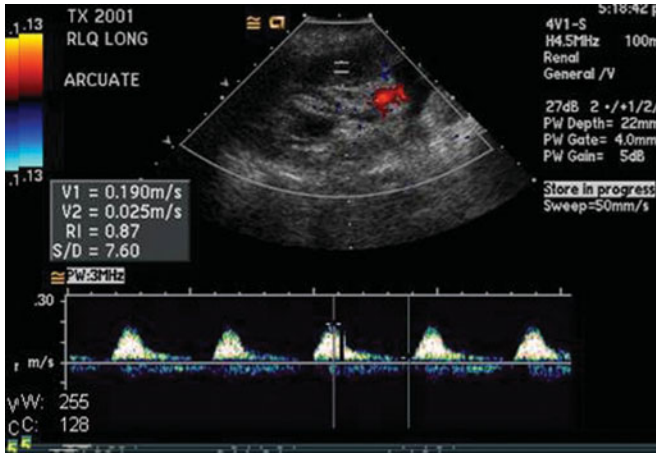
The primary goal of ultrasound in the transplanted kidney is to differentiate between obstructive uropathy and systemic or intrinsic causes of reduced function (acute rejection or acute tubular necrosis) and to identify peritransplant fluid collections.

Acute rejection (AR) and acute tubular necrosis have no specific diagnostic sonographic features. The diagnostic value of ultrasound in ATN or acute rejection is limited. The kidney may be enlarged, with increased cortex hyperechogenecity and occasional distortion of the renal outline. While the differential diagnosis between AR and ATN is not possible with ultrasound, obstruction (hydronephrosis) is very distinctive and is important to rule out (Figure 24.8).

The sonography can promptly identify hydronephrosis and establish indications for surgical intervention or close follow-up. Hydronephrosis has the same appearance in the transplanted kidney as in the native kidney and may be secondary to anastomotic failure or a fluid collection due to urine leak (urinoma), lymphatic leak (seroma), or bleeding (hematoma). Peritransplant fluid collections have been reported in up to 50% of renal transplantation. The clinical relevance of these collections is largely determined by their size, location, and possible growth.[12]



A



B

Figure 24.8. (A) Hydronephrosis of the transplanted kidney. (B) Color Doppler study shows elevated resistive index of the arcuate arteries.

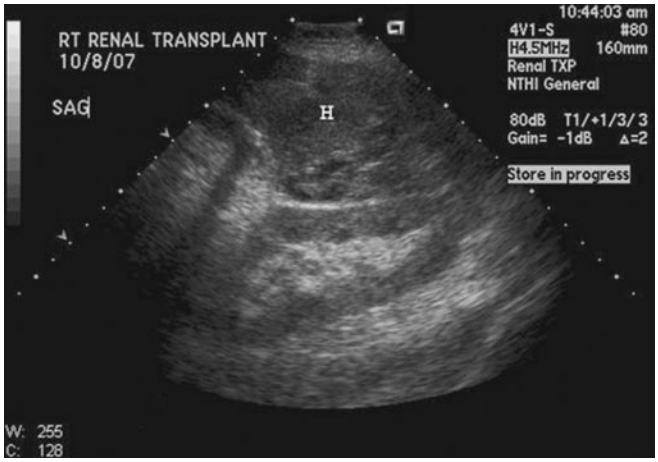
Peritransplant fluid collections are readily detectable by ultrasound. Regardless of their nature (urine, blood, or lymph), sonographically they may appear as well-defined anechoic areas, with or without septation, although acute hematoma may be echogenic. Internal echoes are mostly seen in hematomas where clots have become organized (Figure 24.9).

Color Doppler is used routinely to evaluate the transplanted kidney. However, RI itself is not specific for the differential diagnosis of transplant complications, although it may be helpful to confirm an obstructive hydronephrosis. The most common cause of elevated RI (over 0.70) in the absence of obstruction or infection is acute rejection.[13] The likelihood of an acute rejection increases as the values of RI increase.

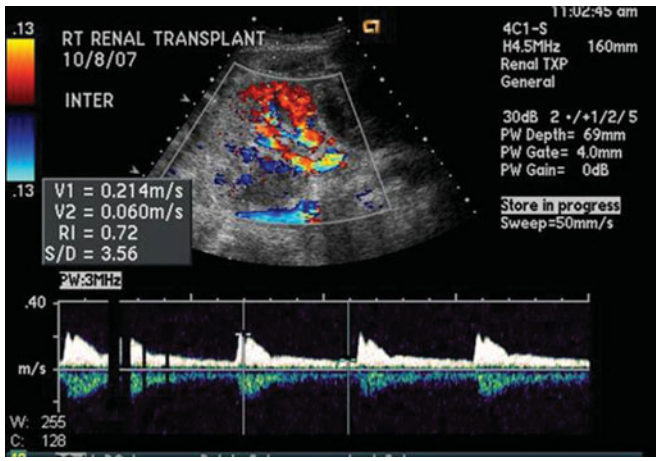
RENAL TRAUMA

Focused assessment with sonography for trauma (FAST) has been an accurate method for detecting hemoperitoneum in unstable patients. However, ultrasound is presently not advocated as a first-line imaging modality in renal trauma because its sensitivity in diagnosing and grading renal injury remains low. Normal findings do not exclude renal injury and major injuries may not be identified.[14,15]

While renal ultrasound may reveal subcapsular or perinephric fluid collections, it cannot provide crucial differentiation between blood, extravasated urine, and other types of free fluid. Color Doppler ultrasound can be helpful in the evaluation of renal blood flow by



A



B

Figure 24.9. (A) Large hematoma anterior to the transplanted kidney, which does not affect kidney function. Renal parenchyma shows normal echogenecity. (B) Normal resistive index of the interlobar arteries.

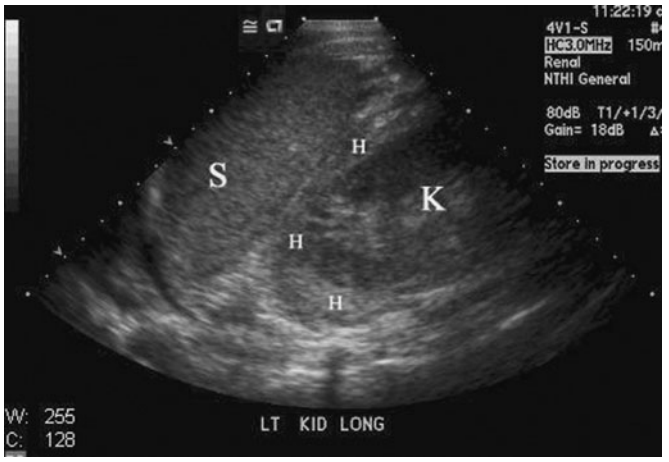


Figure 24.10. Perirenal hematoma in patient with left renal laceration. H indicates hematoma; K, kidney, S, spleen.

assessing perfusion of the whole kidney or any portion. If renal ultrasound suggests injury or if it is negative in patient with the clinical evidence of renal involvement (hematuria), a contrast CT should be provided in stable patient for further evaluation.

However, ultrasound is a very useful tool for a bedside monitoring of the resolution or expansion of hematomas in the critical care setting, as recent management of even severe isolated renal injuries is mostly conservative (Figure 24.10).

UROSEPSIS

Urinary tract infection (UTI) is one of the most common hospital-acquired infections and most frequent nosocomial infection in critically ill patients. Complicated UTI is not uncommon in ICU patients due to multiple predisposing factors including compromised immune systems, associated medical problems, and indwelling urinary catheters. The spectrum of UTI extends from acute pyelonephritis with or without obstructive uropathy to renal and perirenal abscesses.

While pyelonephritis usually has no specific sonographic features, ultrasound can diagnose such abnormalities as hydro- and pyonephrosis, renal and perirenal abscesses, or emphysematous pyelonephritis.

Pyonephrosis represents pus in the obstructed and infected collecting system. It is a medical emergency requiring immediate renal decompression. Pyonephrosis must be suspected in patients with hydronephrosis, urinary tract infection, and ultrasound findings of low-level echoes with occasional layering in the dependent position of the dilated collecting system.

Renal intraparenchymal abscess appears as a complex hypoechoic mass with thick irregular walls and occasional fluid debris level. A perinephric abscess will result in a heterogeneous crescent-shaped fluid collection surrounding the kidney that may deform the renal cortex. While additional tests (CT) may be necessary to differentiate between abscess and renal cancer, ultrasound can be a valuable tool to monitor the diagnosed abscess or focal pyelonephritis during medical treatment (Figure 24.11).

Emphysematous pyelonephritis is an uncommon but life-threatening diffuse infection of the renal parenchyma caused by gas-forming bacteria. Most patients are female, and 90% are diabetic. Patients are usually extremely ill, often toxic, presenting with fever, flank pain, acidosis, hyperglycemia, dehydration, and electrolyte imbalance. Ultrasound typically reveals an enlarged kidney containing high-amplitude echoes within the parenchyma/renal sinus associated with a low-level posterior acoustic shadowing. Computerized tomography scan is necessary to confirm the presence of air in the renal parenchyma.

OTHER SONOGRAPHIC FINDINGS

Screening, observational, or focused ultrasound evaluation of the critically ill patient frequently reveals additional sonographic findings that have no direct impact on the patient's condition and will not change the immediate treatment . However, such incidental findings require further evaluation with additional imaging modalities to establish correct diagnosis when the patient is stable (Figure 24.12).

A solid renal mass is a heterogeneous, isoechoic, or hypoechoic lesion of variable dimensions adjacent to a normal renal parenchyma. Ultrasound is used primarily to differentiate solid masses from simple cysts. All solid renal masses in adults should be considered malignant until proven otherwise. Further evaluation with CT scan is required for appropriate diagnosis.

Nephrolithiasis is one of the most common kidney problems. Kidney stones are intensely hyperechoic linear or arching foci with posterior acoustic shadowing. They can be of different sizes and locations within the kidney. Obstructing stones occasionally can be seen in patients with hydronephrosis and dilated proximal or distal ureter.[16] Nonobstructing kidney stones do not require urgent treatment.

BLADDER ULTRASOUND

The sonographic evaluation of the bladder is uncommon in critically ill patients with an indwelling Foley catheter and collapsed bladder. However, it is a

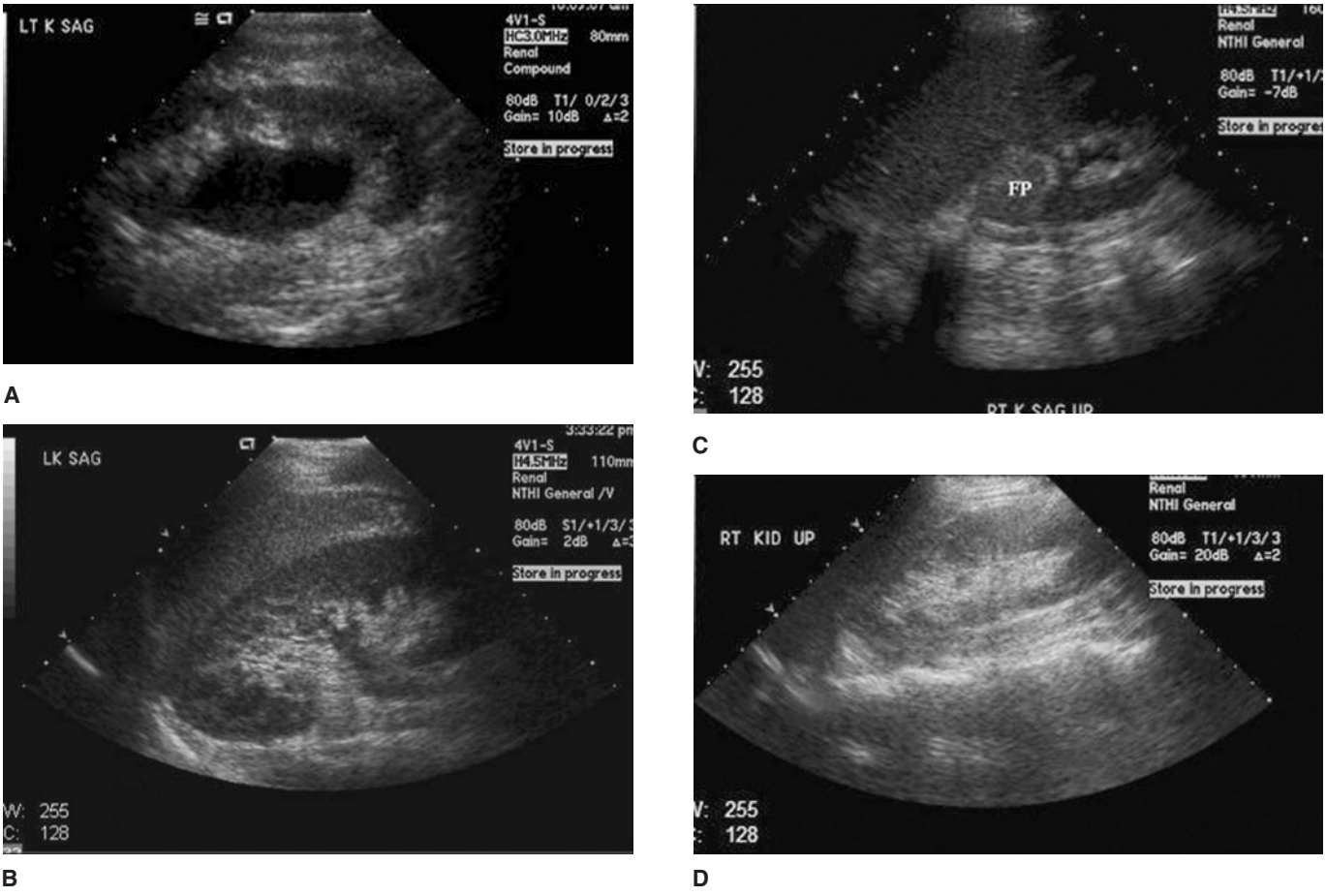


Figure 24.11. (A) Pyonephrosis. Moderate-to-severe hydronephrosis with fine debris in the renal collecting system. (B) Nonspecific finding of the enlarged kidney in patient with clinical picture of acute pyelonephritis. (C) Hyperechoic lesion within renal parenchyma in patient with urosepsis represents area of focal pyelonephritis (FP). (D) Follow-up ultrasound revealed complete disappearance of the lesion after medical treatment.

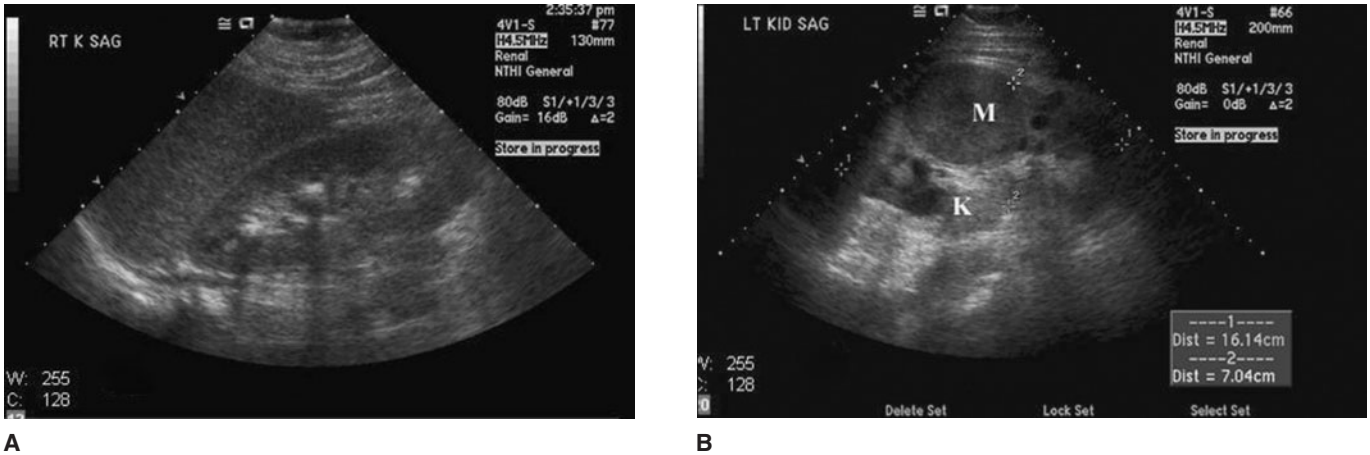
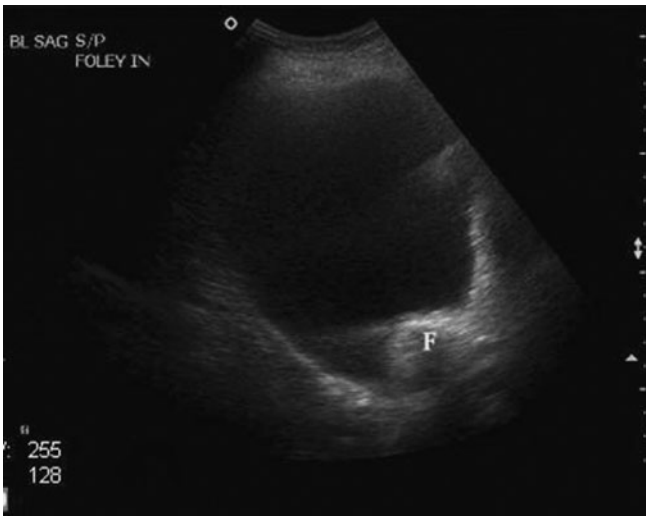
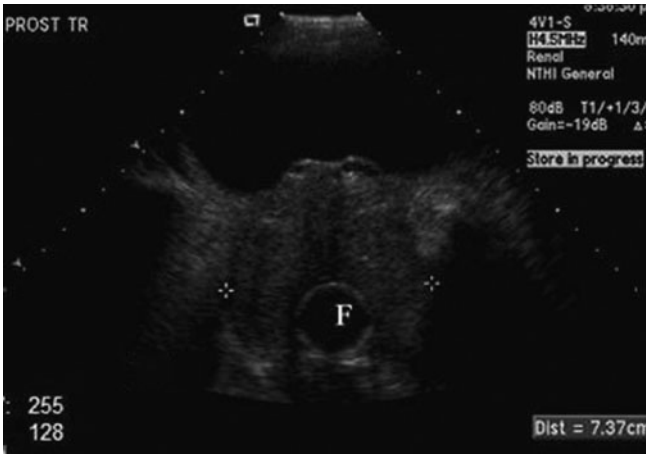


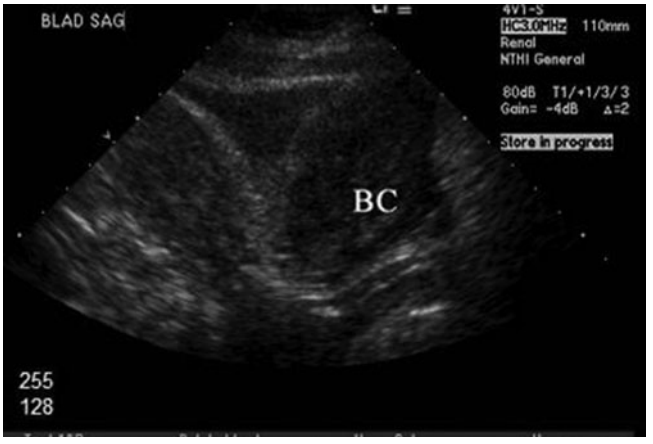
Figure 24.12. (A) Multiple renal stones. Nonobstructed kidney containing hyperechoic (white) calcifications with posterior acoustic shadowing. (B) Large solid renal mass (M) distorting renal collecting system (K).



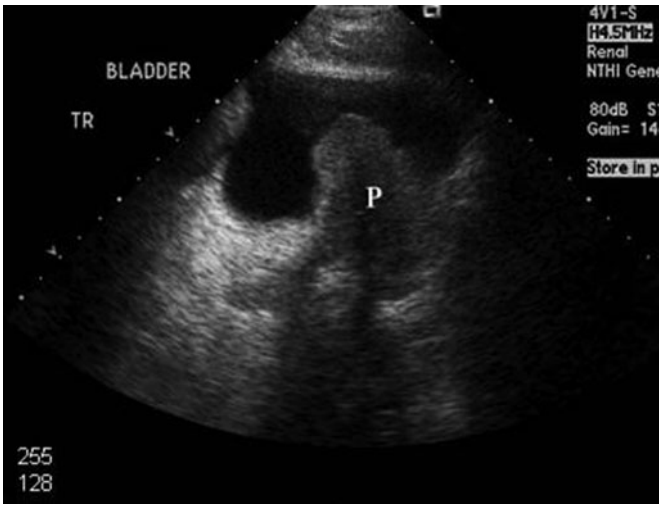
A



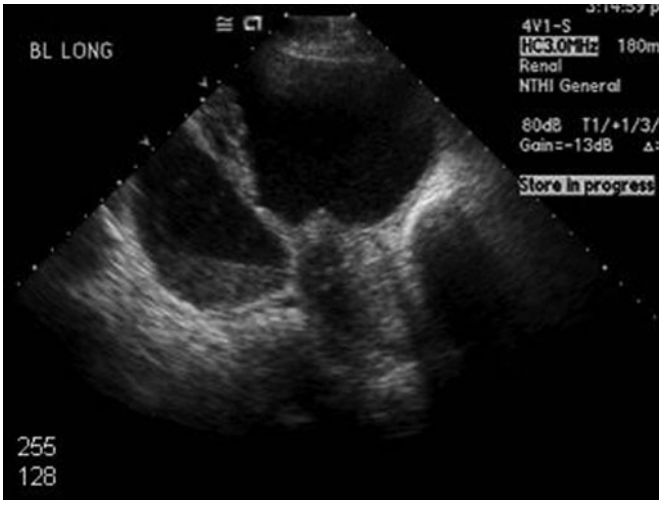
B



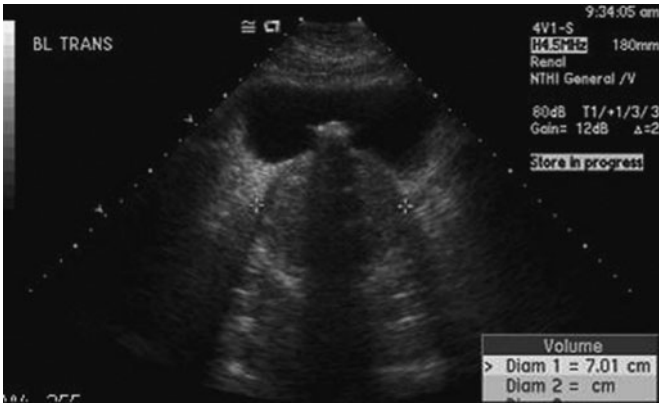
C



D



E



F

Figure 24.13. (A) Portable ultrasound. Distended bladder with obstructed Foley catheter. (B) Dislodged Foley catheter in the prostatic urethra below the bladder. (C) Large blood clot in patient with gross hematuria may simulate bladder tumor. (D) Grossly enlarged prostate protruding into the bladder lumen. (E) Large bladder diverticulum. (F) Bladder stone. BC indicates blood clot; F, Foley catheter.

necessary part of renal ultrasound because urinary retention may cause hydronephrosis. Dislodged or obstructed Foley catheters are a common cause of "anuria" in critically ill patients. Bladder ultrasound allows prompt visualization of a distended bladder and can locate the balloon of the Foley catheter within or outside of a full bladder.[17] Bladder volume can be automatically calculated by measuring horizontal and vertical dimensions of the bladder on a transverse image and maximum longitudinal dimension on longitudinal image. Bladder stones appear hyperechoic with posterior acoustic shadowing. They move with changes in patient position. Blood clots may be visualized in patients with gross hematuria. The differential diagnosis must include bladder tumor, which also appears as a polypoid, hyperechoic projection from the bladder wall. Diverticuli present sonographically as sonolucent masses adjacent to the bladder. Large bladder diverticula may still be visible with an empty bladder. A grossly enlarged prostate may be seen as a round or polypoid mass protrusion at the bottom of the bladder (Figure 24.13).

SUMMARY

The role of ultrasound of kidneys and bladder in critical care has been widely debated based on its usefulness and cost-effectiveness. While bedside urologic ultrasound is not a substitute for standard sonography or other important and informative imaging modalities, it certainly provides properly trained physicians with the unique ability to perform bedside ultrasound-enhanced clinical evaluations of the urinary system of critically ill patients. Whether it is a screening, observational, or emergency evaluation, this approach is helpful in expediting treatment and improving outcome.

References

1. Kirkpatrick AW, Sustic A, Blaivas M. Introduction to the use of ultrasound in critical care medicine. *Crit Care Med*. 2007;35:S123.
2. Barozzi L, Valentino M, Santoro A, Mancini E, Pavlica P. Renal ultrasound in the critically ill patient. *Crit Care Med.* 2007;35:S198.
3. American Institute of Ultrasound in Medicine. AIUM Practice Guidelines for the performance of an ultrasound examination of the abdomen and /or retroperitoneum. *J Ultrasound Med*. 2008;27:319.
4. Heller M, Jehle D. Obstructive uropathy. In: Heller M, Jehle D, eds. *Ultrasound in Emergency Medicine.* Philadelphia, Pa: WB Saunders Co; 1995:65.
5. Noble VE, Brown DFM. Renal ultrasound. *Emerg Med Clin N Am*. 2004;22:641.
6. Pepe P, Motta L, Pennisi M, Aragona F. Functional evaluation of the urinary tract by color Doppler ultrasonography (CDU) in 100 patients with renal colic. *Eur J. Radiol.* 2005;53:131.
7. Gurel S, Acata D, Gurel K, et al. Correlation between renal resistive index (RI) and nonenhanced computed tomography in acute renal colic. How reliable is RI in distinguishing obstruction? *J Ultrasound Med.* 2006;24:1113.
8. Cronan J. Renal failure. In: Bluth EL, Arger PH, Benson CB, Ralls PW, Siegel MJ, eds. *Ultrasound: A Practical Approach to Clinical Problems.* New York, NY: Thieme; 2000:90.
9. Keyserling HF, Fielding JR, Mittelstaedt CA. Renal sonography in the intensive care unit: when is it necessary? *J Ultrasound Med.* 2002;21:517.
10. American College of Radiology Appropriateness Criteria® renal failure. American College of Radiology—Medical Specialty Society. 1995 Available at: www.guideline.gov. Accessed 2005.
11. Huang SW, Lee CT, Chen CH, Chuang CH, Chen JB. Role of renal sonography in the intensive care unit. *J Clin Ultrasound.* 2005;33:72.
12. Park SB, Kim JK, Cho K. Complications of renal transplantation. *J Ultrasound Med.* 2007;26:615.
13. Baxter GM.: Ultrasound of renal transplantation. *Clin Radiol.* 2001;56:802.
14. McGahan PJ, Richards JR, Bair AE, Rose JS. Ultrasound detection of blunt urological trauma: A 6-year study. Injury, Int. *J Care injured* 2005;36:762.
15. Scout LM, Sawyers SR, Bokhari J, Hamper UM. Ultrasound evaluation of the acute abdomen. *Ultrasound Clin.* 2007;2:493.
16. Lin EP, Bhatt S, Dogra VS, Rubens DI. Sonography of urolithiasis and hydronephrosis. *Ultrasound Clin.* 2007; 2:1.
17. McAchran SE, Hartke DM, Nakamoto DA, Resnick MI. Ultrasound of the urinary Bladder. *Ultrasound Clin.* 2007; 2:17.

This page intentionally left blank

CHAPTER 25

Ultrasound Evaluation of the Pelvis

Michael Blaivas

INTRODUCTION

Relevant pelvic pathology that can be encountered in the intensive care unit (ICU) setting, and is amenable to bedside ultrasound evaluation, may be split into three general categories. The first is a source of blood loss and will most typically include ectopic pregnancy, hemorrhagic cyst, or mass. The second is a source of infection and is most likely to include pelvic inflammatory disease and tubo-ovarian abscess. The third is a source of pain apart from the first two categories including ovarian cysts, masses, and ovarian torsion.

RELEVANT ANATOMY

Sonographic pelvic anatomy can be challenging and, depending on the ultrasound technique utilized, anatomical relationships may appear confusing. The uterus, a pear-shaped muscular organ typically measuring 6–8 centimeters (cm) in length and 4 cm in width, is bordered anteriorly by the bladder and posteriorly by the rectum. The uterus comprises the fundus, body, and cervix, where it narrows and protrudes into the vagina (Figure 25.1). The fallopian tubes exit the uterus on either side of the uterine fundus at the level of the cornua. The anterior cul-de-sac is a potential space between the uterus and bladder, while the posterior cul-de-sac (pouch of Douglas) is between the uterus and rectum. With the patient supine, the pouch of Douglas is the most dependent part of the pelvis and is typically the first area to collect fluid such as blood or pus. The fallopian tubes extend laterally from the cornua in the broad ligament. An ovary attaches to the broad ligament posteriorly on each side. The iliac artery and vein run posterior and lateral to the ovaries and the two are a major landmark sonographically.

PROCEDURE

The pelvic ultrasound examination is split into two distinct types that are not mutually exclusive, and one may lead to the other depending on the pathology discovered. The easiest is the transabdominal (TAS) pelvic ultrasound examination. It is performed utilizing a curved linear array with a typical frequency range of 5–2.5 MHz. The broad field of view afforded by this type of transducer is ideal for surveying the pelvis. In general, the TAS pelvic ultrasound examination requires a full bladder. In the case of many ICU patients, this simply means clamping the urinary catheter. An alternative is to fill the bladder with either sterile saline or to simply hold up the catheter bag and have some of the urine flow back into the bladder. The ideal volume that allows the bladder to act as an optimum acoustic window will vary from patient to patient. Approximately 350 milliliters (mL) will be ample in most cases and it is possible to overfill the bladder and actually move organs of interest farther away from the transducer. Similar to the pelvic portion of the focused assessment with sonography for trauma (FAST) examination, the transducer is held in two standard orientations. The transverse orientation is with the marker oriented to the patient's right hip and the longitudinal one is with the probe indicator pointed to the patient's head. It is critical to scan through the bladder in order to best image pelvic organs in the TAS approach. Pelvic organs, like most others, should be scanned in two orthogonal planes. The TAS examination gives limited views of the ovaries and fallopian tubes in most patients. The fallopian tubes are rarely seen on TAS unless filled with fluid. Even then it may be difficult to differentiate them from other fluid collections without using an endovaginal (EV) approach.

The EV ultrasound approach is generally preferred for structures within the true pelvis. The ovaries are visualized with greater detail, pregnancy can be seen at a much earlier date, and ectopic pregnancies can be identified with much greater accuracy than with TAS.[2] However, it is still considered by most as an examination to perform after TAS. The EV ultrasound examination requires an endocavity probe. These are typically microconvex and tend to range from 8 MHz to 4 MHz, with some variation. Color or power Doppler is critical for this type of transducer in order to differentiate blood flowing in vessels from other types of fluid. The endocavity transducer is encased in a nonsterile

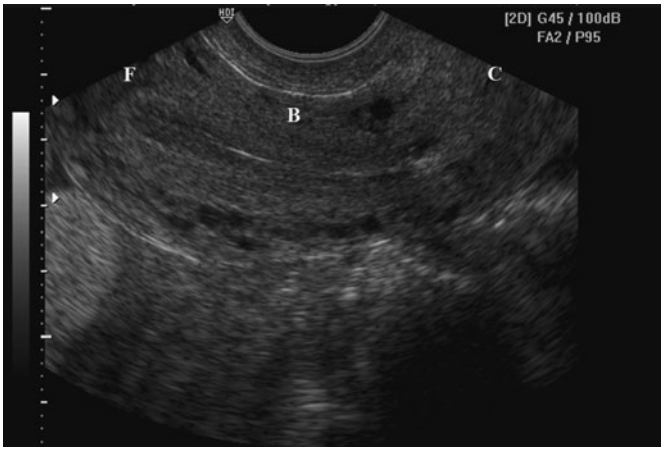


Figure 25.1. A normal uterus is seen in long axis on ultrasound. B indicates body of uterus; C, cervix; F, fundus of uterus.

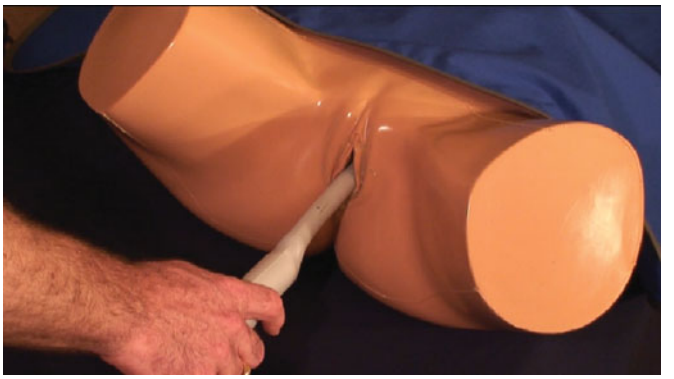


Figure 25.2. An endovaginal probe is being held in the vaginal vault of an ultrasound phantom, with the transducer indicator pointed toward the ceiling. This orientation gives a long-axis view through the uterus similar to that in Figure 25.1.

sheath, such as a condom. Condoms with receptacle tips tend to trap air and are best avoided. The middle finger of a glove can be used as well. Prior to insertion into a sheath, the probe tip is covered with ultrasound gel. Once the sheath is slipped over the probe, the tip should be stretched tight and all air bubbles smoothed out with a finger. Additional gel is used on top of the sheath over the transducer's scanning surface. Unlike the TAS approach, the EV approach requires an empty bladder. Once dressed properly, the probe is ready to insert into the vaginal vault. In modern society, it is best to perform the examination in the constant presence of a chaperone, preferably a female. This will avoid potential questions that may be difficult to defend against if no chaperone was present. Typically, the probe is inserted too far by beginners and it does not need to go beyond the anterior fornix. There are two general imaging planes in endovaginal ultrasound. They are a coronal plane and a longitudinal plane. It may be helpful for beginners to imagine the patient standing on her head as they view the ultrasound machine screen. Anatomy will now make sense, even though the orientation will take some time to adjust to. With the probe indicator pointing toward the ceiling, the empty bladder, long axis of the uterus, and posterior structures are seen (Figure 25.2). A full bladder will make the examination frustrating and has to be corrected. The probe is moved from side to side to obtain images from one adnexa to the other. In addition, the transducer can be angled up and down while the indicator remains pointed toward the ceiling as well as introduced slightly deeper or pulled back in the vaginal vault. The fundus, cervix of the uterus, and surrounding structures can be visualized with good detail using these movements. To obtain a short-axis or coronal view, the transducer is rotated toward the adnexa of interest and moved up and down or from side to side (Figure 25.3). In EV scanning it is critical to image in two orthogonal planes, as structures of interest are periodically seen better in one plane than another. The ovaries are located lateral to the uterus and are almost invariably anterior and medial to the internal iliac artery and vein. These vascular structures are relatively easy to find on the EV examination and are an excellent landmark (Figure 25.4). The fallopian tubes can be seen leaving the cornuate portions of the uterine fundus on either side and may often be tracked nearly to each ovary (Figure 25.5). The tubes are obvious when filled with fluid, but with modern equipment they are still easily seen in most patients.

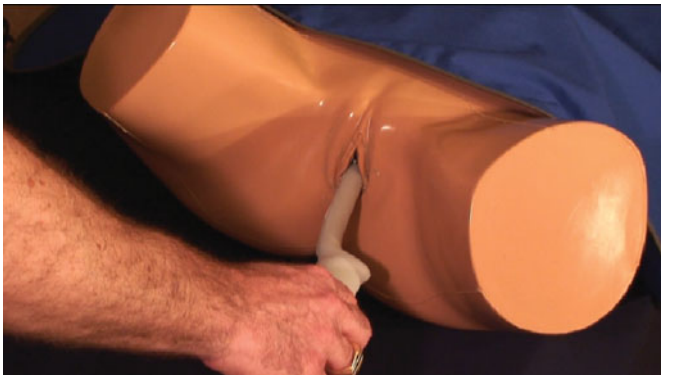


Figure 25.3. The probe inserted into the vaginal vault of the ultrasound phantom has been turned 90° counterclockwise and toward the right adnexa and is now pointed into the adnexa. Short-axis views of the uterus and both ovaries are accomplished in this orientation.

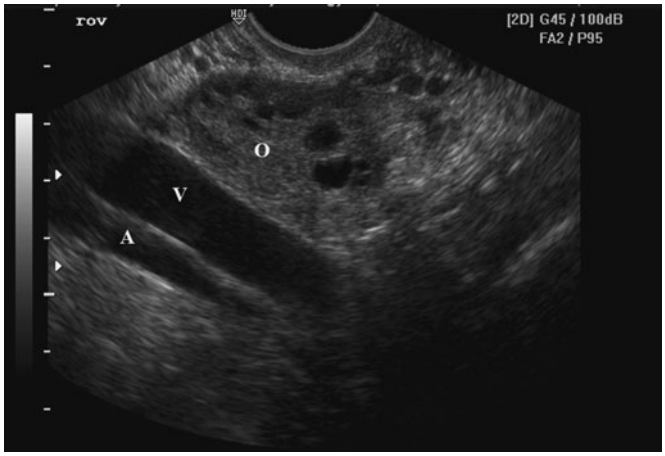


Figure 25.4. The right ovary (O) is seen adjacent to the iliac vein (V) and artery (A).

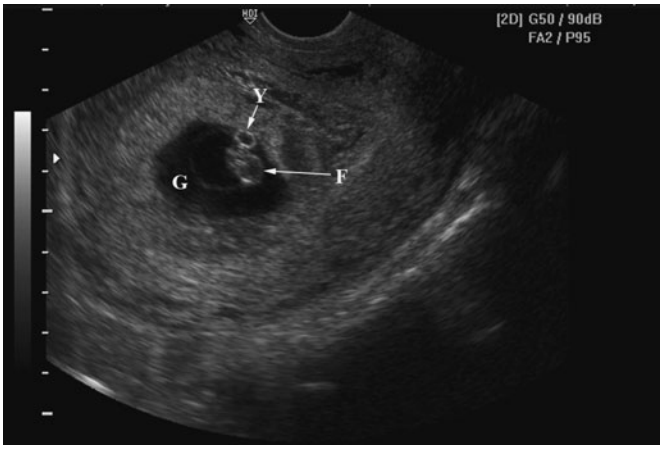


Figure 25.6. This endovaginal ultrasound of a pregnant uterus shows the gestational sac (G), yolk sac (Y), and fetal pole (F).

HEMORRHAGE FROM PREGNANCY

One of the best ways to rule out the presence of an ectopic pregnancy is by ruling in an intrauterine pregnancy (IUP).[2] Although ectopics coexisting with IUPs are encountered, they were traditionally thought to be exceedingly rare in the general population, approximately 1 in 30,000.[3] However, more modern figures show a background incidence closer to 1 in 8000.[4] Patients undergoing any fertility treatment are at a much greater risk for heterotopic pregnancy, with some studies suggesting as high as 1 in 100 for very specialized infertility practices.[5] It is important to note that as many as 70% of all ectopic pregnancies resolve on their own and many others can be treated medically. Thus identifying one does not mean an impending surgical disaster if there is no evidence of rupture and if the ectopic mass is small.[6] While identifying an embryo (fetal pole), especially with a heartbeat, is the best way to diagnose an IUP, earlier embryonic structures can be used confidently to rule in an IUP. Specifically, a yolk sac, the earliest reliable embryonic structure (Figure 25.6). A gestational sac alone is not enough to diagnose an IUP. Having a double decidual sign increases the likelihood of an IUP to over 80%, but still does not reliably rule out an ectopic pregnancy.[7] A double decidual sign refers to the two concentric circles outlying a normal gestational sac, made up of the maternal and fetal decidua. An apparent gestational sac may in actuality be a pseudogestational sac, which is seen in a portion of ectopic pregnancies as fluid collects in the endometrial canal due to hormonal stimulation of the endometrial lining (Figure 25.7).[8]

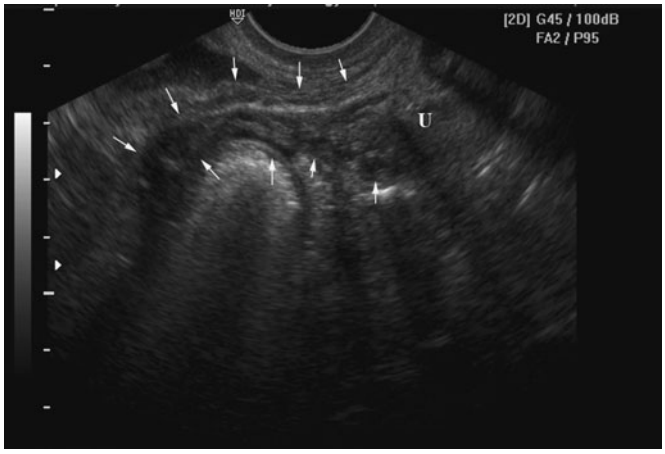


Figure 25.5. The fallopian tube (arrows) is seen leaving the uterus (U) and traveling out into the adnexa toward the ovary (not seen in this image).

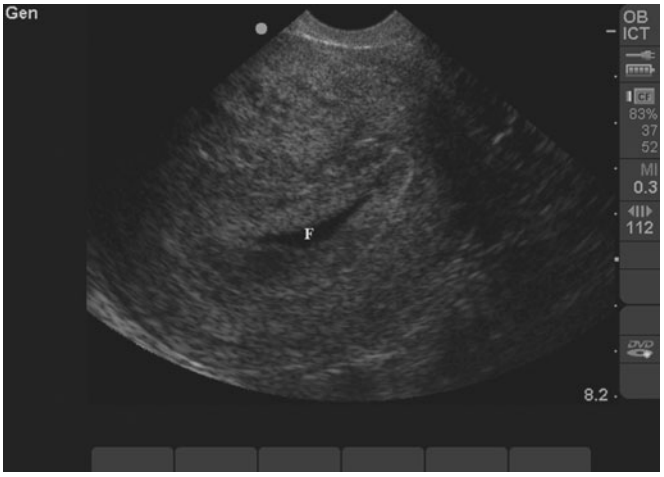


Figure 25.7. A thin collection of fluid (F) is seen in the endometrial canal of this patient with an ectopic pregnancy and represents a pseudogestational sac.

The TAS examination allows for visualization of embryonic structures as soon as six to eight weeks with ideal ultrasound equipment, while the EV approach will typically reliably show a yolk sac by 4.5 weeks' gestational age. The normal location of a gestational sac is in the uterine fundus just off the endometrial midline. The fetal pole is located next to the yolk sac, but if measured, the yolk sac should not be included in the measurement. If IUP is not confirmed on TAS, then an ectopic has not been ruled out. TAS findings suggesting ectopic pregnancy include an absence of IUP (in most cases), presence of echogenic fluid in the pouch of Douglas, a large amount of fluid in the pelvis, and a discrete mass in the adnexa. Actual live ectopics can be seen on TAS and it may even be possible to date an embryo. This is more likely on EV, however (Figure 25.8). Reported rates of live ectopic pregnancy range from 3% to 10%.[9,10] Certainty of ectopic pregnancy depends on the ultrasound findings, coupled with clinical scenario. Obviously, having a positive pregnancy test is important for ectopic diagnosis, but anecdotal reports of ruptured ectopic in the setting of negative pregnancy test do exist. Thus, never say never with ectopic pregnancy. The earliest sign of an ectopic on EV ultrasound is typically a small circular mass with central clearing, located next to the ovary. This structure is called a "tubal ring sign" and represents the fallopian tube with the ectopic pregnancy implanted in it (Figure 25.9). On the opposite end of the spectrum, the tubal ring can be very subtle and may be difficult to differentiate from the corpus luteum of the ovary (which should be located on the same side as the ectopic in the majority of cases). Gently prodding the ovary with

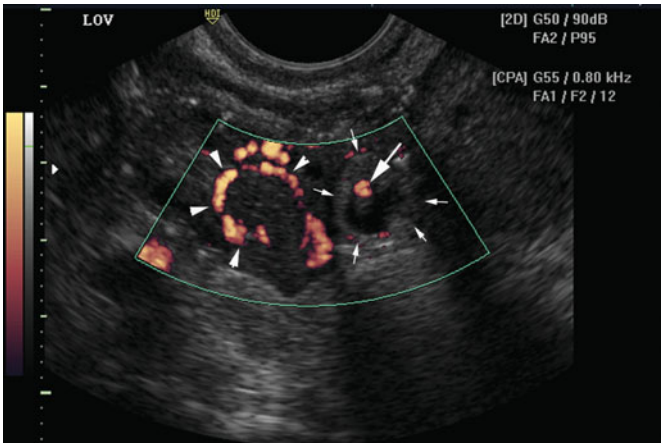


Figure 25.8. Blood flow in the heart of a live ectopic pregnancy is shown (large arrow). The ectopic mass (small arrows) sits adjacent to the left ovary, which contains a solid corpus luteum outlined with blood flow on power Doppler (arrow heads).

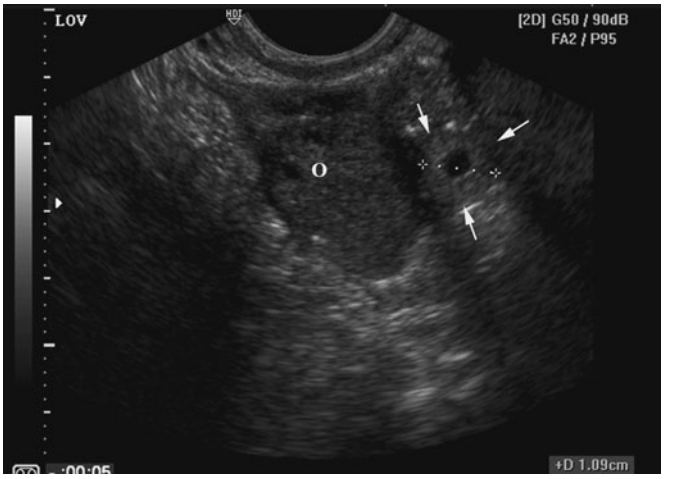


Figure 25.9. A tubal ring (arrows) is shown adjacent to the left ovary (O) and measures just over 1 cm across.

the EV probe will result in movement of the tubal ring and ovary as separate units and is a reliable sign to differentiate a corpus luteum from a tubal ring.[11] A mass next to the ovary and a large amount of echogenic fluid in the pelvis should further raise suspicion for ectopic pregnancy. Occasionally, a yolk sac or embryo may be seen in the tubal ring and this will clinch the diagnosis of ectopic pregnancy.

HEMORRHAGE IN A NONPREGNANT PATIENT

Blood loss can also occur from an ovarian cyst or mass. Hemorrhagic cysts can occasionally result in significant intraabdominal blood loss, especially when arterial bleeding is present. Typical findings include pelvic fluid and a complex ovarian cyst, which may contain evidence of thrombus in it (Figure 25.10). It may occasionally be possible to see blood flow from the offending vessel into the cyst and pelvis. Masses, especially malignant ones, may hemorrhage also, but this is less common. Fibroids can also be the source of significant hemorrhage, but, typically, do not bleed exclusively into the pelvis, and vaginal bleeding will often be present. Fibroids take on a heterogeneous appearance and cast shadows on ultrasound examination. This shadowing can make it difficult to see other structures around the uterus (Figure 25.11). Central liquefaction may be seen in some cases as the fibroid degenerates.

SOURCES OF INFECTION

Although describing the variety of possible pathologies leading to infection is beyond the scope of this chapter,

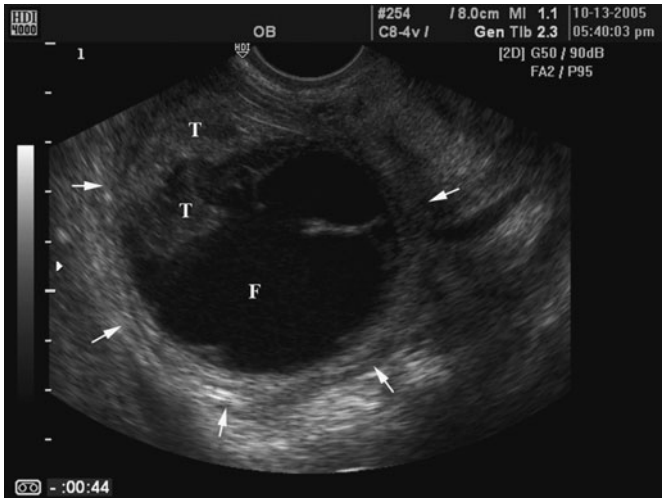


Figure 25.10. A large cyst inside of an ovary stretched around it is shown. Arrows outline the outer edge of the ovary. The cyst contains thrombus of different density (T) as well as free fluid (F) that is liquid blood.

there are several specific aspects to focus on. Sources of infection may arise from the pelvis and will most frequently include pelvic inflammatory disease, and may also involve abscess formation such as tubo-ovarian abscess (TOA). Pelvic inflammatory disease (PID) affects about 10–15% of women in the United States.[12] As many as 275,000 women are hospitalized annually for PID, with more than 100,000 surgical procedures performed.[13,14] Pus in the pelvis appears as echogenic fluid with mixed echoes. It can be confused with blood that is starting to thicken. Small amounts of fluid are

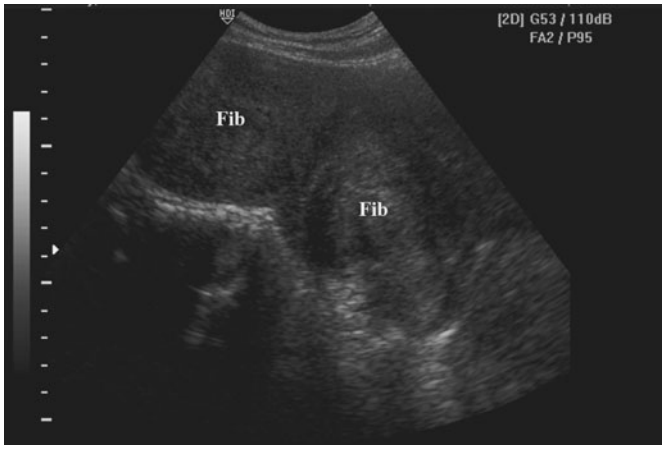


Figure 25.11. A transabdominal longitudinal image of the uterus shows two distinct masses, both fibroids (Fib). Characteristic shadows are cast by both to different degrees. The patient had pelvic pain that was worsening over several weeks.

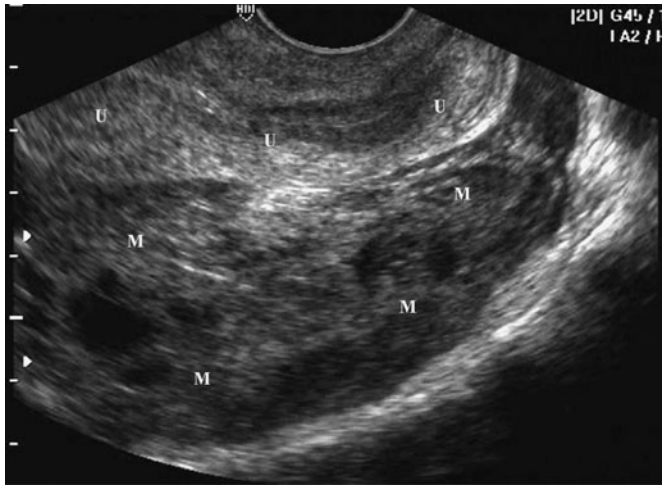


Figure 25.12. The image shows a complex mass (M) from the right adnexa extending behind the uterus (U). The patient required surgical drainage of this TOA.

first seen in the pouch of Douglas and then extend up further along the uterus as the quantity increases. Tubo-ovarian abscesses are often unilateral but occasionally appear as bilateral entities. Ultrasonography is most useful in differentiating TOA from other stages of PID.[15] The most common sonographic findings in PID include thickened, heterogeneous endometrium, an enlarged uterus with fluid in the endometrial cavity, and fluid-filled fallopian tubes.[16,17] A tubo-ovarian complex (TOC) is an inflammatory pelvic mass without any pus within a cavity. A TOC consists of edematous, adherent, infected ovaries and tubes, which can still be visualized but cannot be separated by an endovaginal probe. In TOA, there is a loss of the normal boundaries between the fallopian tube and ovary due to edematous, inflamed tissue that is filled with pus. A variety of sonographic appearances have been described for TOA.[18] The typical sonographic appearance of a TOA is a complex adnexal mass of varying echogenicity with debris, septations, and irregular margins (Figure 25.12).[19–21] The other sonographic markers of TOA are pyosalpinx and loculated or speckled echogenic fluid in cul-de-sac (Figure 25.13).[22–24]

SOURCES OF PAIN

Ovarian cysts, masses, and torsion are likely to be the most common sources of pain encountered. Large fibroids, especially degenerating ones, can cause severe pain and are readily detected on EV ultrasound when small, and on TAS when larger. Typically, larger fibroids cause more pain than smaller ones. Ovarian cysts or

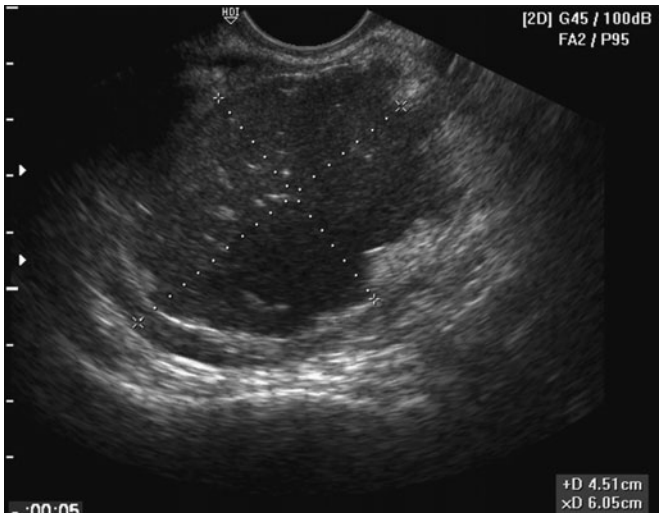


Figure 25.13. This left fallopian tube is dilated and filled with echogenic material. Measurement in the right lower corner shows its size.

masses can cause pain by virtue of mass effect as they grow. In addition, cysts or masses associated with ovaries may occasionally lead to torsion, which will result in severe pain and eventual loss of the ovary and possibly fallopian tube. Typically, a source for torsion is required, and if a sizable cyst or a mass is not present on the ovary or immediately next to an ovary, torsion is unlikely. If a large cyst or a mass is present, then the ovary may be at risk for torsion if clinical symptoms are present. Due to the dual arterial supply of each ovary, both ovarian and uterine arteries, it is easier to rule torsion in than out with ultrasound. If an EV examination with power Doppler shows an absence of blood flow in the ovary containing a large cyst or a mass, then torsion is quite likely. However, if an EV examination shows blood flow in an ovary with a mass or cyst and clinical suspicion is high, laparoscopic evaluation may be necessary to completely rule out torsion. Rupture of a simple cyst may cause intense pain that diminishes in intensity with time and should disappear completely within a day. Endovaginal examination may show a small collection of fluid in the pouch of Douglas and occasionally immediately adjacent to the ovary in question.

References

1. Lambert M, Villa M. Gynecologic ultrasound in emergency medicine. *Emerg Med Clin North Am.* 2004;22:683–696.
2. Kaplan BC, Dart RG, Moskos JM. Ectopic pregnancy: prospective study with improved diagnostic accuracy. *Ann Emerg Med.* 1996;28:10–17.
3. DeVoe RW, Pratt JE. Simultaneous intrauterine and extrauterine pregnancy. *Am J Obstet Gynecol.* 1948;56:1119–1126.
4. Bright DA, Gaupp FB. Heterotopic pregnancy: a reevaluation. *J Am Board Fam Pract.* 1990;3:125–128.
5. Berger MJ, Taymor ML. Simultaneous intrauterine and tubal pregnancies following ovulation induction. *Am J Obstet Gynecol.* 1972;113:812–813.
6. Elson J, Tailor A, Banerjee S, Salim R, Hillaby K, Jurkovic D. Expectant management of tubal ectopic pregnancy: prediction of successful outcome using decision tree analysis. *Ultrasound Obstet Gynecol.* 2004;23:552–556.
7. Yeh HC, Goodman JD, Carr L. Intradecidual sign: a US criterion of early intrauterine pregnancy. *Radiology.* 1986;161:463–467.
8. Dart R, Howard K. Subclassification of indeterminate pelvic ultrasonograms: stratifying the risk of ectopic pregnancy. *Acad Emerg Med.* 1998;5:313–319.
9. Nyberg DA, Hughes MP, Mack LA, Wang KY. Extrauterine findings of ectopic pregnancy of transvaginal US: importance of echogenic fluid. *Radiology.* 1991;178:823–826.
10. Adhikari S, Blaivas M, Lyon M. Diagnosis and management of ectopic pregnancy using bedside transvaginal ultrasonography in the ED: a 2-year experience. *Am J Emerg Med.* 2007;25:591–596.
11. Blaivas M, Lyon M. Reliability of adnexal mass mobility in distinguishing possible ectopic pregnancy from corpus luteum cysts. *J Ultrasound Med.* 2005;24:599–603.
12. Gjelland K, Ekerhovd E, Granberg S. Transvaginal ultrasound-guided aspiration for treatment of tuboovarian abscess: a study of 302 cases. *Am J Obstet Gynecol.* 2005;193:1323–1330.
13. Policy Guidelines for Prevention and Management of Pelvic Inflammatory Disease. U S Department of Health and Human Services, Public Health service, Centers for Disease Control, National Center for Prevention Services, From MMWR 1991;Vol, 40 (RR5).
14. Pastorek JG. Pelvic inflammatory disease and tuboovarian abscess. *Obstet Gynecol Clin North Am.* 1989;16:347–361.
15. Timor-Tritsch IE, Lerner JP, Monteagudo A, Murphy KE, Heller DS. Transvaginal sonographic markers of tubal inflammatory disease. *Ultrasound Obstet Gynecol.* 1998;12:56–66.
16. Ignacio EA, Hill MC. Ultrasound of the acute female pelvis. *Ultrasound Q.* 2003;19:86–98.
17. Horrow MM. Ultrasound of pelvic inflammatory disease. *Ultrasound Q.* 2004;20:171–179.
18. Timor-Tritsch IE, Lerner JP, Monteagudo A, Murphy KE, Heller DS. Transvaginal sonographic markers of tubal

inflammatory disease. *Ultrasound Obstet Gynecol.* 1998;12: 56–66.

19. Timor-Tritsch IE, Lerner JP, Monteagudo A, Murphy KE, Heller DS. Transvaginal sonographic markers of tubal inflammatory disease. *Ultrasound Obstet Gynecol.* 1998;12: 56–66.
20. Ignacio EA, Hill MC. Ultrasound of the acute female pelvis. *Ultrasound Q.* 2003;19:86–98.
21. Webb EM, Green GE, Scoutt LM. Adnexal mass with pelvic pain. *Radiol Clin N Am.* 2004;42:329–348.
22. Timor-Tritsch IE, Lerner JP, Monteagudo A, Murphy KE, Heller DS. Transvaginal sonographic markers of tubal inflammatory disease. *Ultrasound Obstet Gynecol.* 1998;12: 56–66.
23. Ignacio EA, Hill MC. Ultrasound of the acute female pelvis. *Ultrasound Q.* 2003;19:86–98.
24. Varras M, Polyzos D, Perouli E, Noti P, Pantazis I, Akrivis Ch. Tubo-ovarian abscesses: spectrum of sonographic findings with surgical and pathological correlations. *Clin Exp Obstet Gynecol.* 2003;30:117–121.

This page intentionally left blank

CHAPTER 26

Ultrasound Evaluation of the Peripheral Vascular System

James E. Foster, II and Kevin Wiseman

INTRODUCTION

Duplex ultrasound examination of the peripheral arterial and venous systems has been refined to the point where it has become the initial modality of choice for vascular diagnosis. Technical advances have improved diagnostic accuracy such that treatment decisions previously based on angiographic studies can now be based solely on noninvasive studies. This is most evident in the noninvasive diagnosis of deep venous thrombosis (DVT),[1] and is becoming more prevalent in the management of carotid occlusive disease and atherosclerotic peripheral vascular disease.

ULTRASOUND EXAMINATION OF THE PERIPHERAL VENOUS SYSTEM

Risk factors associated with the development of DVT are common in the critical care setting. Virchow's triad of stasis, endothelial injury, and altered coagulation are readily seen in today's intensive care unit (ICU). Clinical factors such as major trauma, which include neurological injury, pelvic and long bone fractures[2,3]; prolonged immobilization due to altered mental status, paralysis, morbid obesity; multiple sites for venous access and central monitoring; and advancing age, all contribute to this increased risk.[4]

The true prevalence of acute DVT in the ICU setting is unknown. Reported incidence varies widely (4–60%) due to patient population, detection methods, and the application of surveillance programs.[5–7] Despite increased awareness and aggressive application of protocols for the prevention of DVT, postmortem studies indicate that subclinical, undetected DVT and pulmonary embolism (PE) continue to exist.[8] In addition, many ICU patients are at risk for rebleeding and are not candidates for anticoagulation.

Invasive hemodynamic monitoring or prolonged central venous access is common in the critical care environment. Catheter-associated thrombosis occurs in response to endothelial injury and the alterations in normal venous flow patterns caused by the catheter. This may be more significant in children, where small diameter veins can be functionally occluded by catheterization.[9]

Although the most common sequelae of DVT are the late problems of venous insufficiency and stasis ulceration, PE is the primary concern in the acute care setting. The present emphasis on DVT prophylaxis arises from the recognition that PE is one of the most preventable causes of death and major morbidity in hospitalized patients. Because most clinically significant PE arises from deep veins of the lower extremities, some centers have advocated routine duplex ultrasound surveillance of patients during their ICU stay.

Continuous-wave Doppler ultrasound technology was introduced to clinical practice in the 1970s. Although no images were possible, these devices allowed the examiner to assess venous flow patterns by auditory waveform analysis. The combination of ultrasound imaging and Doppler spectral analysis provided the basis for current duplex ultrasound technology. By the early 1990s, the venous duplex ultrasound examination replaced contrast venography as the gold standard for the diagnosis of DVT.

Standard practice requires a trained sonographer to transport the ultrasound machine (portable but bulky) to the ICU, where a full lower extremity examination is performed and recorded on videotape or digital media. The study is reviewed by the interpreting physician who generates a report that is transcribed and returned to the patient's chart. This process, although very accurate, is time consuming and may not always serve the needs of the very dynamic and often unstable conditions in the critical care environment.

The recently developed portable, handheld, duplex scanners with multihertz transducers and color-flow Doppler capability bring the possibility of a focused venous examination to the bedside. The clinician is now able to obtain diagnostic information rapidly and interpret these results within the context of the patient's overall clinical condition. Bedside investigation

can eliminate the need for patient transport to the ultrasound department or the computerized tomography (CT) scanner, often an enormous task with critically ill patients that itself has inherent risks. Where results are uncertain or equivocal, a formal diagnostic study can be obtained to assist in a definitive diagnosis. With proper training and experience, a focused venous duplex examination at the bedside can be accomplished by any clinician familiar with venous anatomy, venous flow characteristics, and the basics of duplex ultrasonography.

Venous Anatomy

The venous systems of both the upper and lower extremities are divided into deep and superficial components. The deep venous system is composed of those veins draining the muscle compartments and paired with named arteries. The superficial systems drain cutaneous structures and run in the subcutaneous space. These superficial veins are not associated with adjacent arteries.

In the lower extremity, the deep venous system includes the external iliac, common femoral, superficial and deep femoral, popliteal, anterior and posterior tibial, peroneal, and soleal and gastrocnemius veins (Figure 26.1). All of the deep veins are accompanied by named arteries, except for the soleal and gastrocnemius veins. The two main veins of the superficial venous system are the greater and lesser saphenous veins. The lesser saphenous is located in the lateral calf and drains into the deep system at the popliteal vein. The greater saphenous vein runs along the medial aspect of the leg from ankle to proximal thigh, where it traverses the fossa ovalis and drains into the common femoral vein. An unfortunate consequence of the traditional anatomic nomenclature is that the "superficial" femoral vein is actually a deep venous structure and is often the site of acute DVT leading to all the complications of thrombosis including pulmonary embolism. Therefore, it is important to understand that thrombosis of any segment of the femoral vein (superficial, deep, or common) constitutes a deep venous thrombosis and should be so identified for documentation and treatment purposes.

In the upper extremity, the deep veins include the internal jugular, subclavian, axillary, brachial, radial, and ulnar veins (Figure 26.2). These veins each have a companion artery. The major components of the superficial venous system of the arm are the cephalic vein, running laterally from wrist to shoulder and draining into the subclavian vein, and the basilic vein, running medially from antecubital fossa to the axilla, where it drains into the axillary vein. When available, the superficial veins are the sites of choice for peripheral venous access.

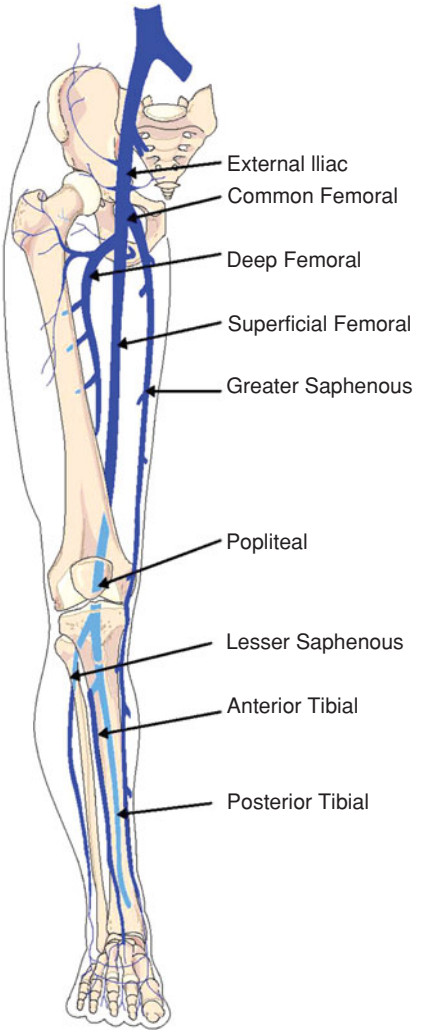


Figure 26.1. Venous anatomy of the lower extremity. (Available at: http://www.google.com/imgres?imgurl=http://www.icvein.com/images/perforators-image.jpg&imgrefurl=http://www.icvein.com/venous-disease-varicose-vein-treatment-iow-city-ia.htm&usg=pC-NUcynW8Bg79T56G2trGjuVxl=&h=384&w=308&sz=41&hl=en&start=4&tbnid=LVqqafQInoStm:&tbnh=123&tbnw=99&prev=/images%3Fq%3Dlower%2Bextremity%2Bvenous%2Banatomy%2Bpictures%26hl%3Den%26sa%DG. Accessed On March 29th, 2009.)

Sonographic imaging is performed with B-mode scanning, often using color-flow imaging to add more information. The external iliac artery and vein exit the pelvis deep to the inguinal ligament to become common femoral vessels. The common femoral vein is medial to the artery and is slightly larger in diameter. These relationships are easily identified when scanning in a transverse plane at the groin crease (Figure 26.3).

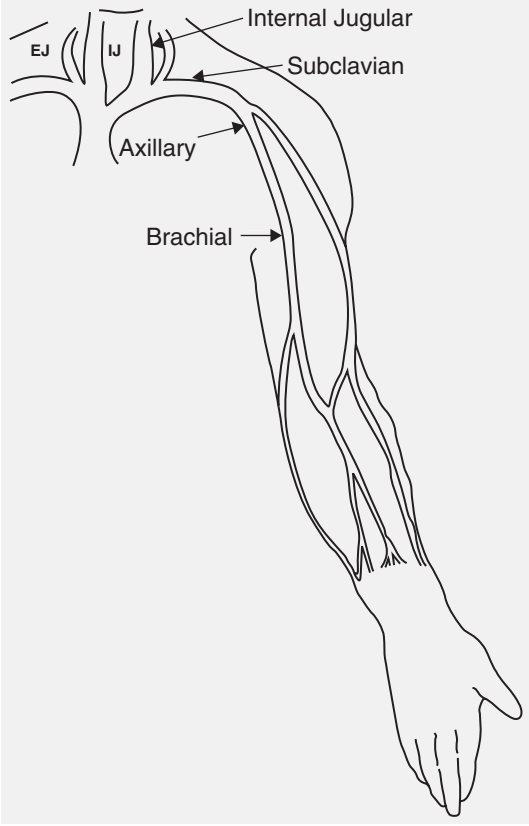


Figure 26.2. Venous anatomy of the upper extremity.

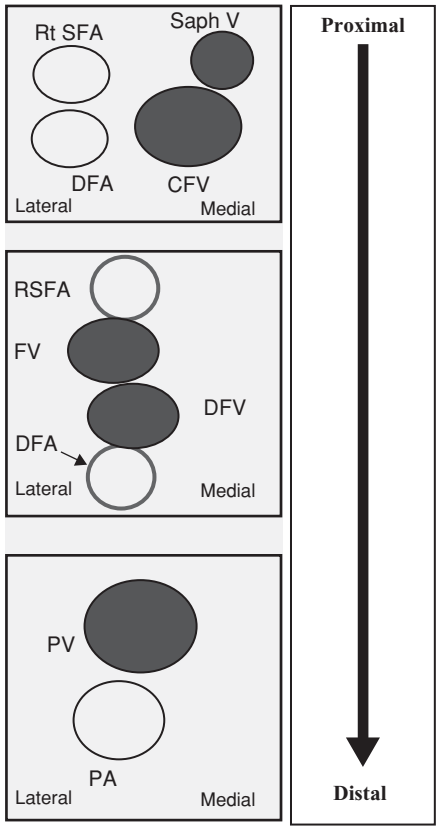


Figure 26.4. Diagrams of vessel relationships at three levels.

The transverse plane is used to assess the veins for compressibility (see below). Moving distally along the common femoral vein, the greater saphenous can be identified as it passes from the superficial plane to drain into the femoral vein. Again, no artery accompanies the greater saphenous vein. Moving slightly more distally, common femoral vessels divide into superficial and deep femoral vessels. At this level, four vessels are seen in cross-section (Figure 26.4). From this point distally, the superficial femoral artery (SFA) and vein continue to the adductor canal, where they enter the popliteal space and are designated as popliteal vessels (Figure 26.1).

Once the distal external iliac vein is identified at the inguinal ligament, turning the transducer 90° provides a longitudinal image of the vessel. This view is best suited for a rapid survey of the veins and allows for assessment of the extent and nature of a thrombus when seen (Figure 26.5). Vein compression is not reliable

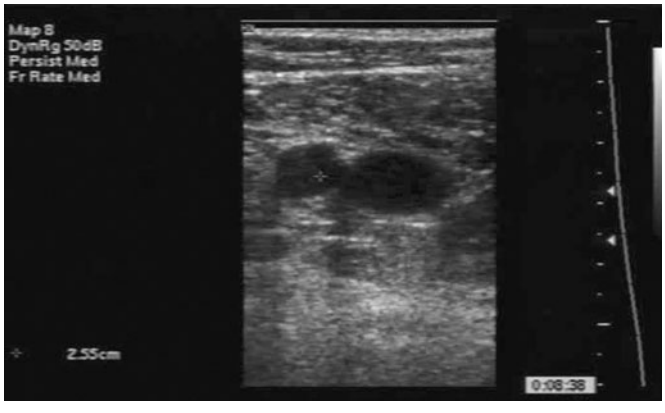


Figure 26.3. Transverse view of common femoral vessels.

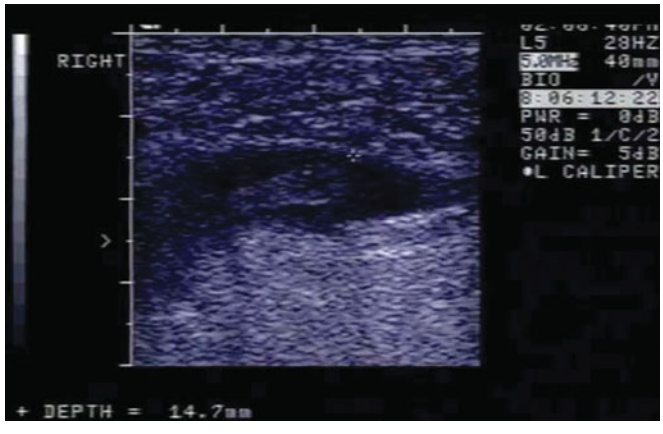


Figure 26.5. Acute deep venous thrombosis (DVT), common femoral vein (longitudinal view).

while scanning in the longitudinal plane. Compressibility must be confirmed in the transverse view.

Diagnostic Criteria

Diagnostic criteria for identifying DVT can be divided into vessel characteristics and flow characteristics. The primary vessel characteristic is compressibility; the ability to demonstrate wall-to-wall apposition of the vein when adequate pressure is applied using the ultrasound transducer in the transverse plane. Adequate pressure is determined by noting mild deformation of the adjacent artery (Figure 26.6). Noncompressiblity indicates that intraluminal thrombus is preventing the vessel walls from collapsing. It is important to remember that fresh, immature thrombus may not be echogenic because newly formed clot has an acoustic impedance similar to blood. The second vessel characteristic is identification of intraluminal echogenic material. This can often be seen on the initial survey of the venous system and should alert the clinician to the presence of thrombus. Intraluminal echogenic material should be confirmed in both imaging planes, and when thrombus is identified, compression should be limited due to the possibility of dislodging the clot. Longitudinal imaging provides the best view for determining the extent or length of thrombus and whether it is adherent to the vessel wall or may have a free floating tip (Figure 26.7). A third vessel characteristic that may be helpful is the assessment of valve function. Occasionally, venous valves are visible in situations where higher frequency transducers can be used (thin patients, children). Normal valves open and close in conjunction with venous flow. However, because valve cusps are often the site of thrombogenesis, an immobile valve cusp may be a clue to the presence of thrombus.

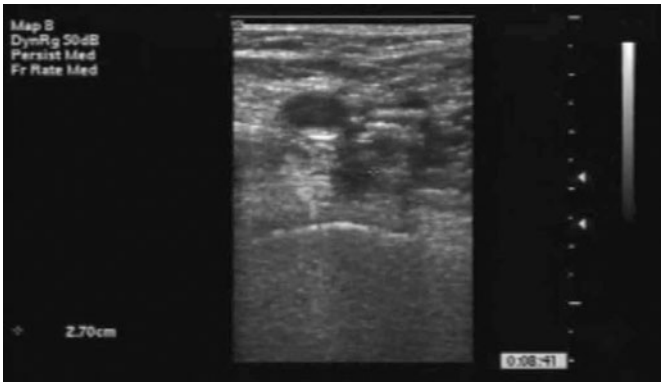


Figure 26.6. Common femoral artery and compressed common femoral vein (compare with Figure 26.3).

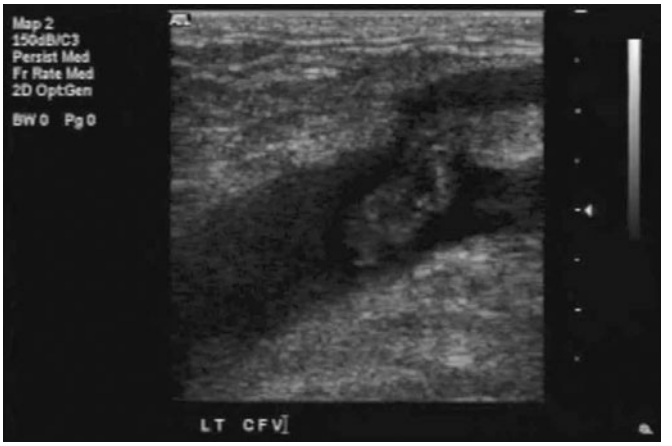


Figure 26.7. Free-floating thrombus in extending from greater saphenous vein into common femoral vein.

Venous blood flow characteristics are also important in assessing the presence of acute DVT. Normal venous flow patterns show a phasicity that varies with respiration. Normal inspirations decrease intrathoracic pressures and cause associated increase in venous flow. Similarly, expiration increases intrathoracic pressure and is reflected in a decrease in venous flow. A Valsalva maneuver increases intrathoracic pressure sufficiently to completely interrupt venous flow that is associated with an augmentation in venous flow when the Valsalva maneuver is released. These changes are easily identified by observing the Doppler waveform or by listening to flow patterns with a continuous-wave Doppler unit. Any obstructive process between the thoracic cavity and the site of insonation of the veins of the lower extremity can alter the normal phasic changes associated with respiration. Absence of phasic changes with a continuous-flow pattern or loss of augmentation with deep inspiration suggest obstruction of the venous system. Additional maneuvers to augment flow include compression of calf muscles and the distal thigh to demonstrate increased flow at the site of insonation. Loss of normal augmentation with compressions also suggests the presence of obstruction in the venous system. The assessment of both vessel characteristics and venous blood flow characteristics lead to highly accurate and reliable detection of DVT. Normal vessel and flow characteristics also provide a very high negative predictive value for DVT.

Performing the Studies

Equipment requirements for venous duplex examinations include high-resolution gray-scale imaging, color Doppler capability, and spectral analysis directional

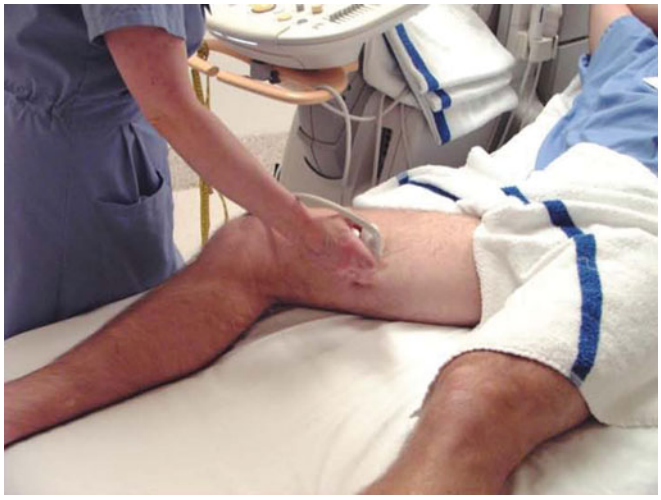


Figure 26.8. Patient position for lower extremity venous examination.

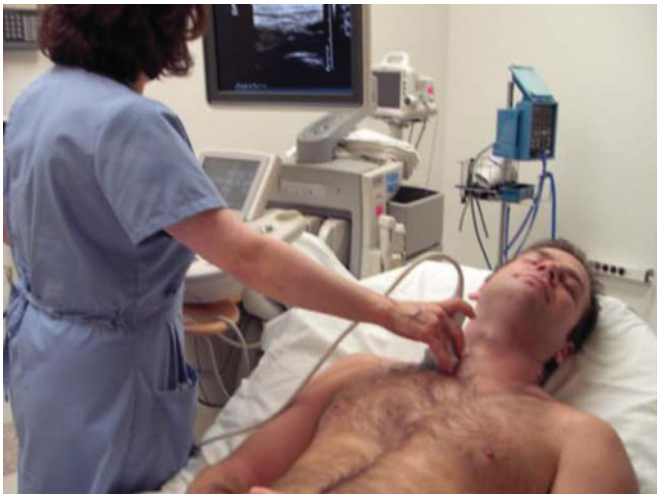


Figure 26.9. Patient position for upper extremity venous examination.

Doppler. The transducer selection should allow for optimal imaging and Doppler analysis, and generally means using the linear-array, high-frequency (7–10 MHz) transducer for most patients. Occasionally, a low-frequency (3–5 MHz), curved-array transducer will be necessary if the patient is obese or there is considerable edema.

Patient positioning is very important, as proper positioning enhances the demonstration of abnormalities, eases the strain on the sonographer, and reduces time to complete the study. For lower extremity examinations, the patient is placed supine with the head elevated approximately 30°, if possible. The leg to be scanned is externally rotated with the knee flexed (Figure 26.8). For upper extremity studies, the patient is also placed supine, with the head turned away from the side being scanned. The chin should be raised (neck extended) slightly, if possible (Figure 26.9).

The first component of the lower extremity protocol involves transverse compressions in gray-scale imaging mode. Beginning at the femoral crease, with the transducer in a transverse (cross-section) orientation, identify the common femoral vein. There should be one artery and the sapheno-femoral junction should be visible. Using gentle-to-moderate probe pressure applied toward the femur, observe the femoral vein compression with wall-to-wall contact. Identification of intraluminal echogenic material mandates gentle pressure only. Visualization of a mobile thrombus within the vein precludes further compression to avoid dislodging an embolus. If compressions are easy and complete, the probe can be moved a few centimeters distally, and the division of the common femoral vein into the deep and superficial femoral veins can be observed. Repeat the compression maneuver every 5 centimeters (cm), proceeding distally along the superficial femoral vein. At the distal thigh, the vein traverses the adductor canal and enters the popliteal space. At this point, position the transducer in the popliteal space, identify the popliteal artery and vein, and compress the vein to assure wall-to-wall contact.

The second phase of the lower extremity venous examination evaluates flow characteristics using color Doppler imaging and Doppler spectral analysis. The transducer is returned to the groin and the common femoral vein is again identified in the transverse view. The transducer is then rotated 90° to obtain a longitudinal (sagittal) image of the vein (Figure 26.10). Observe the Doppler signal for flow that is spontaneous and phasic with respiration. A Valsalva maneuver will halt flow at end inspiration and will demonstrate augmented

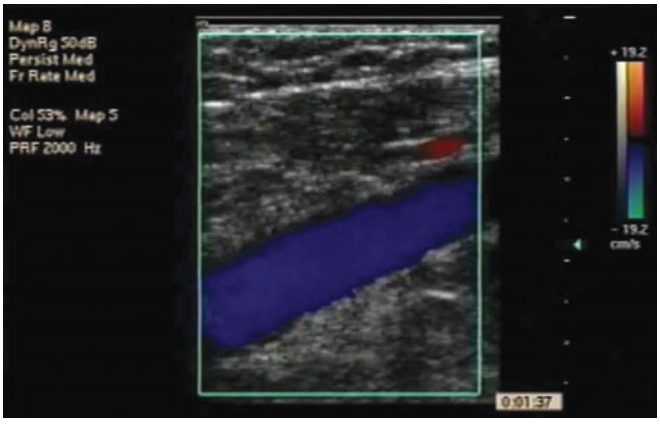


Figure 26.10. Normal color-flow image of common femoral vein. Note: direction of flow is away from transducer.

flow when respiration resumes. Flow can also be augmented by squeezing the calf muscles. This should produce a spike in the spectral signal. These observations are made in the superficial femoral and popliteal veins to complete the study.

A similar protocol is used to evaluate the upper extremities. Transverse compressions of the internal jugular, axillary, and brachial veins should be demonstrated. The subclavian vein cannot be compressed because of the clavicle. Therefore, color Doppler and spectral analysis are used to obtain signals from the subclavian vein and its confluence with the internal jugular vein. Flow in the subclavian and internal jugular should be spontaneous and somewhat pulsatile. These indirect findings confirm the patency of the brachiocephalic veins and the superior vena cava, which cannot be routinely imaged directly on the Duplex examination.

Interpretation of Results

Table 26.1 shows the sensitivity, specificity, positive and negative predictive values for the vessel, and flow characteristics assessed in the venous study.[10] It is readily seen that a combination of noncompressible veins with intraluminal echogenic material and abnormal flow characteristics is diagnostic for DVT. The absence of these abnormal findings correlates with no venous pathology.

The venous duplex study is straightforward and easily accomplished at the bedside. But besides the information on venous pathology, a variety of additional information can be obtained while performing these studies. Associated arterial disease is often recognized because adjacent arteries are easily viewed with the same modalities. Atherosclerosis, arterial injury, pseudoaneurysm, or arterial embolization may be evident and are described more fully below. Flow abnormalities may be a clue to increased central venous pressures or valvular heart abnormalities. Mass effects with extrinsic compression may show altered flow patterns, with no evidence of intraluminal pathology. Central venous catheters are easily recognized and may demonstrate catheter-associated thrombus. The internal jugular exam may suggest associated thyroid abnormalities.

The availability of high-quality portable bedside duplex scanners has advanced the ability to accurately diagnose acute DVT within the capability of every physician dealing with critically ill patients. The education and skills required to become comfortable with the techniques are basically extensions of the physical examination skills. And it is almost axiomatic that the individual who is best able to interpret the findings is the one with daily responsibility for the care of the patient and who can correlate the results within that individual patient's clinical context.

TABLE 26.1. Diagnostic value of venous Duplex parameters for diagnosis of acute DVT

Criteria	*Sensitivity*	*Specificity*	*PPV*	*NPV*
Thrombus seen	50	92	95	37
Incompressible	79	67	88	50
No spontaneous flow	76	100	100	57
Absent phasic flow	92	92	97	79

DVT indicates deep venous thrombosis; NPV, negative predictive value; PPV, positive predictive value.

ULTRASOUND EXAMINATION OF THE PERIPHERAL ARTERIAL SYSTEM

The most common application of ultrasound in the evaluation of the arterial system is to assess the presence and degree of atherosclerotic peripheral vascular disease. Atherosclerotic plaque and calcification of arterial walls are easily demonstrated by gray-scale imaging. Duplex imaging and Doppler waveform analysis provide reliable information related to flow characteristics and arterial stenosis.

Perhaps the most common ultrasound-based assessment is the ankle-brachial ratio (ABI), which utilizes continuous-wave Doppler instruments to identify arterial flow, while blood pressures are obtained to calculate the index. Although the baseline assessment of peripheral vascular disease is valuable, in the ICU setting screening ultrasound examinations are most often directed to determining the presence or absence of flow and indications of arterial injury such as arterial dissection, pseudoaneurysm, or arteriovenous fistula. Diagnosis of these problems is based on the accurate assessment of flow characteristics and requires a basic understanding of arterial anatomy, hemodynamics, and the fundamentals of pulsed Doppler ultrasound to optimize results. Because bedside arterial examinations may be technically challenging, suspected abnormalities are best confirmed by formal complete sonographic examination or by angiographic modalities.

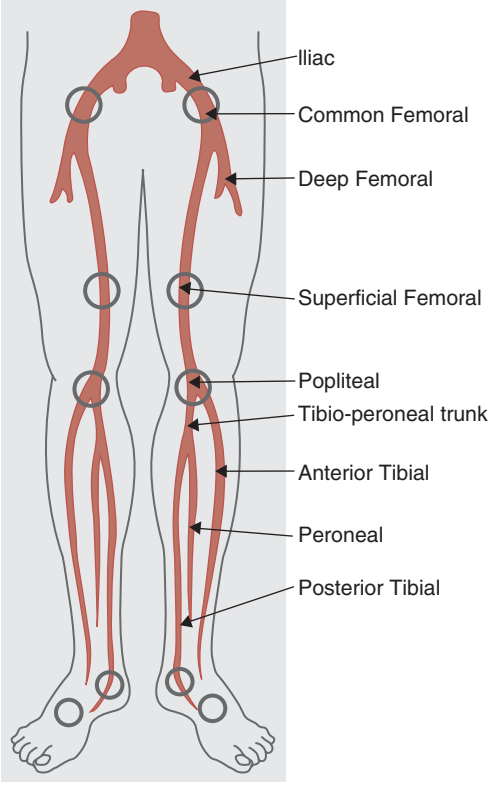


Figure 26.11. Arterial anatomy of the lower extremity.

Arterial anatomy of the upper extremity is consistent and generally accessible to ultrasound examination. Examination of the great vessels is best performed with a transesophageal approach (see Chapter 8). Peripheral vessels amenable to ultrasound evaluation include the carotid and vertebral arteries supplying the head, face and brain, and the subclavian, axillary, brachial, radial, and ulnar arteries in the upper extremities. The arterial supply of the lower extremities consists of the external iliac vessels, the common, deep, and superficial femoral artery in the thigh, the popliteal artery and its branches—the anterior and posterior tibial arteries along with the peroneal artery (Figure 26.11).

The common carotid arteries are readily identified in the cervical region, lateral to the trachea and deep to the internal jugular veins. The right common carotid arises from the innominate artery. The left common carotid arises directly from the aortic arch. At the level of the larynx, the common carotid bifurcates into the internal and external carotid vessels. The external is more medial, has multiple branches, and supplies the face and scalp. The internal carotid is lateral, has no branches in the neck, and supplies the brain.

Normal peripheral arterial hemodynamics are characterized by laminar flow in a high-resistance system

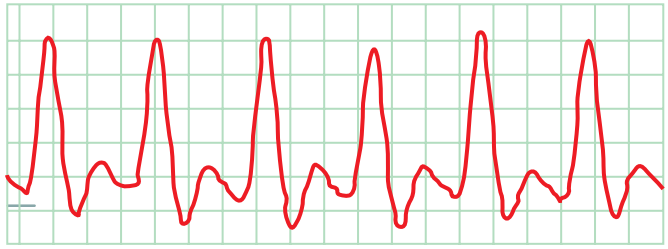


Figure 26.12. Normal triphasic waveform of a peripheral artery.

that generates a characteristic triphasic waveform (Figure 26.12). The initial forward flow is generated by ventricular systole (phase 1). The second phase is a short period of reversed flow that occurs as the aortic valve closes. Phase 3 reflects forward flow generated by the elastic recoil of normal arterial walls. The normal triphasic waveform is easily recognized by Doppler spectral analysis or color-flow imaging. In addition, the characteristic auditory signal is easily recognized when using continuous-wave Doppler instruments without imaging capability. Arterial flow in low-resistance systems such as the internal carotid and vertebral arteries show characteristic changes in the waveform due to the requirement for continuous forward flow to high-demand organs such as the brain. Low-resistance waveforms show forward flow during systole and maintain forward flow through the entire cardiac cycle with no phase 2 flow reversal. These characteristics are helpful in distinguishing internal carotid artery (low-resistance) from the external carotid artery (high-resistance).

Various disease processes produce changes in the flow patterns that form the basis for duplex evaluation and diagnosis. Medial calcinosis of peripheral arteries seen in diabetes leads to decreased vascular elasticity and a loss of phase 3 of the normal arterial waveform. Atherosclerosis can cause similar changes in its early stages. As plaque deposition increases, normal laminar flow becomes more turbulent, leading to spectral broadening seen on ultrasound examination. As stenosis progresses, flow velocities and turbulence increase. Turbulent flow is associated with spectral broadening and generates audible bruits. The ultrasound manifestation of the high-grade stenosis is a Doppler artifact known as “aliasing” (see Chapter 2). Aliasing occurs when the flow velocity exceeds the ability of the pulsed Doppler signal to accurately describe the flow rate (see Chapter 2). The duplex instrument falsely generates an opposite image or color map of the arterial flow. Aliasing is beneficial in quickly directing the sonographer to a point of maximum stenosis. When aliasing is identified, the sonographer should change instrument

settings to eliminate the artifact. Only after appropriate adjustments can accurate flow information be obtained. Critical degrees of arterial stenosis eventually lead to low flow states and occlusion, often as result of thrombosis. Arterial occlusion is characterized by the absence of a Doppler signal or color-flow signal in the vessel distal to the point of obstruction. Duplex findings consistent with arterial occlusion should be confirmed by some angiographic technique, as a "string sign" with extremely low flow can be missed by ultrasound. In addition, embolic events can lead to acute arterial occlusion that can be recognized by duplex examination. Emboli to the carotid arteries can be a cause of stroke, and in the extremities lead to acute arterial insufficiency. These events require a search for the source including mural thrombi from the heart, large vessel aneurysms, and possible paradoxical emboli originating in the venous system and traversing an intracardiac shunt.[11] All of these etiologies can be evaluated using various sonographic techniques.

Arterial injuries also have characteristic ultrasound findings. Aortic dissection is the most common and may be best evaluated by transesophageal echocardiography (TEE) (see Chapter 8). The increase in cervical trauma related to shoulder belt injuries from motor vehicle collisions has also raised the awareness of carotid artery dissection. These injuries are characterized by altered flow in the affected artery. Gray-scale imaging sometimes demonstrates an intraarterial echogenic line that represents the separated intima. Doppler or color-flow analysis may show different flow patterns on either side of the septum.[12,13] Additional imaging with magnetic resonance (MR) angiography or CT angiography may be warranted to confirm the ultrasound findings.[14]

Advances in percutaneous intravascular procedures from coronary angiography to intravascular stent placement have led to an increased incidence of arterial injuries and pseudoaneurysm formation. Iatrogenic femoral artery pseudoaneurysm is reported to occur in up to 6% of diagnostic and therapeutic catheterizations.[15] These pseudoaneurysms can be recognized as extravascular fluid collections that communicate with the arterial system by a narrow tract. Duplex examination demonstrates high-flow velocity with marked turbulence in the tract and a swirling, turbulent flow pattern within the pseudoaneurysm itself (Figures 26.13 and 26.14). Current treatment options include ultrasound-guided thrombin injection.[16] Care must be taken to avoid delivery of thrombin to the native artery. Repeat injections can be performed for persistent or recurrent pseudoaneurysms. Arteriovenous (AV) fistula can result from blunt or penetrating trauma or attempts at vascular access for a variety of purposes. Arteriovenous fistula at the peripheral level changes a high-resistance system to a low-resistance system. As such, the resulting changes in sonographic flow patterns are predictable. Marked turbulence is identified at the site of the abnormal communication. The presence of aliasing artifact is helpful in identifying the site. The arterial waveform takes on the low-resistance characteristic of higher-end diastolic flow. The flow pattern in the adjacent involved vein will be "arterialized," reflecting the pulsatile waveform of the high-pressure artery.

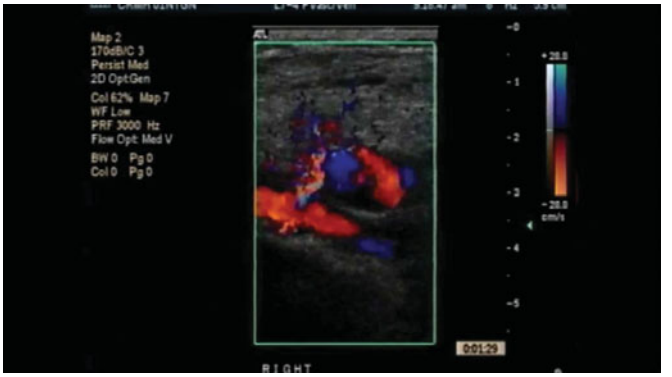


Figure 26.13. Common femoral artery pseudoaneurysm.

Performing the Studies

Carotid Duplex Examination

Equipment: Ultrasound system with high-resolution gray-scale imaging, color Doppler capability, and spectral analysis directional Doppler for velocity measurements.

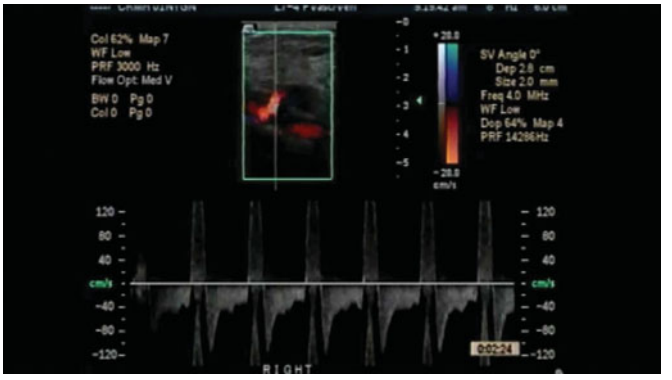


Figure 26.14. High-velocity, turbulent flow seen in tract of pseudoaneurysm.

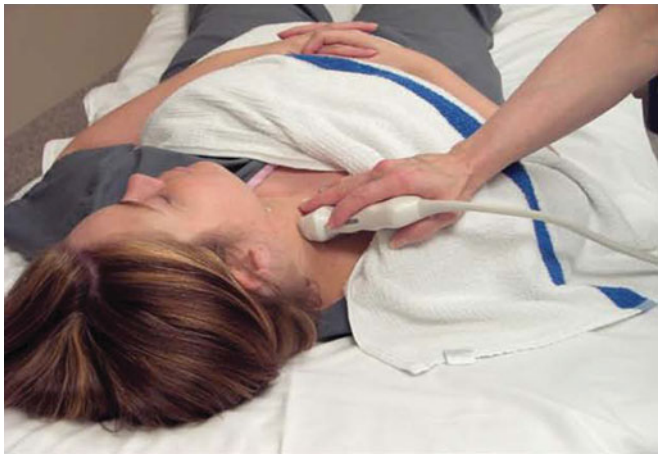


Figure 26.15. Patient position for carotid arterial examination.

Transducer: Linear-array, high-frequency transducer (7–10 MHz), with the occasional use of curved-array transducer (3–5 MHz) for deep vessels.

The patient is placed supine, with the head elevated slightly without a pillow and turned away from the side of the examination; the chin should be raised slightly. This approach helps to extend the neck. The patient's head position can and should be altered during the examination to obtain the best image (Figure 26.15).

Examination Protocol

Step 1: Gray-Scale

Examine the carotid arteries in at least two long-axis views and one transverse view. Identify the bifurcation of the common carotid into internal and external carotid arteries. Document the extent and severity of plaque formation (location, characteristics, luminal reduction).

Step 2: Color Doppler

Examine the carotid arteries in a transverse plane with color Doppler to demonstrate the patency of the vessels and to determine the flow disturbances caused by plaque formation or other abnormalities.

Step 3: Spectral Analysis

Obtain a velocity spectrum from the following locations:

- Subclavian artery (high-resistance waveform)
- Proximal and distal common carotid artery
- Proximal external carotid artery (high-resistance waveform)
- Proximal internal carotid artery (low-resistance waveform)
- Vertebral artery: the vertebral is found by rotating the transducer laterally and identifying the vessel between the transverse processes of the cervical vertebrae.

Note: All velocity recordings should be made with an angle of insonation of 60° or less, parallel to the vessel wall, and in the center of the vessel where flow is highest. Obtain a velocity spectrum at the level of any stenosis as well as immediately distal to the lesion to assess flow disturbance. The aliasing artifact can be very helpful at this time in identifying the areas of maximum flow velocity/stenosis.

Arterial Duplex Examination of the Lower Extremities

Equipment: Ultrasound system with high-resolution, gray-scale imaging, color Doppler capability, and spectral analysis directional Doppler for velocity measurements.

Transducer: Linear-array, high-frequency transducer (7–10 MHz), with the occasional use of a curved-array transducer (3–5 MHz) for deep vessels.

Patient Positioning: Supine, with head slightly elevated. Examined leg is flexed at the knee and externally rotated.

Examination Protocol

Step 1: Gray-Scale

Beginning in the groin with a transverse probe position, identify the common femoral artery (CFA). Rotate to a longitudinal probe position in line with the CFA. Follow the CFA distally to identify the bifurcation into profunda and superficial femoral arteries (SFA). Continue to follow the SFA to the distal thigh. Change probe position to the popliteal fossa. Begin again in the transverse probe position to identify the popliteal artery. Rotate to a longitudinal probe position in line with the popliteal artery. All arteries should be examined in gray-scale first to look for acute emboli, plaque formation, or other abnormalities.

Step 2: Color Doppler and Spectral analysis

Repeat step 1 following the CFA, SFA, and popliteal arteries, using color-flow and spectral Doppler modes. Signals should be obtained in the CFA, SFA (proximal, mid, and distal segments), and the popliteal artery.

Note: All velocity recordings should be made with an angle of insonation of 60° or less, parallel to the vessel wall, and in the center of the vessel, where flow is highest. Obtain a velocity spectrum at the level of any stenosis as well as immediately distal to the lesion to assess flow disturbance. The aliasing artifact can be very helpful at this time in identifying the areas of maximum-flow velocity/stenosis.

Examination of the upper extremities follows a similar pattern to that described above for the lower extremities.

Interpreting the Findings

The diagnosis of arterial stenosis depends on elevated flow velocities when seen in the appropriate clinical setting. In high-resistance vessels, a doubling of the flow velocity at a site of stenosis when compared with the arterial flow velocity immediately prior to the suspicious area suggests a hemodynamically significant stenosis when accompanied by characteristics of Doppler spectral broadening and turbulent flow.[17] Such findings may warrant a formal arterial duplex exam for confirmation.

Diagnosis of hemodynamically significant stenosis in the internal carotid artery is somewhat more refined. Peak systolic velocity >145 cm/sec is associated with stenosis >50%. An associated end-diastolic velocity >140 cm/sec identifies the critical stenosis in the range of 80–99%. An additional criterion is the ratio of peak internal carotid artery velocity divided by the peak velocity in the common carotid artery; the internal carotid artery/common carotid artery (ICA/CCA) ratio. A ratio >3.7 helps confirm the critical stenosis. The ICA/CCA ratio can also be helpful when flow in the carotids is restricted, as in the case of associated aortic stenosis or conditions of decreased cardiac output.[10]

Vascular occlusion is identified by complete absence of color-flow or Doppler signal in the examined artery. Intraluminal emboli may be seen on gray-scale imaging (Figures 26.16 and 26.17). Patent collateral vessels may be identified; however, these are not often seen in the acute setting. Suspected internal carotid artery occlusion should be confirmed by another angiographic modality prior to deciding the patient is not a candidate for endarterectomy.

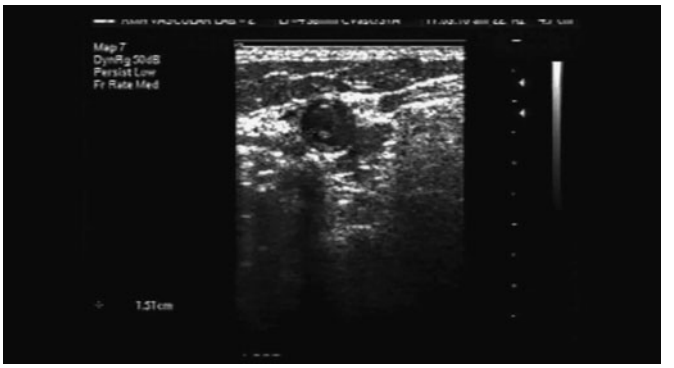


Figure 26.17. Intraarterial (carotid) embolus. Transverse view.

Additional Information

Duplex arterial exams can yield associated information that can be helpful in recognizing pathologic conditions. Generally, reduced arterial flow may be an indication of left ventricular failure (Figure 26.18). Valvular heart disease can be suspected from alterations in the arterial waveforms. Ventricular and valve function can then be evaluated with transthoracic echocardiography. Associated venous disease can be recognized, along with nonvascular problems such as cysts and tumors.

TRANSCRANIAL DOPPLER

The application of transcranial Doppler ultrasound technology to the evaluation of the cerebral circulation was introduced by Aaslid and colleagues in 1982.[18] The initial technique involved a handheld, low-frequency pulsed-wave transducer and relied on waveform analysis and depth of insonation to identify cerebral vessels. With current duplex technology, color-flow imaging serves as an adjunct to vessel location,

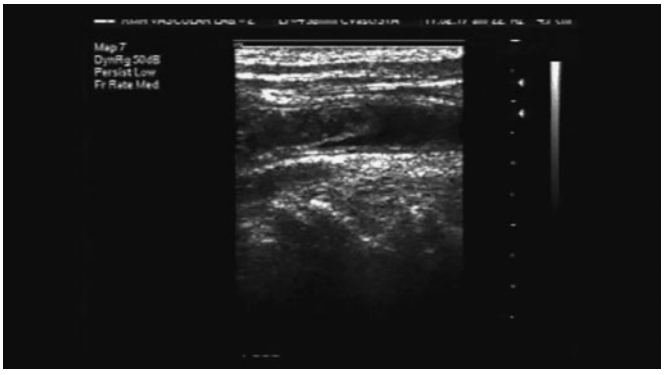


Figure 26.16. Intraarterial (carotid) embolus. Longitudinal view.

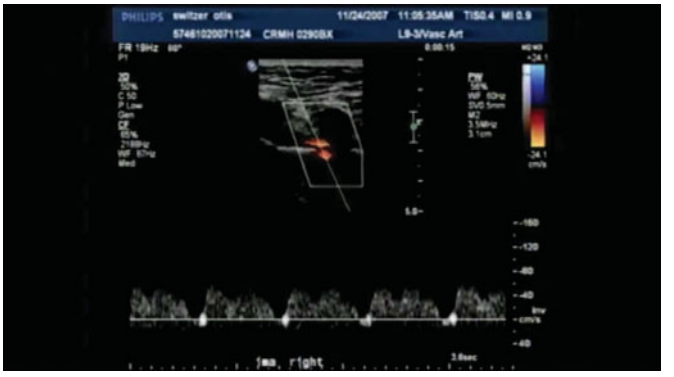


Figure 26.18. Double systole waveform of a patient on intraaortic balloon assistance.

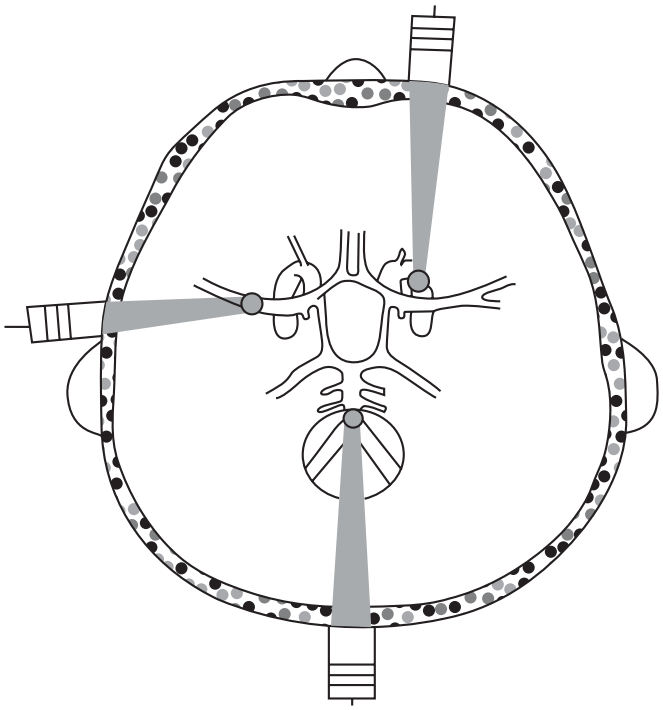


Figure 26.19. Transcranial doppler: acoustic windows.

identification, and flow assessment. Areas of clinical application include assessment of arterial stenosis and occlusion, identification of vascular anomalies, assessment of blood flow in the posterior cerebral circulation, intraoperative monitoring during carotid endarterectomy, assessment of vasospasm in relation to subarachnoid hemorrhage (SAH) and in sickle cell anemia, autoregulation of cerebral blood flow, and as an adjunct in assessment of brain death.[10] Compared with other duplex vascular examinations, transcranial Doppler (TCD) is more technically demanding due to limited acoustic access to the cerebral circulation and requirements to alter power levels and Doppler parameters to insure an optimal study with reliable and reproducible results.

Study protocols are generally standardized. A 2 MHz pulsed-wave signal is used for insonation. Access to the cranial vessels is obtained via any of three common acoustic windows: transtemporal, transforaminal, and transorbital (Figure 26.19). The transtemporal window allows evaluation of the middle cerebral, anterior cerebral, and posterior cerebral arteries (Figure 26.20).

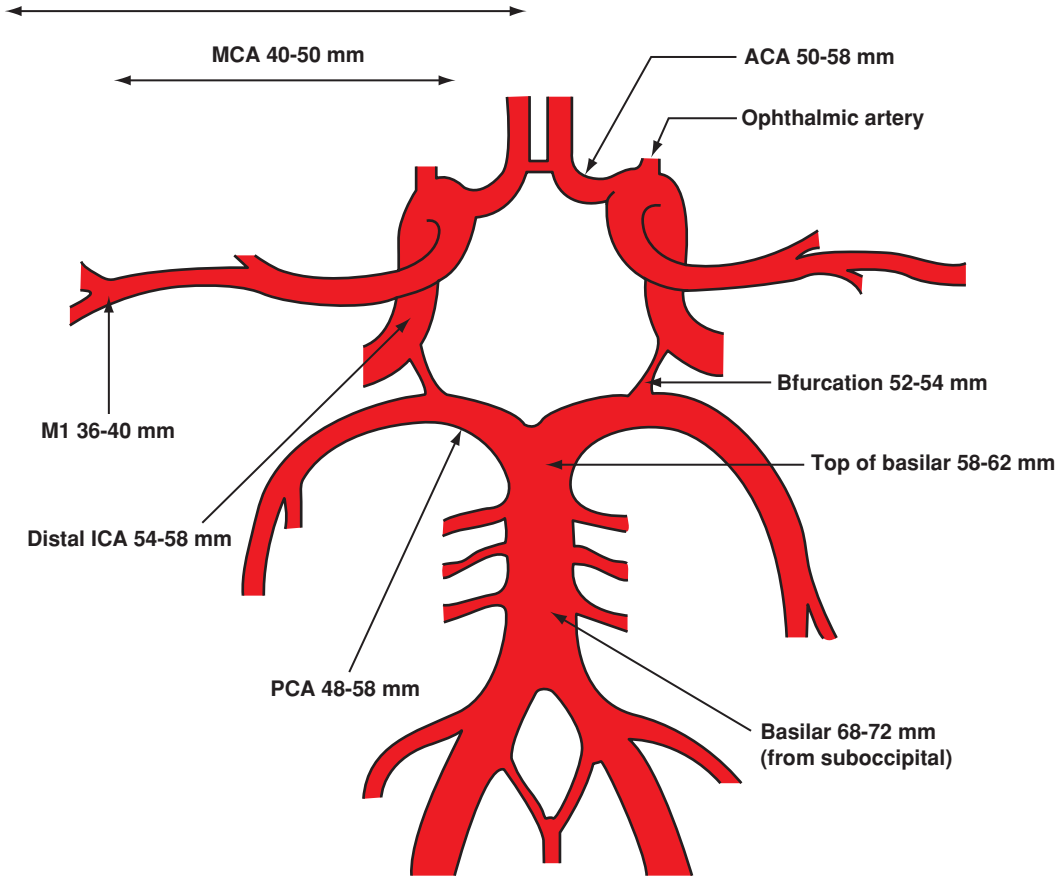


Figure 26.20. Diagram of circle of Willis with depths of insonation from ipsilateral transtemporal window.

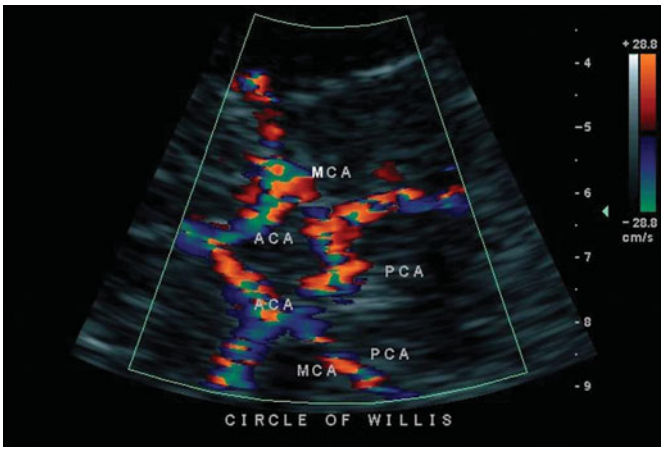


Figure 26.21. Transcranial color image of the circle of Willis. ACA indicates anterior cerebral artery; MCA, middle cerebral artery; PCA, posterior cerebral artery.

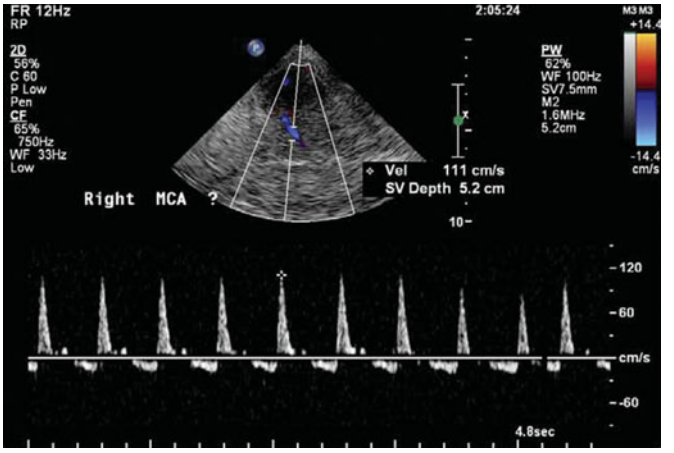


Figure 26.22. Abnormal middle cerebral artery (MCA) waveform seen in brain death. Note the high-resistance pattern with end-diastolic flow reversal.

Ipsilateral and contralateral vessels may be insonated from the same window. It should be noted that 8–12% of patients may have inadequate acoustic windows.[19] The transforaminal window allows access to the vertebral and basal arteries via the foramen magnum. The transorbital window is used to insonate the ophthalmic artery and the carotid siphon. Care must be exercised to reduce power settings to prevent adverse bioeffects to the eye. Several parameters are combined to confirm the vessel being insonated. These include depth of the sample volume in relation to the acoustic window being utilized; orientation of the transducer; direction of blood flow in relation to the transducer; and relationship of the examined artery to the junction of middle cerebral and internal carotid arteries. Duplex machines add the benefit of color imaging, which can assist in vessel identification and demonstration of the circle of Willis (Figure 26.21). Flow velocities are calculated as time-average means and normal values vary by vessel. Tables of norms are published,[20] and it is suggested that each vascular laboratory validate its own results in comparison with these norms (Table 26.2). Perhaps the most significant application in the ICU setting is monitoring middle cerebral artery (MCA) vasospasm following SAH. In a recent review, Springborg et al. reported a high sensitivity and specificity for serial TCD studies when compared to arteriography.[21] Sharp increases in MCA flow velocities when compared with baseline values predicted the development of MCA vasospasm. The role of TCD in the diagnosis of brain death remains ill defined. Several patterns of altered waveforms have been described, but the most accurate has not been determined (Figures 26.22 and 26.23). Sensitivity >90% and specificity of 100% have been reported.[22] However,

TABLE 26.2. Transcranial Doppler parameters

Artery	*Depth (mm)*	*Mean velocity*	*Direction*	*Peak systolic*	*End diastolic*	*Children mean vel*
MCA	45–65	32–82	toward	63–110	23–52	<170
ACA	62–75	18–82	away	53–93	20–45	<150
ICA siphon	60–64	20–77	bidirectional	—	—	<130
OA	50–62	20 ± 6	toward	38 ± 11	11 ± 4	—
PCA	60–68	16–58	bidirectional	—	—	<100
Basilar	80–100	12–66	away	32–64	12–32	<100
Vertebral	60–80	12–66	away	32–64	12–32	<80
		MCA > ACA > ICA > PCA ≥ Basilar > Vertebral				

ACA indicates anterior cerebral artery; ICA, internal carotid artery; MCA, middle cerebral artery; OA, ophthalmic artery; PCA, posterior cerebral artery.
Source: Modified from Aaslid R et al. (Ref. 18)

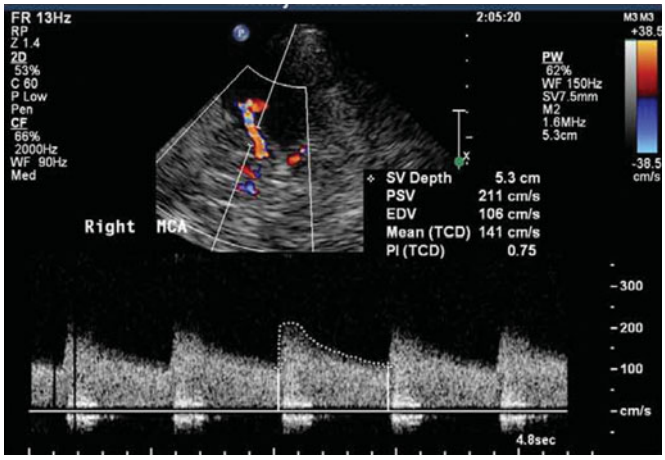


Figure 26.23. Normal middle cerebral artery (MCA) waveform. This is a low-resistance pattern with flow throughout the cardiac cycle.

limitations to the diagnostic value of TCD still present problems. Premorbid TCD studies must be available to confirm that an absent signal is not due to the absence of a suitable acoustic window. Isolated anterior or posterior circulation blood flow may be demonstrated despite clinical evidence of brain death. Although TCD may be useful in patients known to have received sedative drugs, its current role in the diagnosis of brain death remains adjunctive.

CONCLUSION

The availability of high-quality, portable bedside duplex scanners has brought the ability to accurately diagnose acute DVT within the capability of every physician dealing with critically ill patients. The education and skills required to become comfortable with the techniques are basically extensions of the physical examination skills. And it is almost axiomatic that the individual who is best able to interpret the findings is the one who has daily responsibility for the care of the patient and who can correlate the results within the clinical context for that individual patient. A working knowledge of arterial anatomy and a basic understanding of arterial hemodynamics provide the bedside sonographer a solid foundation on which to perform and interpret the arterial duplex examination. Current portable duplex equipment provides high-quality information and puts this modality in the armamentarium of every clinician willing to gain the necessary experience with this modality.

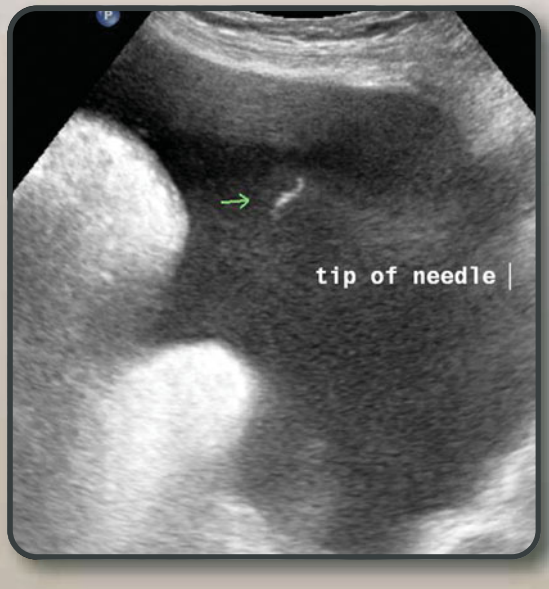
References

1. Blaivas M. Ultrasound in the detection of venous thromboembolism. *Crit Care Med.* 2007;35(suppl 5):s224.
2. Knudson MM, Collins JA, Goodman SB, et al. Thromboembolism following multiple trauma. *J Trauma.* 1992;32:2–11.
3. Kudsk KA, Fabian TC, Baum S, et al. Silent deep vein thrombosis in immobilized multiple trauma patients. *Am J Surg.* 1989;158:515–519.
4. Cook D, Attia J, Weaver B, et al. Venous thromboembolic disease: an observational study in medical-surgical intensive care unit patients. *J Crit Care.* 2000;15:127–132.
5. Burns GA, Cohn SM, Frumento BJ, et al. Prospective ultrasound evaluation of venous thrombosis in high-risk trauma patients. *J Trauma.* 1993;35:405–408.
6. Harris LM, Curl GR, Booth FV, et al. Screening for asymptomatic deep vein thrombosis in surgical intensive care patients. *J. Vasc Surg.* 1997;26:764–769.
7. Marik PE, Andrews L, Maini B. The incidence of deep venous thrombosis in ICU patients. *Chest.* 1997;111:661–664.
8. Stein PD, Henry JW. Prevalence of acute pulmonary embolism among patients in a general hospital and at autopsy. *Chest.* 1995;108:978–981.
9. Massicotte MP, Dix D, Monagle P, et al. Central venous catheter related thrombosis in children: analysis of the anadian Registry of Venous Thromboembolic Complications. *J Pediatr.* 1998;133:770–776.
10. Strandness DE. Ultrasonic duplex scanning. In: Ascher E, ed. *Haimovici's Vascular Surgery.* Blackwell Science, Blackwell Publishing Co; 2004:7–34.
11. Beaulieu Y, Marik PE. Bedside ultrasonography in the ICU: Part 2. *Chest.* 2005;128:1770.
12. Panetta TF, Sales CM, et al. Natural history, duplex characteristics, and histopathologic correlation of arterial injuries in a canine model. *J Vasc Surg.* 1992;16:867–874.
13. Tola M, Yurdakul M, Cumhur T. B-flow imaging in low cervical internal carotid artery dissection. *J Ultrasound Med.* 2005;24:1497–1502.
14. Davison BD, Polak JF. Arterial injuries: a sonographic approach. *Radiol Clin North Am.* 2004;42:383–396.
15. Imsand D, Hayoz D. Current treatment options of femoral pseudoaneurysms. *Vasa.* 2007;36:91–95.
16. Hanson JM, Atri M, Power N. Ultrasound-guided thrombin injection of iatrogenic pseudoaneurysm: Doppler features and technical tips. *Br J Radiol.* 2008;81:154–163.
17. Kohler TR et al. Duplex scanning for diagnosis of aortoiliac and femoropopliteal disease: a prospective study. *Circulation.* 1987;76:1074–1080.
18. Aaslid R, Markwalder TM, Nornes H. Noninvasive transcranial Doppler ultrasound recording of flow velocity in the casal cerebral arteries. *J Neurosurg.* 1982;57:769–774.

19. Otis SM, Ringelstein FB. Transcranial Doppler sonography. In: Zweibel WJ, ed. Introduction to vascular ultrasonography, Philadelphia: WB Saunders, 145–171, 1992.
20. White H, Venkatesh B. Applications of transcranial Doppler in the ICU: a review. *Intensive Care Med.* 32:981–994, 2006.
21. Springborg JB, Frederikson HJ, Eskesen V, et al. Trends in monitoring patients with aneurysmal subarachnoid haemorrhage. *Br J Anaesthesia* 94(3):259–70, 2005.
22. Ducrocq X, Braun M, Debouverie M, et al. Brain death and transcranial Doppler: experience in 10 cases of brain dead patients. *J Neurol Sci.* 160:41–46.

SECTION IV

ULTRASOUND GUIDANCE FOR PROCEDURES



This page intentionally left blank

CHAPTER 27

Ultrasound-Guided Transthoracic Procedures

Peter Doelken and Paul H. Mayo

INTRODUCTION

Ultrasonographic guidance of thoracic drainage and biopsy procedures is an attractive alternative to computerized tomography (CT) or fluoroscopic guidance. While CT scanning is the standard for overall imaging of the chest in all cases of malignancy and many cases of nonmalignant conditions, ultrasound may subsequently be used for procedure guidance. Ultrasound guidance eliminates further radiation exposure and is often less time consuming and more comfortable for the patient. The intensivist–sonographer who engages in thoracic interventions must possess the cognitive and manual skills required for pleural and lung sonography; and, in the case of anterior mediastinal biopsy, must be familiar with the ultrasound anatomy of the mediastinal organs. The ability to visualize inserted hardware and to recognize and interpret reverberation artifact associated with hardware is required for all but thoracentesis and some simple biopsy procedures.

HARDWARE

Convex-array or sector-scanning probes with frequencies between 2 and 5 MHz (typically 3.5 MHz) are most suitable for thoracic sonography and are also the most versatile for thoracic procedure guidance.[1] Higher-frequency transducers do have better near-field resolution at the expense of penetration depth. We do not recommend probes with a biopsy channel for thoracic interventions due to the common problem of ribcage interference with imaging. Instead, an approach combining imaging with traditional bony landmark detection, i.e., finding the upper rib margin with a finder needle prior to insertion of other hardware, is generally preferred. As in general thoracic sonography, the mark on the probe is oriented cephalad and the corresponding mark on the screen is placed at the upper left of the image. Thus, the orientation of standard views, which is imaging in the longitudinal axis, is cephalad left and caudal right on the screen. However, during procedure planning and visualization of hardware, nonstandard imaging planes are routinely used.

POSITIONING FOR PROCEDURES

Proper patient positioning is essential in interventional chest sonography. Free-flowing pleural effusions follow the gravitational gradient and collect in the most dependent part of the thoracic cavity. In the patient sitting upright, an effusion will collect in the inferior and posterior chest and is most easily accessed from a position behind the patient. The approach to positioning the critically ill patient varies with the size of the effusion, presence of obesity, number and type of support devices, and physiologic compromise such as hemodynamic instability. Large effusions may be accessed with the patient in the supine position, which presents few problems during access. However, lateral access may be impossible in the very obese even with very large effusions. Adduction of the ipsilateral arm across the chest greatly improves lateral access and should uniformly be attempted. Posterior access may be facilitated by having the patient held in a sitting position but also by placing the patient at the very edge of the bed or even in the full lateral decubitus position. These positions require assistants to assure safety and prevent inadvertent movement of the patient. Occasionally, simply elevating the head of the bed allows lateral access even in cases where this was not possible in the fully supine position. Careful monitoring of endotracheal tubes and vascular access devices is required at all times during patient positioning.

Patient positioning for biopsy of lung or pleural lesions depends on the location of the lesion. The ability of the patient to comfortably maintain position for the duration of the procedure is essential for successful performance of the procedure and for maintaining sterility throughout. The overall guiding principle for positioning for sonographically guided procedures is

the individualized approach that fully exploits the flexibility of sonographic imaging.[2]

THORACENTESIS

The objectives of the addition of sonographic guidance to the thoracentesis procedure are to increase the success rate and to decrease complications. The success rate and likely complication rate is intimately related to diagnostic sonography of pleural effusion by eliminating inadvertent attempts to drain fluid when no fluid is present. Proficiency in diagnostic sonography of pleural effusion is therefore a prerequisite for the successful use of sonographically guided thoracentesis (See Chapter 21). The issues pertaining to sonographic guidance in particular, such as patient positioning and access site selection, are described here. The more common complications of thoracentesis include pneumothorax, pain, shortness of breath, cough, and vasovagal reactions. Other complications that have been described are reexpansion pulmonary edema, inadvertent liver or splenic injury, hemothorax, infection, subcutaneous emphysema, air embolism, and chest wall or subcutaneous hematoma. Of all the complications, sonographic guidance appears to result in lower rates of traumatic pneumothorax; with rates between 5–18% for clinically guided vs. 1–5% for sonographically guided thoracentesis. Obviously, the likely reduction of traumatic pneumothorax is especially important in the mechanically ventilated patient who is at much higher risk for tension pneumothorax than the spontaneously breathing patient.

In patients not receiving mechanical ventilation, the risk of pneumothorax associated with thoracentesis performed by a radiologist has been reported to be 2.7%[3]; in a surgical intensive care unit setting, the complication rate was found to be 2.4%.[4] Ultrasound guidance for therapeutic thoracentesis appears to almost eliminate needle trauma as the immediate cause of postprocedure pneumothorax in spontaneously breathing patients. Pneumothorax in this setting has been reported to be associated with unexpandable lung and not laceration of the visceral pleura in almost all cases.[5] The incidence of pneumothorax in mechanically ventilated patients has been reported by radiologists to be higher than in spontaneously breathing patients, with an overall rate of only 2%,[6] but with a rate of 7% in intubated patients. However, Godwin and Sahn reported a similar risk of pneumothorax in patients receiving mechanical ventilation when compared with spontaneously breathing patients.[7] Other studies have reported low pneumothorax rates in the mechanically ventilated patient.[8–10] Although no direct comparison between thoracentesis with versus without ultrasound guidance has been performed, Diacon et al. found that use of ultrasonography for site location for thoracentesis was more accurate than standard physical examination.[11]

In addition to the apparent safety of sonographically guided thoracentesis in regard to pneumothorax, physician-performed bedside ultrasound guidance may obviate the need to transport the critically ill to interventional radiology, thus eliminating the indirect risks related to the transport of the critically ill.

Because the pulmonary and critical care physician is already familiar with the basic procedure, thoracentesis is ideally suited for the initial adoption of sonographic guidance for thoracic interventions. While diagnostic sonography of pleural effusion is concerned with the detection and characterization of pleural fluid and the size of the effusion, sonographic guidance has the selection of a suitable access site and avoidance of organ puncture as the primary goals.

For ultrasound-guided thoracentesis to be performed safely and successfully, particular attention must be directed toward patient and operator positioning relative to the ultrasound machine to allow unencumbered use of the device without compromising sterility. The procedure field needs to be free of monitoring and support devices. Determination of a suitable access site requires demonstration of pleural fluid immediately adjacent to the parietal pleura and sufficient distance from organs throughout the respiratory cycle. The diaphragm, liver, or spleen should be identified unequivocally. This is necessary so as not to confuse the curvilinear line of Morrison's pouch, located between liver and kidney, with the diaphragm (Figure 27.1). On the left, a curvilinear line may also be seen between spleen and kidney, and this line may also be mistaken for the diaphragm by the inexperienced sonographer. Misidentification of these lines can result in inadvertent hepatic or splenic injury. After marking the access site, position of the mark should once again be sonographically verified. Sonography allows the measurement of the distances between the parietal pleura and the various underlying organs. Provided that the patient does not change position between the ultrasound examination and needle insertion and does not cough during the procedure, these distance measurements are reliable and are not subject to compression artifact as are measurements of chest wall thickness.[2,12–14] As the depth of the parietal pleural surface is indicated by pleural fluid return when the needle is advanced under aspiration, the measured distances between parietal

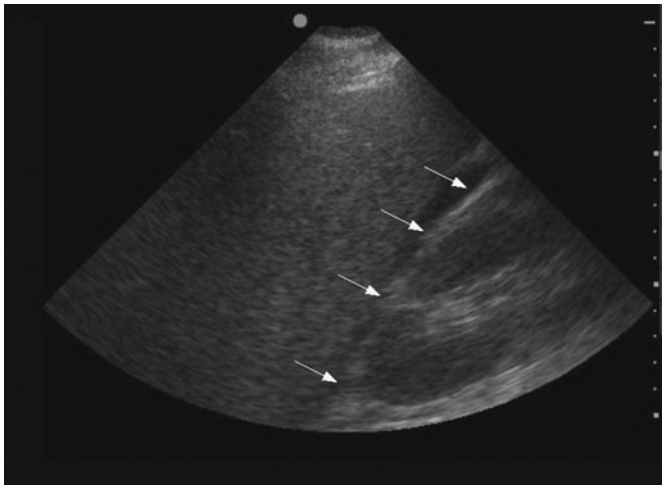


Figure 27.1. The ultrasound image shows a curvilinear structure lying between the liver and kidney. The inexperienced ultrasonographer may misidentify the hepatorenal recess as the diaphragm, and the liver as an hyperechoic pleural effusion. This could lead to inadvertent subdiaphragmatic device insertion.

pleural surface and underlying organs are measures of how much further and at which angle the needle may be safely advanced. An important caveat is related to possible occlusion of the needle lumen by clot or the solid contents of a complex effusion. This pitfall may be avoided by remembering that fluid return is expected approximately within 5 millimeters (mm) after passing the upper margin of the inferior rib. The needle lumen may also be cleared by injecting small amounts of local anesthetic if occlusion is suspected.

We do not routinely use real-time visualization of the thoracentesis needle during insertion. Real-time visualization adds complexity due to the need for a sterile sleeve, and may interfere with maintaining the proper needle insertion angle.[15]

After achieving proper local anesthesia and pleural fluid return, the needle is withdrawn and a small incision is made to allow insertion of the thoracentesis catheter if large volume thoracentesis is performed. Care must be taken that no air is inadvertently introduced during catheter insertion and connection to the drainage system.[16] In case only a diagnostic sample is required, we withdraw the needle into the rib interspace until fluid return stops. We then exchange the syringe containing the local anesthetic with a clean syringe and reinsert the needle to the same depth at which pleural fluid was obtained before and begin aspiration of the sample. Anterior lung sliding may be documented prior to the procedure, and its continued presence after the procedure will reliably exclude an immediate postprocedure pneumothorax.

ULTRASOUND-GUIDED TUBE THORACOSTOMY

Indications for sonographically guided tube thoracostomy include complicated parapneumonic effusion, empyema, malignant pleural effusion, and pneumothorax. The type and size of the chest tube is dictated by the underlying condition. Large bore tubes are typically inserted for acute hemothorax or for pneumothorax and bronchopleural fistula in the mechanically ventilated patient, whereas small-bore pigtail catheters are the most versatile of chest tubes and are suitable for most other conditions. For chronic outpatient management of malignant pleural effusion, tunneled catheters may be used. Regardless of the type of tube used, the principles of ultrasound guidance are similar to those of simple ultrasound-guided thoracentesis. We routinely image a guide wire in real time, if applicable, to ascertain proper placement prior to use of dilators or catheter insertion. In order to image hardware, the transducer is rotated along its long axis to bring the wire or catheter into the sonographic image plane. The tip of a typical J-type guide wire can easily be seen with ultrasonography.

As the drainage devices remain in place, consideration has to be given to patient comfort whenever feasible and the device preferably should be inserted more laterally than during typical thoracentesis. However, this is not always possible and occasionally a pigtail catheter must be inserted posteriorly.

The use of sonography in catheter placement for pneumothorax is limited, as the distance between parietal and visceral pleura cannot be visualized in the presence of intrapleural air. However, sonography is useful in order to exclude underlying organs touching the parietal pleura immediately at the intended insertion site. We recommend CT guidance for pneumothorax in a complex pleural space due to adhesions from previous pleural injury or disease.

TRANSTHORACIC BIOPSY PROCEDURES

Ultrasound-guided needle biopsy is suitable for peripheral lung lesions, anterior mediastinal masses, and lesions of the pleura itself. However, any lesion must abut an accessible area of the parietal pleura and must be readily visualized by sonography (Figure 27.2). Critical

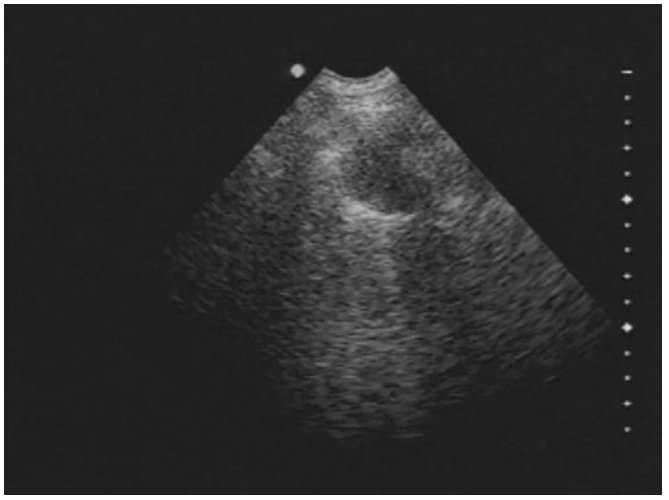


Figure 27.2. The ultrasound image shows a peripheral lung mass.

to the success of the biopsy is operator proficiency in chest ultrasonography and the ability to correlate CT images with ultrasound findings. Computerized tomography imaging is essential for procedure planning in order to characterize the lesion and document extent and local topography, such as proximity of the heart or other structures. However, sonographic guidance is preferred to CT guidance of device insertion due to lack of radiation exposure and better patient comfort. Accessibility of the lesion with ultrasound guidance is the determined by imaging of the lesion and confirmation of a clear needle path without intervening air, bone, organs, or vasculature. Angle of needle entry and penetration depth is then determined. Determination of penetration depth is susceptible to skin compression artifact, and this requires employment of strategies similar to the ones described above (see Thoracentesis).

Although the needle may be tracked in real time after insertion by keeping the scanning plane parallel to the needle path, this is difficult due to the anatomy of the chest wall especially during biopsy of a superficial lesion. Needle guides may help keep the needle in the scanning plane, thereby simplifying real-time visualization. However, this benefit is outweighed by limitations imposed on needle angulation due to the more cumbersome device.[12–14,17] We do not routinely use real-time visualization during biopsy of superficial lesions of the lung or pleura.

Eighteen or 20 gauge needles are appropriate for fine needle aspiration. After local anesthesia, and once the lesion is entered, suction is applied and the needle is moved in a to-and-fro motion in a fanlike pattern. Suction is then released and the needle withdrawn. It is important to release suction before the needle is withdrawn to avoid contamination of the specimen and to avoid aspiration of the specimen into the syringe used to apply suction. Specimens are immediately transferred to microscopy slides and air-dried or fixed in alcohol. Media for flow cytometry or cell block preparations should also be readily available.

Core biopsies of pleural or lung lesions require the use of cutting needle devices. The standard coaxial cutting needle technique is suitable for the purpose, and kits are commercially available. The kits contain an introducer with stylet and a spring-loaded cutting needle. Cutting needles are available with and without adjustable throw. We use needle sizes 16–20 G and reserve the largest size for pleural biopsy.

After site verification by ultrasound, the introducer assembly is advanced into the intercostal space. The patient is asked to hold breath while the introducer assembly is introduced into the target lesion. Resistance may be encountered upon entering the lesion. Once desired position is attained, the introducer assembly is locked in place. Whenever the stylet or biopsy needle is removed from the introducer lumen, the patient is asked to hold breath to prevent inadvertent air entry. With the introducer assembly in place, the stylet is removed, the charged cutting needle advanced and locked and immediately discharged. If applicable, desired throw is adjusted prior to insertion of the needle. The cutting side of the needle is always oriented caudally to help avoid injury to the intercostal vasculature. Core biopsies allow histologic diagnosis. On-site cytopathologic examination to determine specimen adequacy is possible by rolling the tissue cylinder on a microscopic slide prior to transfer into fixative.[18–20]

There are few absolute contraindications to biopsy. These include an uncooperative patient, a patient who cannot be maintained in position for the duration of the procedure, and intractable coughing. Mechanical ventilation and severe pulmonary hypertension are relative contraindications. The feasibility of biopsy has to be considered on a case-by-case basis. Other relative contraindications are coagulopathies, thrombocytopenia (platelet count <50 K), and uremic platelet dysfunction. Some of these abnormalities can be corrected prior to the procedure.

The risks of biopsy of lung lesions are similar to the risks of fiberoptic transbronchial biopsy. The most common complication is pneumothorax, with an occurrence rate of about 3%, and is typically associated with pleuritic chest pain. Unfortunately, when pneumothorax does occur, the ultrasound image is lost, and the

procedure has to be aborted. This need to stop mid-procedure is a disadvantage of using ultrasonography when compared with CT guidance.[21,22] Obviously, underlying lung disease or having only a single lung increases the risk for complications of pneumothorax. In this case, a pneumothorax often requires immediate treatment.[23–27]

Hemoptysis occurs in up to 10% of cases after core biopsy but is typically self limited. Massive hemoptysis may occur and result in poor outcome. Management of massive hemoptysis is no different from management of hemoptysis from transbronchial biopsy: tamponade with the bronchoscope, endobronchial balloon occlusion, and selective intubation of the contralateral side.[27] Coughing may cause tearing of the pleura and air embolism, which can also occur without coughing. The risk of air embolism may be lower with ultrasound-guided than with CT-guided biopsy due to the generally peripheral location of lesions accessible to ultrasound guidance. Occluding the introducer cannula between passes of the needle may prevent this complication. If air embolism is suspected, the patient should be placed in the left lateral decubitus position and given 100% oxygen. Hyperbaric oxygen treatment is an option to be considered. Other risks include inadvertent puncture of other vital organs, which should be an extremely rare occurrence with an experienced operator.[28]

BIOPSY OF LUNG LESIONS

A requirement for ultrasound guidance for lung biopsy is the ability to visualize the target. Any intervening aerated lung will make sonographic visualization impossible and initial determination of suitability for sonographic guidance may be made by careful examination of the chest CT (Figures 27.3–27.9). One of the advantages of sonographic guidance when compared with CT guidance is the superior ability of sonography in distinguishing solid from liquid in a partially necrotic lesion. Using ultrasound, the biopsy needle may be confidently directed toward solid areas.[29] Cavitary lesions containing air need to be considered individually. Chronic cavitating or bronchiectatic lesions are commonly highly vascularized and have a higher risk of bleeding. It has been shown recently that important staging information in primary lung cancer can be derived by determination of presence or absence of movement of lung lesions relative to the parietal pleura. With higher-frequency transducers, interruption of the pleural reflection and invasion of the ribs or chest wall may be demonstrated. Any of these findings indicates chest wall invasion and may affect staging of non-small cell lung cancer.[30]

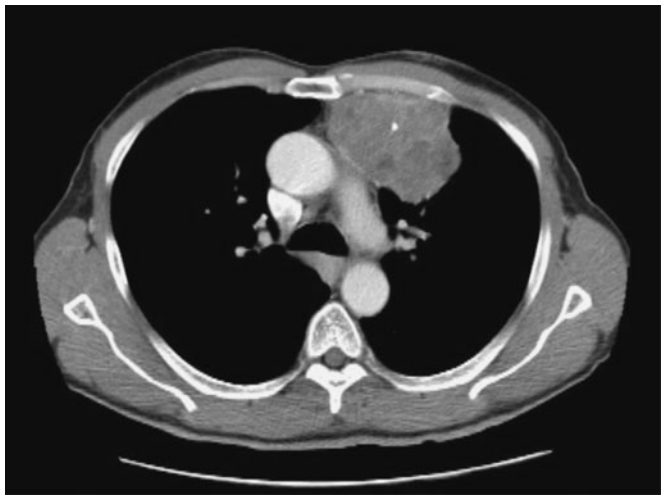


Figure 27.3. The chest CT scan shows an anterior mediastinal mass that is well situated for ultrasound-guided biopsy. CT indicates computerized tomography.

PLEURAL BIOPSY

Indications for sonographically guided pleural biopsy include suspected pleural malignancy, especially when pleural fluid cytology is negative; suspected malignant mesothelioma; and suspected tuberculous pleural effusion. Ultrasonography may detect abnormal areas of the pleura that may be targeted for biopsy. Yield is improved with this approach when compared with random pleural sampling.[18,31] For the diagnosis of malignant pleural mesothelioma, a tissue sample

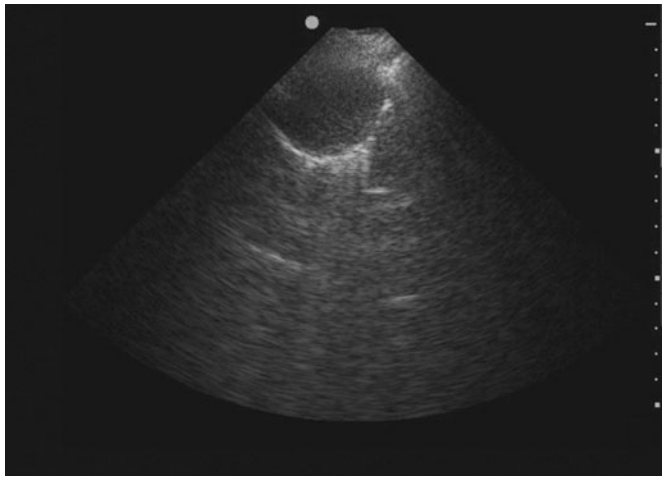


Figure 27.4. The ultrasound image shows the anterior mediastinal mass seen on chest CT scan. CT indicates computerized tomography.

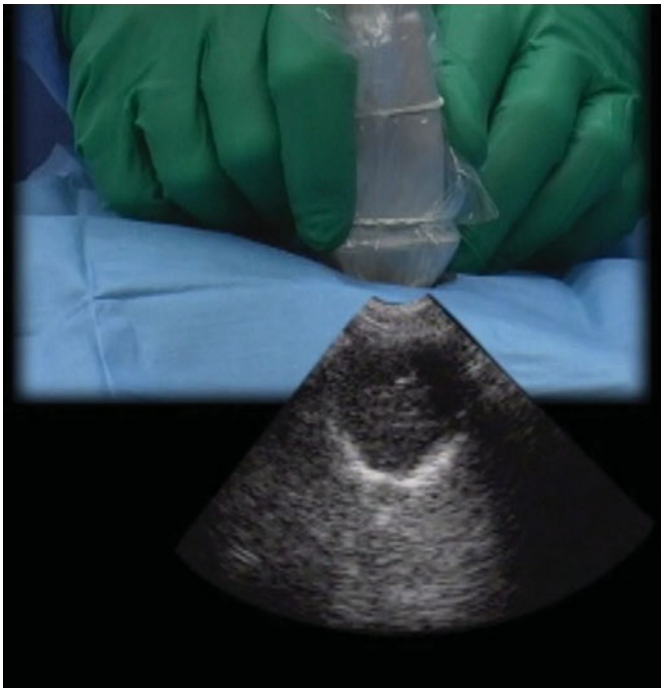


Figure 27.5. The ultrasound image of a mediastinal mass is shown in relation to the transducer.

demonstrating invasiveness is often required and ultrasound-guided biopsy should be considered as the initial diagnostic procedure. We include part of the chest wall in the biopsy. Demonstration of invasiveness requires inclusion of chest wall tissue with the biopsy core. This may be achieved by partial withdrawal of the introducer assembly into the rib interspace prior to discharge of the cutting apparatus. The cutting aspect of the needle is oriented caudally, away from the neurovascular bundle. A sensitivity of 77% and

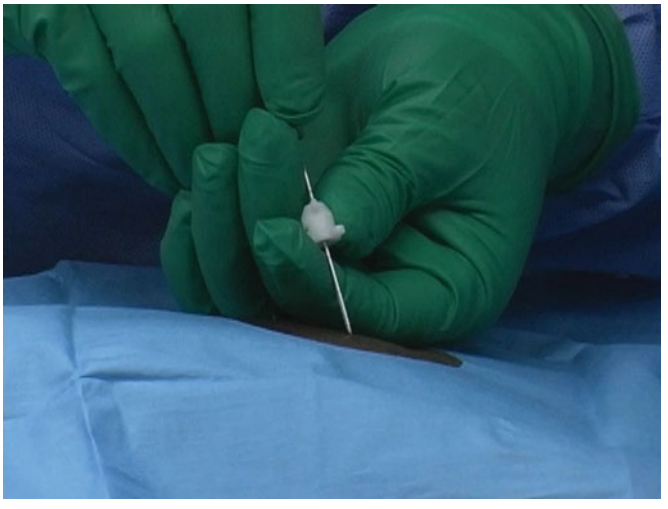


Figure 27.6. The introducer is inserted at the site, depth, and angle determined by ultrasonography.

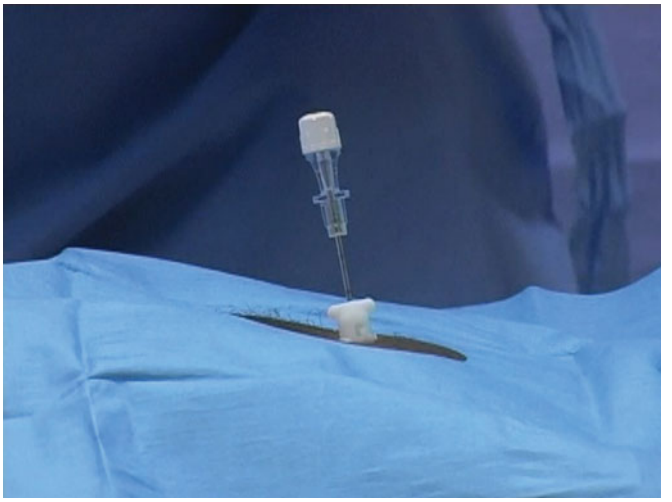


Figure 27.7. The introducer is positioned for insertion of the spring-loaded cutting needle.

a specificity of 88% may be attained with sonographically guided core biopsy in cases of malignant pleural mesothelioma.[32]

ANTERIOR MEDIASTINAL BIOPSY

Anterior mediastinal masses may be accessible to sonographically guided biopsy provided that aerated lung is displaced sufficiently to open a sonographic window through the anterior rib cage. It is essential that the needle path is well clear of the mammary vessels as well as the aorta, pulmonary artery, and the heart. The procedure is performed in the supine position after careful review of the chest CT. Generally, core biopsy is necessary for the diagnosis of anterior

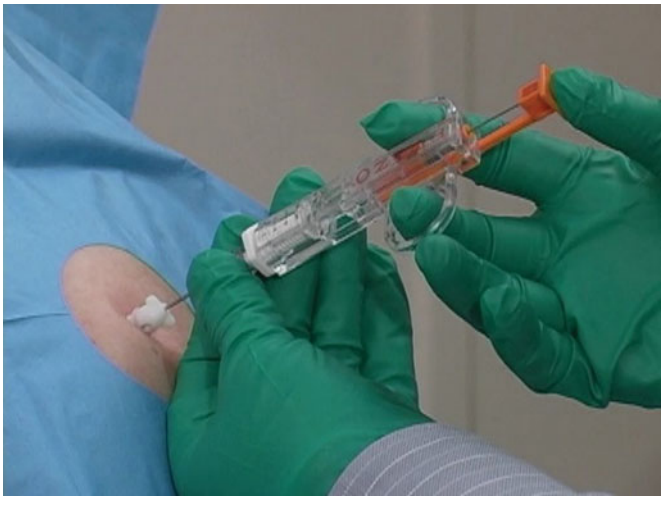


Figure 27.8. A spring-loaded cutting needle is inserted into the lung lesion.

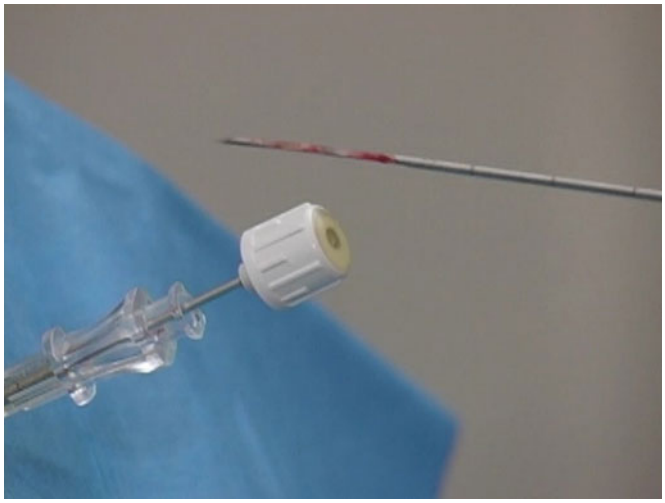


Figure 27.9. The cutting needle has obtained a good-quality biopsy.

mediastinal masses, unless metastatic carcinoma is suspected, in which case fine needle aspiration may be sufficient.[33–36]

SUMMARY

The use of ultrasound for guidance in procedures involving the pleural space has become routine and has an excellent safety record. Once understanding of the pleural anatomy as seen on ultrasonography is learned, the techniques for intervention can be easily mastered by the physician–operator. Ultrasound-guidance is in many cases preferable to CT guidance, as transport of the critically ill is avoided. Sonography does not subject the patient to the radiation effects of CT and comes at a lower cost. The operator must be familiar with all the possible complications of these procedures and their treatment. The need to understand pleural ultrasonography, especially its role in interventions, will continue to grow and become important part of the critical care and pulmonary physician's armamentarium.

PERICARDIOCENTESIS

Ultrasonography allows safe performance of pericardiocentesis. Seward et al. described 1127 serial pericardiocenteses with a very low complication rate.[37] Ultrasound-guided pericardiocentesis should completely replace fluoroscopic guidance. Flouroscopic guidance requires subcostal needle insertion during which the position of the liver is unknown; and the relationship of the needle to the myocardium is uncertain, as fluoroscopy is a two-dimensional technique. Ultrasound-guided pericardiocentesis is so clearly superior to fluoroscopic guidance, that the critical care ultrasonographer should develop proficiency in the procedure to improve patient safety.

Overview of Procedure

Ultrasonographic guidance of pericardiocentesis has features in common with thoracentesis and paracentesis. The operator identifies the fluid collection, and then chooses a safe site, angle, and depth for needle insertion. While the principles are the same, pericardiocentesis is different because of its intrinsic hazard. Laceration of the myocardium or of a coronary artery is a catastrophic complication of pericardiocentesis. The operator needs to be highly skilled at image acquisition and interpretation as well completely proficient with the manual skills related to hardware insertion. Pericardiocentesis is not a procedure for the entry-level ultrasonographer. The teacher attending is advised to supervise the fellow very closely during the procedure.

Equipment Requirements

An ultrasound machine equipped with cardiac transducer is required. Doppler capability is not required for ultrasound guidance of pericardiocentesis. Regarding equipment for the procedure itself, there are kits that are marketed specifically for pericardiocentesis. Another approach is to use a generic wire insertion approach. Central venous insertion kits can be used for pericardiocentesis, as can widely available cavity drainage systems. Manufacturers also market as stand-alone items, wire/catheter combinations. These are supplemented with appropriate equipment of the operator's choice. Whatever equipment is chosen, the clinician should choose the shortest possible needle length for the initial pericardial penetration. Some kits provide a 20 centimeter (cm) needle. These are difficult to manipulate, and therefore dangerous to use. Some clinicians prefer to insert an angiocath into the space, followed by the wire. Others prefer to use a wire-through-needle approach. Either way, a general principle always holds. The needle is inserted to the most minimal depth possible to obtain free flow of fluid, and for the shortest time possible time. Once the wire is in place and the needle removed, the risk of myocardial of coronary artery or myocardial laceration is no longer present. Whatever hardware is used for the procedure, the operator must be completely proficient in all aspects of wire placement, dilatation over the wire,

and insertion of a catheter over the wire. This chapter will not discuss the technical aspects of hardware use, but will concentrate on the use of ultrasonography to guide the procedure.

Site Selection and Preparation

With fluoroscopic guidance, the operator is limited to the subcostal approach with liver and other vital organs in close but uncertain proximity to the needle path. Using ultrasonography, the best site is determined by where the most fluid is found. This may be subcostal or at any point on the anterior or lateral chest. Frequently, the best site is identified on the lateral chest using the apical four-chamber view. If the effusion is very large, a parasternal view may offer a good approach. Frequently, the effusion will be predominately posterior in location. This being the case, changing the patient's body position may distribute the fluid into a more favorable position. For example, the semisupine position may improve the subcostal view, while a left lateral decubitus position may move the fluid for improved apical view.

The heart is a highly mobile organ. It changes in size throughout the contractile cycle, it is subjected to respiratory effects that are accentuated if the patient has respiratory distress, and cardiac "swinging" is a common phenomena in severe tamponade. The apparent thickness of the pericardial effusion may rapidly change to major extent during cardiac movement. The distance between the site of needle penetration into the pericardium and the heart itself is a major determinant of safety. There is no absolute rule for how much fluid must be present to allow safe needle entry. A reasonable approach is to require at least 1 cm of fluid depth between the heart and the proposed needle entry point into the pericardial space, while taking into account the change in this distance that occurs during cardiac and respiratory cycle.

An important element in safe site selection is the avoidance of injury to adjacent structures. The lung is of concern. Fortunately, aerated or consolidated lung is easy to identify and therefore to avoid (see Chapters 21 and 22). The same holds for the liver, which may be readily identified and therefore avoided when using the subcostal approach. If using a parasternal approach, the internal mammary artery is an issue. Color Doppler of the proposed needle track is mandatory when using the parasternal approach in order to avoid the internal mammary vessels. Pleural effusion may be identified in association with the pericardial effusion, and may be large enough to interfere with pericardial access.

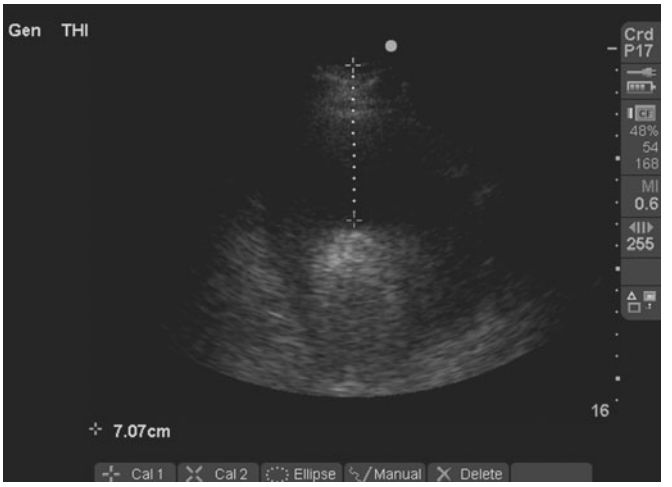


Figure 27.10. The ultrasound image shows a large pericardial effusion from the apical four-chamber view, with depth measurement for needle penetration made in preparation for pericardiocentesis. This defines the "no go" depth that would result in cardiac injury.

In this case, the best approach is to drain the pleural effusion first, followed by rescanning to determine best approach to the pericardial effusion.

The depth of needle penetration is a critical element to safe pericardiocentesis. The ultrasound machine allows accurate measurement of the distance required for needle penetration image (Figures 27.10 and 27.11). However, skin compression artifact is a concern. The transducer is pressed into the patient's skin with some degree of force in order to obtain good image quality. In

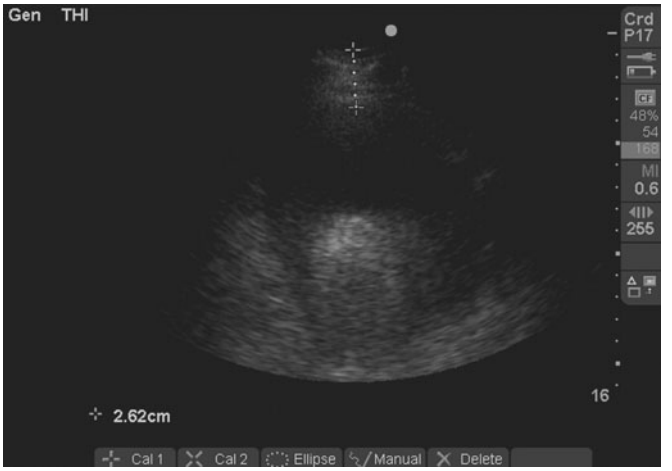


Figure 27.11. The ultrasound image shows a large pericardial effusion from the apical four-chamber view, with depth measurement for needle penetration made in preparation for pericardiocentesis. This defines the depth required for entry into the pericardial space.

the edematous or obese individual, this compression artifact may be several centimeters in depth. This results in a significant underestimation of the distance required to access the pericardium during the actual procedure. The last factor in determining safe needle trajectory for pericardiocentesis is to ascertain the best angle of approach. This is determined by the angle at which the transducer is held to obtain the best site of access.

Site Preparation

It is advisable to scan the patient before sterile skin preparation. The best site and angle of needle insertion is determined and the site is marked. The skin may be marked with ink. Alternatively, it may be indented with a needle cap. Depth measurement includes estimate of skin compression artifact. The depth of needle penetration is measured from a frozen image on the ultrasound screen using the calipers function. The skin is then prepared for sterile procedure. It is strongly recommended that the patient be covered with a full body drape, and the team use sterile coverings (mask, gown, eye protection, and caps). The transducer should be covered with a sterile sleeve and be part of the sterile field setup.

Before the final confirmatory scan, all aspects of equipment setup should be completed, such as needle–syringe assembly, lidocaine draw-up, wire layout, and tubing connections. This minimizes the time between the final scan and needle insertion. Following full equipment set-up, the operator once again confirms the site and depth of needle penetration using the transducer with sterile probe cover, and notes the angle of the transducer. This angle will be carefully duplicated by the angle of the needle–syringe assembly during device insertion. The operator proceeds with needle–wire insertion, followed by catheter insertion. Confirmation of wire or catheter insertion may be accomplished by direct visualization using 2D ultrasonography. If there is question of position, several cc's of agitated saline may be injected through the catheter to document catheter position. Malignant pericardial effusions are frequently hemorrhagic. The team may be apprehensive about this result, so documentation of pericardial placement with bubble study is a reassuring result.

Similar to thoracentesis and paracentesis, pericardiocentesis does not require real-time guidance with ultrasonography. The largest published study on the subject did not use real-time guidance.[1] However, it is important to have the transducer with sterile cover in place for immediate use throughout the procedure, in case there is need to rescan and document successful device insertion.

Pitfalls: Common and Uncommon

Skin compression artifact is a common problem (see above). It causes an underestimation of the depth for needle insertion. During needle insertion, the operator is appropriately concerned, if there is no fluid obtained at the depth measured from the ultrasound machine screen. After all, coronary or ventricular laceration may result in lethal outcome to the patient. The solution to this problem is to rescan the patient, confirm angle of insertion, and estimate compression artifact more accurately. Another cause for difficulty is movement of the mark that designates the appropriate site for needle insertion. Skin is movable, so that the injudicious force application by the operators hand may shift the skin mark. The needle should be inserted at the mark without any tension applied to the area that might shift mark position. Similarly, a "dry tap" might result from inaccurate duplication of the angle at which the transducer was held or inaccurate skin mark. The solution remains to rescan the patient to recheck angle and site. Generally, it is easier to duplicate a perpendicular transducer angle than one that is acutely angled. This favors an anterior or lateral chest wall approach (if fluid is accessible), as the transducer is often perpendicular to the chest wall when scanning in these areas. This is not the case in the subcostal approach.

An unusual cause of a "dry tap" is a blocked needle, resulting in overly deep needle insertion. Clotted blood or skin plug may be the culprit. Overly vigorous probing of the anterior costal cartilage (if using a parasternal approach) may also block the needle with cartilage so that the operator inserts too deeply with potential complication to the patient.

A large anterior pericardial fat pad may be mistaken for a pericardial effusion by the inexperienced ultrasonographer, with potentially catastrophic consequences to the patient. Pericardial fat has some element of echogenicity and moves in synchrony with cardiac contraction. In addition, it is very uncommon for a consequential pericardial effusion to occur anterior to the heart without a significant posterior pericardial effusion also being present.

An uncommon pitfall of pericardiocentesis occurs when the anesthesia needle penetrates the pericardium after having traversed the pleural space. The pericardial effusion may then drain into the pleural space through the defect in the pericardium made by the anesthesia needle. This may occur if there is a delay before definitive pericardial device insertion. The operator is unpleasantly surprised by the lack of pericardial

effusion and the presence of a new pleural effusion. To avoid this situation, device insertion should promptly follow infiltration of the local anesthesia.

Summary

Ultrasonography permits safe pericardiocentesis. Ultrasonography allows the intensivist to select a safe site, angle, and depth for needle and device insertion. Meticulous attention to image acquisition and interpretation allows the operator to avoid the serious complication of myocardial or coronary artery laceration. The critical care ultrasonographer is strongly encouraged to develop proficiency in ultrasound guidance of pericardiocentesis, as it is superior to subcostal fluoroscopic guidance.

References

1. Reuses J. Interventional chest sonography. In: Mathis G, Lassa KD, eds. *Atlas of Chest Sonography.* Berlin, Germany: Springer-Verlag; 2003:147–162.
2. Lichtenstein DA. Interventional ultrasound. In: Lichtenstein DA, ed. *General Ultrasound in the Critically Ill*. Berlin, Germany: Springer-Verlag; 2005:170–174.
3. Jones PW, Moyers JP, Rogers JT, Rodriguez RM, Lee YC, Light RW. Ultrasound-guided thoracentesis: is it a safer method? *Chest.* 2003;123:418–423.
4. Petersen S, Freitag M, Albert W, Tempel S, Ludwig K. Ultrasound-guided thoracentesis in surgical intensive care patients. *Intensive Care Med.* 1999;25:1029.
5. Heidecker J, Huggins JT, Sahn SA, Doelken P. Pathophysiology of pneumothorax following ultrasound-guided thoracentesis. *Chest.* 2006;130:1173–1184.
6. Gervais DA, Petersein A, Lee MJ, Hahn PF, Saini S, Mueller PR. US-guided thoracentesis: requirement for postprocedure chest radiography in patients who receive mechanical ventilation versus patients who breathe spontaneously. *Radiology.* 1997;204:503–506.
7. Godwin JE, Sahn SA. Thoracentesis: a safe procedure in mechanically ventilated patients. *Ann Intern Med.* 1990; 113:800–802.
8. Lichtenstein D, Hulot JS, Rabiller A, Tostivint I, Meziere G. Feasibility and safety of ultrasound-aided thoracentesis in mechanically ventilated patients. *Intensive Care Med.* 1999;25:955–958.
9. Mayo PH, Goltz HR, Tafreshi M, Doelken P. Safety of ultrasound-guided thoracentesis in patients receiving mechanical ventilation. *Chest.* 2004;125:1059–1062.
10. McCartney JP, Adams JW 2nd, Hazard PB. Safety of thoracentesis in mechanically ventilated patients. *Chest*. 1993; 103:1920–1921.
11. Diacon AH, Brutsche MH, Soler M. Accuracy of pleural puncture sites: a prospective comparison of clinical examination with ultrasound. *Chest.* 2003;123:436–441.
12. Beckh S, Bolcskei PL, Lessnau KD. Real-time chest ultrasonography: a comprehensive review for the pulmonologist. *Chest.* 2002;122:1759–1773.
13. Dubs-Kunz B. Sonography of the chest wall. *Eur J Ultrasound.* 1996;3:103–111.
14. Reuss J. Sonographic imaging of the pleura: nearly 30 years experience. *Eur JUltrasound.* 1996;3:125–139.
15. Tsang TS, Enriquez-Sarano M, Freeman WK, et al. Consecutive 1127 therapeutic echocardiographically guided pericardiocenteses: clinical profile, practice patterns, and outcomes spanning 21 years. *Mayo Clin Proc.* 2002; 77:429–436.
16. Doelken P, Sahn SA. Thoracentesis. In: Ayres SM, ed. *Textbook of Critical Care*. Philadelphia, Pa: Elsevier Saunders; 2005:1845–1848.
17. Matalon TA, Silver B. US guidance of interventional procedures. *Radiology*. 1990;174:43–47.
18. Chang DB, Yang PC, Luh KT, Kuo SH, Yu CJ. Ultrasound-guided pleural biopsy with Tru-Cut needle. *Chest*. 1991; 100:1328–1333.
19. McLoud TC. Should cutting needles replace needle aspiration of lung lesions? *Radiology.* 1998;207:569–570.
20. Yang PC, Lee YC, Yu CJ, et al. Ultrasonographically guided biopsy of thoracic tumors. A comparison of large-bore cutting biopsy with fine-needle aspiration. *Cancer.* 1992;69:2553–2560.
21. Brown KT, Brody LA, Getrajdman GI, Napp TE. Outpatient treatment of iatrogenic pneumothorax after needle biopsy. *Radiology.* 1997;205:249–252.
22. Yankelevitz DF, Davis SD, Henschke CI. Aspiration of a large pneumothorax resulting from transthoracic needle biopsy. *Radiology.* 1996;200:695–697.
23. Charboneau JW, Reading CC, Welch TJ. CT and sonographically guided needle biopsy: current techniques and new innovations. *AJR Am J Roentgenol.* 1990;154: 1–10.
24. Heilo A. US-guided transthoracic biopsy. *Eur J Ultrasound.* 1996;3:141–151.
25. Klein JS. Interventional techniques in the thorax. *Clin Chest Med.* 1999;20:805–826, ix.
26. Moore EH. Technical aspects of needle aspiration lung biopsy: a personal perspective. *Radiology.* 1998;208:303–318.
27. Weisbrod GL. Transthoracic percutaneous lung biopsy. *Radiol Clin North Am.* 1990;28:647–655.
28. Moore EH, Shepard JA, McLoud TC, Templeton PA, Kosiuk JP. Positional precautions in needle aspiration lung biopsy. *Radiology.* 1990;175:733–735.
29. Pan JF, Yang PC, Chang DB, Lee YC, Kuo SH, Luh KT. Needle aspiration biopsy of malignant lung masses with necrotic

centers. Improved sensitivity with ultrasonic guidance. *Chest.* 1993;103:1452–1456.

30. Bandi V, Lunn W, Ernst A, Eberhardt R, Hoffmann H, Herth FJ. Ultrasound vs. CT in detecting chest wall invasion by tumor: a prospective study. *Chest.* 2008;133:881–886.
31. Diacon AH, Theron J, Schubert P, et al. Ultrasound-assisted transthoracic biopsy: fine-needle aspiration or cutting-needle biopsy? *Eur Respir J.* 2007;29:357–362.
32. Heilo A, Stenwig AE, Solheim OP, Malignant pleural mesothelioma: US-guided histologic core-needle biopsy. *Radiology.* 1999;211:657–659.
33. Pedersen OM, Aasen TB, Gulsvik A. Fine needle aspiration biopsy of mediastinal and peripheral pulmonary masses guided by real-time sonography. *Chest.* 1986;89:504–508.
34. Saito T, Kobayashi H, Sugama Y, Tamaki S, Kawai T, Kitamura S. Ultrasonically guided needle biopsy in the diagnosis of mediastinal masses. *Am Rev Respir Dis.* 1988; 138:679–684.
35. Wernecke K, Vassallo P, Peters PE, von Bassewitz DB. *Mediastinal tumors: biopsy under US guidance. Radiology.* 1989;172:473–476.
36. Yu CJ, Yang PC, Chang DB, et al. Evaluation of ultrasonically guided biopsies of mediastinal masses. *Chest.* 1991;100:399–405.
37. Tsang TM, Enriquez-sarano M, Freeman WK, et al. Consecutive 1127 therapeutic echocardiographically guided pericardiocenteses: clinical profile, practice patterns, and outcomes spanning 21 years. *Mayo Clin Proc.* 2002;77: 429–436.

This page intentionally left blank

CHAPTER 28

Ultrasound Guidance for Abdominal and Soft Tissue Procedure

Sameh Aziz, William J. Brunelli, Jr., and James S. Cain

INTRODUCTION

Ultrasound technology is readily available and can provide guidance for invasive abdominal procedures in multiple planes without ionizing radiation. Further, real-time guidance of the needle tip position allows the operator to avoid inadvertent puncture of vital structures during the performance of invasive and soft tissue procedures of the abdominal cavity. Recently, this technology has a demonstrated benefit in abdominal procedures such as paracentesis, liver and kidney biopsy, percutaneous gallbladder drainage, and abscess drainage.

Transducer selection for abdominal procedures most commonly includes a curved or phased array. A curved array allows the user to visualize a larger field, but the actual transducer size is larger than a phased array. Curved array is the transducer of choice for most abdominal procedures since a larger field can be visualized during the exam. A sector array is smaller and is most commonly used for abscesses that are close to the diaphragm and allows for an intercostal approach.

PARACENTESIS

Ultrasound use in paracentesis improves both the ease and efficiency of the procedure and reduces unnecessary abdominal punctures in patients with scant fluid. It is particularly helpful in obese patients to assess the depth to the peritoneum and in patients with loculated effusions to define the largest locule for drainage. Ultrasound use can identify intrabdominal pathology that may increase the risk of bowel perforation during the procedure.[1]

Indications

Diagnostic paracentesis is indicated as a part of the initial evaluation of patients with new onset ascites and in patients with a known history of ascites secondary to liver cirrhosis who develop clinical deterioration, including the signs and symptoms of fever, abdominal pain, rapid worsening of renal function, worsened hepatic encephalopathy, leukocytosis, acidosis, gastrointestinal bleeding or sepsis, and to rule out underlying spontaneous bacterial peritonitis.[2] Paracentesis can also be used for the evaluation of intraabdominal fluid in trauma patients. Therapeutic paracentesis is performed in patients with tense or diuretic-resistant ascites to alleviate difficulty with breathing or abdominal pain.

Contraindications

Paracentesis is contraindicated in patients with disseminated intravascular coagulation, a platelet count $<50 \times 10^9$/ liter, an international normalized ratio (INR) >2, and a local skin infection, visible scar, hematoma, or cutaneous vein at the site of needle entry.[3] Patients with underlying renal insufficiency who have an increased tendency for bleeding should be carefully evaluated prior to the procedure.

Equipment and Procedure Tray

A 3.5–5 MHz broadband curved array is preferred for the evaluation of ascites and for assistance with ultrasound-guided paracentesis.[1,4] Prepackaged paracentesis procedure kits are commercially available and contain all necessary equipment including an aspiration catheter, catheter bag, blood collection tubing, 19-gauge introducer needle with 5 French catheter, lidocaine for skin anesthesia, skin preparation solution, and dressing pack with sterile draping (Figure 28.1). For real time ultrasound guided procedures a sterile sleeve is used to cover the ultrasound probe.

Site

The optimal site for paracentesis is where the depth of ascitic fluid is maximal and the abdominal wall is the thinnest.[5] It is common practice to perform a

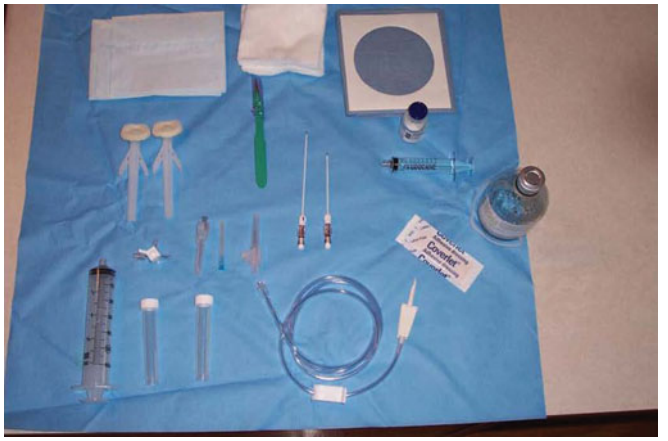


Figure 28.1. Paracentesis tray showing equipment including 5 French catheter.

paracentesis in the left lower quadrant (LLQ). Paracentesis in the right lower quadrant is generally avoided because of an increased risk of bowel perforation in patients with a distended cecum. The best location for paracentesis is 5 centimeters (cm) superior and medial to the anterior superior iliac spine. One prospective study of 52 patients with ascites secondary to cirrhotic liver disease demonstrated that the LLQ was the optimal location for paracentesis because it was easier and safer than an infraumbilical, midline approach because of a lower incidence of "dry tap" and bleeding.[5] Preprocedure scanning with ultrasound is recommended to avoid complicating factors like cutaneous veins, the inferior epigastric artery, and areas with previous scars and operations (Figure 28.2).

Procedure Description

After obtaining informed consent, the patient is asked to empty the bladder prior to the procedure to decrease the risk of urinary bladder perforation (Figure 28.3). The patient is positioned supine. Prior to starting the procedure, the four abdominal quadrants are scanned to evaluate the extent of the ascites. The liver and spleen should also be evaluated. Scanning the entire abdomen allows the identification of complicating factors for intraabdominal paracentesis like intraperitoneal septation (Figure 28.4). Noncomplicated ascites usually has a characteristic anechoic contrast on ultrasound (Figure 28.5). An abnormal density, loculation, or septation may be identified on ultrasound and presents visually as an echogenic mass.[6]

After screening the abdominal cavity with ultrasound, the optimal site is identified, cleaned, and

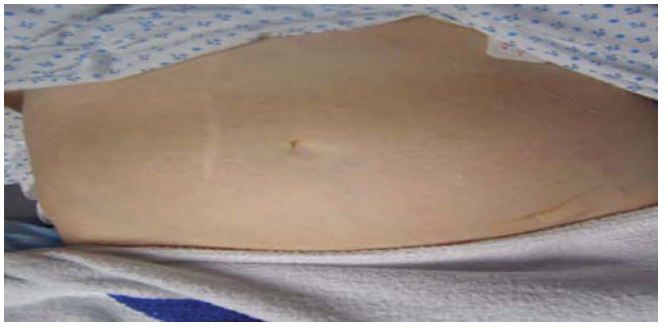


Figure 28.2. The abdomen of a patient with malignant ascites is shown. This patient had a history of a previous cholecystectomy, with a paraumbilical scar. Paracentesis was performed in the left lower quadrant, as demonstrated by the bandaid, to avoid possible adhesions in the right lower quadrant.

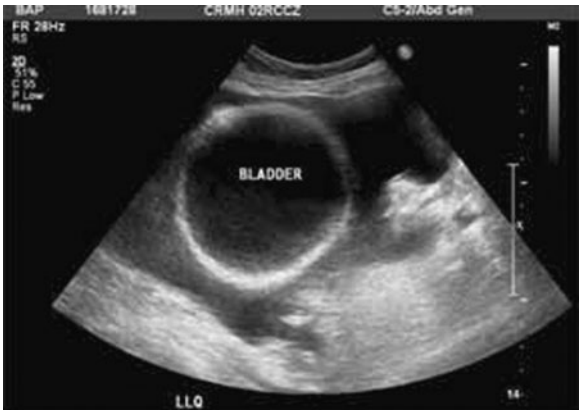


Figure 28.3. Ultrasound examination of the left lower quadrant in a patient with ascites demonstrates a collection of ascites adjacent to a full urinary bladder. A full bladder poses an increased risk of bladder perforation; therefore, the patient should be asked to empty bladder prior to the procedure.

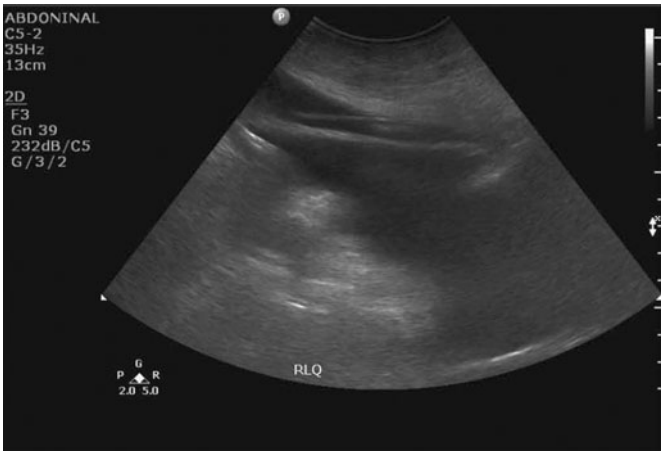


Figure 28.4. Right lower quadrant ultrasound examination for the evaluation of ascites demonstrates the presence of peritoneal fluid with intraabdominal separation representing complicated intraabdominal ascites.

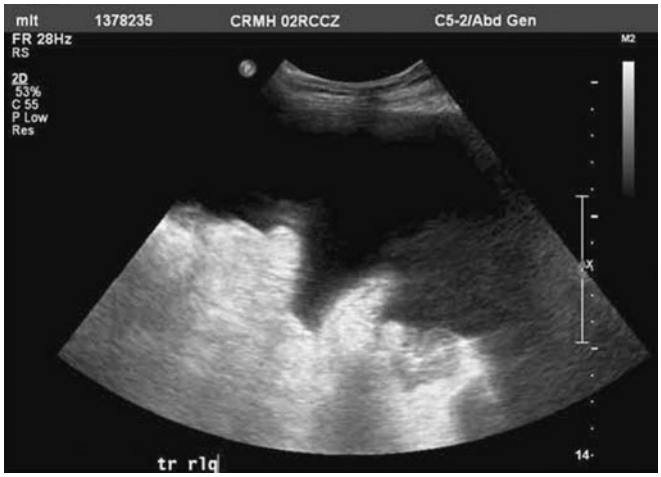


Figure 28.5. Ultrasound evaluation of the right lower quadrant demonstrated clear anechoic peritoneal fluid.

draped. The ultrasound probe is covered with a sterile sheath and the abdomen is rescanned to confirm the site of needle entry. Using a 25–30 gauge needle, the skin is infiltrated with local anesthetic using 1% or 2% lidocaine. A combination of lidocaine and sodium bicarbonate may be used to reduce the burning associated with the local injection of lidocaine. Next, a 22-gauge 1.5 cm needle can be used to infiltrate the local anesthetic to the subcutaneous tissues and anterior abdominal wall. A 19-gauge needle with an echogenic tip is then introduced into the peritoneal cavity. To avoid precipitating a peritoneal fluid leak, a "Z-line technique," which introduces the needle at an angle and avoids creating a direct tract, is recommended. A 5 French catheter can be used for drainage of the peritoneal fluid.

A free-hand technique, in which paracentesis is guided by real-time ultrasound, is recommended to avoid direct injury to the bowel for a small pocket of peritoneal fluid (Figure 28.6). In this approach, the needle is introduced into the peritoneal cavity and fluid collected under direct vision (Figure 28.7). A sample of fluid is submitted to the clinical laboratory for appropriate diagnostic studies.

For a therapeutic tap of a large volume of ascites, a catheter-over-the-needle assembly can be used. In this approach, a needle is introduced into the peritoneal cavity, and once the needle passes through the ascitic collection, a catheter is introduced over the needle. Follow-up imaging with ultrasound can document the proper and safe position of the tip of the catheter. Then, drainage tubing connected to a vacuum device can be attached to the catheter. On occasion, the bowel will

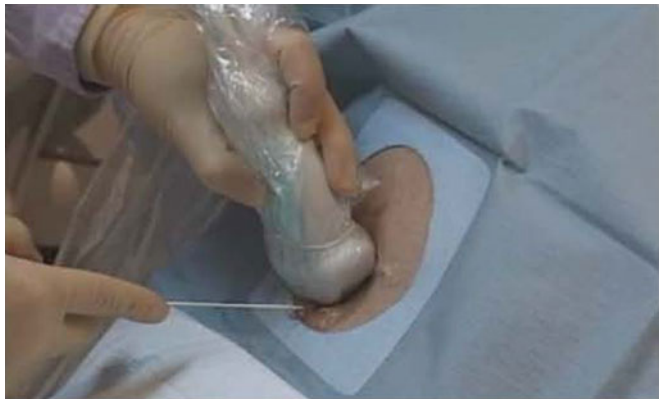


Figure 28.6. A demonstration of the "Free Hand Technique" during ultrasound-guided paracentesis.

obstruct the catheter; when this occurs, the drainage tubing can be clamped with hemostats and the tubing separated from the catheter. This allows the suction to be temporarily removed from the catheter. The catheter can then be pulled back a few millimeters until ascites again begins to escape from the catheter. The drainage tubing is reattached and the hemostats removed to continue draining the ascites.

Fluid Analysis

A number of diagnostic tests can be performed on the ascitic fluid to assist with clinical

Interpretation.[7–9] These include:

- *Gram stain and culture:* A gram stain and culture is recommended to rule out infection of the peritoneal fluid and may identify secondary peritonitis.

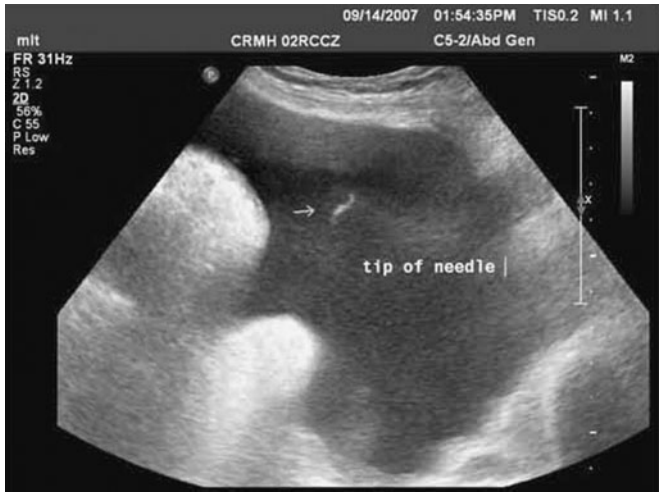


Figure 28.7. Ultrasound examination of the abdomen during real-time paracentesis demonstrates the position of the needle tip.

- *Cell count and differential:* This test allows for the diagnosis of inflammation or spontaneous bacterial peritonitis (SBP). The sensitivity and specificity depends on the polymorphonuclear (PMN) count. A PMN count >500 cells/mm^3 has a sensitivity of 70–100% and a specificity of 86–100%.
- *Glucose:* Ascitic fluid glucose is low in secondary peritonitis.
- *Albumin:* Albumin levels allow for a determination of the etiology of the ascitic fluid. The serum-ascites albumin gradient (SAAG) is the absolute difference between the serum albumin and the ascitic albumin level. A SAAG >1.1 grams/dl identifies ascites due to portal hypertension. A SAAG <1.1 grams/dl indicates an exudative ascites that can be due to infection or malignancy.
- *Total protein:* An ascitic fluid protein level >2.5 grams/dl supports a diagnosis of an exudative peritoneal effusion, while a protein level <2.5 grams/dl supports a diagnosis of a transudative effusion. The differential diagnosis associated with a transudate includes cirrhosis, congestive heart failure, portal vein thrombosis, and myxedema, and that associated with an exudative effusion includes peritoneal carcnomatosis, tuberculosis, infection, connective tissue disease, bowel obstruction, and infarction.
- *Amylase:* A high amylase level in ascitic fluid >1000 indicates leakage of pancreatic enzymes into the peritoneal cavity.
- *Triglycerides:* Chylous ascites with triglycerides level >200 mg/dl is highly suggestive for intraabdominal malignancy or infection (filariasis, tuberculosis).
- *Bilirubin*: High ascites bilirubin >6 mg/dl indicates choleperitoneum (bile in the peritoneal cavity).
- *Cytology:* Malignant cells may be present in the peritoneal fluid and indicate carcinomatosis.[7–9]

Complications

The incidence of bleeding from diagnostic paracentesis is estimated at approximately 0.2%. Hemorrhage related to large volume therapeutic abdominal paracentesis >4 liters is estimated at approximately 2%.[10] An inferior epigastric artery pseudoaneurysm has been reported in two cases after large volume paracentesis. These were treated by percutaneous embolization.[11] This complication can be avoided by scanning of the abdomen with ultrasound to define the optimal entry site that is lateral to the rectus abdominis muscles and avoids large intrabdominal veins.

Other complications may include intestinal or urinary bladder perforation, secondary peritonitis, and reaccumulation of peritoneal fluid. Large volume paracentesis, >10 liters, is usually avoided because it may lead to a reduction in central venous pressure and associated hypotension. In addition, activation of the renin-angiotensin-aldosterone system occurs, which is extremely sensitive in patients with liver cirrhosis. This phenomenon is worse in patients who have liver cirrhosis with ascites but without peripheral edema, and may precipitate hyponatremia, renal insufficiency, and hepatorenal syndrome if the patient does not receive simultaneous intravenous albumin.[12,13] This is due to the absence of edema, which can functionally reequilibrate with plasma.

ABSCESS DRAINAGE

Drainage of an abscess by using puncture and aspiration was described in the 1930s.[14]

Role of Ultrasound Guidance

Real-time sonography has the advantages of being portable, readily available at the bedside, and a success rate of approximately 90% for intraabdominal abscesses.

Ultrasound is able to differentiate between a solid mass and cyst in approximately 95% of cases.[15] Using a gray scale, the cyst will appear bright white, while the adjacent organs appear gray. This is referred to as the "light bulb" sign.[16] Ultrasound provides real-time imaging during catheter placement. When compared with computerized tomography (CT), ultrasound is more accurate in localizing and guiding the drainage of small, deep abscesses (Figure 28.8).

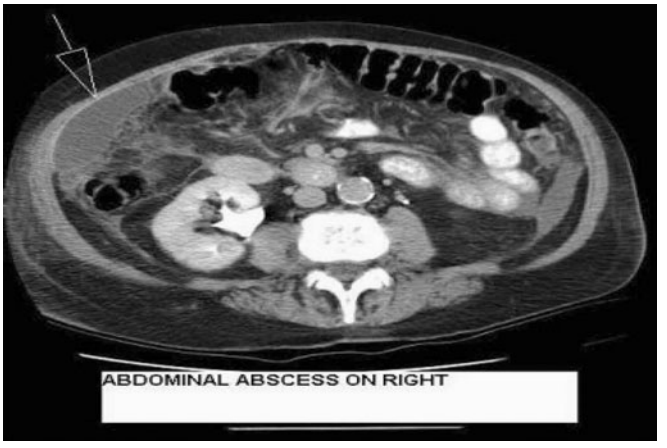


Figure 28.8. Computerized tomography (CT) scan of the abdomen demonstrating an intraabdominal abscess.

Indications

The drainage of abnormal fluid collections is recommended for fluid that is suspicious for infection. Percutaneous fluid drainage is a safe and expeditious option for critically ill patients who are too unstable for an operative intervention or need a temporizing measure to improve their condition and reduce morbidity. Percutaneous drainage provides a rapid, inexpensive, and immediate treatment in elderly, unstable patients. While this procedure is usually performed in the radiology department with the assistance of CT or fluoroscopy, the role of bedside ultrasound is important for those patients who are too sick for transportation to the radiology suite and whose transportation conveys additional risks for potential complications.

Contraindications

Percutaneous abscess drainage is contraindicated if the catheter path is through a vital organ (lack of safe route). If possible, consider correction of coagulopathy prior to the procedure.[1,16] Ultrasound may be limited in differentiating between an abscess and other abdominal fluid collections, such as blood. Bowel gas may reduce image quality. Surgical dressing or drains may limit ultrasound evaluation.

Site

The site of the abscess must be evaluated by ultrasound prior to the procedure by using a 5–7 MHz transducer; for very superficial abscesses a 10 MHz transducer may provide better resolution.[15] One needs to evaluate a safe path for catheter placement, the size of the abscess, the content of the abscess (light bulb sign), and the proximity to the skin surface.[16,17] The key to a successful procedure is a careful, well planned needle path and an appropriate angle of entry prior to starting.

Equipment

The catheter chosen for the procedure depends on the indication, with smaller catheters (6–8 French) used for aspiration of fluid collections, while larger-caliber catheters (10–14 French) may be needed for more viscous fluid collections.

Procedure Description

The procedure site is sterilized and draped. Lidocaine 1–2% is used to anesthetize the skin. A sterile sheath is used to cover the ultrasound probe. The catheter is introduced into the abscess under direct ultrasound visualization.

Different techniques are available for abscess drainage. The trocar technique is the preferred method for a large abscess with thick fluid; however, this method is associated with more severe complications due the large size of the introducer needle. In the Seldinger technique, a needle, guide wire, and dilators are sequentially introduced prior to the introduction of the catheter.

For a superficial abscess, a 22-gauge needle can be used; for deeper collections, a larger bore needle is recommended. For deeper abscesses using a Seldinger technique, the skin is prepped and draped and the ultrasound probe is covered with a sterile sleeve. The probe is held in parallel with the needle tract and the skin is anesthetized. The 18-gauge introducer needle is advanced along the parallel ultrasound plane (beam), carefully observing and avoiding surrounding anatomical structures. After placing the needle tip into the collection, a syringe is attached to the needle and a sample of aspirate is removed for cultures. The syringe is removed and a 0.035 guide wire is placed through the needle lumen into the collection. Direct visualization of the wire can be achieved by carefully tugging on the wire while observing for movement within the collection. The tract is dilated to the desired size of the catheter, most commonly a10 French, 30 cm, multipurpose "pigtail" catheter. Starting with a 6 French dilator, sequential dilation can occur with 8 and 10 French dilators. The drainage catheter is advanced over the wire and into the abscess (Figure 28.9). The metal stiffener (inner portion of the catheter mechanism) should only be advanced a few millimeters passing through

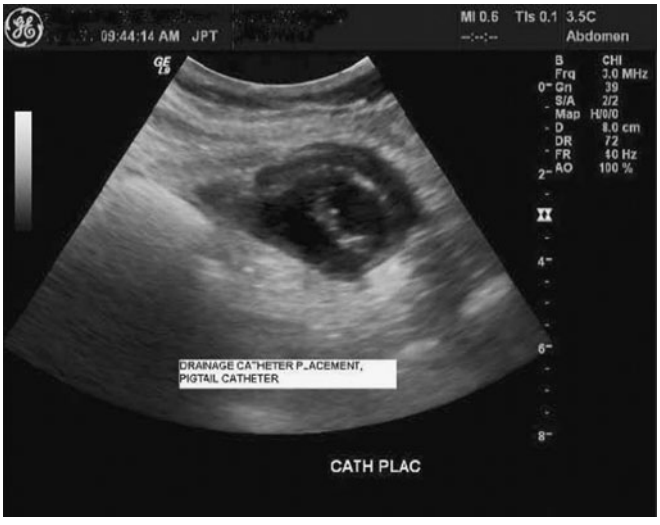


Figure 28.9. Pigtail catheter drainage of an abscess.

the border of the collection; the catheter is slightly longer than the stiffener. After passing through the wall of the collection, hold the stiffener while advancing the catheter over the stiffener into the collection. Remove the stiffener from the catheter, remove the wire, and pull the string, which causes the distal end of the catheter to coil (pigtail). The catheter is secured using either suture or an adhesive device. The catheter is attached to the drainage bag and a dressing placed over the catheter entry site. If it is difficult to visualize catheter placement, push a combination of 3 cc of sterile saline with 1 cc of air through the catheter. Perform this technique while scanning; you should see air bubbles floating within the collection.

Aggressive manipulation of the catheter after placement is discouraged to prevent bacteremia.[18] The abscess contents should be aspirated as completely as possible, followed by gentle irrigation with sterile saline. Once the catheter is in place, it should be secured and allowed to drain to gravity. Irrigation of the catheter with normal saline is recommended every four hours until the drainage fluid clears. A precise recording of daily output of the drainage is important.

Once the patient becomes more stable and the condition improves, a follow-up imaging study, such as a CT, is recommend to evaluate the position of the catheter, the extent of the abscess, and identification of nondraining areas. After completion of antibiotics, a trial of catheter lock for 24 hours is recommended prior to removal of the catheter.

Complications

Risks range from minor bleeding in 9.8% of the cases to serious complications such as perforation and bacteremia in 5% of the cases. Uncooperative patients increase the risk of complications.[18]

LIVER BIOPSY

Percutaneous liver biopsy was first described at the end of the 19th century. A second liver biopsy was described by Menghini in the 1950s, and ultrasound guidance for percutaneous biopsy was introduced in the late 1970s.[19] Ultrasound can provide a real-time evaluation of the liver, the lesion, and needle placement. The addition of color Doppler improves the ability to visualize and avoid blood vessels that may be in the track of the needle.[20] While diagnostic yield may not be improved with ultrasound-guided liver biopsy, it often results in fewer attempts, fewer complications, less pain, and shorter procedure times.[21]

Indications

The most common indications for liver biopsy are:

- Diagnosis and staging of hepatitis B or C
- Chronic alcoholism
- Diagnosis of Wilson's disease
- Alpha 1-antitrypsin deficiency
- Hemochromatosis
- A liver mass
- A focal liver lesion
- Elevated liver enzymes after liver transplantation
- Unexplained abnormal liver biological tests.[2]

Contraindications

Liver biopsy is considered a safe procedure when performed by skilled practitioners. The reported incidence of bleeding ranges from 0.06% to 1.7%, with an associated mortality rate between 0.009% and 0.33%.[21] Most of the bleeding complications are caused by a penetrating injury to a branch of the hepatic artery, portal vein, or puncture of the gallbladder. There is controversy about the need to withhold aspirin or nonsteroidal antiinflammatory drugs prior to the procedure due to their potential affect on platelets. Relative contraindications to liver biopsy include ascites, empyema, or infection below the right diaphgram. Absolute contraindications include coagulopathy, an INR >1.4, a platelet count $<60{,}000/mm^3$, a prolonged bleeding time, a liver mass with suspicion of a hemangioma, or an uncooperative patient.

Role of Ultrasound Guidance in Liver Biopsy

Ultrasound-guided biopsy of the liver can be performed either by the free-hand technique, in which the operator holds the probe in one hand and the biopsy needle in the other, or by using a biopsy adaptor, which is a tool that attaches to the ultrasound probe and guides the needle to the target area.

For the diagnostic accuracy of fibrotic liver disease, a biopsy sample of >25 mm is needed. After evaluation of the liver lesion with B-mode, gray-scale sonography, a color Doppler sonogram is used for evaluation to view the anatomic course of blood vessels and biliary ducts and to provide a precise evaluation of vasculature of

the liver mass. Highly vascular liver lesions like hemangiomas may be echo-poor and show heterogeneity.[22]

Ultrasound-guided liver biopsy improves specimen adequacy regardless of operator experience.[23] By providing guidance for needle angulation, ultrasound helps to guide the operator with positioning the needle.[24] This is particularly helpful because selective clinical criteria like obesity, difficult liver percussion, or chest deformity alone are often insensitive selection criteria for determining which patients may need an ultrasound-guided biopsy.[25–27] In children, ultrasound-guided percutaneous liver biopsy has been more accurate, has had a higher success rate and fewer complications likely due to a reduced number of passes, and has allowed the needle to be directed away from large intrahepatic vessels, gallbladder, lungs, and kidneys.[27–28] Ultrasound also allows the operator to detect significant postprocedure complications, such as hepatic hematomas, following percutaneous liver biopsy.

Tense ascites often prevents adequate tissue sampling because the liver may "bounce" away during the procedure. In addition, bleeding may be difficult to control. Ultrasound guidance positively influences these outcomes by directly visualizing the liver; avoiding intervening structures within the procedure track such as the lung, gallbladder, a large central vessel, or colonic loop; andreducing the incidence of bleeding. In one study, ultrasound guidance led to a change in procedure site in 15.1% of patients.[29]

Equipment and Needles

There are three types of needles that can be used in liver biopsy: (1) suction needles (like Menghini needle), (2) cutting needles (like Tru-cut needles), and (3) spring-loaded cutting needles.

Fine needle aspiration biopsy (FNAB) is preferred for focal liver lesions because it has a high sensitivity and specificity for the detection of malignancy. A Tru-cut needle has a high diagnostic yield in patients with liver cirrhosis because of the ability to cut through the liver, which results in better preservation of tissue architecture.[30,31] However, there is a higher risk of bleeding with Tru-cut needles.

Procedure Description

Ultrasound-guided, percutaneous liver biopsy is usually performed with the patient in the supine position, with the right arm raised above the head. The patient is asked to take a deep breath and hold it. Percussion is performed between the anterior and midaxillary lines during deep inspiration beginning under the right breast and progressing in a caudal direction to the point of first maximal dullness; a site for biopsy is marked on the skin in the interspace below the percussion line, usually at the midaxillary line. The liver biopsy is performed in expiratory apnea. A transthoracic approach is preferred. First, a longer needle (22-gauge) is used to administer local anesthetic along the needle track, a small skin incision is then performed to alleviate skin resistance. After identifying the safe needle tract to the lesion that avoids the bowel, major vessels, diaphragm, and gallbladder, the needle is introduced adjacent to the transducer into the peritoneal cavity and into the biopsy site while maintaining visualization of the needle in the middle of the ultrasound field using a slow, rocking motion of the transducer. A steering attachment can be applied to the transducer to help guide the needle into the exact location. Alternatively, the coaxial technique allows the lesion to be visualized and focused in the center of the field and the needle is introduced along the longitudinal axis of the transducer as a straight arrow entering the biopsy site (Figure 28.10). Postprocedure ultrasound evaluation is recommended to rule out acute hematoma formation. After the procedure, the patient is positioned on the right side for at least two hours, with frequent monitoring of vital signs. Prophylactic antibiotics are recommended for patients with underlying heart disease who are at risk for endocarditis.

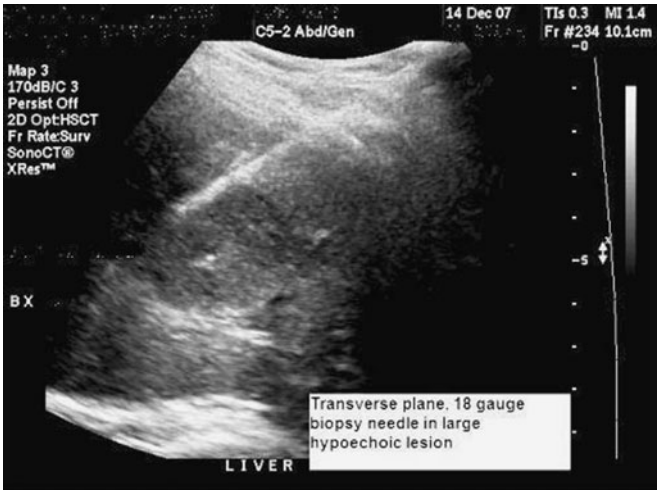


Figure 28.10. Liver biopsy of hypoechoic liver lesion.

Complications

Pain, ranging from moderate to severe in intensity, is the most frequent complication of percutaneous liver biopsy occurring in approximately 30% of cases, and

is responsible for approximately 4% of postprocedure hospital admissions.[32–34] The most common source of postprocedure pain is related to injury to the lung, irritation to the pleural, and subcapsular bleeding.

The mortality related to percutaneous liver biopsy is 0.01–0.1%, and is mainly related to bleeding or biliary peritonitis due to puncture of the gallbladder. The risk of major bleeding secondary to injury to intra- or extrahepatic vasculature is 0.12–0.34%. Intrahepatic bleeding is associated with pain due to stretching of the capsule. Severe intrahepatic bleeding requires evaluation with angiography and potentially embolization of the bleeding artery. Resection of a portion of the liver may be needed if bleeding is uncontrollable. One reported case of a delayed hemorrhage after percutaneous liver biopsy occurred 17 days postprocedure and was related to a pseudoaneurysm of a branch of the right hepatic artery and associated with an arterioportal venous fistula.[33]

GALLBLADDER DRAINAGE

Indications

Acute cholecystitis is one of the main indications for drainage of the gallbladder and achieves similar clinical outcomes to percutaneous cholecystostomy.[35,14] Percutaneous cholecystostomy is recommended in critically ill patients when surgical intervention is considered risky. Percutaneous cholecystostomy can provide easy access for cholangiography prior to surgery, allowing for identification of the anatomy of the biliary duct and the pathogenesis of the underlying disease.[36] The failure to drain the gallbladder may lead to empyema, perforation, abscess, peritonitis, and sepsis. There is a high positive predictive value to diagnosing cholecystitis when ultrasound evaluation shows gallstones, gallbladder wall thickening, and a positive Murphy's sign.[14]

Acute acalculous cholecystitis (AAC) is a serious disease with high mortality and morbidity in contrast to calculous cholecysitis. Percutaneous cholecystostomy is the best therapy for critically ill patient with AAC.[37] Surgical consultation for patients that undergo percutaneous cholecystostomy but do not improve within 24 hours is important to ensure that early surgical intervention can be provided if needed.[38]

Contraindications

The contraindications for the procedure include coagulopathy, INR above 1.2, platelets $<80.000/mm^3$, prolonged bleeding time, liver masses with suspension of hemangioma and an uncooperative patient.[39]

Equipment and Procedure Tray

A 0.035 guide wire such as a 3 mm J-type, 18-gauge needle with echogenic tip (7 cm, 10 cm, or 15 cm), 6 and 8 French dilators, 8 French multipurpose catheter, 3.5 MHz or 5.0 MHz transducer with curved array.

Procedure Description

The procedure is usually performed with the patient in the supine position, with right arm raised above the head. The fundus of the gallbladder is the preferred entry point to be used for the needle and the catheter. The gallbladder may be drained through a transhepatic or direct transperitoneal approach. The transhepatic approach is preferred because of the lower risk of bowel perforation, peritonitis, and loss of access after decompression of the gallbladder. In patients with acute cholecystitis, the distended gallbladder usually extends below the liver margin and is easier to access for direct puncture.[38]

The entry point for the needle is marked, the skin is cleaned, and appropriate drapes are placed. The skin is anesthetized and a 3 mm incision is made with a #11 blade. Placing the transducer parallel to the needle tract, the needle is traversed through the tissues and into the gallbladder, assuring that the gallbladder is entered through the fundus. About 5 milliliters (mL) of bile is removed to partially decompress the gallbladder and the fluid is sent for gram stain and cultures. Placement of a drainage catheter can be performed using a trocar device or for placement of pigtail-shaped drainage using the Seldinger technique.

In the Seldinger technique, a guide wire is placed through the needle into the gallbladder, with real-time observation of the ultrasound monitor. The needle is removed, and the tract is dilated sequentially using first a 6 French and then an 8 French dilator. If there is resistance when advancing the dilator, smaller dilators can be used and sequential one-size increments can be attempted. The catheter and stiffener are then advanced over the wire, into the lumen of the gallbladder. Direct visualization is necessary to ensure that the catheter has advanced past the gallbladder wall. While holding the stiffener, advance the catheter into the gallbladder. It is essential to advance the catheter far enough so that the drainage ports are inside the gallbladder. After placing the catheter, the stiffener and wire are removed and the distal end of the catheter is coiled by pulling the string. The catheter is then secured and attached

to a drainage bag. A final scan should be performed and an image recorded. If catheter placement is not achieved, a contrast study may be performed. Up to 90% of patients with cholecystitis show an improvement in symptoms after drainage.[38]

Complications

The most common complications of the procedure are bleeding, bile leakage, and catheter dislodgment. Failure to develop a mature track may occur in critically ill patients with underlying medical problems that intervene with the healing process. Other complications such as bowel perforation and vasovagal reactions have been reported.[39]

RENAL BIOPSY IN THE INTENSIVE CARE UNIT

Indications

The renal biopsy is used to diagnose and guide the treatment of kidney disease. In the intensive care unit (ICU) setting, renal biopsy should be performed rarely to determine the cause of renal dysfunction in situations where the added information will guide therapy. The cause of acute renal failure in ventilated patients can be determined without biopsy in most cases.[40]

The most likely ICU scenarios calling for a kidney biopsy are the pulmonary renal syndromes (PRS) due to Goodpasture syndrome, the antineutrophil cytoplasmic antibody (ANCA) vasculidites, or rapidly progressive glomerulonephritis.[41,42] The combination of respiratory failure with acute renal failure can rapidly lead to death,while the diagnostic differential of the condition leads to therapeutic dilemmas (antibiotics vs. immunusuppression). Biopsy can provide the correct information for treating the condition. Histologic confirmation of diagnosis should be obtained, if possible, prior to initiating therapy.[40]

Biopsy should be performed when:

1. The diagnosis cannot be obtained by other tests
2. The disease appears to be renal and diagnosable (this presumes a high likelihood of renal disease and pathology support to rapidly process and interpret the biopsy)
3. The patient will benefit from the information obtained
   - The interpretation will be timely
   - There are therapeutic choices that depend on the result
   - The patient agrees to the potential treatment change
4. There are no contraindications to biopsy[43,44]

Nephrotic Syndrome

The nephrotic syndrome, edema with heavy proteinuria, and low serum albumin could potentially require renal biopsy in the ICU. Usually, however, the nephrosis of diabetes, membranous, IgA, human immunodeficiency virus (HIV)-associated nephropathy, heroin-associated nephropathy (HAN), or focal and segmental glomerulosclerosis (FGS) can await stabilization and biopsy can be done in the routine manner.

Acute Nephritic Syndrome

Rapidly progressive glomerulonephritis (RPGN) requires renal biopsy to diagnose and guide therapy. The diseases involved are often aggressive and fatal. The primary diagnoses to be considered are systemic lupus erythematosus (SLE) and the vasculitides (Wegener's granulomatosis and their variants). In the ICU setting, these syndromes may be confused with severe pneumonia.[41,42] Immunosuppressive treatment is indicated for SLE and Wegener's granulomatosis (with or without plasmapheresis). It is contraindicated for an infectious process.

Acute Renal Failure

A renal biopsy for the diagnosis of the commonly diagnosed acute renal failure, the result of low blood flow or pressure in the setting of infections, nephrotoxins, or shock does not usually require renal biopsy. The occasional patient with unexpected failure to recover can usually be stabilized and biopsied in a controlled setting.[43]

Chronic Renal Failure (CRF)

Several chronic renal diseases have the potential to complicate acute critical care, and renal biopsy may be considered to guide therapy. Diabetic nephropathy (DN) is the most common cause of chronic kidney disease (CKD) in the United States (US). Additional renal insults can result in additional loss of renal function. The resulting "acute on chronic" renal failure can cloud the diagnosis and treatment of critical illness, especially when the diagnosis of RPGN must be excluded. Renal biopsy is rarely required.

Allograft Dysfunction

Biopsy of a dysfunctional renal allograft in the ICU is easier than the biopsy of native kidneys. The

allograft is closer to the surface and is relatively immobile. Acute rejection must be ruled out, while other causes of acute renal failure, such as acute tubular necrosis, cyclosporine toxicity, and recurrent primary glomerulonephritis, are considered. Ultrasound guidance is helpful in determining the depth to the organ and the method for avoiding other structures.

Risks

The primary risk from the standard percutaneous renal biopsy is bleeding. Gross hematuria is present in low percentages of patients.[44] Properly performed renal biopsies can result in hematuria, both gross and microscopic. The uncomplicated renal biopsy in the past was admitted for at least 24 hours of observation, but recent advances in biopsy preparation and technique have allowed this to become an outpatient procedure in most cases. Some recommend a 24-hour period of observation due to the late appearance of some complications.[44] These improvements apply to the ICU setting as well, allowing much more flexibility and safety in the procedure. The advent of ultrasonography allows bedside real-time guidance in the ICU, as opposed to the former practice of finding the general location and depth of the kidney for the US-guided operator, and the use of intravenous pyelogram (IVP)-guided or open renal biopsy in the past.

In addition, the use of the "free hand" biopsy gun allows more flexibility in patient position, as experienced user of the ultrasound machine can guide the needle safely to the target organ. These guns have also smaller needles, which results in less bleeding while still obtaining adequate specimens for diagnosis. Voss and colleagues reported no reduction in bleeding compared with a Vim Silverman needle, apparently with excellent results, but did report shorter hospital stays.

The use of a trocar to relocate the kidney for second and third biopsy has reduced the time and number of passes required to perform the procedure. This should facilitate biopsy of ventilated patients.

In some institutions, the use of transvenous renal biopsy has been advocated in the patient with bleeding problems. These improvements allow more aggressive diagnostic approaches to be taken without compromising patient safety.[45]

Other risks to consider in addition to bleeding include pain, infection, transfusion, loss of renal function, loss of limb or organ function, and loss of life. In the ICU setting, with PRS as a diagnosis, these are managable, relatively less significant issues.

Contraindications

In the past,several renal conditions have been considered too risky to biopsy due to the danger in outcome, but the low rate of complication for renal biopsy in general coupled with recent technical advances have allowed most of these conditions to be biopsied. The physician is responsible for weighing the risks and benefits and determining the best course of action.[40,43,45,46]

Solitary Kidney, Horseshoe Kidney

The risk for a solitary or horseshoe kidney is that controlling bleeding could require a nephrectomy. Newer, intraarterial techniques to occlude bleeding arteries and avoid loss of organ can be used in most cases.[44]

Urinary Tract Infection (UTI)

The presence of a UTI is a relative contraindication to renal biopsy due to the risk of bacterial spread outside of the urinary tract. The use of antibiotics may allow a biopsy of the patient with a UTI. In general, the biopsy of anyone with pyelonephritis is contraindicated.[43,44]

Renal Tumor

The risk of spreading malignant cells to extrarenal sites must be considered. The usual practice is to perform a nephrectomy for suspected malignancy if the patient's renal function is adequate and age and condition do not preclude it. Urologic consultation is often helpful in these circumstances. In the setting of a PRS, pulmonary metastasis may be the cause of the problem.[44]

Bleeding Disorders

Correction of a bleeding disorder from a factor deficiency, anticoagulation, or platelet deficiency or dysfunction before the biopsy will allow the procedure to proceed safely. Uremic platelet dysfunction can be controlled with estrogen therapy or transfusion,[47] thrombocytopenia with transfusion. An open biopsy would have been the primary recommendation in the recent past, but the advent of transjugular biopsies[45] and biopsy guns[9,10] has made these the preferred approaches.

Anemia

Anemia, especially a hematocrit $<30\%$, is associated with prolonged postbiopsy bleeding. Transfusion and erythropoietin therapy will often correct the problem. The correction of ongoing losses and evaluation for the cause of the anemia are advised.

Hypertension

Correction of uncontrolled hypertension is advised prior to renal biopsy due to an increased risk of hemorrhage. Donadio and Buxo report a higher incidence of postbiopsy bleeding in hypertensive patients.[48]

Patient Unable to Cooperate with Procedure

The noncompliant patient is a contraindication to the procedure; the patient is asked to hold breath briefly to stop the movement of the kidney, which occurs normally with respiration. The patient must be awake and cooperative. Light sedation to relieve anxiety is administered; general anesthesia is not routinely used.

The intubated patient can be fully anesthetized. Assistance to provide an expiratory hold to complete the biopsy will be needed. In both of these settings, the option of transjugular biopsy or open biopsy should be chosen if that is in the best interest of the patient.

The original position for performing a renal biopsy was sitting up on the edge of the bed, and the right kidney was usually biopsied. The usual procedure now is to have the patient prone, and the left organ is usually the target of choice. There is no contraindication to creative positioning of the patient to allow for mechanical ventilation. No other requirements are necessary, as long as the procedure is performed by the experienced sonographer using real-time guidance and reasonable caution.

Procedure Description

Prebiopsy Preparation

Review of the history and physical, removal or correction of anticoagulation, control of blood pressure, review for and correction of urinary tract infection. A renal ultrasound, if not previously done, may be performed at the time of the procedure. Review for obstruction, mass, polycystic kidney disease, or nephrolithiasis. Gross hematuria, if present, should be explained and other causes that may mimic PRS ruled out. Rule out pregnancy with a beta human chorionic gonadotropin (beta-hCG). Proper permission must be obtained. Appropriate laboratory testing must be performed, including a prothrombin time, a partial thromboplastin time, a basic metabolic panel, complete blood count; type and screen two units of packed red blood cells. Anemia should be corrected as needed prior to the procedure. Sedation may be given 30 minutes prior to the procedure.

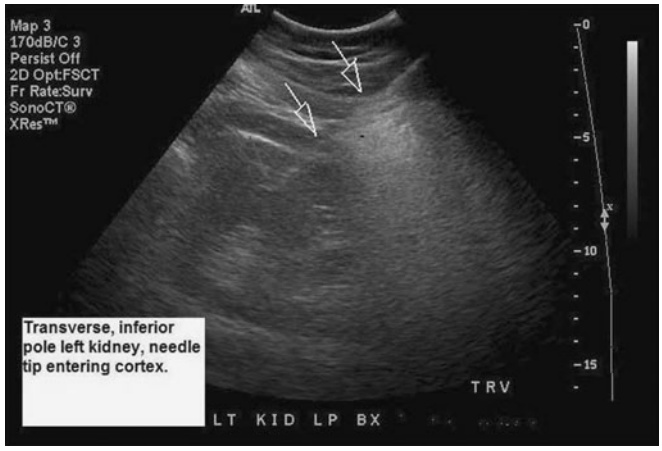


Figure 28.11. Renal biopsy showing the position of the needle tip entering the cortex.

The Procedure

The patient is placed face down; this may be difficult for a ventilated patient, and pulmonary or anesthesia support may be needed to ensure the patient's safety. The kidney is localized, and the presence of two kidneys free of obstruction, mass, or tumor is documented. With ultrasound guidance, the skin over the lower pole of the kidney is marked at end inspiration. The skin is prepped in a sterile manner and lidocaine injected superficially. A nick in the skin with a small blade is made to pass the needle through. Bleeding is controlled with direct pressure.[44]

A small-gauge spinal needle is introduced to the surface of the kidney and the location is verified either by direct visualization or using ultrasound (Figures 28.11 and 28.12). The needle will move in a vigorous arc cephalad to caudad with respiration when it is in the

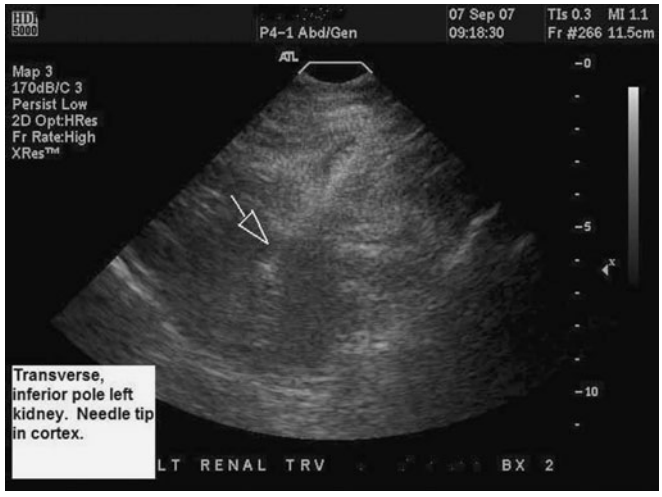


Figure 28.12. Renal biopsy showing the position of the needle tip in the cortex.

renal parenchyma. The operator's hands should not impede movement of the needle. When renal tissue has been reached, the surface of the kidney is anesthetized with 1% lidocaine. The depth of the needle needs to be marked closely and lidocaine injected as the spinal needle is withdrawn. The patient needs to be told to stop breathing when the operator has contact with the needle and the operator needs to be mindful of the patient's respiratory status because discomfort and incomplete exhalation may occur during a biopsy pass. The danger here is that a firmly held needle could tear the renal tissue when the kidney moves with respiration. The spinal needle is withdrawn and a biopsy needle inserted to the same depth. Once again, the location of the needle at the surface of the organ is verified by movement with respiration or by real-time ultrasound (Figures 28.11 and 28.12). Respirations are held and the biopsy device, Tru-cut needle or biopsy gun, is activated. The needle is withdrawn and the patient instructed to breathe. The patient should be asked how he or she is doing, which allows the operator to assess pain and also verifies responsiveness. The tissue is removed from the needle and the presence of glomeruli verified by direct inspection. A magnifying lens or dissecting microscope is helpful here. This procedure is repeated until adequate tissue for diagnosis is obtained. The tissue is submitted to nephropathology for light microscopy, immunofluorescent microscopy, and electron microscopy. The light microscopy specimen is placed in formaldehyde, the electron microscopy specimen is submitted in glutaraldehyde, and the immunofluorescence is submitted on ice for immediate frozen sections or is placed in Michel's fixative.[44]

Postbiospy Care

After the biopsy, vital signs should be closely monitored for hypotension, tachycardia, and signs of internal bleeding. Urinalysis to detect hematuria and serial hematocrit determinations is performed. Most complications are evident within 8 hours. A total of one third of complications occur after the first 8 hours, so patients must continue to be watched closely.[49] Most light microscopy and immunofluoresence can be processed and interpreted within 24 hours. Direct and clear communication with the pathologist will be helpful.

SUMMARY

Overall, there are a number of benefits to the use of ultrasound for abdominal and soft tissue procedures that make it an ideal technique for the critically ill patient. The technique improves the safety and efficiency of bedside procedures and prevents the difficult and often dangerous transportation of the critically ill patient to the radiology suite. In trained and experienced hands, it is a powerful diagnostic tool.

References

1. Shameem R. Nazeer, Dewbre H, Miller AH. Ultrasound-assisted paracentesis performed by emergency physicians vs the traditional technique: a prospective randomized study. *Am J Emerg Med.* 2005;23:363–367.
2. Thomsen TW, Shaffer RW, White B, et al. Paracentesis. *N Eng J Med.* 2006;355:e21.
3. Mc Vay PA, Toy PT. Lack of increased bleeding after paracentesis and thoracentesis in patients with mild coagulation abnormalities. *Transfusion.* 1991;31:164–171.
4. Nicolaou S, Talsky A, Khashoggi K, et al. Ultrasound-guided interventional radiology in critical care. *Crit Care Med.* 2007;35(suppl 5):S186–S197.
5. Sakai H, Sheer TA, Mendler MH, et al. Choosing the location for non-image guided abdominal paracentesis. *Liver Intern.* 2005;25:984–986.
6. Blavias M. Emergency diagnostic paracentesis to determine intraperiotneal fluid identity discovered on bedside ultrasound of unstable patients. *J Emerg Med.* 2005;29: 461–465.
7. McGibbon A, Chen GI, Peltekian KM, et al. An evidence-based manual for abdominal paracentesis. *Dig Dis Sci.* 2007;52:3307–3325.
8. Mittal R, Dangoor A. Paracentesis in the management of ascites. *Br J Hosp Med.* 2007;68(9):M162–M165.
9. Lipsky MS, Sternbach MR. Evaluation and initial management of patients with ascites. *Am Fam Phys.* 1996;15:1327–1333.
10. Webster ST, Brown KL, Lucey MR, et al. Hemorrhagic complications of large volume abdominal paracentesis. *Am J Gastroenterol.* 1996;91:366–368.
11. Lam EY, Mclafferty RB, Taylor LM, et al. Inferior epigastric artery pesudoaneurysm a complication of paracentesis. *J Vasc Sur.* 1998;28:566–569.
12. Schouten J, Michielsen PP. Treatment of cirrhotic ascites. *Acta Gastroenterol Belg.* 2007;70(2):217–222.
13. Zakim, Boyer. *Hepatology*. 4th ed. Philadelphia: Saunders: 2003.
14. Rumack CM, Wilson SR, Charboneau JW. *Diagnostic Ultrasound.* 4th ed. Philadelphia: Elsevier Science; 2004.
15. Tibbles CD, Porcaro W. Procedural applications of ultrasound. *Emerg Med Clin N Am.* 2004; 22:797–815.
16. Conard MR, Sanders RC, James E. The sonolucent "Light Bulb" sign of fluid collections. *J Clin Ultrasound.* 1976;4:409–415.

17. McGahan JP. Aspiration and drainage procedures in the intensive care unit: percutaneous sonographic guidance. *Radiology.* 1985;154:531–532.
18. Irwin RS, Rippe JM, Curley FJ, Heard SO. *Procedures and Techniques in Intensive Care Medicine.* 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2003.
19. Bravo AA, Sheth SG, Chopra S. Liver biopsy. *N Engl J Med.* 2001;344:495–500.
20. JPolakow J, Ladny JR, Dzieciol J, et al. Ultrasound guided percutaneous fine-needle biopsy of the liver: efficacy of color Doppler sonography. *Hepato-Gastroenterology*. 1998;45:1829–1830.
21. Farrell RJ, Smiddy PF, Pilkington RM, et al. Guided versus blind liver biopsy for chronic hepatitis C: clinical benefits and cost. *J Hepatol.* 1999;30:580–587.
22. Duysburgh I, Michilesen P, Fierens H, et al. Fine needle trucut biopsy of focal liver lesions. *Dig Dis Sci.* 1997;42:2077–2081.
23. Shiffman AK. Percutaneous liver biopsy in clinical practice. *Liver Int.* 2007;27:1166–1173.
24. Caturelli E, Giacobbe A, Facciorusso D, et al. Percutaneous biopsy in diffuse liver disease: increasing diagnostic yield and decreasing complications rate by routine ultrasound assessment of puncture site. *Am J Gastroenterol.* 1996;91:1318–1320.
25. Terjung B, Lemnitzer I, Ludwig F, et al. Bleeding complications after percutatneous liver biopsy. *Digestion.* 2003;67:138–145.
26. Ahmed M, Riley T. Can one predict when ultrasound will be useful with percutaneous liver biopsy? *Am J Gastroenterol.* 2001;96:547–549.
27. Nobili V, Comparcola D, Sartorelli MR, et al. Blind and ultrasound-guided percutaneous liver biopsy in children. *Pediatr Radiol.* 2003;33:772–775.
28. Michielsen PP, Duysburgh IK, Francque SM, et al. Ultrasonically guided fine needle puncture of focal liver lesions: review and personal experience. *Acta Gastro-Enterologica Belgica.* 1998;61:158–163.
29. Chevallier P, Ruitort F, Denys A, et al. Influence of operator experience on performance of ultrasound-guided percutaneous liver biopsy. *Eur Radiol.* 2004;14:2086–2901.
30. Colombo M, Del Ninno E, De Franchis R, et al. Ultrasound-assisted percutaneous liver biopsy: superiority of the Tru-Cut over the menghini needle for diagnosis of cirrhosis. *Gastroenterology.* 1988;95:487–489.
31. Lindor KD, Bru C, Jorgensen RA, et al. The role of ultrasonography and automatic-needle biopsy in outpatient percutaneous liver biopsy. *Hepatology.* 1996;23:1079–1083.
32. Riley T. How often does ultrasound making change the liver biopsy site? *Am J Gastroenterol.* 1999;94:3320–3322.
33. Sherlock S. Aspiration liver biopsy. *Lancet.* 1945;29:397–401.
34. Kowdley KV, Aggarwal AM, Sachs PB. Delayed hemorrhage after percutaneous liver biopsy. *J Clin Gastroenterol.* 1994;19:50–53.
35. Chopra S, Dodd GD III, Mumbower AL, et al. Treatment of acute cholecystitis in non-critially ill patient at his surgical risk: comparison of clinical outcomes after gallbladder aspiration and after percutaneous cholecystostomy. *AJR.* 2001;176:1025–1031.
36. Ralls PW, Colletti PM, Lapin SA, et al. Real-time sonography in suspected acute cholecystitis, prospective evaluation of primay and secondary signs. *Radiology.* 1985;155:767–771.
37. Wong S-R, Lee K, Kuo K-K, et al. Ultrasound-guided percutaneous transhepatic drainage of gallbladder followed by cholecystectomy for acute cholecystitis. 10 years' experience. *Kaohsiung J Med Sci.* 1998;14:19–24.
38. Hultman, C. Scott, Herbst, Chales A. The efficacy of percutaneous cholecystostomy in critically ill patients. *Am Surg.* 1996;62(4):263–269.
39. Kadir S. *Teaching atlas of interventional radiology, nonvascular interventional procedures*. Thieme Medical Publishers: New York, New York; 2005.
40. Conlon PJ, Kovalik E, Schwab SJ. Percutaneous renal biopsy of ventilated intensive care unit patients. *Clin Nephrol.* 1995;43:309–311.
41. Falk RJ. ANCA-associated renal disease. *Kidney Int. Vol 38*, 1990;38:998–1010.
42. Urizar RE, Mcgoldrick D, Cerda J. Pulmonary Renal Syndrome Its clinicopatholigic approach in 1991. *NYS Med J Med.* 1991;91:212–221.
43. Parrish AE. Complications of percutaneous renal biopsy. *Clin Nephrol.* 1992;38:135–141.
44. Radford MG, Donadio JV, Holley KE, Bjornsson J, Grande JP. Renal Biopsy in Clinical Practice. *Mayo Clin Proc.* 1994;69:983–984.
45. Rychlik I et al. Transjugular renal biopsy. Our experience with 67 cases. *Kidney Blood Press Res.* 2001;24:207–212.
46. Voss DM, Lynn KL. Percutaneous renal biopsy: an audit of a 2 year experience with the Biopty gun. *New Zealand Med J.* 1995;25(108):8–10.
47. Sloand JA, Schiff MJ. Beneficial effect of low dose transdermal estrogen on bleeding time and clinical bleeding in uremia. *Am J Kidney Dis.* 1995;26:22–26.
48. Diaz-Buxo JA, Donadio JV. Complications of percutaneous renal biopy: an analysis of 1000 consecutive biopsies. *Clin Nephrol.* 1975;4:223–227.
49. Whittier WL, Korbe SM. Renal biopsy update. *Curr Opinion in Nephrol Hypertens.* 2003;13:661–665.

This page intentionally left blank

CHAPTER 29

Peripheral and Central Neuraxial Blocks in Critical Care Medicine

Santhanam Suresh

INTRODUCTION

The role of neuraxial blocks and peripheral nerve blocks in the critical care setting has vastly improved due to the use of ultrasonography. Despite the available technology, the use of these techniques in critical care remains rare. This chapter will provide examples of current and potential uses for the use of central and peripheral nerve blocks using ultrasound in a critical care setting.

EQUIPMENT

Although the physics and the use of equipment has been described previously (Chapter 2), specific discussion of the use of particular transducers for certain procedures is important. The use of a linear probe can help to localize nerve using ultrasonography. Sterile precautions should always be exercised prior to the performance of these blocks. Although the use of a sterile sheath can be very helpful, in an acute setting, a sterile Tegaderm™ can be used to cover the probe and effectively place nerve blocks in an intensive care unit (ICU). Nerves can appear anechoic, hypoechoic, or hyperechoic, depending on the particular plexus. Unlike vascular structures, they are not always hypoechoic and therefore color is unable to delineate them. A portable ultrasonography machine that can be brought to the patient's bedside to scan the patient and place the blocks is most useful in the ICU. Although sedation may be required in some instances, especially if infants and children are involved, most blocks can be performed with the superficial subcutaneous injection of local anesthetic. The advantage of ultrasonography is the ability to have a single pass directly to the proximity of the nerve structure and provide the block without the need for nerve stimulation. Nerve blocks are performed for a variety of reasons in the ICU, including diagnostic reasons, pain control, and managing vascular insufficiency (Table 29.1).

LOCAL ANESTHETIC SOLUTION

Any long-acting local anesthetic solution, mostly amides, are used for pain control using a regional anesthetic technique.[1] Although the commonly used, long-acting, local anesthetic bupivacaine is a dextro-enantiomer and may have greater cardiovascular toxicity compared with the levo-enantiomer, it is still routinely used in most clinical practices.[2,3] The dose of local anesthetic solution has to be contained within the toxic dosage allowable. A dose of <4 mg/kg will ensure a reasonable degree of safety, although careful aspiration should be carried out prior to injection. Ultrasonography has advanced our ability to identify vascular structures prior to injection. Newer levo-enantiomers, ropivacaine and levobupivacaine, although safe, cannot be considered completely immune to the cardiovascular and neurotoxicity of local anesthetic solutions. A detailed description of local anesthetics and their toxicity can be found in many standard pharmacology and anesthesia textbooks. A rule of thumb is that toxicity varies for different blocks decreasing in the progression from intercostal block, caudal blocks, epidural blocks, to peripheral nerve blocks. Local anesthetic toxicity includes seizures and cardiovascular collapse. A newer modality of treating the toxicity with intravenous intralipid is gaining popularity.[4] In the ICU, it may be reasonable to have the availability for lipid rescue in the event there is accidental injection of local anesthetic solution into the intravascular compartment.

CENTRAL NEURAXIAL BLOCKS

Central neuraxial blocks are performed for diagnosis or for pain control. A common central neuraxial procedure in the ICU is a diagnostic lumbar puncture. Although this can be performed with ease in most patients, the depth of the epidural space and the dura from the skin may be difficult to ascertain both in younger populations, such as in infants and children,[5]

TABLE 29.1. Ultrasonography in critical care

Diagnostic
Lumbar puncture
Pain control
Epidural analgesia
Upper extremity blocks
Lower extremity blocks
Truncal blocks
Vascular insufficiency
Epidural analgesia
Upper extremity blocks
Lower extremity blocks

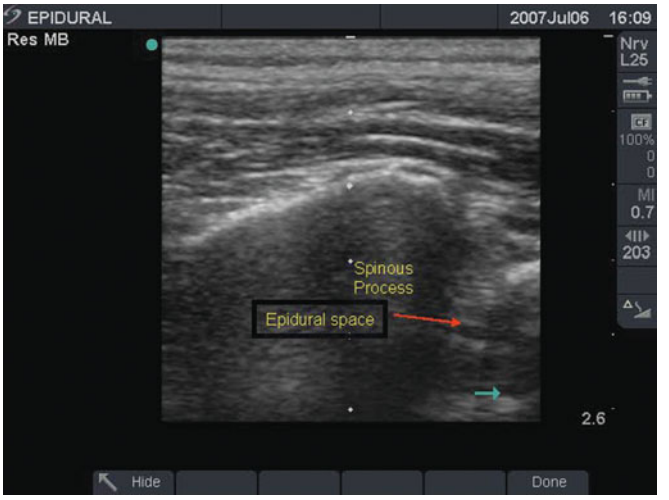


Figure 29.1. Ultrasound image of the epidural space.

and in the obese older patient, particularly in the obstetrical suite. The use of ultrasound may be a helpful diagnostic tool to determine the exact depth of the epidural space and the dura in children and in adults.[6,7] Although the curvilinear probe may be helpful in the older adult, a transverse probe capable of scanning deeper (7 MHz) may be helpful in children and infants. The epidural and dural area can be scanned for depth using a transverse axial approach and in a saggital longitudinal plane. A cadaver-based teaching model for ultrasonography has recently been described for learning ultrasound imaging for central neuraxial sonoanatomy.[8] It is important to understand that the transverse axial plane can be used to discern the sonoanatomy of the vertebral column, while the longitudinal saggital plane can be used for recognizing the spinous, articular, and transverse processes. Real-time use of the sonoanatomy for placement of epidural catheters can be applied in children, where there is less calcification and an improved ability to visualize structures.[7] The use of epidural analgesia for pain control has traditionally been reserved for patients in the postoperative period. The use of this technique for pain control in the ICU has been reported anecdotally for vascular insufficiency by providing a sympathetic blockade.[9] Epidural analgesia can be provided for pain control following vascular crisis in sickle cell disease in an intensive care setting.[10]

The technique for epidural analgesia is presented in Figure 29.1.

Axial Plane

1. Place the ultrasound probe between the spinous process.
2. Determine the location of the spinous and transverse processes.
3. Gently slide the probe cephalad or caudad until the dura and the epidural space can be located.
4. Use the depth indicator to measure the exact distance of the epidural space from the skin.
5. Mark the ends of the probe bilaterally and in the midline.
6. A line intersecting these two lines will provide a point of entry of the needle.
7. The exact distance for the needle to enter into the epidural space to the predetermined area allows the operator to advance the needle to the specified depth.
8. If real-time ultrasonography is used, it is imperative to use loss of resistance with saline or local anesthetic solution for determining the depth of the space.

UPPER EXTREMITY BLOCKS

The brachial plexus supplies the pain fibers to the upper extremity. It is derived from the cervical roots C5, C6, C7, C8, and T1. It is important to understand the multiple approaches to the brachial plexus for a variety of surgical procedures, depending on the area that is being operated on (Table 29.2). This technique can also be used for pain relief in critically ill trauma victims in the ICU.[11] This block has the advantage of increasing blood supply, thereby potentially improving perfusion to the compromised upper extremity.[12] The approach to the brachial plexus is at the interscalene (roots), supraclavicular (trunks), infraclavicular

TABLE 29.2. Brachial plexus block

Interscalene block: For shoulder pain

Supraclavicular block: For pain relief from fractures, vascular insufficiency

Infraclavicular: For longer duration of pain relief with catheter

Axillary: Single shot for immediate pain relief and vascular insufficiency

Dose: Adults: 15 mL, 0.2% ropivacaine or 0.25% bupivacaine
Children: 0.2 mL/kg, 0.2% ropivacaine or 0.25% bupivacaine

(divisions and cords), and axillary (branches) levels of the brachial plexus in the arm (Tables 29.2 and 29.3).[13,14] If an indwelling catheter is placed for trauma or vascular insufficiency, we prefer using the infraclavicular approach.[15,16] The supraclavicular approach is an easier approach, especially if there is significant trauma in the upper extremity, because this can reduce arm movement during performance of the block. The ultrasound technique for each one of these approaches is well described. Often, operator preference determines the approach used. We tend to use the supraclavicular approach for most fractures. For larger limb salvage procedures that require multiple days of intense pain control, an indwelling infraclavicular approach is preferred. The volume of local anesthetic solution varies, depending on the access. For most pain control, however, a volume of 15 mL of local anesthetic solution (0.2% ropivacaine or 0.25% bupivacaine) can provide adequate analgesia. In children, a dose of 0.2 mL/kg of local anesthetic solution is used.

Supraclavicular Approach

Ultrasound-guided approach to the supraclavicular plexus is an easy and rapidly achieved block in the ICU. The ultrasound probe is placed above the clavicle, and the nerves are seen surrounding the subclavian artery.

TABLE 29.3. Approaches and probes for Brachial plexus blocks

Axillary block	Linear probe	In-plane approach
Infraclavicular	Linear probe	In-plane approach
Supraclavicular	Linear probe	In-plane or out of plane
Interscalene	Linear probe	In-plane

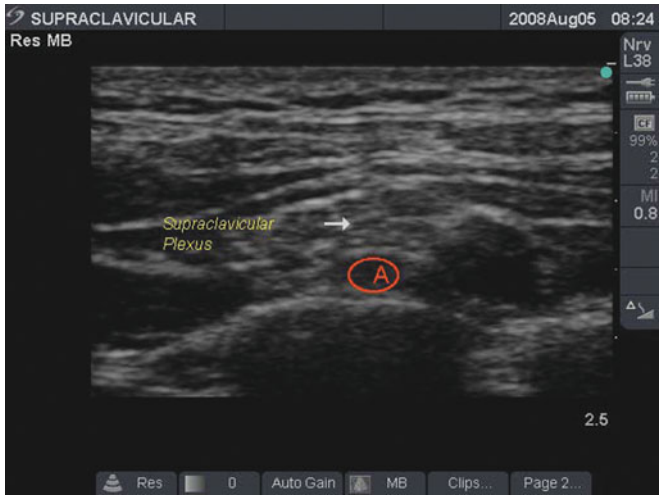


Figure 29.2. Supraclavicular plexus. Note the arrow pointing to the plexus surrounding the subclavian artery.

We prefer using an in-plane approach to the nerves. Local anesthetic solution is deposited and the spread of local anesthetic is seen in real time as the injection proceeds. A "donut sign" of the nerves surrounded by the local anesthetic solution denotes correct placement of the local anesthetic solution (Figure 29.2).

Infraclavicular Approach

The infraclavicular approach is preferred for placement of a catheter for pain control.[17] A linear ultrasound probe is placed below the clavicle and medial to the acromial process. The cords are seen surrounding the subclavian artery; the medial, lateral, and posterior cords can be easily identified (Figure 29.3). The

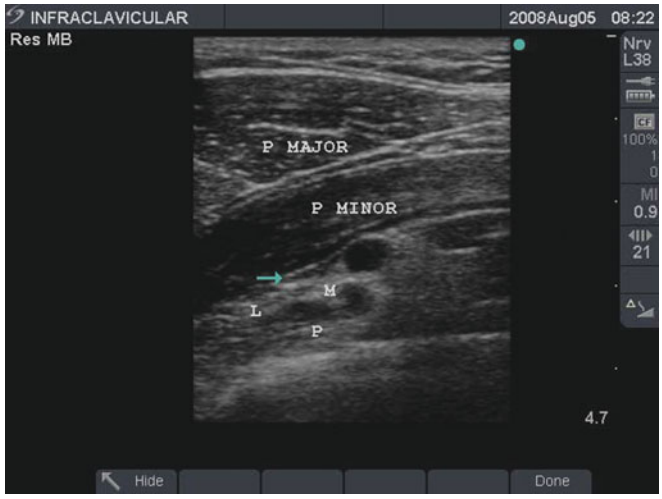


Figure 29.3. Infraclavicular plexus. Note the lateral, medial, and posterior cords surrounding the subclavian artery.

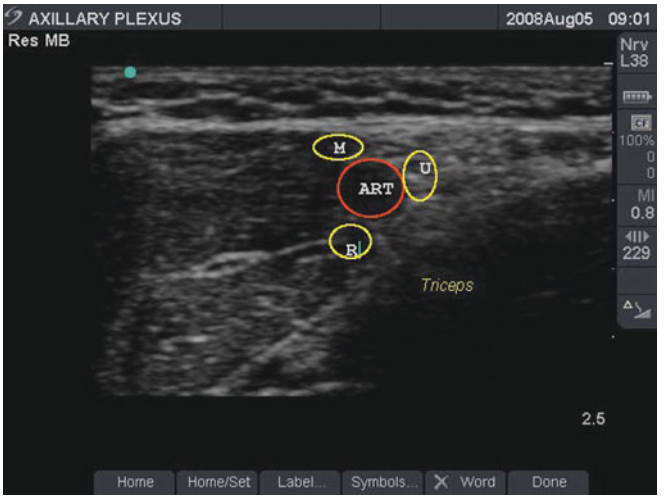


Figure 29.4. Axillary plexus ultrasound image.

pectoralis major and minor have to be traversed prior to access to the cords. If a catheter is placed in the area for continuous infusion of local anesthetic, it can be introduced using an 18-G Touhy needle with the tip of the catheter close to the posterior cord. If a continuous infusion is sought postoperatively, an infusion rate of 3 mL/hour is usually adequate for providing pain relief and also a sympathetic blockade for vascular insufficiency.

Axillary Block

The axillary approach to the brachial plexus is easy and can be performed in most ICU patients with ease using a linear probe or a hockey-stick probe in younger children and infants. The probe is placed with the orientation of the median, radial, and ulnar nerves in close proximity to the axillary artery (Figure 29.4). The plexus is very superficial even in obese individuals. If there is any doubt about the position of the nerves, color Doppler is used to check the position of the vessels. A total volume of about 15 mL of local anesthetic solution is injected into the area providing adequate relief of pain. The efficacy of the block is increased if the local anesthetic solution is seen surrounding the nerves completely.

Complications from Brachial Plexus Blocks

Intravascular injection is an inherent problem due to the close proximity of the great vessels. Injection into the vertebral artery and the intrathecal space can be a complicating factor while performing the supraclavicular approach.

TABLE 29.4. Femoral nerve block

Indications: Femur fracture; vascular insufficiency

Technique: In-plane approach, linear probe

Landmarks: Vein, artery, and nerve from medial to lateral orientation; color used to identify vascular structures

Complication: Intraneural injection, intravascular injection

LOWER EXTREMITY BLOCKS

The lumbar (L2, L3, L4) and sacral roots (S1, S2, S3) supply the sensory and motor supply to the lower extremity. Although it is of academic importance to understand the regional anesthetic techniques for lower extremity in the ICU, it is important to understand its utility for major trauma, particularly femur fractures, and for patients with vascular insufficiency.[18]

Femoral Nerve Block (Table 29.4)

The femoral nerve is located lateral to the femoral artery as it leaves the femoral triangle. The nerve supplies the anterior portion of the thigh and the femur and is blocked for managing pain. A nerve stimulator can be used to localize the nerve, eliciting a "patellar snap" denoting a quadriceps muscle contraction. Ultrasonography can be used for performing this block.

Technique

A linear ultrasound probe is placed below the ilioinguinal ligament along the crease. The femoral artery is identified. The femoral nerve is located lateral to the femoral artery. It can be visualized as a hyperechoic structure (Figure 29.5). With slight angling of the probe, the fascicles of the nerve can be seen under ultrasound imaging.[19] A needle is then placed using an in-plane technique to block the femoral nerve. Adequate blockade is noticed when the nerve is encircled with local anesthetic solution (donut sign). A volume of 15 mL of 0.2% ropivacaine or 0.25% bupivacaine can provide adequate blockade of the femoral nerve. In children and infants, a dose of 0.2 mL/kg is used to provide blockade of the femoral nerve. A catheter can be left in place for managing severe pain due to trauma in adults and children. This may facilitate a reduction in the use of opioids and associated adverse effects including somnolence, nausea, and vomiting.

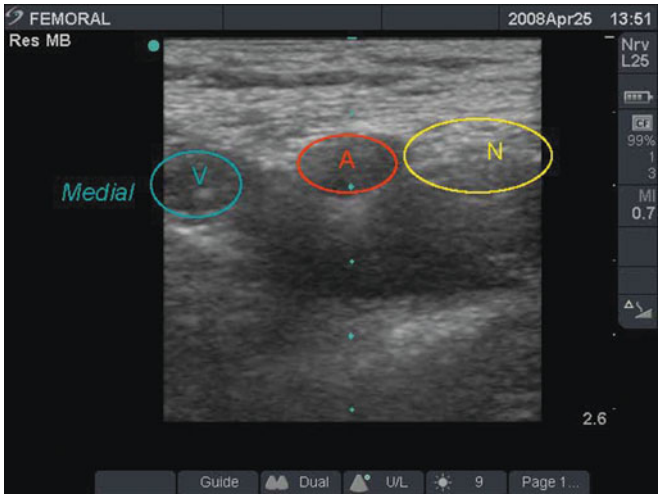


Figure 29.5. Ultrasound image of femoral nerve. Note the vein, artery, and nerve located in a single plane.

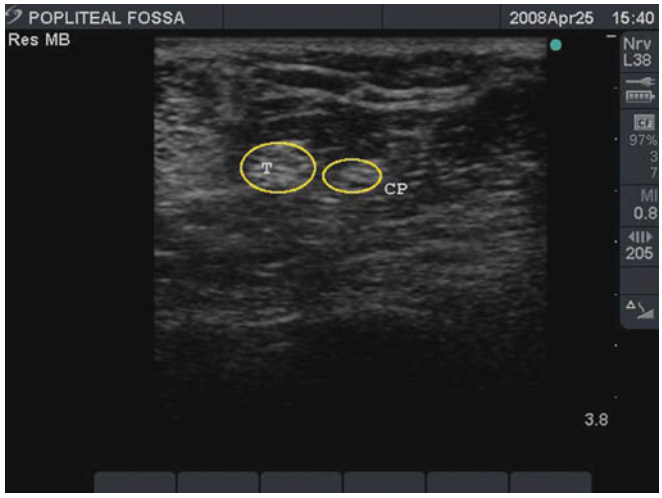


Figure 29.6. Ultrasound image of the popliteal fossa. Note the common peroneal (CP) and tibial nerve (T) in the popliteal fossa distal to their bifurcation.

Complications

Intravascular placement of needle and catheter, injury to femoral nerve due to intravascular injection.

Sciatic Nerve Block

The sciatic nerve is derived from the lower lumbar and the upper sacral roots. It supplies the sensory and motor block to the leg and foot. Sciatic nerve blockade is easy to perform under ultrasound guidance, although the need for sciatic nerve block in the ICU is less than the femoral nerve. In our institution, we have used ultrasound-guided sciatic nerve blocks for a child with severe acute respiratory distress syndrome (ARDS) and hemodynamic instability who required a fasciotomy for compartment syndrome. The block can be performed at the popliteal fossa or at the subgluteal area.

Technique

Popliteal Fossa Block. The patient is kept prone or supine depending on his or her hemodynamic stability. A linear ultrasound probe is placed in the popliteal fossa. The popliteal artery is identified. The common peroneal and the tibial nerves are identified. The ultrasound probe is moved cephalad until the coalition of the two peripheral branches is seen. A needle is inserted using the in-plane approach. A total of 15 to 20 mL of local anesthetic solution is deposited around the nerve to completely encircle the nerve (donut sign) (Figure 29.6).

Subgluteal Approach. The subgluteal approach to the sciatic nerve is another approach to the nerve as it exits the pelvis. This is another easy approach using ultrasonography and can be utilized for leaving an indwelling catheter for pain control after major trauma. The ultrasound probe is placed along the gluteal crease and the biceps femoris and the semitendinosus are identified. The sciatic nerve is seen as a hyperechoic shadow at this level. Using an in-plane approach we block the sciatic nerve at this site (Figure 29.7). Usually a volume of 15 to 20 mL of local anesthetic solution is needed to block the nerve effectively (Figure 29.7).

TRUNCAL BLOCKS

Truncal blocks can be of value inpatients who may have significant pain secondary to rib fractures. In fact, we

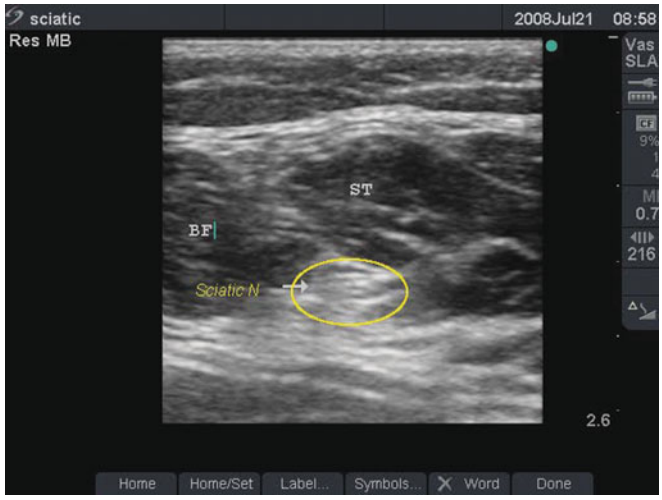


Figure 29.7. Subgluteal approach to sciatic nerve. Note the biceps femoris and the semitendinosus. Arrow points to the sciatic nerve.

notice that the use of these blocks may enhance the ability of these patients to take deep breaths and may reduce the incidence of atelectasis due to decreased efforts at breathing.

Intercostal Block

Anatomy

The intercostal nerves are derived from thoracic nerves T1 to T12. As the nerves emerge from their respective intervertebral foramina, they divide into four branches:

1. The first branch is the paired gray-and-white anterior ramii communicans, which pass anteriorly to the sympathetic ganglion.
2. The second branch is the posterior cutaneous branch, which supplies the skin and muscle in the paravertebral region.
3. The third branch is the lateral cutaneous branch, which then branches to an anterior and posterior branch to supply the midline portion of the chest and abdomen.
4. The ventral ramus, which is the intercostal nerve.

The intercostal nerves carry both sensory and motor fibers and pierce the posterior intercostal membrane distal to the intervertebral foramen to enter the subcostal grove and continue to run parallel to the rib. Its course within the thorax is between the parietal pleura and innermost intercostals muscle and the external and internal intercostals muscles.

Indications

Intercostal nerve blocks (ICBs) can be usedfor chest trauma associated with rib fractures,[18] thoracotomy,[20] and upper abdominal surgery including cholecystectomy and appendectomy, and for pain control following breast surgery. The 10th intercostal nerve (umbilical nerve) can also be blocked for umbilical hernia repair.[21] Intercostal nerve blocks with the use of neurolytics can be used to manage chronic pain conditions such as postmastectomy pain, herpetic neuralgia, and postthoracotomy pain syndromes.[22]

Technique

Ultrasound guidance has significantly improved the performance of this block. Although not described in detail in the literature, there is growing interest in using ultrasound guidance to perform this block. We prefer using a midaxillary approach to the intercostal nerve. A linear ultrasound probe is placed alongside the midaxillary line overriding two ribs. The pleura can be seen moving with every breath. The innermost intercostal muscle is identified. Using an off-plane approach, a 27-gauge needle is introduced into the innermost intercostal muscle; local anesthetic solution is injected after careful aspiration to rule out intravascular placement (Figure 29.8).

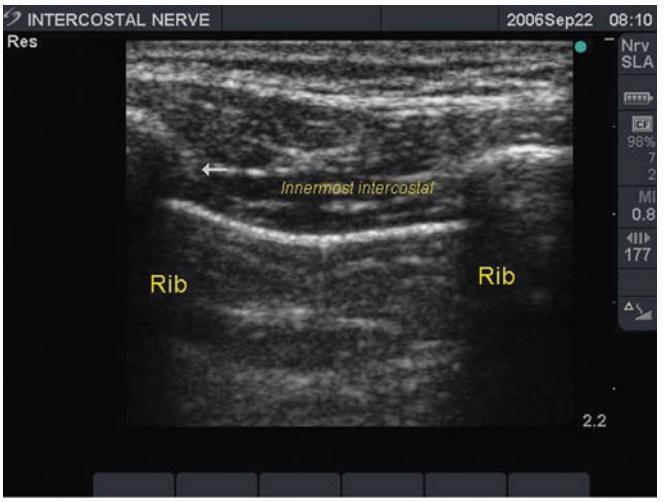


Figure 29.8. Intercostal space. The arrow points to innermost intercostal muscle, where the neurovascular bundle is seen.

Complications

The major complication is pneumothorax, the rate of which is below 1%.[23] Tension pneumothorax is rare, and the need for chest tube placement is dictated by presence of respiratory or cardiovascular compromise. Absorption of the local anesthetic from the intercostal space is rapid and has a higher incidence of toxicity when compared with other nerve blocks.[24] Peak plasma concentrations develop rapidly and toxicity is a concern, especially with multiple or continuous intercostal injections. Peak arterial plasma concentration develops rapidly and toxicity is always a concern with multiple or continuous intercostal injections.[24] The use of more dilute local anesthetic concentrations, smaller volumes, and incremental injection with frequent aspiration may reduce the probability of excess vascular absorption. The peritoneum and abdominal viscera are at risk of penetration when the lower intercostal nerves are blocked. A high subarachnoid blockade is possible when a posterior approach to the intercostal space is utilized.

CONCLUSION

The use of ultrasonograpy to provide analgesia may provide a safe and standardized method for critically

ill patients. The techniques are still early in their development, but demonstrate promise in clinical care to relieve pain outcomes of ICU patients. Prospective randomized, controlled trials can help to determine the efficacy of these nerve blocks in critically ill patients to the current approaches to pain control in the ICU.

References

1. Mather LE, McCall P, McNicol PL. Bupivacaine enantiomer pharmacokinetics after intercostal neural blockade in liver transplantation patients. *Anesth Analg.* 1995;80:328–335.
2. Foster RH, Markham A. Levobupivacaine: a review of its pharmacology and use as a local anaesthetic. *Drugs*. 2000;59:551–579.
3. McClellan KJ, Faulds D. Ropivacaine: an update of its use in regional anaesthesia. *Drugs*. 2000;60:1065–1093.
4. Weinberg G. Lipid rescue resuscitation from local anaesthetic cardiac toxicity. *Toxicol Rev.* 2006;25:139–145.
5. Suresh S, Wheeler M. Practical pediatric regional anesthesia. *Anesthesiol Clin North Am.* 2002;20:83–113.
6. Arzola C, Davies S, Rofael A, Carvalho JC. Ultrasound using the transverse approach to the lumbar spine provides reliable landmarks for labor epidurals. *Anesth Analg.* 2007;104:1188–1192.
7. Willschke H, Bosenberg A, Marhofer P, et al. Epidural catheter placement in neonates: sonoanatomy and feasibility of ultrasonographic guidance in term and preterm neonates. *Reg Anesth Pain Med.* 2007;32:34–40.
8. Tsui B, Dillane D, Pillay J, Walji A. Ultrasound imaging in cadavers: training in imaging for regional blockade at the trunk. *Can J Anaesth.* 2008;55:105–111.
9. Chiafery MC, Stephany RA, Holliday KJ. Epidural sympathetic blockade to relieve vascular insufficiency in an infant with purpura fulminans. *Crit Care Nurse.* 1993;13:71–76.
10. Yaster M, Tobin JR, Billett C, et al. Epidural analgesia in the management of severe vaso-occlusive sickle cell crisis. *Pediatrics.* 1994;93:310–315.
11. Clark F, Gilbert HC. Regional analgesia in the intensive care unit. Principles and practice. *Crit Care Clin.* 2001;17:943–966.
12. Szili-Torok T, Paprika D, Peto Z, et al. Effect of axillary brachial plexus blockade on baroreflex-induced skin vasomotor responses: assessing the effectiveness of sympathetic blockade. *Acta Anaesthesiol Scand.* 2002;46:815–820.
13. Brull R, Perlas A, Chan VW. Ultrasound-guided peripheral nerve blockade. *Curr Pain Headache Rep.* 2007;11:25–32.
14. Perlas A, Chan VW, Simons M. Brachial plexus examination and localization using ultrasound and electrical stimulation: a volunteer study. *Anesthesiology.* 2003;99:429–435.
15. Sandhu NS, Manne JS, Medabalmi PK, Capan LM. Sonographically guided infraclavicular brachial plexus block in adults: a retrospective analysis of 1146 cases. *J Ultrasound Med.* 2006;25:1555–1561.
16. Ilfeld BM, Morey TE, Enneking FK. Continuous infraclavicular brachial plexus block for postoperative pain control at home: a randomized, double-blinded, placebo-controlled study. *Anesthesiology.* 2002;96:1297–1304.
17. Sandhu NS, Capan LM. Ultrasound-guided infraclavicular brachial plexus block. *Br J Anaesth.* 2002;89:254–259.
18. Burton AW, Eappen S. Regional anesthesia techniques for pain control in the intensive care unit. *Crit Care Clin.* 1999;15:77–88, vi.
19. Oberndorfer U, Marhofer P, Bosenberg A, et al. Ultrasonographic guidance for sciatic and femoral nerve blocks in children. *Br J Anaesth.* 2007;98:797–801.
20. Cook TM, Riley RH. Analgesia following thoracotomy: a survey of Australian practice. *Anaesth Intensive Care.* 1997;25:520–524.
21. B DEJM, Gotzens V, Mabrok M. Ultrasound-guided umbilical nerve block in children: a brief description of a new approach. *Paediatr Anaesth.* 2007;17:44–50.
22. Doi K, Nikai T, Sakura S, Saito Y. Intercostal nerve block with 5% tetracaine for chronic pain syndromes. *J Clin Anesth.* 2002;14:39–41.
23. Moore DC, Bridenbaugh PO. Pneumothorax: Its incidence following intercostal nerve block. *JAMA.* 1960;174:842.
24. Behnke H, Worthmann F, Cornelissen J, et al. Plasma concentration of ropivacaine after intercostal blocks for video-assisted thoracic surgery. *Br J Anaesth.* 2002;89:251–253.

This page intentionally left blank

CHAPTER 30

Ultrasound Guidance for Vascular Access

Christian Butcher

INTRODUCTION

Vascular access procedures, such as central venous and arterial catheterization, are commonly performed in the critical care setting. An estimated 5 million central venous catheters (CVCs) are placed annually in the United States[1] in a variety of settings, including critical care units, emergency departments, operating rooms, and even in the outpatient arena. The usual indications for CVC placement are to assist in hemodynamic monitoring, as a route for the administration of vasoactive medications, total parenteral nutrition (TPN), or other vascular irritants, and as a route for drawing blood. In addition, oxymetric central line placement may play a future role in the management of septic shock, which could ultimately lead to increased utilization of central venous catheters.

Arterial catheters are an important tool in the management of many intensive care unit (ICU) conditions, including shock, severe hypertension, and other circumstances in which blood pressure monitoring is important. For a number of reasons, it seems that the role for arterial catheterization in the ICU may also increase. First, with the introduction of "minimally invasive" techniques now available to help estimate cardiac output, arterial catheter placement is becoming increasingly important for the management of selected patients with heart failure. Second, arterial catheterization can be used to assess the response to therapy in patients with pulmonary hypertension. Finally, there has been a significant amount of attention focused recently on respiratory variation of the peak arterial pressure as a means to predict fluid responsiveness in shock states.[2]

Peripherally inserted central venous catheters (PICCS) and peripherally inserted catheters sited in a midline position (midlines) have gained increased popularity as an alternative to CVCs in the care of selected patients because of their ease of insertion, longevity, and low rate of early complications. They are becoming an important component of the central venous access armamentarium.

Vascular access is associated with a relatively low rate of serious complications.[1] However, an improved understanding of complications and why they occur may help the provider to reduce their risk. Complications associated with vascular access procedures are well described,[1] and can be categorized as patient or operator dependent (Table 30.1). Patient-dependent factors include body habitus, coagulopathy, and anatomic variation. Operator-dependent factors include the operator's level of experience, time allotted to perform the procedure, and human factors like fatigue and lack of ultrasound guidance.[3–5] The most common complications of CVC placement include accidental arterial puncture, failed placement, malposition of the catheter tip, hematoma, pneumothorax, and hemothorax, the frequency of which vary depending on the site of catheter insertion (Table 30.2). Arterial catheter placement can be complicated by venous puncture, multiple arterial punctures, significant hematoma, and failed placement. Peripherally inserted central venous catheters and midline placement are also associated with hematomas and arterial insertions. A common complication of PICC line placement is malposition of the catheter tip into the ipsilateral internal jugular vein, or coiling in the subclavian vein or a thoracic branch such as the thoracodorsal vein (Figure 30.1).

Complications from these procedures are associated with excess direct costs derived from prolonged hospital and ICU lengths of stay (LOS) and additional procedures, such as chest tube insertion or hematoma evacuation, to treat the complications. For example, a single episode of iatrogenic pneumothorax has an attributable LOS of 3–4 days.[6] Indirect costs such as additional provider time and patient suffering are also important considerations.

TABLE 30.1. Patient and operator-associated risk factors for central line complications

Patient-dependent	*Operator-dependent*
Body habitus	Experience
Coagulopathy	Time allotted for procedure
Vascular anatomic variation	Fatigue
Prior surgery with distortion of anatomy	Lack of ultrasound use

ULTRASOUND USE FOR VASCULAR ACCESS

There are several studies that assess the impact of ultrasonography in improving the success of vascular access procedures. In 1984, Legler, and colleagues published a brief report describing the use of Doppler ultrasonography to locate the internal jugular vein for cannulation.[7] Since then, two metaanalyses investigating the use of ultrasound for CVC placement,[8,9] several review articles, standardized procedure guidelines,[10,11] and the SOAP-3 trial[12] have been published. The body of evidence from these and other studies demonstrates that the use of 2D ultrasound during central venous access is associated with fewer complications, fewer attempts before successful cannulation, shorter procedure times, and fewer failed procedures when compared with a landmark-based approach. As a result, the Agency for Healthcare Research and Quality (AHRQ) and the British National Institute of Clinical Excellence (NICE) have issued statements advocating the use of ultrasound guidance in central venous access procedures.[13,14]

Despite these evidence-based guidelines, some providers continue to resist and use ultrasound only in potentially "difficult to cannulate" patients, such as the morbidly obese, or in cases of failed cannulation.[15] Unfortunately, it is difficult to predict which patients will be difficult to cannulate, and the recognition of a failed attempt, as may arise from an occluded vessel, can only be viewed retrospectively after the failure has occurred and the patient has been adversely affected.[16] Some complications from CVC are considered preventable medical errors (PME), which refers to either mistakes or poor outcomes that could potentially have been prevented or hospital-acquired conditions (HAC), which is a medical problem not present on admission.[17] Ultrasound is a noninvasive tool to help prevent these complications and assists the operator in achieving optimal care for patients with less discomfort and fewer risks. Therefore, the consideration of ultrasound to improve safety in all central venous access procedures is recommended.

TABLE 30.2. The most common complications of central venous catheterization by site of insertion

	Internal jugular	*Subclavian*	*Femoral*
Pneumothorax	0–1%	2–3%	N/A
Hemothorax	0	<1%	N/A
Arterial puncture	5–10%	3–5%	5–15%
Failed attempt	15–20%	5–15%	15–40%

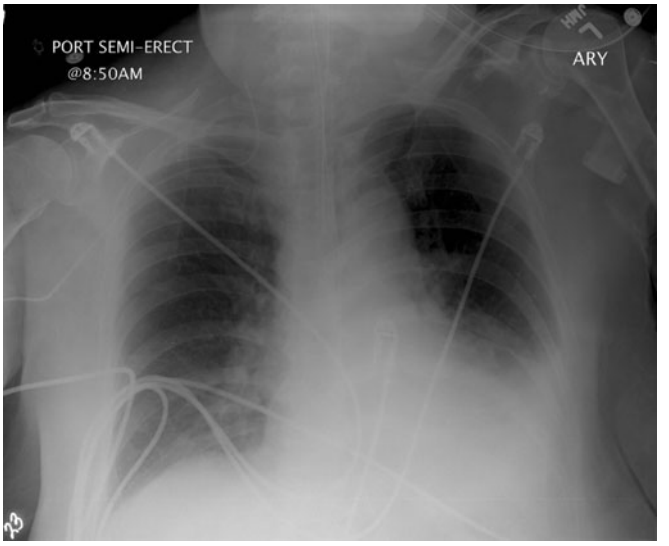


Figure 30.1. Peripherally inserted central venous catheter (PICC) tip malposition. The catheter tip is visualized in the ipsilateral internal jugular vein.

REVIEW OF ULTRASOUND

Transducer Selection

As described above (see Chapter 2), transducers come in a variety of frequencies, each with different properties and clinical applications. Two important concepts are important for ultrasonography in central venous access and need to be reviewed here. First, the relationship between ultrasound frequency and the depth of tissue penetration is an inverse relationship. This implies that low-frequency ultrasound (1–3 MHz) penetrates more deeply than high-frequency ultrasound

(7–10 MHz). Second, the relationship between frequency and image detail, or resolution, is *proportional*. This means that low-frequency ultrasound has poorer resolution than high-frequency ultrasound. Therefore, high-frequency ultrasound provides a very detailed image of superficial structures, to a depth of approximately 5 centimeters (cm), but cannot penetrate into deeper tissues. Alternatively, lower-frequency ultrasound is capable of reaching into deeper structures, but provides a less detailed image. These relationships form the basis for transducer selection. For percutaneous vascular access, which is a procedure that is superficial, higher-frequency transducers are ideal.

Modes

A-mode ultrasound has very few clinical applications and is not discussed further here. B mode ultrasound creates recognizable two dimensional (2D) images. B-mode is the most common mode currently employed in diagnostic medical ultrasound. M-mode ultrasound uses information obtained with B-mode to create an image that demonstrates the movement of structures over time (Figure 30.2). The most common application of M-mode is to assess valve leaflet movement and wall motion in cardiac ultrasound.

Doppler mode also has several forms. The simplest produces no image; there is only an audible signal that varies in intensity with the velocity of the structure being studied (e.g., blood) (Figure 30.3). Recently available ultrasound equipment uses Doppler in combination with B-mode to both create an image and give information about velocity (Figure 30.4). Color Doppler takes velocity information obtained by the Doppler shift and applies color to it. The Doppler is then superimposed on the B-mode image (Figure 30.5). Color Doppler is very commonly used in vascular applications, such as vascular access. The strength of the Doppler signal is related to the velocity of the target tissue (e.g., blood) and the angle of incidence. The best estimate of velocity occurs at an angle approaching zero (Figure 30.6). However, if the same vessel is imaged at 90°, there is no perceived motion of blood either

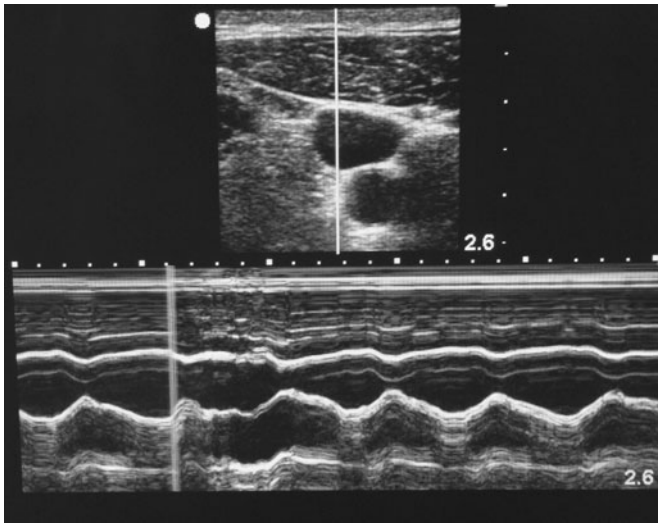


Figure 30.2. 2D image through the internal jugular (IJ) vein transversely, with the common carotid artery inferior and to the right (top). M-mode image through the IJ (see vertical line in 2D image) showing changes in vessel diameter with respiration (bottom). IJ indicates internal jugular.

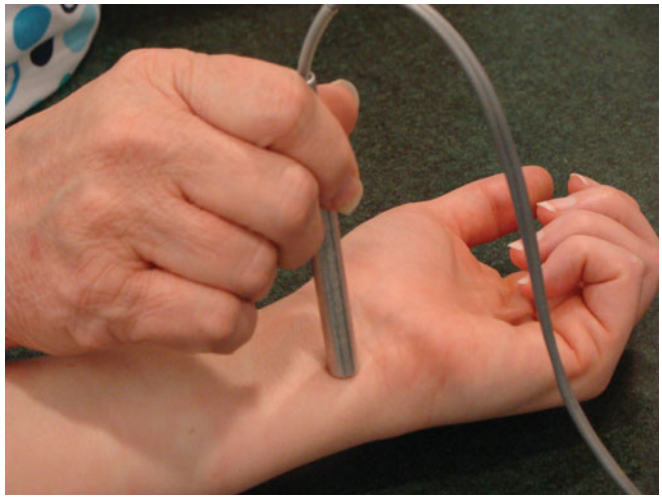


Figure 30.3. Continuous-wave Doppler "wand" seen during evaluation of the ulnar artery.

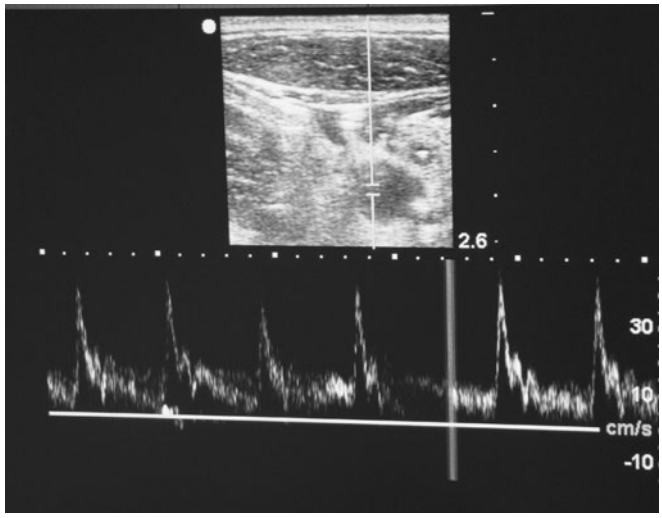


Figure 30.4. Doppler mode showing target of sample in the common carotid artery. Bottom image shows typical arterial waveform.

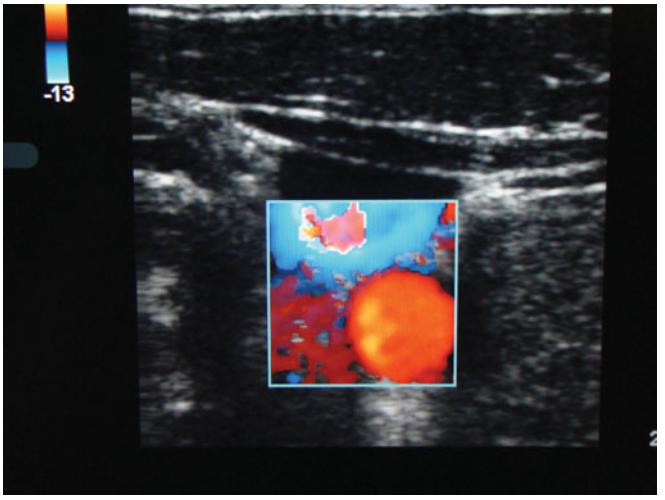


Figure 30.5. Transverse view through right internal jugular vein and common carotid artery, showing color Doppler. Vein is superior to the artery, and is depicted as blue.

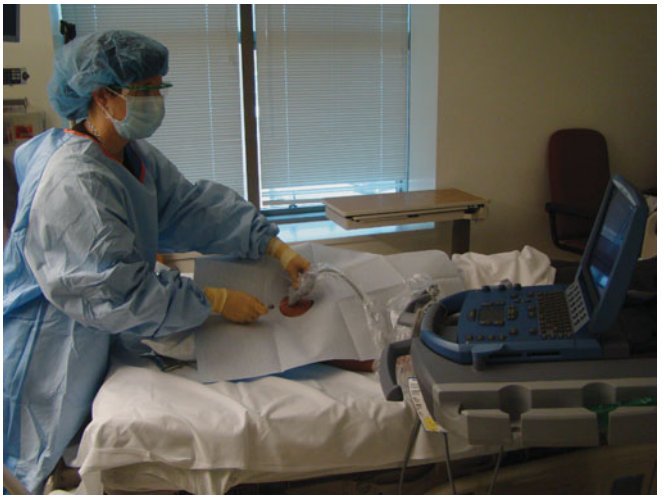


Figure 30.7. A combination of landmark-based and ultrasound-based techniques is optimal. Paying particular attention to the ultrasound screen, and ignoring the patient, can be disastrous.

toward or away from the transducer, and the Doppler signal fades. When the angle of incidence changes from one "side" of the 90° mark to the other "side," the color of the blood within the target vessel changes (from red to blue). This is very important and a potential source of error when a beginner is becoming familiar with orientation and selecting a vessel for cannulation.

Techniques of Ultrasound Guidance

Ultrasound is not a substitute for a thorough knowledge of the landmark-based technique for central venous cannulation. Frequently, the beginner may focus on the image on the screen and be inattentive to anatomic landmarks and the position of the needle (Figure 30.7).

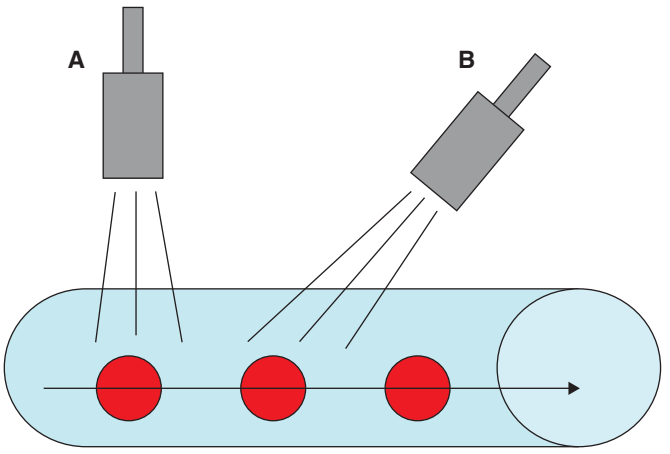


Figure 30.6. Relationship between angle of incidence of the ultrasound beam and the strength of the Doppler signal. As the angle approaches zero, the signal strength is maximized; as the angle approaches 90°, the strength is reduced.

Ultrasound-guided procedures can be categorized as static or dynamic. Static guidance refers to the use of ultrasound to localize and mark a site on the skin to facilitate a subsequent percutaneous procedure, much like a traditional landmark-based approach. B-mode or Doppler ultrasound is used to locate the internal jugular vein, assess its patency, and mark a suitable site on the skin for cannulation. The cannulation itself is not performed with ultrasound. Dynamic guidance refers to performing the procedure in "real time," with ultrasound imaging viewing the needle puncturing the vessel wall. For vascular access, static guidance appears to be inferior to dynamic, but still better than the landmark-based technique alone.[12] This is due to the time interval between marking with static guidance and the puncture, during which patients may move, or marks removed during skin preparation, both of which can lead to complications. Table 30.3 provides a comparison between static and dynamic guidance techniques. Dynamic guidance is more technically demanding because it requires significant eye–hand coordination.

Planes and Views

For our purposes, there are two planes to be considered: transverse and longitudinal, which refer to the orientation of the ultrasound transducer and the image to the vessel axis. A transverse view is a cross-section and provides the operator with information about

TABLE 30.3. Differences between static and dynamic guidance techniques for central line placement

Dynamic guidance	*Static guidance*
Ultrasonic localization and image-guided cannulation	Ultrasonic localization and marking of landmarks only
More precise and "real-time"	Cannulation is not image guided
More difficult to maintain sterility	Time delay between marking and cannulation
Requires significant hand–eye coordination	Less difficult to maintain sterility
	Less technically demanding

structures that lay adjacent to the vessel of interest. For example, a cross-sectional view of the internal jugular vein will enable visualization of the adjacent common carotid artery and, perhaps, the vagus nerve, thyroid gland, and trachea (Figure 30.8).

A longitudinal view will depict structures anterior and posterior to the vessel of interest and may allow for visualization of the entire needle during cannulation, but does not allow simultaneous visualization of structures lateral to the vessel (Figure 30.9). All commonly utilized central venous and peripheral arterial sites can be visualized in either orientation. As a general rule, transverse views tend to be easier for the novice to learn ultrasound-guided cannulation.

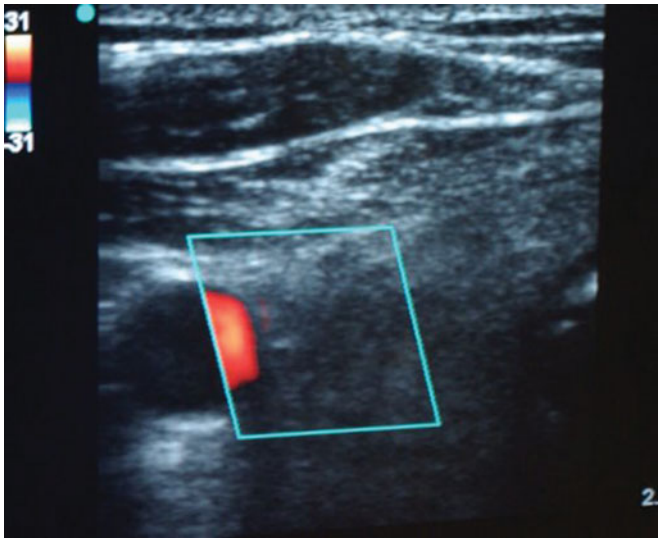


Figure 30.8. Transverse (short axis) view at the level of the internal jugular vein (to the left of the carotid artery and not shown). The right carotid artery and right thyroid lobe can be seen. The lateral wall of the trachea can be seen to the far right of the picture.

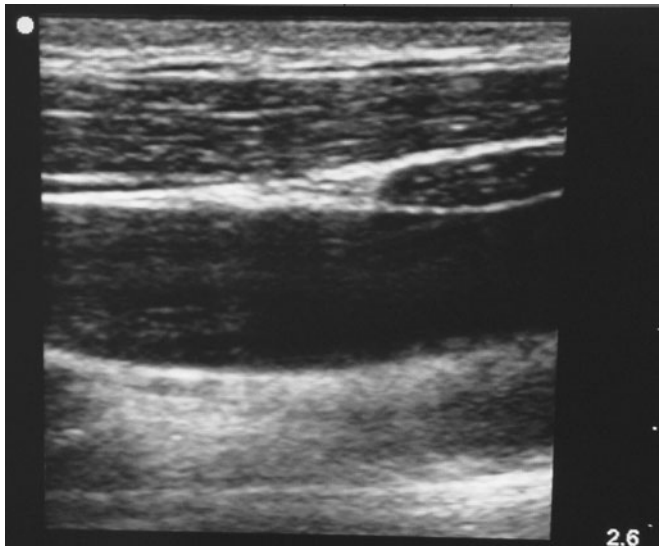


Figure 30.9. Longitudinal view through the internal jugular vein. Information regarding the location of surrounding structures is limited when compared with the transverse view.

Methods of Orientation

Orientation is probably the most important step to a successful procedure. Most transducers have an identifiable mark, known as a "notch," on one side. This corresponds to a mark displayed on one side of the image, and allows right–left, or lateral, orientation (Figure 30.10). In rare instances, where the orientation is

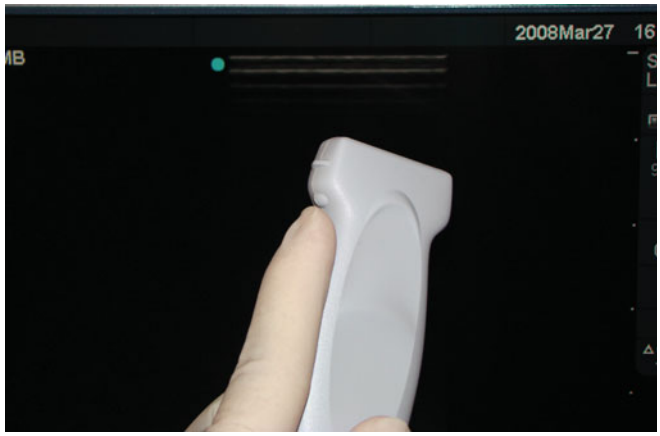


Figure 30.10. To gain orientation, the "notch" on the transducer (just distal to fingertip) should be matched to the "dot" on the screen (blue circle, upper left side).

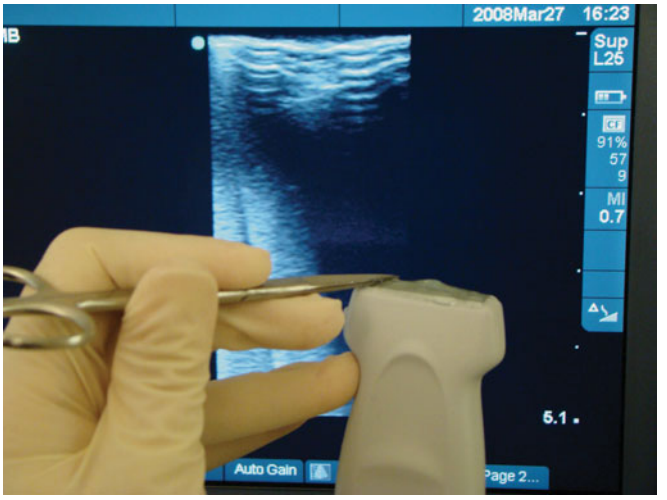


Figure 30.11. During a procedure, the probe surface can be rubbed with a finger or instrument to determine orientation; in this case, the scissors are placed on the left side of the probe and are seen on the screen as an artifact on the left side. This can also be easily accomplished while the probe is on the patient.

uncertain, a finger can be rubbed on one side of the transducer surface to produce an image and confirm the orientation (Figure 30.11).

Problems with orientation can largely be prevented by ensuring proper patient, transducer, and ultrasound console positioning adjacent to each other. The operator, transducer, and console should be arranged in a straight line (Figure 30.12). In this way, the vessel to be cannulated and the image screen will be in the direct line of sight of the operator. When accessing the internal jugular vein, the console should be on the *same* side as the vessel to be cannulated, usually at the level of the patient's, to ensure that transducer orientation—the right side of the transducer, the right side of the patient, and the right side of the image—are all aligned. When cannulating the subclavian or axillary vein, the console should be on the *opposite* side of the patient, directly across from the operator, again, in the direct line of vision of the operator. In this example, the right side of the transducer corresponds to the inferior aspect of the patient, but everything else is the same.

Once the proper orientation is assured, the area of interest is scanned and the operator needs to be able to differentiate an artery from vein, which can be done in several ways. The first and easiest method is to assess vessel compressibility by applying downward pressure with the transducer while visualizing the vessel on the monitor. Veins will typically compress at a lower applied pressure than arteries, unless a clot is present (Figure 30.13). The second technique is to assess for the influence of respiratory variation on vessel diameter. Veins usually have easily identifiable respiratory variation as compared with arteries. The third technique is to apply standard Doppler or color Doppler to the vessel and listen to the audible signal or observe the character of the color "pulsation," both of which give an estimation of blood velocity inside the target vessel. Remember, as previously discussed, the color (red versus blue) of the blood in the vessel is dependent on transducer position. It is useful to compare

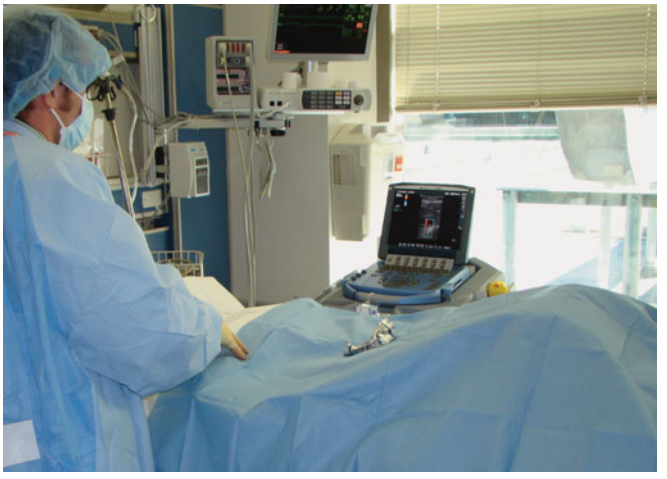


Figure 30.12. To optimize comfort, the patient, the target vessel/probe, and the screen should be in the line of vision of the operator; this minimizes operator movement during the procedure.

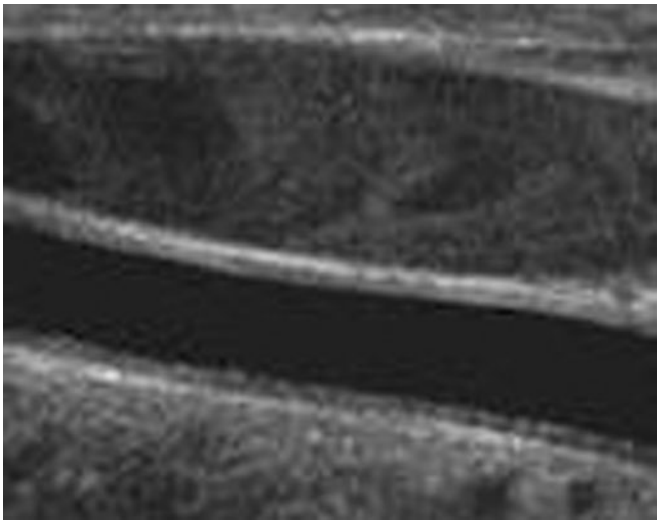


Figure 30.13. Longitudinal view through the internal jugular vein (top) and carotid artery (bottom) showing thrombus in the internal jugular vein.

color Doppler signals of all vessels in the area of interest, paying close attention to the angle of incidence; with a little practice, arterial flow is easily differentiated from venous flow. Large, rapid fluctuations in intrathoracic pressure can create very high venous blood flow velocities that can mimic arterial flow, which may require the use of the other two methods of respiratory variation and compressibility to help differentiate the vessel type.

Occasionally, the vein cannot be visualized. The most common reason for this is hypovolemia with associated venous collapse, which can be remedied by placing the patient in the Trendelenburg position or applying a valsalva maneuver or fluid administration. Other less common causes are agenesis, chronic occlusion or scarring of the vessel, and clot that is completely occluding the lumen. Clot may be difficult to distinguish from the surrounding tissue, and appears similar to that of an absent vessel. In this case, a thorough examination of the proximal and distal parts of the vessel should be performed and a formal venous Doppler should be performed to evaluate for deep venous thrombosis prior to any attempted central venous cannulation. If access is critical and vessel presence or patency cannot be assured, a different vessel should be cannulated.

HOW TO PERFORM ULTRASOUND-GUIDED CANNULATION

Internal Jugular Vein

The first step in successfully cannulating the internal jugular vein is proper positioning of the patient. The head should be rotated slightly contralaterally, with the neck extended. Severe rotation of the neck and head should be avoided because this may lead to significant distortion of the anatomy, and may increase the amount of overlap of the carotid artery and jugular vein. The bed should be placed in Trendelenburg position and the ultrasound machine should be placed by the ipsilateral side of the bed, at about the level of the patients' waist.

An initial examination of the landmarks, without ultrasound, should be performed, followed by selection of an insertion site. The site should then be confirmed with ultrasound. There are two reasons for this. Not only does it provide the operator with immediate feedback regarding landmark-based positions, but it also facilitates teaching both the landmark-based approach and ultrasound-guided approach. During this process, proper orientation, both transverse and longitudinal, should be ensured. The target vessel and surrounding structures should be identified and the patency of the vessel should be confirmed.

The patient's skin can now be prepped in the usual manner, and full barrier precautions should be used to maintain sterility and reduce the incidence of catheter-related infections.[18] Ultrasound use introduces another piece of equipment onto the sterile field, making the maintenance of sterility more difficult. While learning, special attention should be paid to this issue in order to develop good habits. A sterile ultrasound sheath should be placed on the sterile field for when an assistant hands you the ultrasound transducer.

After the patient is prepped and draped, the catheter is set up as per normal routine. All ports should be flushed with bacteriostatic saline to remove air and to test for occlusion caused by manufacturing defects. The components needed for catheter insertion, including needles, wire, dilator, scalpel, and catheter, should be arranged in an orderly fashion and placed within easy reach. The assistant holds the transducer, with ultrasound gel applied (can be nonsterile gel), in a position such that the operator can both acquire the transducer and place it in the sterile sheath in one motion (Figure 30.14). Note that instead of utilizing an assistant, the transducer can be "picked up" by the operator,

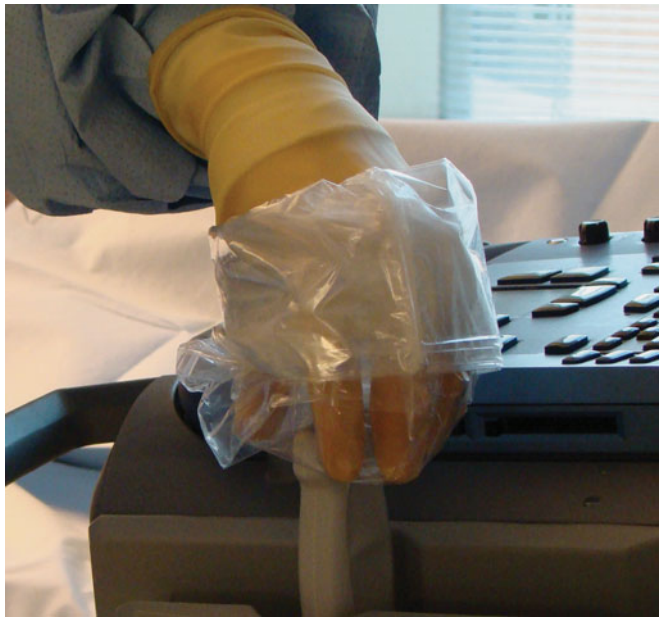


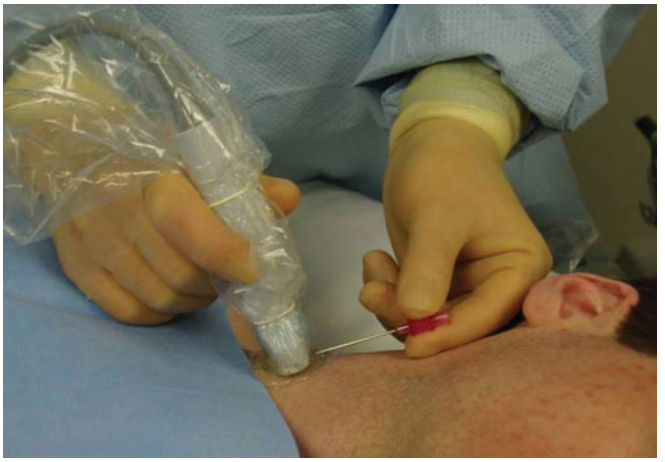
Figure 30.14. Retrieving the probe using the single-hand technique to maintain sterility. Once covered, the probe is placed on the sterile field.

whose hand is inside the sterile sheath. The sheath is then extended to cover the transducer cord, and sterile rubber bands are applied to secure the sheath in place.

A second ultrasound examination should be performed to ensure that the original insertion site is still viable. Remember that proper orientation every time the probe is applied to the patient is essential for assuring an appropriate procedure.

When cannulating the vessel, use the same insertion site and needle trajectory that you would if you were using the landmark-based approach (lateral, medial, etc.). If using the transverse plane for ultrasound guidance, which is especially good for novices, be sure to center the vessel lumen on the screen; remember that if the vessel is centered on the screen, it is directly underneath the middle of the transducer head. Sometimes, it is useful to perform a "mock poke" to confirm your proposed insertion site relative to the underlying vessel (Figure 30.15). This is done by laying the needle on the skin surface, then placing transducer over it. The acoustic shadow produced by the needle should be visualized directly over, or superimposed on, the target vessel (Figure 30.15). The skin puncture should be approximately 1 cm proximal to the transducer, which in most cases will result in visualization of the needle tip entering the vessel without having to move the probe much. If the needle tip cannot be visualized indenting either the subcutaneous tissue overlying the vessel or the vessel itself, move the probe along the axis of the vessel while slightly "agitating" the needle; this will accentuate the image of the needle and tip. The point of the "V" caused by indenting the subcutaneous tissue above the vein with the needle tip should be directly over the vessel (Figure 30.16). Be sure to visualize the tip of the needle at all times; it is very easy to misinterpret the shaft of the needle as the tip; be sure to move the probe axially along the vessel frequently to maintain imaging of the tip. If done properly, the needle tip should be seen entering the lumen at about the same time as the flash of blood is obtained in the syringe.

Once the vessel has been successfully cannulated, the transducer can be set aside and the procedure can



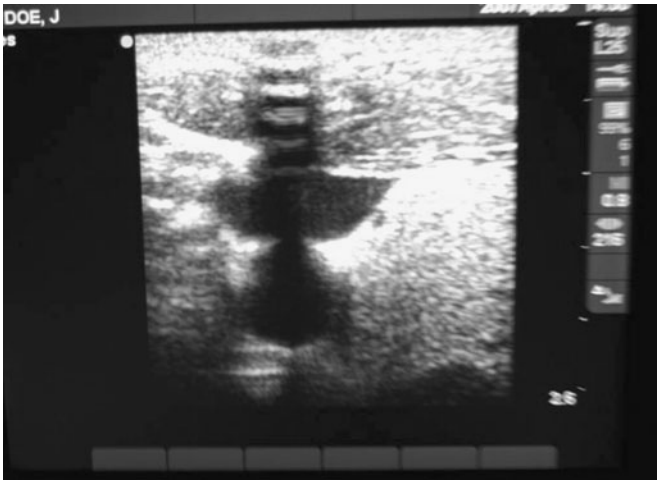


Figure 30.15. Technique of performing a "mock poke"; the needle is placed on the skin surface, and then imaged with ultrasound (top). The needle will cast an acoustic shadow on underlying structures. If the needle directly overlies the vein, the acoustic shadow will bisect the vein (bottom).

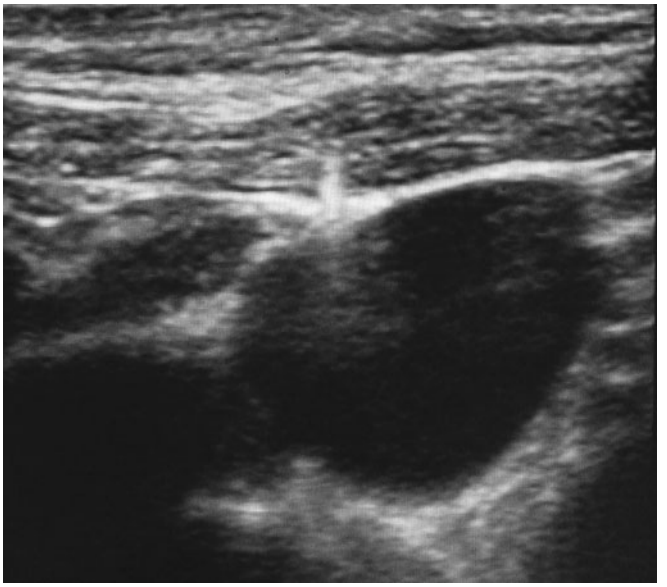


Figure 30.16. Transverse view of the internal jugular vein during cannulation. The needle tip (bright white) is seen penetrating the internal jugular vein at about 11 o'clock. Note that the probe is typically placed approximately 1–2 cm distal to the needlestick to ensure that the tip will remain in the ultrasound view. The probe can be moved proximally or distally to keep the needle tip in view.

proceed normally with wire placement. Intravascular position of the wire can be confirmed with ultrasound, which can be saved for documentation in the medical record. Advance the wire slowly; there should be little to no resistance. Based on practical experience, the distance from the insertion site to the distal superior vena cava (SVC) in the average person is no more than 17–18 cm, so the wire should not be advanced farther than this. Once the wire is in place, the needle is removed, and a "stab" incision is made through the dermis at the point where the wire enters the skin. The tract is then dilated. Care should be taken not to insert the dilator too far, as this can potentially cause vessel perforation. After removal of the dilator, the catheter is then advanced to the desired distance, which is usually no more than 17–18 cm. Once the line is in place, flushed, secured, and dressed, a quick ultrasound examination of the anterior chest wall can be performed to evaluate for a pneumothorax (see Chapters 21 and 22).[19]

The use of ultrasound should be documented in the medical record. Typically, a statement regarding the use of ultrasound to assess the location and patency of the vessel and an image of the wire or catheter in the vessel lumen is sufficient for documentation and often provide sufficient documentation for reimbursement. Additionally, a statement about the presence or absence of sliding pleura should be included.

Subclavian Vein

Typically, the subclavian vein is more difficult to visualize ultrasonographically than the internal jugular, axillary, or femoral veins. This is due to its position under the clavicle, which requires significant angulation and manipulation of the transducer to acquire a useful image (Figure 30.17). Two additional challenges are the difficulty of visualizing the vein in obese patients using an infraclavicular view and the inability to externally compress the vein, making it difficult to adequately assess the vein for clot.

In our experience, it is usually easier to visualize the subclavian with a longitudinal, supraclavicular view because an adequate transverse view is often technically challenging. Considering the ease with which the internal jugular and axillary veins are visualized, we have largely abandoned the subclavian vein in our practice, except for specific clinical situations, such as for long-term TPN administration or for emergency central venous access.

Figure 30.17 shows the typical transducer placement for imaging the subclavian vein and Figure 30.18 provides the ultrasound image. Note that the patient is

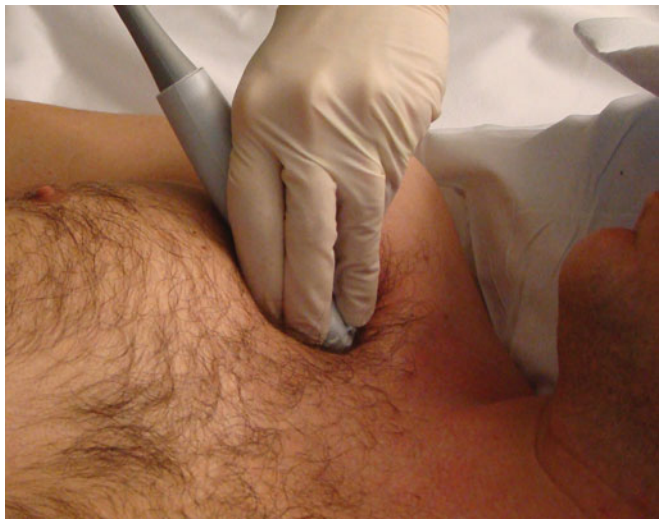


Figure 30.17. Probe position required to image the subclavian vein longitudinally. Note the cephalad angulation that is necessary to obtain a good view, which can become cumbersome during image-guided cannulation.

relatively thin, so an infraclavicular window is used. Except for the relative difficulty in cannulating the subclavian under dynamic guidance and the longer "learning curve" associated with it, the procedure itself is

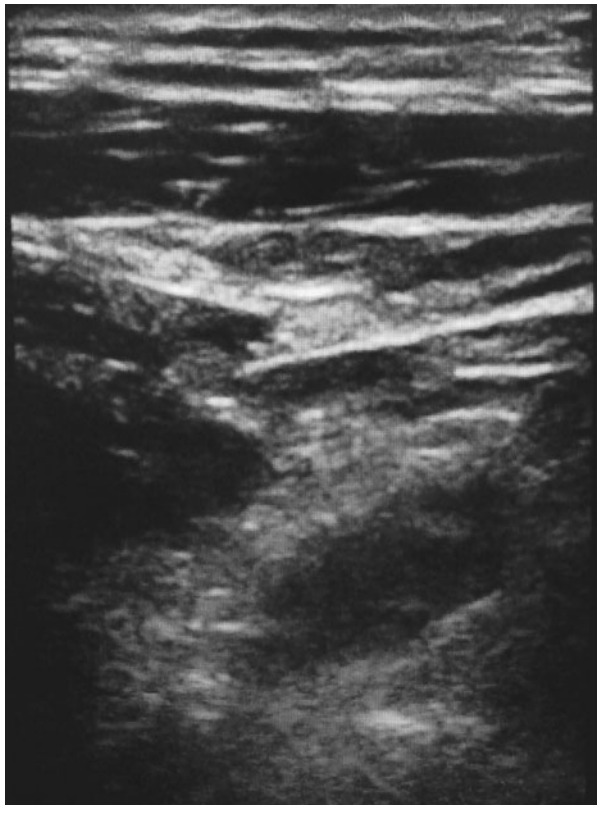


Figure 30.18. Longitudinal view of the subclavian vein (dark, round structure just left of center) and subclavian artery (bottom toward the right).

largely the same as that outlined above under "Internal Jugular Vein" cannulation, except for the use of the longitudinal view as described.

Axillary Vein

Using the axillary vein for central venous access has many unique advantages over other sites.[20–23] Although not well studied, because the insertion site is on the anterior chest, axillary catheterization likely shares a lower incidence of catheter-related infections than the subclavian approach. Unlike the subclavian vein, using the axillary vein may be associated with fewer complications, such as pneumothorax, hemothorax, and chylothorax. The axillary vein is usually easier to compress than the subclavian vein and allows for an easier recognition of clots. There is, however, the additional potential complication of causing a brachial plexus injury, particularly if a far lateral approach is used.[22] One distinct disadvantage of the axillary approach is the unique dependence on ultrasound to ensure localization and subsequent cannulation; landmark techniques are not as effective as with the other common sites used to access the central venous system. Figure 30.19 shows proper transducer placement for viewing the axillary vein transversely. As with internal jugular (IJ) and subclavian access approaches, a quick postprocedure scan of the chest should be performed to ensure sliding pleura, which essentially eliminates the possibility of pneumothorax.[19]

Femoral Vein

Femoral cannulation remains a popular approach due to its relatively low incidence of life-threatening complications. However, several clinically important complications may occur that lead to significant morbidity. Accidental (or intentional) femoral arterial cannulation, especially in coagulopathic patients, may cause life-threatening retroperitoneal hemorrhage and hematoma. Inadvertent stimulation of the femoral nerve with the cannulation needle can cause intense pain. A puncture site that is too proximal can also result in inadvertent puncture of intraperitoneal structures. Ultrasound can help avoid some of these important complications.

As with IJ, subclavian, and axillary cannulation, the first step in successful femoral access is achieving proper orientation. The ultrasound machine should be placed on the contralateral side of the patient, directly across from the operator. The entire area should be scanned, with identification of all vascular structures, including the femoral artery, common femoral vein, and saphenous or profunda femoris vessels if possible. Once the vein is identified, it should be evaluated for the presence of clot. Additionally, a longitudinal view of the vein should be obtained as it dives under the inguinal ligament, and the ligament itself should be marked on the skin (Figure 30.20). This ensures that an intraperitoneal puncture will not occur.

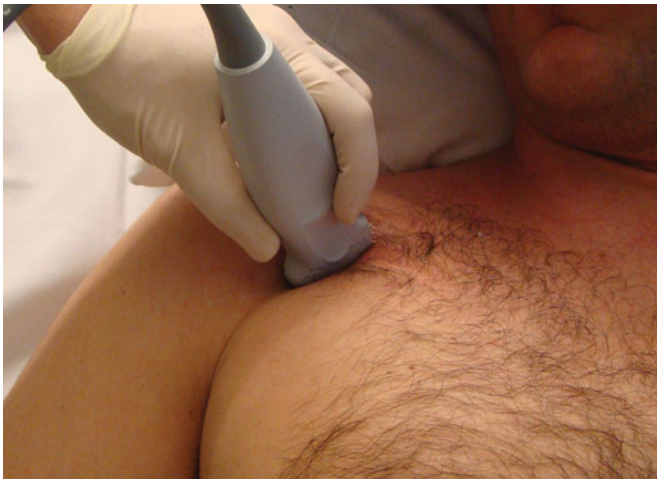


Figure 30.19. Probe position during visualization of the axillary vein.

ARTERIAL CATHETER PLACEMENT WITH ULTRASOUND GUIDANCE

The principles and techniques of ultrasound guidance for central venous catheter insertion can be easily adapted to the placement of arterial cannulae. From an ultrasound guidance perspective, the procedures are very similar. There are, however, some factors associated with arterial catheter placement that deserve special consideration.

The most commonly cannulated arteries include the radial, axillary, and femoral approaches. The radial approach significantly exceeds the others in terms of popularity. The reasons for this are easy accessibility of the wrist, the presence of a dual circulation of the hand (in most patients), and the fact that the wrist is a relatively clean site. It is important to understand, however, that radial artery catheterization is not risk-free.

In 1929, Dr. Edgar van Nuys Allen described a maneuver in which the dual palmar circulation could be tested by obstructing both radial and ulnar arterial flow, then releasing either ulnar or radial to see if palmar circulation was restored. The test was repeated to assess flow in the other of the two arteries. The importance of this test is to ascertain the duality of the circulation, so that

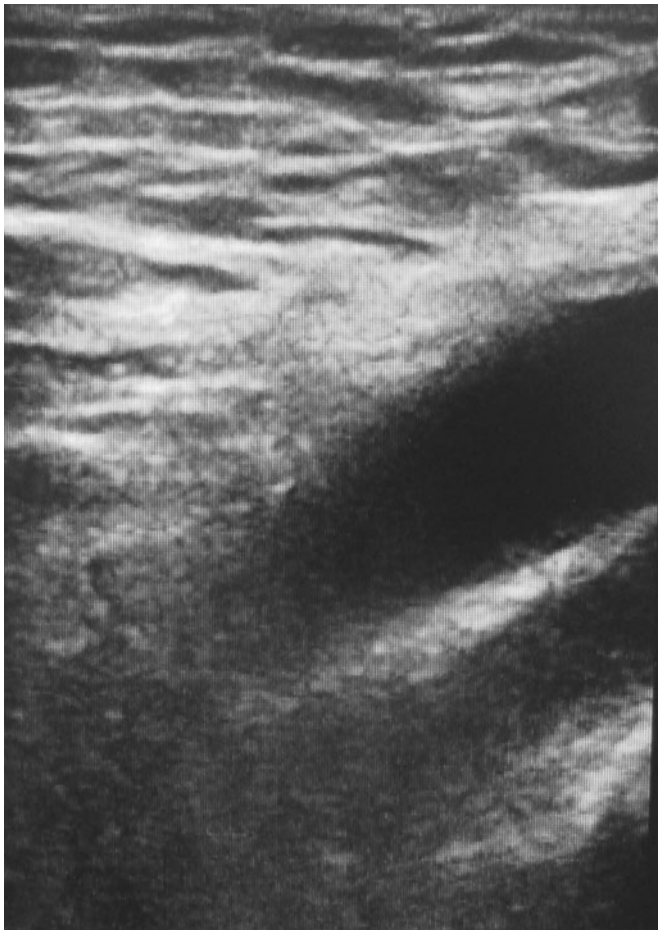


Figure 30.20. Longitudinal view of the femoral vein as it dives posteriorly under the inguinal ligament. The artery is superior to the vein in this example. The inguinal ligament is seen as a bright area to the left.

if one of the arteries was obstructed (from thrombus or spasm after puncture), the palmar circulation would not be compromised. Although there is some debate as to the value of Allen's test in predicting who is at risk of hand ischemia, the test continues to be performed on a routine basis, especially in the setting of radial artery harvesting for coronary bypass grafting. Ultrasound use may improve the accuracy of Allen's test, first reported in 1973.[24] This test requires ultrasonic localization of the palmar arteries with Doppler, followed by occlusion of the radial artery; if flow is maintained, there is adequate dual circulation, suggesting that radial artery cannulation or harvesting is safe (Figure 30.21).

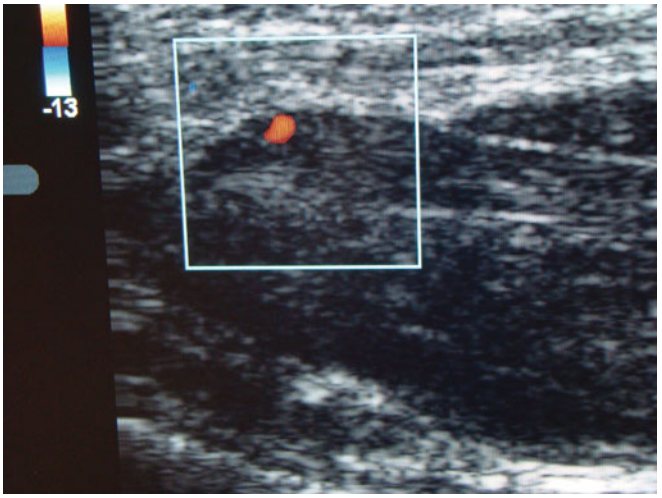
Unsuccessful attempts at radial artery catheterization can be associated with hematoma formation. While this is usually insignificant and without clinical consequence, hematomas can seriously impair further attempts at cannulation by obscuring the arterial pulsation during palpation. As a result, procedure times are prolonged, pain is increased, and procedures fail. Using either static guidance to mark a suitable site for cannulation or cannulation under dynamic guidance reduces the number of unsuccessful attempts (Figure 30.22).[25–27] If a hematoma occurs while using ultrasound, arterial flow is still readily apparent with application of Doppler or color Doppler to the 2D image, enabling subsequent attempts.

Interestingly, Yokoyama and colleagues demonstrated anatomic variations using ultrasound in 11 of 115 (2.6%) patients scheduled to undergo percutaneous coronary intervention via a radial artery approach. Of these, only three were inaccessible for cannulation. These findings confirm that although anatomic variations exist, ultrasound guidance can identify many of these in anticipation of the procedure.[28]

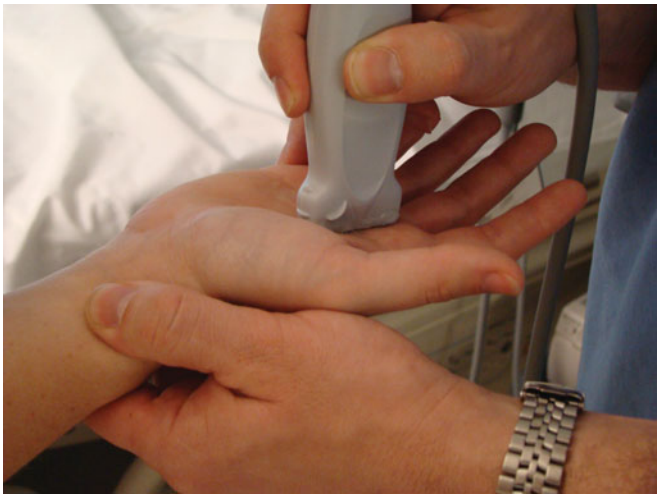
PICC LINES/MIDLINES

Peripherally inserted central venous catheter lines have gained significant popularity in recent years, presumably because of a low incidence of complications from insertion, improved patient comfort as compared with standard central venous catheters, safety and ease of care in the outpatient setting, and a relatively low incidence of catheter-related infections.[29,30] First described as an alternative to central venous catheters placed in the IJ, subclavian, or femoral veins, PICCs are placed in peripheral veins of the upper extremities and "threaded" into the central venous system (Figure 30.23).

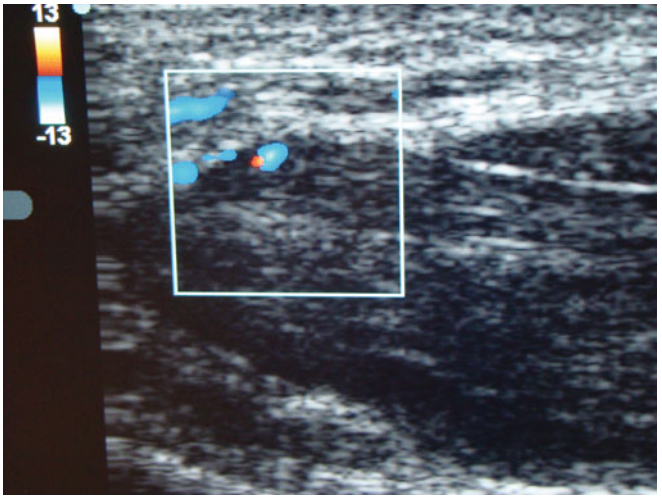
There are a fair amount of data on long-term complications of these catheters. The most common complications include thrombosis, catheter-related infection, catheter tip malposition or migration, vessel or heart chamber perforation, deep venous thrombosis, and malfunction.[29–32] Factors associated with a higher risk of thrombosis include larger catheter size, cephalic vein placement, "peripheral" placement (outside of the vena cava), duration of catheterization, and presence of underlying solid-tumor malignancy or hypercoagulation disorders. Note that the best catheter tip position is the distal third of the superior vena cava at the superior vena cava–right atrial junction. This position causes the catheter tip to "float" within the lumen, which is associated with a lower incidence of thrombus formation.[31] Also, the superior vena cava has a higher flow rate compared with the axillary, subclavian, or brachiocephalic veins, which has implications for thrombus formation and damage to the vessel from infusion of caustic substances.[31]



A



B



C

Figure 30.21. (A) Color Doppler image of the palmar arch. (B) occlusion of the radial artery while imaging the palmar arch. (C) reversal of flow in the palmar arch after occlusion, which indicates flow is maintained by the ulnar artery.

The risk of catheter-related infection with PICCs is substantially lower than that with central venous catheters, but is still a significant problem.[27] Factors associated with higher infection rates are use of any skin prep other than 2% chlorhexidine, lack of full barrier precautions (cap, mask, gown, gloves, and large drape), and use of catheters with more than a single lumen (the more lumens, the higher the risk). Antimic robial PICC lines may reduce this risk, but the evidence at this point is inconclusive.

There are several PICC line kits on the market. It is important to review the needs of your particular patient when selecting a catheter. A "power PICC," which is capable of handling high-pressure infusions, such as may be used with intravenous contrast agents, may be indicated. Peripherally inserted central venous catheters also come with one, two, or three lumen, which should be selected depending on the patient's needs.

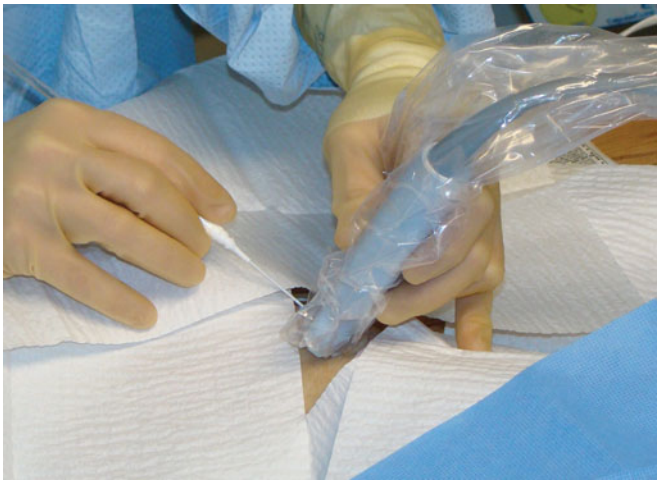


Figure 30.22. Cannulation of the radial artery under dynamic guidance.

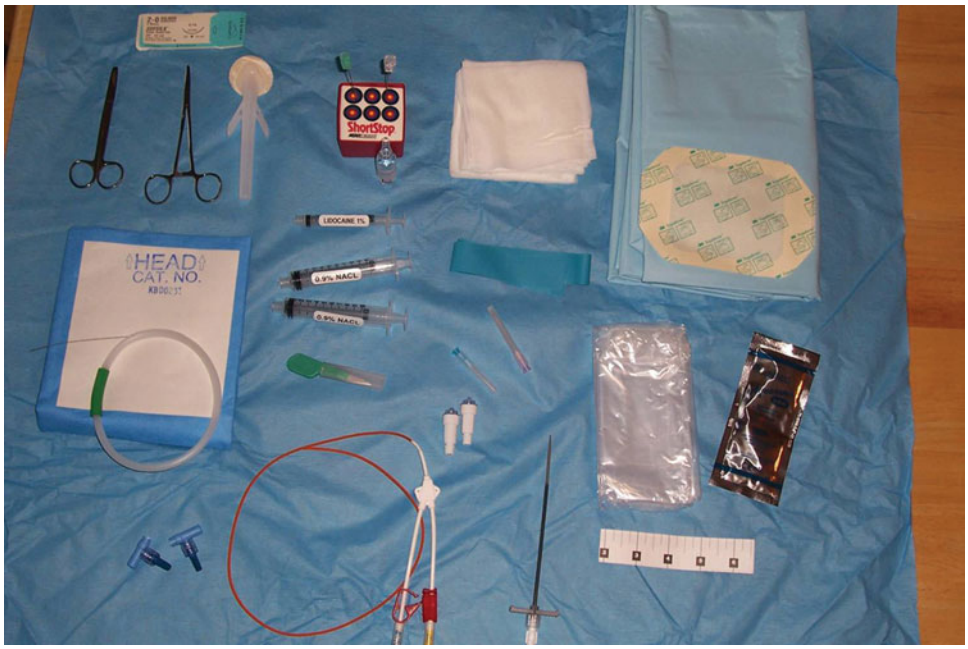


Figure 30.23. Typical PICC line kit. Note the catheter at the bottom center of the photo. This is an example of a guide wire–based introducer system (peel-away introducer is to the bottom right). PICC indicates peripherally inserted central venous catheter.

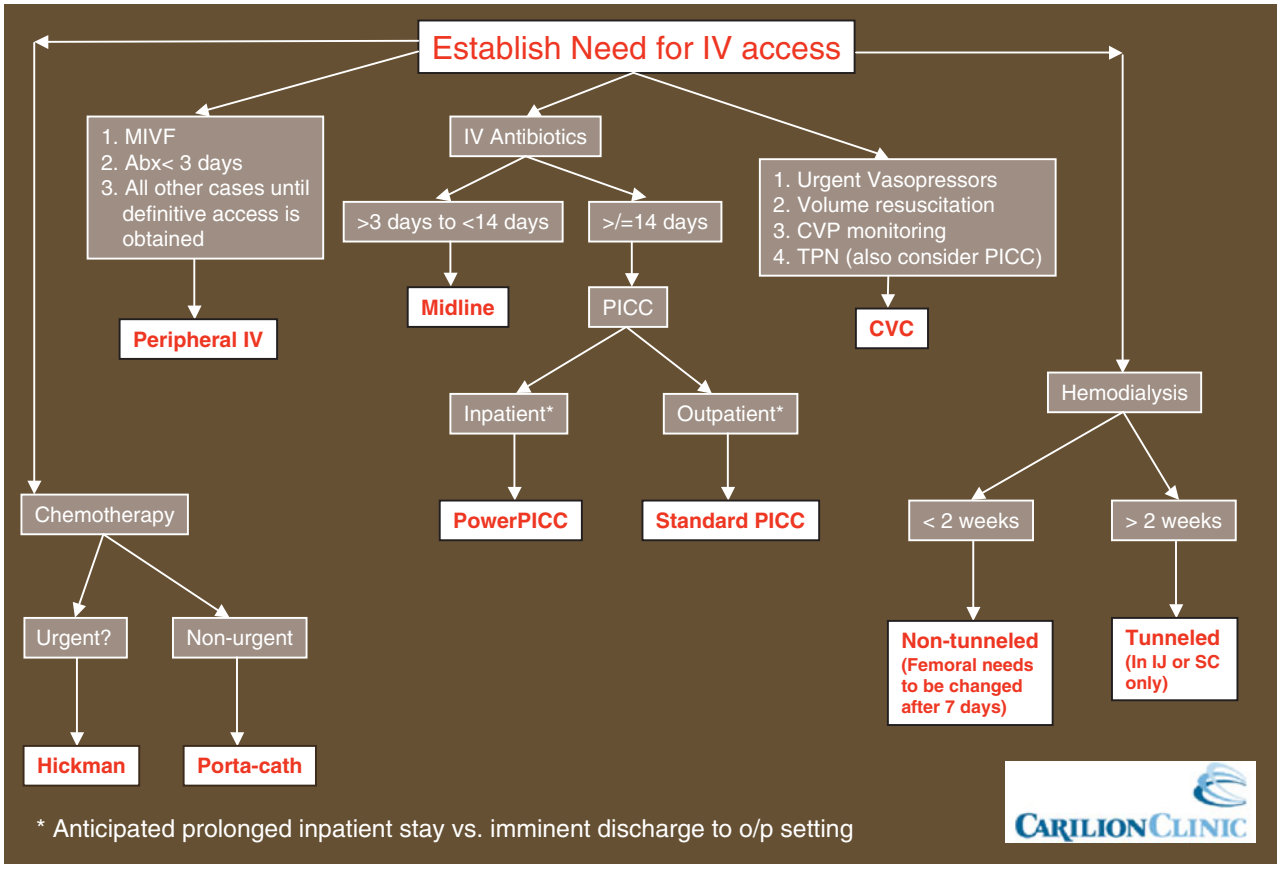


Figure 30.24. An example of an IV access algorithm. IV indicates intravenous.

There are two basic methods of PICC placement. First, the Seldinger technique, where the vessel is cannulated with a needle, a wire is threaded through the needle followed by needle withdrawal, and a dilator/tear-away introducer is then inserted. The dilator is removed from the introducer, and the PICC is inserted to the appropriate position, followed by removal of the introducer. The second method requires cannulation with a device similar to an angiocath, where the vessel is cannulated by a needle/catheter combination, and then the catheter is advanced over the needle into the vessel. The PICC is advanced through this catheter, which is then "torn away." This method tends to be more cumbersome.

An institutional algorithm that governs intravenous (IV) access taking into consideration indications, patient factors, and alternatives when deciding on the type of vascular access device may avoid excessive and inappropriate PICC line use. One approach is shown in Figure 30.24.

For PICC line insertion, 2D and color Doppler ultrasound is used to "map" the extremity of interest. All superficial vascular structures of the distal brachium are identified, paying particular attention to differentiating artery from vein, and assessing vein size. Figure 30.25 shows the typical venous anatomy of the upper extremity. After mapping is complete, a candidate vein is selected for insertion and marked. Patency should be assessed, by ensuring compressibility, as well as venous flow.

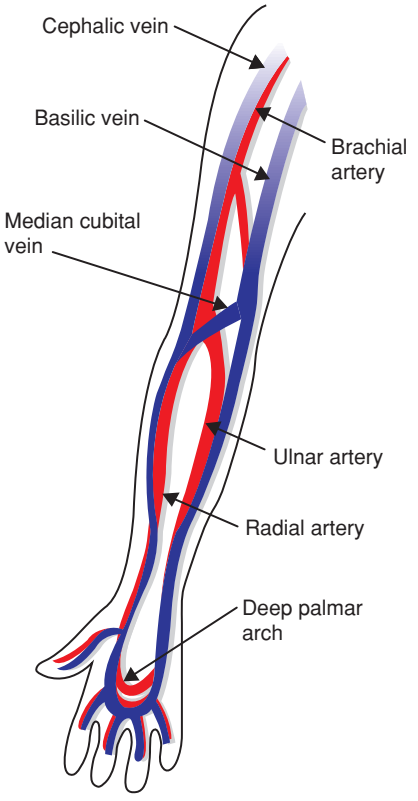


Figure 30.25. Typical venous anatomy of the upper extremity.

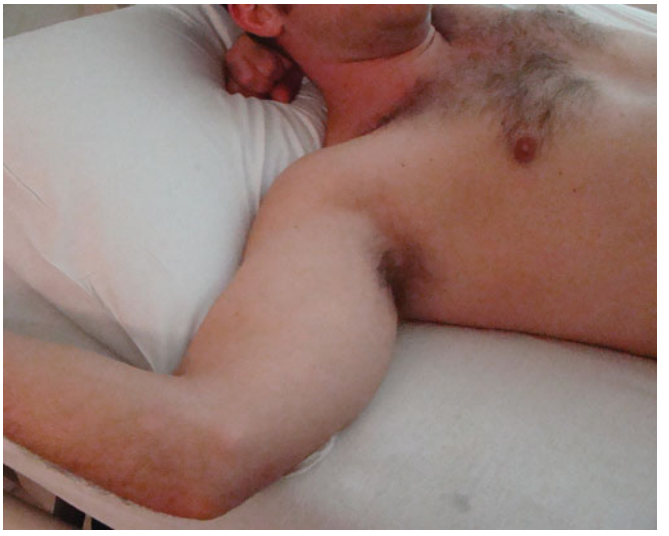


Figure 30.26. Optimal arm position during PICC line insertion. Shoulder abducted and externally rotated, arm flexed 90°. This maneuver exposes the basilic vein.

Once all the necessary equipment is ready, the patient is positioned and sterilely prepped and draped. The ideal position for successful catheter insertion is depicted in Figure 30.26. The right arm is preferable due to the higher incidence of catheter-tip malposition when inserted in the left arm. Note that the extremity is abducted, slightly externally rotated, and secured; this allows easy access to the basilic vein, and may help reduce catheter tip malposition by forming a straight line from the insertion site to central venous system. If the arm is left at the patient's side, the catheter tip must negotiate a turn when entering the subclavian; this increases the risk of the catheter either entering the ipsilateral internal jugular vein or coiling in the subclavian. There are no data on the risk of air embolization with PICC or midline placement; the risk is likely to be negligible and roughly the same as that with peripheral IV insertion. The Trendelenburg position, therefore, is not necessary.

Next, the desired PICC kit is opened and the line itself is prepared. Usually, these catheters have a long metallic obturator that provides stiffness during insertion; this should be partially withdrawn to allow for catheter trimming. The desired catheter length is estimated by measuring the distance from the proposed insertion site to the glenohumoral joint, adding the distance from the glenohumoral joint to the sternal notch,

then adding about 6 cm to allow for proper positioning in the distal superior vena cava. Once this distance is determined, the catheter should be trimmed to length. *The obturator should not be cut because this will produce a sharp point capable of puncturing the vessel.*

Rescan the area and confirm the position of the target vein. Cannulate the vessel under dynamic guidance as described above in the central venous catheter section. When access to the vein is obtained, remove any dilators that may be present with the introducer, and advance the catheter slowly to the hub. Quickly advancing the catheter increases the risk of catheter tip malposition. By slowing the rate of advancement, the catheter becomes more "flow directed" and follows the flow into the correct position. Remember that the catheter was trimmed to an appropriate length already, so advancing the hub will ensure correct tip position. When the catheter is fully advanced, remove the inner stylette or obturator, attach a syringe, and aspirate blood to confirm an intravascular position. Ultrasound can also be used to evaluate for catheter tip malposition by scanning the ipsilateral internal jugular vein and contralateral subclavian, if possible. The line can then be secured by a suture or one of several commercially available adhesive devices and dressed appropriately. Of course, a portable chest radiograph should be obtained to confirm correct placement.

Peripheral IV Access

One of the most common reasons cited for placing PICC and midline devices is difficulty obtaining adequate peripheral access. This can, in part, be avoided by providing nursing and support personnel with ultrasound guidance principles for peripheral IV access.

SUMMARY

Vascular access can be made safer and easier with ultrasound guidance. The basic technique is the same regardless of the procedure being performed. Once the technique of dynamic guidance is mastered, it can be applied to almost any procedure. Ultrasound should not be used as a substitute for a proper understanding of the landmark-based technique. Rather, it should be used to augment the knowledge of the venous system and vascular access procedures. Remember to appropriately document the use of ultrasound in the medical record.

References

1. McGee DC, Gould MK. Preventing complications of central venous catheterization. *N Engl J Med.* 2003;348:1123–1133.
2. Brennan JM, Blair JE, Hampole C, Goonewardena S. Radial artery pulse pressure variation correlates with brachial artery peak velocity variation in ventilated subjects when measured by internal medicine residents using hand-carried ultrasound devices. *Chest.* 2007;131:1301–1307.
3. Polderman KH, Girbes AJ. Central venous catheter use. Part 1: mechanical complications. *Intensive Care Med.* 2002;28:1–17.
4. Merrer J, De Jonghe B, Golliot F, et al. Complications of femoral and subclavian venous catheterization in critically ill patients: a randomized controlled trial. *JAMA.* 2001;286:700–707.
5. Mansfield PF, Hohn DC, Fornage BD. Complications and failures of subclavian vein catheterization. *New Engl J Med.* 1994;331:1735–1738.
6. Light RW. *Pleural Diseases.* 5th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2007.
7. Legler D, Nugent M. Doppler localization of the internal jugular vein facilitates central venous cannulation. *Anesthesiology.* 1984;60:481–482.
8. Randolph AG, Cook DJ, Gonzales CA, et al. Ultrasound guidance for placement of central venous catheters: a meta-analysis of the literature. *Crit Care Med.* 1996;24:2053–2058.
9. Hind D, Calvert N, McWilliams SR, et al. Ultrasonic locating devices for central venous cannulation: meta-analysis. *BMJ.* 2003;327:361.
10. Feller-Kopman D. Ultrasound-guided internal jugular access. *Chest.* 2007;132:302–309.
11. Maecken T, Grau T. Ultrasound imaging in vascular access. *Crit Care Med.* 2007;35:s178–s185.
12. Milling TJ Jr, Rose J, Briggs WM, et al. Randomized, controlled clinical trial of point-of-care limited ultrasonography assistance of central venous cannulation: the third sonography outcomes assessment program (SOAP-3) trial. *Crit Care Med.* 2005;33:1764–1769.
13. NICE Guidelines. Available at: http://www.nice.org.uk/nicemedia/pdf/Ultrasound_49_GUIDANCE.pdf. Accessed March 3, 2009.
14. Rothschild J. AHRQ Evidence based practice. Available at: http://www.ahrq.gov/clinic/ptsafety/pdf/chap21.pdf. Accessed March 3, 2009.
15. Muhm M. Ultrasound guided central venous access (letter). *BMJ.* 2002;325:1374–1375.
16. Forauer A, Glockner J. Importance of US findings in access planning during jugular vein hemodialysis catheter placements. *J Vasc Interv Radiol.* 2000;11:233–238.

17. Available at: http://www.cms.hhs.gov/HospitalAcqCond/Downloads/HAC-POA-Listening12-17-2007.pdf. Accessed:.
18. Mermel LA. Prevention of intravascular catheter-related infections. *Ann Intern Med.* 2000;132:391–402.
19. Mayo PH, Doelken P. Pleural Ultrasonography. *Clin Chest Med.* 2006;27:215–227.
20. Sandhu NS. Transpectoral ultrasound-guided catheterization of the axillary vein: an alternative to standard catheterization of the subclavian vein. *Anesth Analg.* 2004;99:183–187.
21. Mackey SP, Sinha S, Pusey J. Ultrasound imaging of the axillary vein-anatomical basis for central access (Letter). *Br J Anaesth.* 2003;93:598–599.
22. Galloway S, Bodenham A. Ultrasound imaging of the axillary vein-anatomical basis for central venous access. *Br J Anaesth.* 2003;90:589–595.
23. Sharma S, Bodenham AR, Mallick A. Ultrasound-guided infraclavicular axillary vein cannulation for central venous access. *Br J Anaesth.* 2004;93:188–192.
24. Mozersky DJ, Buckley CJ, Hagood CO Jr, et al. Ultrasonic evaluation of the palmar circulation. A useful adjunct to radial artery cannulation. *Am J Surg.* 1973;126:810–812.
25. Maher JJ, Dougherty JM. Radial artery cannulation guided by Doppler ultrasound. *Am J Emerg Med.* 1989;7:260–262.
26. Levin PD, Sheinin O, Gozal Y. Use of ultrasound guidance in the insertion of radial artery catheters. *Crit Care Med.* 2003;31:481–484.
27. Shiver S, Blaivas M, Lyon M. A prospective comparison of ultrasound-guided and blindly placed radial artery catheters. *Acad Emerg Med.* 2006;13:1275–1279.
28. Yokoyama N, Takeshita S, Ochiai M, Koyama Y. Anatomic variations of the radial artery in patients undergoing transradial coronary intervention. Catheter *Cardiovasc Interv.* 2000;49:357–362.
29. Schmid MW. Risks and complications of peripherally and centrally inserted intravenous catheters. *Critical Care Nursing Clinics of North America.* 2000;12:165–174.
30. Maki DG, Kluger DM, Crnich CJ. The risk of bloodstream infection in adults with different intravascular access devices: a systematic review of 200 published prospective studies. *Mayo Clin Proc.* 2006;81:1159–1171.
31. National Association of Vascular Access Networks (NAVAN). Tip Location of Peripherally Inserted Central Catheters. NAVAN Position Statement. *JVAD.* 1998;3:9–10.

APPENDIX A

Glossary

Absorption: Conversion of ultrasound energy into heat.

Active element: Integral part of all ultrasound transducers. Also called a crystal, it is made of piezoelectric material (lead zirconate titanate or PZT) that converts electrical energy into ultrasound and vice versa.

Acoustic variables: Parameters that define a sound wave, such as pressure and density that change rhythmically.

AIUM: American Institute of Ultrasound in Medicine.

AIUM 100 mm test object: Standard phantom used for quality assurance.

Akinetic: Organ or its part that should be moving, but is not.

ALARA **A**s **L**ow **A**s **R**easonably **A**chievable: AIUM principle, limiting possible bioeffects of acoustic radiation.

Aliasing: Sampling error characteristic of the inability of pulsed-wave Doppler to accurately measure high-flow velocities.

Ambiguity (range): Characteristic of continuous-wave Doppler describing its inability to define the position of the sample. Caused by an overlap between transmitting and receiving beams.

A-mode ultrasound: Antiquated mode of ultrasound used to depict the position of a reflector as well as the strength of the returning echo by its amplitude. Seldom used in modern practice.

Amplification (receiver gain): Increases signal strength in the receiver of the ultrasound system and therefore overall brightness of the image.

Amplitude: The difference between the average value of the acoustic variable and its maximum value through the duration of the sound wave; the "loudness" of the ultrasound.

Analog image: Image on the screen of the cathode-ray tube (TV screen) prior to any computer processing.

Anechoic: Area producing no-echo reflections and appearing black on the ultrasound image.

Archiving: Storage of images.

Array transducer: Transducer with multiple active elements, arranged in a certain order.

Artifact: Image errors or any image that differs from true anatomy of the reflector. Can be caused by malfunction of the ultrasound system, physical limitations of ultrasound, or operator error.

Attenuation: Reduction of amplitude of an ultrasound wave, as it propagates through the medium.

Attenuation coefficient: Attenuation in negative decibels per one centimeter (cm) travel. In soft tissues, **0.5 dB/cm/MHz**.

Augmentation: Increase in venous flow with distal compression; a sign of venous patency.

Axial resolution: The minimal distance between two objects positioned along a line parallel to the ultrasound beam where both can be distinguished as separate objects. Defines longitudinal or depth resolution or the distance between two reflectors, measured in millimeters (mm), at which the reflectors are still imaged as separate. It is measured as a half of the ultrasound pulse length, with typical values in diagnostic ultrasound of **0.05–0.5 mm**.

Backing material: Backing also known as damping material consists of the layer of epoxy resin impregnated with tungsten and placed behind the active element of the ultrasound transducer. It improves axial resolution by decreasing pulse duration (after-ringing), much like a hand placed on a guitar string.

Banding: Hyperechoic artifact within the focal zone. Appears as a bright, horizontal stripe.

Beam (Ultrasound beam): Bundle of acoustic radiation transmitted by the transducer, caused by wavelet interactions, and shaped like an hourglass.

Bernoulli equation (simplified): Converts maximal flow velocity into a pressure gradient: used to assess

the severity of either valvular or vascular stenosis. **Pressure gradient (mm Hg) = 4 × [(Max flow velocity (m/sec)]^2.**

Bioeffects: All patient-related effects of acoustic radiation.

Bistable image: Black-and-white image characterized by excessively high contrast and a narrow dynamic range (see **Dynamic range**).

B-mode ultrasound: Imaging mode where echoes are represented by dots with the brightness corresponding to the strength of the signal. Though two-dimentional ultrasound is often called B-mode, this use of the terminology is technically incorrect.

Case: The outer shell of the transducer that prevents electrical injury to the patient and the operator.

Cavitation: Biological effect of the ultrasound on the tissues, caused by expansion and bursting of air bubbles in tissue.

Color Doppler: Pulsed Doppler technique that converts flow velocity information into color. Colored Doppler measures the "mean" velocity of the moving reflector.

Color map: Depicts the direction and velocity (sometimes also the variance) of the flow with relationship to the transducer. It is presented as a colored stripe in the corner of the image. The upper color represents maximal flow velocity toward the transducer; the lower color represents maximal flow velocity away from the transducer.

Compensation (also known as TGC or DGC (time or distance gain compensation): Image processing technique that is used to selectively amplify distant (deeper), and therefore weaker, echoes, making all similar reflectors look the same irrespective of the depth.

Compression: Image-processing technique that diminishes the difference between the strongest and the weakest echo signal (the brightest and the darkest parts of the image) by reducing the dynamic range.

Constructive interference: Summation of two in phase sound waves to form a wave with greater amplitude.

Continuous-wave Doppler (CW): Nonimaging ultrasound modality measuring flow velocity by Doppler shift. One active element continuously emits and the other receives ultrasound signals. CW measures maximal (peak) flow velocity, but cannot measure velocity at a selected point of flow due to signal overlap (range ambiguity).

Convex (curved)-array transducer: Transducer with active elements arranged in an arc and activated in the same manner, as in a linear-array transducer. Curved-array transducers tend to be lower-frequency abdominal transducers characterized by a large image both in the near and far field with a blunted or trapezoid sector image.

Crosstalk: Doppler mirror-image artifact.

Crystal: Active element of the ultrasound transducer.

Curie point (temperature): The temperature (360 C°) at which the active element irreversibly loses its piezoelectric properties. As a result, the transducer should never be exposed to heat sterilization.

Decibel dB (0.1 Bell): Unit of amplitude or intensity. In audible sound, it is perceived as loudness. The decibel scale is logarithmic and relative such that a 3 dB difference indicates a 2-fold change in the intensity or loudness of a sound, while a 10 dB difference indicates a 10-fold change in the intensity or loudness of a sound.

Demodulation: Image-processing technique that makes echo signals suitable for screen display.

Destructive interference: Summation of two counter-phase sound waves to form a wave with lesser amplitude.

Diffraction: The ability of sound to spread in more or less concentric circles in all directions. Higher-frequency sounds (ultrasound) diverge less than lower frequency sounds. Diffraction allows a listener to hear sound around corners.

Digital converter: Converts images into digital format for archiving and display.

Diskinetic: Organ or its part moving in the direction opposite to what is expected (e.g., aneurysmal dilatation during systole).

Display (screen, glass): That part of the ultrasound system where the image is observed.

Divergence: Spreading out of the ultrasound beam beyond the focal point. Higher-frequency transducers produce less divergence.

Doppler effect: Change in frequency of the emitted or reflected sound produced by the moving object. If the object is moving toward the receiver, the frequency increases (positive Doppler shift); if it is moving away, the frequency decreases (negative Doppler shift).

Doppler packets (ensembles): Series of multiple pulses in colored Doppler.

Doppler transducer: Utilizes Doppler effect (frequency difference between emitted and reflected ultrasound) to measure the velocity of the moving reflector.

Dosimetry: Study of the biological effects of acoustic radiation.

Duplex imaging: Modality providing an anatomical image and Doppler flow information simultaneously.

Duty factor (DF): Percentage of time when the transducer emits sound (usually **0.1–1%** in imaging and pulsed Doppler transducers). If DF is 0%, the system is off; if it is 100%, continuous-wave Doppler is on.

dV/dP: Compliance.

Dynamic frequency tuning: An imaging technique utilizing higher-frequency signals to visualize superficial structures and lower -requency signals to image deeper structures.

Dynamic range: The ratio of the strongest to the weakest signal in the ultrasound system (image gray scale). The narrower the dynamic range, the higher the image contrast.

Echo: Any reflected sound.

Echocardiography (Echo): Ultrasound study of the heart, so named by cardiologists to differentiate the cardiac examination from other applications of ultrasonography.

Echoencephalography: Archaic A-mode technique used to detect the position of midline brain structures in head trauma.

Energy (acoustic): Amount of energy delivered by the sound beam into the tissue; proportional to the bioeffects of the ultrasound radiation.

Enhancement: Low-attenuation artifact resulting in a hyperechoic (bright) image distal to a hypoechoic structure.

Five-chamber view: Apical echocardiographic view that visualizes both atria, ventricles, and the aorta. Useful for measurement of stroke volume and aortic flow velocity.

Focus or focal zone: Narrowest area (waist) of the hourglass-shaped ultrasound beam. Technically, the focus is a single point in the middle of the focal zone. The narrower the focus, the better the lateral resolution of the image.

Focusing: Techniques diminishing the size of the focus (acoustic lenses in single-crystal transducers [fixed focus] or electronic focusing in phased-array probes [adjustable focus]). Focusing improves lateral resolution.

Footprint (acoustic footprint): Area of the direct contact between the transducer and the surface of the skin. Curved-array probes, used in the abdominal ultrasound, have the largest footprint.

Fourier transform: Form of spectral analysis of Doppler signal.

Frame: One complete sweep of the mechanical or the phased-array 2D transducer. The frame is a basic element of the movie of the mobile reflector.

Frame rate: Number of frames produced by the ultrasound system per unit of time. Measured in Hz, it should not be confused with the frequency of the ultrasound wave. The higher the frame rate, the more fluid the motion and the more "real time" the 2D image. Higher frame rates result in better temporal resolution.

Fraunhofer or far zone: The area of the ultrasound beam distal to the focus where there is beam divergence.

Frequency: The number of sound oscillations (periods) occurring per unit of time (1 second). It is measured in Hertz (one period per second). This parameter is reciprocal to period (frequency $\times$ period $= 1$). Any sound with frequency of $>$20,000 Hz is an ultrasound; diagnostic ultrasound frequency is between 2,000,000 Hz and 20,000,000 Hz (2–20 MHz). Sound with frequency $<$20 Hz is an infrasound. Neither ultrasound nor infrasound is audible.

Fresnel or near zone: The area of the beam between the transducer and the focus (where the beam is converging).

Gain (receiver gain): A knob controlling amplification. Higher gain increases screen brightness (see **Amplification**).

Ghosting: Doppler artifact caused by registering the movements of adjacent structures rather than the flow of blood.

Harmonic imaging (tissue harmonics, THI): Technique utilizing echoes with frequencies that are multiples of that of the emitted signal for image formation. The frequency of the emitted sound is known as fundamental (Ff); therefore, harmonic frequency will be the fundamental frequency $\times$ 2, $\times$ 4, etc. (i.e., if Ff $=$ 2 MHz,

then with THI the image will be formed from the echoes with the frequency of 4 MHz). THI signals are generated in the tissues, eliminating some artifacts and very often (but not always) improving overall quality of the image.

Heterogeneous: Displaying multiple echo characteristics throughout the image or area of an image.

Homogeneous: Displaying the same echo characteristics throughout the image or area of an image.

Huygens' principle: Explains the formation of the hourglass shape of the ultrasound beam by the algebraic sum of the constructive and destructive interference of the individual wavelets within the beam.

Hyperechoic: Containing more echoes than usual or expected resulting in a brighter image.

Hyperkinetic: Moving more than expected.

Hypoechoic: Containing fewer echoes than usual or expected resulting in a darker image.

Hypokinetic: Moving less than expected.

Impedance: Calculated by multiplying density and propagation speed, and measured in rayls. Impedance describes the sound-transmitting and sound-reflecting properties of the medium. The boundary between two mediums with different impedance will produce reflection, but the boundary between two mediums with identical impedence will produce no reflection. The greater the difference in impedance, the greater the reflective property of the boundary. Impedances of 1,200,000–1,800,000 rayls are usual at human tissue boundaries.

Intensity: Power over area (measured in watts/cm^2). Power correlates with bioeffects. Multiple ways of measuring intensity exist, but spatial peak, temporal average (SPTA) best predicts thermal energy transfer, and therefore thermal bioeffects.

Jellyfish sign: Chest ultrasound term that describes compressed lung visualized floating in pleural fluid, undulating with the respiratory cycle.

Jet: High-velocity Doppler flow signal (high pitch and amplitude) due to valvular or vascular (arterial) stenosis.

Knobology: Knowledge of the particular controls of the ultrasound. Controls differ greatly from one ultrasound system to another, and require specific training that is unique to each device.

Laminar (parabolic) flow: Bullet-shaped, orderly flow of blood through a vessel, where the blood at the center of the vessel moves faster than that at the periphery, but the movement is in parallel lines. Associated with a spectral Doppler envelope with a thin outer line that delineates a clear space or spectral window. Distinguished from disturbed or turbulent flow associated with an obstructive lesion.

Lateral resolution (angular or transverse resolution): The minimal distance between two objects positioned along a line perpendicular to the ultrasound beam where both can be distinguished as separate objects.

Line density: The number of ultrasound beams (lines) per unit of surface forming two dimensional images. Increased line density improves spatial but reduces temporal resolution.

Linear array: Common transducer design that uses a series of piezoelectric elements arrayed in a straight line. Neighboring elements are excited simultaneously, resulting in individual scan lines parallel to one another. Often used in vascular transducers, linear arrays are usually high-frequency probes designed to visualize relatively shallow structures. They are characterized by a square image.

Lobes (side and grating): Artifacts caused by the echoes of ultrasound beams transmitted in a secondary direction (other than that of the main axis).

Long-axis plane: In echocardiography, the ultrasound plane parallel to the long axis of the left ventricle (LV). This is defined by a line that goes through the LV apex and the center of the base of the LV intersecting with the center of the aortic valve (AoV). In vascular and general ultrasound, the plane that parallels the longest dimension of the anatomical structure.

Lung flapping: Chest ultrasound term that describes compressed lung visualized floating in pleural fluid, undulating with the respiratory cycle.

McConnell sign: Diffuse hypokinesis of the right ventriculatr (RV) free wall sparing the apex. It is an echocardiographic finding suggestive of pulmonary embolism.

Mirror-image artifact: In 2D imaging, where an object adjacent to a curved-tissue plane is duplicated on the other side of the curved surface in mirror orientation; most commonly seen adjacent to the diaphragm or other highly reflective boundary (mirror). In Doppler imaging, a symmetric spectral image on the opposite side of the baseline from the true signal (cross talk).

M-mode: An early application of diagnostic ultrasound, utilizes a single line of ultrasound interrogation with the signal-plotting reflector position against the time. Useful for high temporal resolution of rapidly moving cardiac structures (i.e., valves).

Moderator band: A normal right ventricular (RV) structure housing the right bundle that can be confused with a mural RV thrombus.

Nyquist frequency limit: Pulsed-wave Doppler frequency at which aliasing occurs. Nyquist frequency limit (kHz) = PRF/2.

Oscillation: A rhythmic change in a parameter that may produce a wave.

PACS: **P**icture **A**rchiving and **C**ommunication **S**ystem; digital archiving.

Period: Time needed to complete one wave cycle. This parameter is reciprocal to frequency (frequency × period = 1). A typical value in diagnostic ultrasound is $1–5 \times 10^{-7}$ sec.

Phased-array transducers: Transducer design where the image sector is triangular and both focusing and steering is achieved electronically. Relatively high-frequency phased-array transducers offer good real-time images of moving structures. This transducer type has a small acoustic footprint, so it is useful for imaging through the intercostal spaces as in echocardiography.

Pixel: The smallest distinct element of the digital picture or movie. Increased pixel density of the image improves image quality (spatial resolution).

Power Doppler: Colored Doppler modality detecting presence of flow regardless of direction or velocity (used to detect presence or absence of flow in ischemic organs). The only colored Doppler modality not susceptible to aliasing.

Processing (signal processing): Conversion of the ultrasound signal into the image.

Pulse repetition frequency (PRF): The number of pulses emitted by imaging or pulsed-wave (PW) transducer per unit of time (usually one second); measured in Hz. Not to be confused with the frequency of the ultrasound waves.

Pulsed-wave Doppler (PW): Single crystal Doppler modality offering range resolution but subject to aliasing.

Range equation: Distance to the boundary (mm) = time of flight (μsec) × 0.77 (mm/μsec); used by the ultrasound system to position the object on the screen (13 μsec flight = 1 cm depth).

Range resolution: Ability to identify the location of the pulsed-wave Doppler sample.

Rayleigh scattering: Equal reflection in all directions occurring when the reflector is significantly smaller than the wavelength of the ultrasound.

Reflection: Return of ultrasound beam (energy) from the reflective boundary to the source in a form of an echo.

Refraction: Change in the direction of the ultrasound beam when it encounters a boundary with different propagation speed at an angle. Governed by Snell's law.

Refraction artifact: Side-by-side copy of the anatomical structure.

Regional wall motion abnormalities (RWMA): Echocardiographic term indicating segmental ventricular wall contractile dysfunction; often associated with coronary artery disease.

Reverberation artifact: Multiple, equally spaced hyperechoic horizontal lines ("Venetian Blinds") perpendicular to the direction of the ultrasound beam. Caused by the presence of two strong adjacent reflectors (i.e., parietal and visceral pleura).

Ring-down (comet-tail) artifact: Solid hyperechoic vertical line (form of reverberations).

Ringing: Internal vibrations of the active element that continue after the echo signal has been received. Ringing deteriorates the image quality. Ringing is reduced by backing of "damping" material in the transducer.

Saggital view: Long axis.

SAM: **S**ystolic **A**nterior **M**otion of the mitral leaflet (sign of hypertrophic cardiomyopathy).

Scattering: Reflection of sound in all directions.

Sector: Imaging area in two-dimensional studies. Limiting sector size improves temporal resolution.

Segmental Doppler, Doppler segmental pressures (DSP) analysis: Flow velocity detection in specific places usually utilized in arterial studies to detect the location of a stenotic area.

Snell's Law: Governs refraction (see **Refraction**). Sine (transmission angle): Sine (incident angle) = Propagation speed A: propagation speed B, where A and B are two layers at the boundary.

Spatial resolution: Ability to show image in more detail (see **Pixel**).

Spectral waveform analysis: Graphic display of flow velocity against time.

Speed of sound: Propagation speed (in soft tissue = 1540 m/sec).

Shadowing: Hypoechoic vertical linear artifact caused by the ultrasound beam encountering a high-attenuation reflector (i.e., gallstone).

Short-axis plane: Plane perpendicular to the long axis also referred to as the tranverse or cross-section plane. In echocardiography and vascular ultrasound, the imaged organ appears round.

Transcranial Doppler (TCD): Doppler study designed to detect the flow velocity of intracranial arteries (can be used to diagnose vasospasm after intracranial trauma or to document brain death).

Transmission: Onward propagation of the unreflected portion of the ultrasound beam at the reflective boundary.

Transverse view: Short-axis plane.

Turbulent flow: Chaotic disorganized flow pattern indicative of vascular stenosis or valvular heart disease. In colored Doppler echocardiography, turbulent flow is also called **mosaic** flow pattern.

Two-dimensional imaging (2D): Two-dimensional images offering gray-scale "slices" of anatomic structures in the plane of the steered or serially activated ultrasound beam. Sometimes called B-mode imaging, which is technically incorrect.

Two-dimensional phased-array transducers: Are used to form three and three-dimensional real-time (four-dimensional) images.

Velocity: Directional speed.

Vortex shed: Area distal to the jet where laminar flow becomes disturbed.

VTI: **V**elocity **t**ime **i**ntegral. Used in calculation of the stroke volume and CO.

Wave: Rhythmical transmission of energy (measured as an oscillating parameter) through the medium.

Wavelength: Length of the single cycle within the wave, measured in the units of distance (mm). Wavelength (mm) = 1.54 (mm)/frequency (MHz) in human tissues (0.1 − 1 mm is typical).

Window (acoustic window): The part of the body surface through which the ultrasound image is obtained.

Z transform: Algorithm for spectral analysis.

Zone: Ultrasound image for fixed-focus transducers (**near** = from the transducer to the focus, **far** = beneath or deeper then the focus). For multifocus transducers, the zone division is not as well defined. In most portable ICU systems, there are two separate knobs that control near-field and far-field gain. These gain areas correspond to the near and far zones.

Zoom: The ability to enlarge the image of the structure for close-up view. Preprocessing zoom increases the number of pixels per cm^2 and does not deteriorate the spatial resolution; postprocessing zoom increases the size of the individual pixels and worsens the resolution.

APPENDIX B

Draft Ultrasound Reports by Body Region

The technical and interpretive skills of the physician–sonographer will be judged by peers, including radiologists and cardiologists, and potentially in a court of law if misinterpretations occur and adverse events result. The ultrasound report is an important tool for assuring that physicians document their findings on the data gathered, the interpretation of those data, their decision about what, if anything to do with the acquired information, and the actions, if any, that were taken for the patient's benefit.

Reports should be constructed in such a manner as to provide the necessary information to those interested in understanding the procedure within the context of the patient's condition. The report serves not only for documentation, but as a communication tool from one provider or group of providers to another.

This Appendix includes several report templates that can be useful in assuring that the reports of intensive care unit (ICU) ultrasound procedures for different body regions contain the relevant information. Ultimately, each physician–sonographer will find his or her own method of relating findings in the chart to the patient's family and to other medical professionals; however, the following examples provide a reasonable starting point.

I. Ultrasound of the neck or larynx

A. General information

- State the indication for the examination (e.g., neck mapping prior to percutaneous tracheostomy or evaluation of endotracheal tube placement)
- Provide patient identifying information (name, age, and medical record number)
- Time the beginning and the end of the examination

B. Specific information

- Report your findings:
  - Begin with an overall assessment of the technical quality of the study. For example, were all views obtained? Can all the appropriate structures be visualized? Is the study adequate to answer the clinical question?
  - The physician should pay particular attention to the stated reason for the examination. The examination details should be described including the anatomic or physiologic findings. For example, the neck was mapped prior to percutaneous tracheostomy. There was a bridging anterior jugular vein at the level of the second tracheal cartilage, and a midline inferior thyroid artery, visualized in transverse and longitudinal views, with 2D and color Doppler. A suitable window for the puncture was located in between the second and third tracheal ring, and the site was marked on the skin.
- For neck mapping prior to tracheostomy, make note of the tracheal anatomy itself, such as the number of tracheal rings visible superior to the sternal notch, the width of the trachea at the proposed insertion site, which may impact the size of the inserted tube, and the angle that the trachea makes with the skin surface (e.g., parallel vs. "diving").
- Examine the entire trachea for overlying vascular structures, paying particular attention to any aberrant thyroid vessels or bridging jugular vessels. Note the depth of the trachea from the skin.
- For endotracheal tube (ETT) placement, assure that the tip is in the trachea and not the esophagus. If the tip is seen, note its distance from the cricoid cartilage and the sternal notch. Perform a bilateral pleural examination to evaluate for sliding pleura.
- If anything else is seen during the examination, note it. For example, "the neck was examined for ETT placement. The visualized

portions of the thyroid gland, carotid arteries, and jugular veins were unremarkable. There were no clots present in the jugular veins."
- Summarize the findings in a report and place the report on the chart.

II. Ultrasound examination of the chest

A. General information
- State the indication for the examination (e.g., to determine the presence, size, and characteristics of a pneumothorax or pleural effusion)
- Provide patient identifying information (name, age, and medical record number)
- Time the beginning and the end of the examination

B. Specific information
- Report your findings:
  - Begin with an overall assessment of the technical quality of the study. For example, were all views obtained? Can all the appropriate structures be visualized? Is the study adequate to answer the clinical question?)
  - The examination details should be described next including the anatomic or physiologic findings. The physician should pay particular attention to the stated reason for the examination. For example, "The patient underwent ultrasonography of the chest. Interrogation along the midclavicular and anterior axillary lines in a supine position along X to X intercostal spaces demonstrated presence (absence) of the lung sliding consistent with absence (presence) of pneumothorax." The operator should state when the sliding was noted (lung point). There was an anechoic (state ultrasound description) area consistent with the presence of pleural fluid (define anatomical borders of fluid collection, i.e., diaphragm, atelectatic lung, chest wall)
- State the condition of the lung parenchyma (i.e., local or global increased echogenicity consistent with consolidation, alveolar filling)
- Describe other important findings (e.g., lymph nodes, vascular abnormalities)
- Summarize you findings in final report
- Define the plan (e.g., proceed with thoracentesis or chest tube placement)

III. Focused transthoracic echocardiography report

A. General information
- State the indication for the examination (e.g., volume status assessment, differential diagnosis of shock)
- Provide patient identifying information (name, age, and medical record number)
- Time the beginning and the end of the examination
- Type of examination: 2D, Doppler, color flow, M-mode

B. Specific information
- Report your findings:
  - Begin with an overall assessment of the technical quality of the study. For example, were all views obtained? Can all the appropriate structures be visualized? Is the study adequate to answer the clinical question?
  - The examination details should be described next including the anatomic or physiologic findings. The physician should pay particular attention to the stated reason for the examination describing the findings in a systematic fashion. For example, the patient underwent a two-dimensional, Doppler, and M-mode examination (state only the examinations actually performed and recorded). The technical quality of the study was (e.g., optimal, suboptimal, technically limited, uninterpretable)
  - If M-mode was used to measure chamber dimensions and wall thicknesses, present the numbers and state any abnormalities ("the left atrium was enlarged to 5 centimeters")
  - Proceed with the description of the two-dimensional examination. A two-dimensional study was performed in parasternal, apical, subcostal and suprasternal views (assess technical quality of each view and state which views could be analyzed and interpreted (e.g., "only the subcostal views could be analyzed due to technical limitations") (Figure B.1)
  - Begin with the left ventricle: State if regional wall motion abnormalities (RWMA) were encountered and in which segments; correlate the abnormalities to the coronary artery anatomy and distribution/territory of blood flow (Figure B.2). For example: "RWMAs consistent with

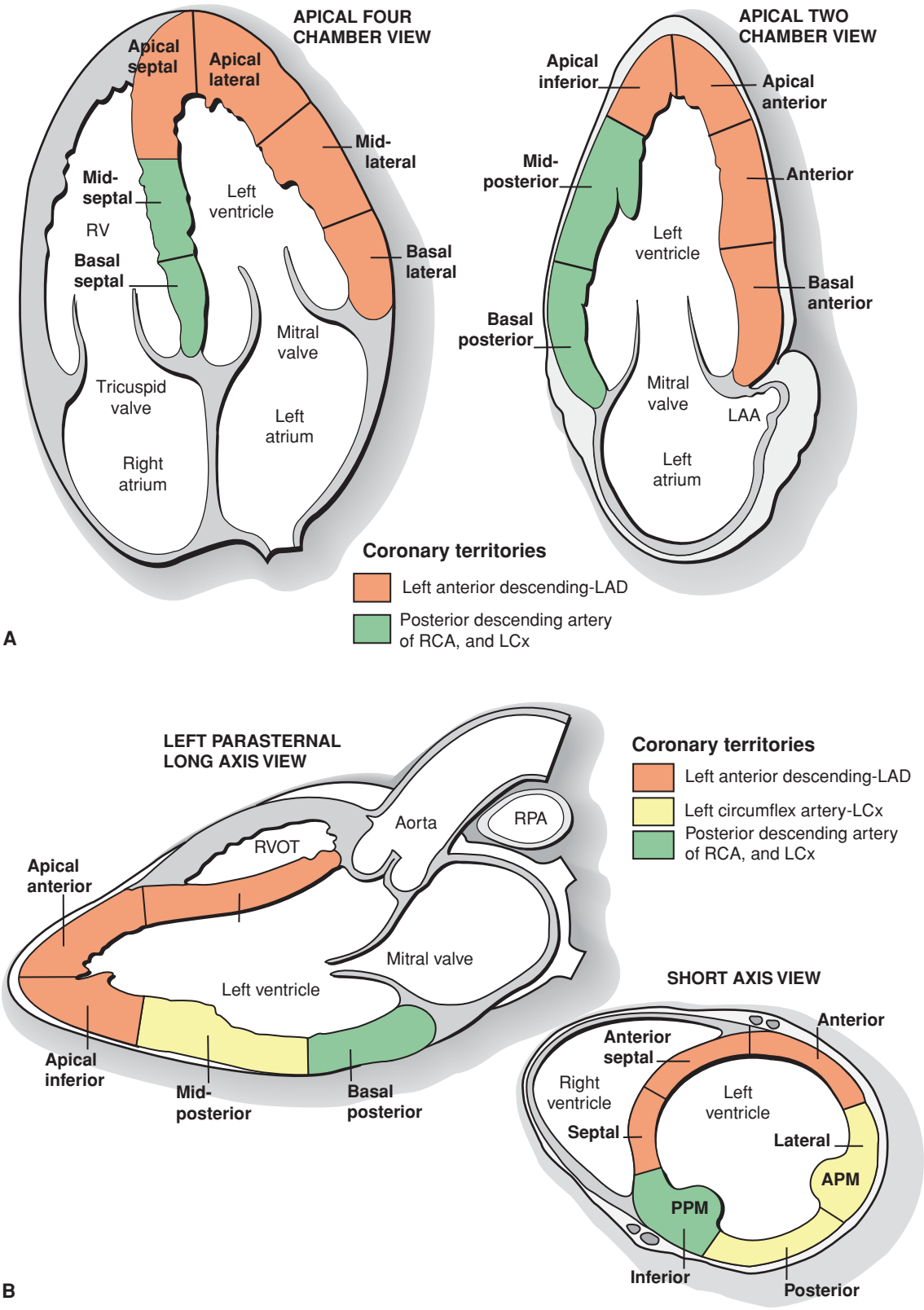


Figure B.1. Standard transthoracic echocardiographic views and regional wall motion abnormalities chart. *Reproduced with permission from Yale University Echocardiography laboratory educational website http://www.med.yale.edu/intmed/cardio/echo_atlas/contents/index.html.*

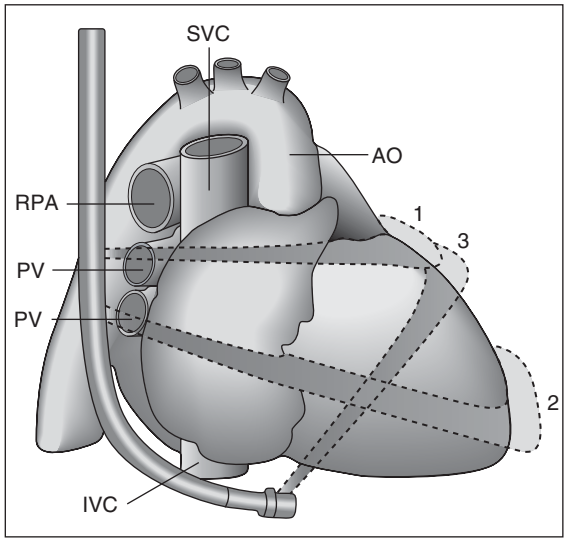


Figure B.2. Standard transesophageal echocardiographic views.

segmental disease were present, involving the mid and apical segments of the anterior free wall of the left ventricle (LV) and adjacent apical segments of intraventricular septum." Describe the overall left ventricular function and, if necessary, link it to the previous paragraph ("stated RWMA resulted in a reduction of overall left ventricular performance"). Absence of RWMA may be an important negative ("there were no RWMAs, but overall LV function was decreased"). Define left ventricular ejection fraction (LVEF) and clearly state how the number was estimated (shortening fraction, bi-plane Simpson). Link it to the prior paragraph ("Left ventricular function was reduced with estimated LVEF of 45%")

- Characterize hydration status by describing left ventricular diastolic dimensions and, if possible, left ventricular compliance. For example, "LV diastolic diameter is normal (decreased, increased), and diastolic dysfunction with decreased diastolic compliance was noted"
- Describe the visual condition of the mitral and aortic valves, including the number of aortic cusps. "The mitral valve apparatus appears normal (calcified) with good (decreased) leaflet separation. If prolapsed, identify which leaflet and give possible explanations. Are abnormal echo densities present or absent? Remember that the presence of any vegetation is a pathologic diagnosis
- Describe the right ventricle (RV) in the same systematic manner. Address RV function and condition of the ventricular septum with regard to right ventricular pressure (flatten, paradoxical septal motion). If secondary RV failure is likely, a follow-up venous study to define the possible source of pulmonary embolism may be necessary
- Characterize the condition of great vessels including the inferior and superior vena cava and aorta
- Pericardium: Is a pericardial effusion present? Assure that you define the reasons why it is pericardial and not pleural. Are there findings of pericardial tamponade (e.g., diastolic collapse of RV)? If a pleural effusion is present, some consideration should be given to proceeding with chest ultrasound and thoracentesis.
- Use Doppler studies to corroborate the findings and link them to previous findings. For example, a Doppler study demonstrated moderate mitral regurgitation consistent with previously described abnormal appearance of mitral valve. You may allow a hypothetical diagnosis here. (The presence of mitral regurgitation and an abnormal echo density on the flow side of the posterior leaflet of the mitral valve makes the diagnosis of mitral valve endocarditis likely)
- Summarize you findings in a final report. Start by answering the initial reason for the examination in the most direct manner (e.g., decreased RV and LV diastolic dimensions with good preservation of systolic function consistent with hypovolemic shock or right ventricular dilatation, increase in right ventricular systolic pressure with decrease of RV systolic function in all but apical segments, makes the diagnosis of pulmonary embolism a likely explanation for the patient's hypotension)
- Define the future plan. Remember that you are not attempting to perform a definitive diagnostic study but rather incorporating the examination into an overall care plan (e.g., will reassess cardiac function and hydration status after an attempt at fluid resuscitation using 30 cc/kg isotonic fluids or will proceed with anticoagulation and computerized tomography (CT)

angiogram of the chest). May have to link to a procedure or other bedside examination as stated previously (e.g., will use ultrasonic guidance for pericardiocentesis or, because sepsis with an abdominal source is highly suspected, will follow with bedside examination of the abdomen.

IV. Transesophageal echocardiography (TEE) report

A. General information

- State the indication for the examination (e.g., volume status assessment, differential diagnosis of shock)
- Provide patient identifying information (name, age, and medical record number)
- Time the beginning and the end of the examination
- Personnel: Physician performing study
- Medication used to facilitate the procedure (sedatives, analgesics)
- Equipment and modalities used including which TEE probe and system, 2D imaging, color flow, spectral Doppler
- Complications: Clearly state if none were encountered

B. Specific information

- Report your findings:
  - Begin with an overall assessment of the technical quality of the study. For example, were all views obtained (esophageal, transgastric)? Can all the appropriate structures be visualized (great vessels, cardiac chambers, valves)? Is the study adequate to answer the clinical question?
  - The examination details should be described next, including the anatomic or physiologic findings. The physician should pay particular attention to the stated reason for the examination, describing the findings in a systematic fashion. For example, the patient underwent a two-dimensional, Doppler, and M-mode examination (state only the examinations actually performed and recorded). The technical quality of the study was (e.g., optimal, suboptimal, technically limited, uninterpretable)
  - Procedural information should begin with the left ventricle and comment on:
    - Size: (normal, reduced, mildly dilated, moderately dilated, severely dilated)
    - Global systolic function: (normal, hyperdynamic, mildly reduced moderately reduced, severely reduced)
    - Regional wall motion abnormalities: (present or absent, and characterize)
  - Doppler findings may be included here including:
    - Color-flow Doppler: Valvular regurgitation may be qualitatively described as well as semiquantitated based upon visualization (absent/none, mild regurgitation, moderate regurgitation, severe regurgitation)
    - Spectral Doppler: Report pertinent findings of a focused examination (e.g., pulmonary vein flow)
    - Summarize the pertinent findings and specifically state how the findings relate to the primary indication for the study. Include a statement about chamber sizes, the left ventricle, and the presence or absence of significant valvular disease. Other categories, such as great vessels and pericardium, may be included if positive findings are noted

V. Ultrasound examination of the abdomen and retroperitoneal space

A. General information

- State the indication for the examination (e.g., differential diagnosis of pain, including location or abnormal laboratory values or other test results)
- Provide patient identifying information (name, age, and medical record number)
- Time the beginning and the end of the examination
- Personnel: Physician performing study
- Medication used to facilitate the procedure (sedatives, analgesics)
- Equipment used: Scan performed using ultrasound instrument and a ( ) MHz curved transducer

B. Specific information

- Report your findings:
  - Begin with an overall assessment of the technical quality of the study. For example, were all views obtained? Can all the appropriate structures be visualized? Is the study adequate to answer the clinical question?
  - The examination details should be described next, including the anatomic or physiologic findings. Table B.1 provides an opportunity to present the findings in a tabular rather than descriptive form. This

TABLE B.1. An example of a templated report for detailing an abdominal and pelvic ultrasound in the intensive care unit

Structure	*Normal*	*Abnormal*	*Not seen*	*Comments*
Liver				
Gallbladder				❒ stones, ❒ sludge, ❒ wall thickness __ mm ❒ CBD dilated __ mm, Other:
Spleen				
Left kidney				
Left adrenal				
Left ovary				
Bladder				
Pelvic region				
Right kidney				
Right adrenal				
Right ovary				
IVC/aorta				
Pancreas				
Duodenum				
Ascites	❒ Absent	❒ Present		
Pericardium				
Left costophrenic angle				
Right costophrenic angle				
Other				

CBD indicates common bile ducts; IVC, inferior vena cava.

approach adds the value of assuring that all elements of the examination are addressed in the template and that appropriate descriptors are used between different physicians in a given department. The physician should pay particular attention to the stated reason for the examination describing the findings in a systematic fashion. The technical quality of the study was (e.g., optimal, suboptimal, technically limited, uninterpretable)

- ◦ Summarize you findings in a final report and suggest repeat examinations if necessary

VI. Venous duplex examination of the extremities

A. General information

- State the indication for the examination (e.g., assess for presence of deep venous thrombosis in a patient with significant oxygen requirement and possible pulmonary embolism)
- Provide patient identifying information (name, age, and medical record number)
- Time the beginning and the end of the examination
- Personnel: Physician performing study
- Equipment used: Scan performed using ultrasound instrument and a ( ) MHz curved transducer

B. Specific information

- Report your findings:
  - ◦ Begin with an overall assessment of the technical quality of the study. For example, were all views obtained? Can all the appropriate structures be visualized? Is the study adequate to answer the clinical question?
  - ◦ The examination details should be described next, including the anatomic or physiologic findings
    - ◦ Venous Duplex examination of the venous system of the (right, left, both)

lower (upper) extremity (extremities) was/were performed using the Duplex ultrasound instrument and a ( ) MHz (linear/curved) phased-array transducer
- Gray-scale imaging demonstrated (good) incomplete compressibility of the (name the abnormal vessels) veins. Intraluminal echogenic material was seen in the (location) veins. The proximal tip of the intraluminal material was (mobile/stable)
- Doppler waveform analysis demonstreated (did not demonstrate) loss of phasicity with respiration/no flow signal) suggesting outflow obstruction
- Color-flow imaging demonstrated (did not demonstrate) abnormal/absent flow

- Summarize your findings in final report and suggest repeat examinations if necessary (e.g., duplex ultrasound findings consistent with deep venous thrombosis involving (name the veins) of the (right/left/both) lower extremity(extremities) or no evidence of deep venous thrombosis)

VII. Procedures

A. Vascular access procedures:

i. General information
- State the indication for the examination (e.g., ultrasound-guided vascular access for shock)
- Provide patient identifying information (name, age, and medical record number)
- Time the beginning and the end of the examination
- Personnel: Physician performing study
- Equipment used: Scan performed using ultrasound instrument and a ( ) MHz curved transducer
- Complications: Clearly state if none were encountered

ii. Specific information
- Preprocedure ultrasound assessment: Patient's (state vein location, e.g., internal jugular, common femoral, deep veins of upper extremities) were assessed with ultrasound for patency and position, and marked
  - If static guidance was used, that is all that should be reported, and representative image should be retained for the chart
  - If dynamic guidance was used, the following report may be helpful: The patient was placed in (state position), prepped in the usual manner, and placed under sterile drapes. An ultrasound probe was placed into the sterile sheath and sterile ultrasound gel was applied to the site of the procedure. (Name vein) was adequately (or not) visualized. Under direct ultrasound visualization, the needle was positioned into the vein, good blood return assured. A J-wire easily passed, with the position of the wire in the vein, was (was not) confirmed ultrasonographically. Proceed with the remaining description of the modified Seldinger technique.
  - Remember that this may link with an ultrasound of the chest if axillary, subclavian, or internal jugular veins were cannulated to rule out procedure-related complications (i.e., pneumothorax), particularly if immediate use of the line is highly desirable

B. Thoracentesis, paracentesis, pericardiocentesis

i. General information
- State the indication for the examination (e.g., suspected empyema or spontaneous peritonitis)
- Provide patient identifying information (name, age, and medical record number)
- Time the beginning and the end of the examination
- Personnel: Physician performing study
- Equipment used: Scan erformed using ultrasound instrument and a ( ) MHz curved transducer
- Complications: Clearly state if none were encountered

ii. Specific information
- Preprocedure ultrasound assessment: Patient was placed in (state position). A collection of pleural (peritoneal) fluid was visualized via ultrasound (state anatomical boundaries and ultrasound characteristics of the fluid and its estimated volume) and marked. For pericardiocentesis, state which echo view was used.

- If static guidance was used, that is all that should be reported, and representative image should be retained for the chart
- If dynamic guidance was used, the following report may be helpful: The patient was placed in (state position), prepped in the usual manner, and placed under sterile drapes. An ultrasound probe was placed into the sterile sheath and sterile ultrasound gel was applied to the site of the procedure. A fluid collection was adequately (or not) visualized. Under direct ultrasound visualization, the needle was positioned into the collection and good fluid return assured. Proceed with describing procedure. If a pigtail catheter or other device is retained, remember to state if its position was confirmed by the ultrasound.

Index

U