

ACC/AHA PRACTICE GUIDELINES—FULL TEXT

ACC/AHA/ASE 2003 Guideline Update for the Clinical Application of Echocardiography

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASE Committee to Update the 1997 Guidelines for the Clinical Application of Echocardiography)

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PREAMBLE

It is clearly important that the medical profession play a significant role in the critical evaluation of the use of diagnostic procedures and therapies in the management or prevention of disease. Rigorous and expert analysis of the available data documenting relative benefits and risks of those procedures and therapies can produce helpful guidelines that improve the effectiveness of care, optimize patient outcomes, and impact the overall cost of care favorably by focusing resources on the most effective strategies.

The American College of Cardiology (ACC) and the American Heart Association (AHA) have jointly engaged in the production of such guidelines in the area of cardiovascular disease since 1980. This effort is directed by the ACC/AHA Task Force on Practice Guidelines. Its charge is to develop and revise practice guidelines for important cardiovascular diseases and procedures. Experts in the subject under consideration are selected from both organizations to examine subject-specific data and write guidelines. The process includes additional representatives from other medical practitioner and specialty groups as appropriate. Writing groups are specifically charged to perform a formal literature review, weigh the strength of evidence for or against a particular treatment or procedure, and include estimates of expected health outcomes where data exist. Patient-specific modifiers, comorbidities, and issues of patient preference that might influence the choice of particular tests or therapies are considered, as well as frequency of follow-up and cost-effectiveness.

These practice guidelines are intended to assist physicians in clinical decision making by describing a range of generally acceptable approaches for the diagnosis, management, or prevention of specific diseases or conditions. These guidelines attempt to define practices that meet the needs of most patients in most circumstances. The ultimate judgment regarding care of a particular patient must be made by the physician and patient in light of all of the circumstances presented by that patient.

The 1997 Committee to Develop Guidelines on the Clinical Application of Echocardiography was chaired by Melvin D. Cheitlin, MD, MACC, and included the following members: Joseph S. Alpert, MD, FACC, FAHA; William F. Armstrong MD, FACC, FAHA; Gerard P. Aurigemma, MD, FACC, FAHA; George A. Beller, MD, FACC, FAHA; Fredrick Z. Bierman, MD, FACC; Thomas W. Davidson, MD, FAFAP; Jack L. Davis, MD, FACC; Pamela S. Douglas, MD, FACC, FAHA, FASE; Linda D. Gillam, MD, FACC, FAHA; Richard P. Lewis, MD, FACC; Alan S. Pearlman, MD, FACC, FAHA, FASE; John T. Philbrick, MD, FACP; Pravin M. Shah, MD, FACC; and Roberta G. Williams, MD, FACC. The document update used the 1997 work as its basis. The Committee to Update the ACC/AHA/ASE Guidelines on Clinical Application of Echocardiography was chaired by Melvin D. Cheitlin, MD, MACC, and included the following members: William F. Armstrong MD, FACC, FAHA; Gerard P. Aurigemma MD,

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The ACC/AHA Task Force on Practice Guidelines makes every effort to avoid any actual or potential conflicts of interest that might arise as a result of an outside relationship or personal interest of a member of the writing panel. Specifically, all members of the writing panel are asked to provide disclosure statements of all such relationships that might be perceived as real or potential conflicts of interest. These statements are reviewed by the parent task force, reported orally to all members of the writing panel at the first meeting, and updated as changes occur. (See appendix for writing committee member relationships with industry.)

The ACC/AHA/ASE 2003 Guideline Update for Echocardiography was approved for publication by the ACC Board of Trustees in May 2003, the AHA Science and Advisory Coordinating Committee in May 2003, and the American Society of Echocardiography in May 2003. The summary article is published in the September 2, 2003 issue of *Circulation*, the September 3, 2003 issue of the *Journal of the American College of Cardiology*, and the October 2003 issue of the *Journal of the American Society of Echocardiography*. The full-text guideline is posted on the ACC (www.acc.org), AHA (www.americanheart.org), and ASE (www.asecho.org) World Wide Web sites. Copies of both the full text and the summary article are available from all three organizations. These guidelines will be reviewed 1 year after publication and yearly thereafter and considered current unless the Task Force on Practice Guidelines revises or withdraws them from circulation.

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I. INTRODUCTION, GENERAL CONSIDERATIONS, AND SCOPE

The previous guidelines for the use of echocardiography were published in March 1997. Since that time there have been significant advances in the technology of echocardiography and growth in its clinical use and in the scientific evidence leading to recommendations for its proper use. Each section has been reviewed and updated both in evidence tables and, where appropriate, changes made in recommendations. A new section on the use of intraoperative transesophageal echocardiography (TEE) is being added to

update the guidelines published by the American Society of Anesthesiologists and the Society of Cardiovascular Anesthesiologists (SCA). There are extensive revisions especially of the sections on ischemic heart disease; congestive heart failure, cardiomyopathy, and assessment of left ventricular function; and screening and echocardiography in the critically ill. There are new tables of evidence and extensive revisions in the ischemic heart disease evidence tables.

The committee to update the echocardiography guidelines was composed of both university-affiliated and practicing physicians, those with specific echocardiographic expertise, and senior clinicians who use the technique. Two general physicians (one general internal medicine and one family practitioner) and a cardiac anesthesiologist also served on the committee. The document was reviewed by two outside reviewers nominated by the ACC, two outside reviewers nominated by the AHA, and two outside reviewers nominated by the American Society of Echocardiography (ASE).

For this guideline update, literature searching was conducted in MEDLINE, EMBASE, Best Evidence, and the Cochrane Library for English-language meta-analyses and systematic reviews from 1995 through September 2001. Further searching was conducted for new clinical trials on the following topics: echocardiography in adult congenital heart disease, echocardiography for evaluation of chest pain in the emergency department, and intraoperative echocardiography. The new searches yielded more than 1000 references that were reviewed by the writing committee.

The original recommendations of the 1997 guidelines are based on a MEDLINE search of the English literature from 1990 to May 1995. Echocardiography was cross-referenced with the following terms: antineoplastic agents, aortic or dissecting aneurysm, arrhythmias, athletes, atrial fibrillation, cardioversion, Marfan syndrome, bacterial endocarditis, myocardial infarction, myocardial ischemia, coronary disease, chest pain, cardiomyopathies, cerebrovascular disorders or cerebral ischemia, embolism, heart neoplasms, heart valve disease, heart murmurs, hypertension, mitral valve prolapse, pericarditis, pericardial effusion, cardiac tamponade, pericardium, pulmonary embolism or pulmonary heart disease or cor pulmonale, screening, shock or aortic rupture or heart rupture, syncope, transplantation, unstable angina, congenital heart disease in the adult, specific congenital lesions, arrhythmias in children, pediatric echocardiography, and fetal echocardiography.

The original search yielded over 3000 references, which the committee reviewed. This document includes recommendations for the use of echocardiography in both adult and pediatric patients. The pediatric guidelines also include recommendations for fetal echocardiography, an increasingly important field. The guidelines include recommendations for the use of echocardiography in both specific cardiovascular disorders and in the evaluation of patients with frequently observed cardiovascular symptoms and signs, common presenting complaints, or findings of dyspnea, chest discomfort, and cardiac murmur. In this way the guidelines will provide assistance to physicians regarding the use of echocardiographic techniques in the evaluation of such common clinical problems.

The recommendations concerning the use of echocardiography follow the recommendation classification system (eg, Classes I, II, and III) used in other ACC/AHA guidelines:

Class I: Conditions for which there is evidence and/or general agreement that a given procedure or treatment is useful and effective.

Class II: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.

Class IIa: Weight of evidence/opinion is in favor of usefulness/efficacy.

Class IIb: Usefulness/efficacy is less well established by evidence/opinion.

Class III: Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful/effective and in some cases may be harmful.*

*Because it is not likely that harm will occur by performing an echocardiogram, the reason for the Class III designation in this guideline is almost exclusively that there is no evidence that performing an echocardiogram has been shown to be helpful.

Evaluation of the clinical utility of a diagnostic test such as echocardiography is far more difficult than assessment of the efficacy of a therapeutic intervention, because the diagnostic test can never have the same direct impact on patient survival or recovery. Nevertheless, a series of hierarchical criteria are generally accepted as a scale by which to judge worth (1-3).

A. Hierarchical Levels of Echocardiography Assessment

- Technical capacity
- Diagnostic performance
- Impact on diagnostic and prognostic thinking
- Therapeutic impact
- Health-related outcomes

The most fundamental criterion is technical capacity, including adequacy of equipment and study performance. The next is diagnostic performance, which encompasses much of traditional diagnostic test assessment, including delineation of the range of clinical circumstances in which a test is applicable, as well as test sensitivity, specificity, and accuracy for individual applications. The third criterion is the capability of a test to alter diagnostic and prognostic thinking, ie, to offer added value. This level depends on the context in which the test is performed and is therefore affected by such factors as what is already known, the judged value of

confirmatory data, and the importance of reassurance in a particular clinical situation. Impact on diagnostic and prognostic thinking is an important link between test results and patient treatment. Subsequent criteria include therapeutic impact and health-related outcomes. Because there are essentially no randomized trials assessing health outcomes for diagnostic tests, the committee has not ranked the available scientific evidence in an A, B, C fashion (as in other ACC/AHA documents) but rather has compiled the evidence in tables. All recommendations are thus based either on this evidence from observational studies or on the expert consensus of the committee.

Two-dimensional echocardiography can provide excellent images of the heart, paracardiac structures, and the great vessels. Because it depends on satisfactory examining windows from the body surface to the cardiovascular structures, there may be limitations on its use for adult patients. For patients with chronic obstructive pulmonary disease, the interposition of air-filled lung between the body surface and the heart severely limits access, and complete examination may not be possible. Other circumstances limit the use of transthoracic echocardiography (TTE), especially for patients in the intensive care unit. For example, patients on ventilators, those who cannot be rotated into a lateral position, and those with incisions may not have satisfactory precordial or apical windows. TEE may avoid most of these limitations because there is no interposed lung tissue between the transducer and the heart.

The definition of echocardiography used in this document incorporates Doppler analysis, M-mode echocardiography, two-dimensional TTE, and, when indicated, TEE. Intravascular ultrasound is not considered but is reviewed in the ACC/AHA Guidelines for Percutaneous Coronary Intervention (515) (available at <http://www.acc.org/clinical/guidelines/percutaneous/dirIndex.htm>) and the Clinical Expert Consensus Document on intravascular ultrasound (516) (available at <http://www.acc.org/clinical/consensus/standards/standard12.htm>). Echocardiography for evaluating the patient with cardiovascular disease for noncardiac surgery is considered in the ACC/AHA Guidelines for Perioperative Cardiovascular Evaluation for Noncardiac Surgery (517). The techniques of three-dimensional echocardiography are still in the developmental stages and also are not considered here.

New techniques that are experimental or are still evolving and for which there is no agreement on their clinical usefulness as well as improvements that are purely technological in echo-Doppler instrumentation, such as harmonic imaging are also not going to be discussed separately in the clinical recommendations addressed in this document. Tissue Doppler imaging, both pulsed and color, which detects low Doppler shift frequencies of high energy generated by the contracting myocardium and consequent wall motion, is still being evaluated and may prove useful in assessing systolic and diastolic function. However, these technological advances will also not be discussed separately in the clinical recommendations (518,519). Echocardiographic-contrast

injections designed to assess myocardial perfusion to quantify myocardium at risk and perfusion beds also are not addressed.

With the development of Doppler echocardiography and proof that the modified Bernoulli equation permitted the conversion of instantaneous velocities of blood flow into instantaneous pressure gradients across obstructions, it became possible to precisely localize and quantify obstruction in the cardiovascular system. This information, when considered with flow volume information provided by Doppler flow velocity integrals, allows a plethora of physiological and functional information to be obtained noninvasively. The differing capabilities of the several types of available Doppler echocardiographic techniques are outlined in Table 1. Recognizing the strengths of each technique will enable the physician to order the appropriate study. Generally a complete transthoracic echocardiogram and Doppler study is called for unless otherwise specified.

When faced with a patient needing cardiovascular evaluation and testing, the clinician must choose among available tests. Echocardiography, nuclear testing, magnetic resonance imaging (MRI), and positron emission tomography can yield overlapping if not identical information, often with similar or comparable accuracy. Decisions concerning which technique to use must then be based on such factors as local expertise in performance and interpretation, test availability, cost, and patient preference. Therefore, it is impossible in this document to judge competing tests or recommend the use of one over another.

TTE is associated with little if any patient discomfort, and no risks with this procedure have been identified. Moreover, the use of TTE with exercise or vasoactive drugs such as dipyridamole or dobutamine involves the minimal risks of arrhythmia, ischemia, and hypotension seen with exercise and the aforementioned drugs. In TEE, the echocardiographic transducer is mounted on a flexible endoscope and passed into the esophagus and stomach. This involves some discomfort and minimal but definite risk of pharyngeal and esophageal trauma and even rarely esophageal perforation. Rare instances of infective endocarditis have been associated with the use of TEE. An occasional patient has a reaction to either the sedative or the local anesthesia used.

The ability of echocardiography to provide unique noninvasive information with minimal discomfort or risk without using contrast material or ionizing radiation, coupled with its portability, immediate availability, and repeatability, accounts for its use in virtually all categories of cardiovascular disease. However, echocardiography is best used after a careful history, physical examination, appropriate electrocardiogram (ECG), and chest radiograph have been obtained so that the appropriate questions can be asked. Indiscriminate use of echocardiography or its use for "screening" is not indicated for two principal reasons. First, the cost of echocardiography is not trivial. Second, the current echocardiographic techniques reveal details of structure and function such as filamentous strands on valves, valvular prolapse, and jet velocities representing minimal and at

Table 1. Doppler Echocardiography Capabilities in the Adult Patient

	Echocardiography		Spectral Doppler	Color Doppler	TEE
	M mode	2 D			
<i>Anatomy-Pathology</i>					
Chamber size	++++	++++	-	-	++
Thickness of walls	++++	+++	-	-	+++
Relation of chambers	+	++++	-	-	+++
Early closure of MV	++++	+	-	-	+
Systolic anterior motion of MV	++++	+++	-	-	+++
LV mass (g)	++++	++++	-	-	-
LV masses (tumor, clot, vegetation)	+	+++	-	-	++++
Masses in atria and right ventricle	+	++	-	-	++++
Anatomic valvular pathology	++	++++	-	-	++++
Septal defects	+	++++*	++	++++	++++
Pericardial effusion	++	++++	-	-	++
<i>Function</i>					
Global LV systolic function (EF)	++	++++	++	-	+++
Regional wall motion	+	+++	-	-	++++
Severity of valve stenosis	+	++	++++	+++	++
Severity of valve regurgitation	+	+	+++	+++	+++
Site of left-to-right, right-to-left shunt	-	++++* (together)	+++	++++ (together)	+++
RV and PA systolic pressure	-	-	++++	-	-
LV filling pressure	-	-	++	-	-
Stroke volume and cardiac output	+	++ (together)	+++	-	-
LV diastolic function	+	+	+++	-	-
Identify ischemia and viable myocardium with exercise or pharmacological stress	-	+++	-	-	-
Diseases of the aorta	-	++	-	++	++++
Prosthetic valve evaluation	+	++	++++	+++	++++

++++ indicates most helpful; +, least useful; -, not useful; 2D, two-dimensional; EF, ejection fraction; LV, left ventricular; MV, mitral valve; PA, pulmonary artery; RV, right ventricular; TEE, transesophageal echocardiography.

*With contrast (intravenous injection of agitated saline).

Note: The committee recognizes that this table is a subjective evaluation. The magnitude of usefulness is indicated by the number of plus (+) signs. It is assumed that M-mode, two-dimensional echocardiography, spectral and color flow Doppler, and TEE will be available in the ultrasound laboratory. A given examination will potentially use most or all of these modalities to some extent. It is assumed that TEE will incorporate Doppler. Where transthoracic echocardiography is inadequate, TEE frequently can obtain the desired information.

times transient valvular insufficiency that could generate unnecessary further testing or inappropriate and potentially detrimental therapy.

These guidelines contain recommendations concerning not only recommendations for the use of these techniques but also specific circumstances when echocardiography adds little or nothing to the care of the patient and is therefore not indicated. An example is the evaluation of the patient with a clearly innocent murmur in the opinion of a qualified, knowledgeable examining physician. Another example is the use of echocardiography in diagnosing mitral valve prolapse (MVP) in a patient with chest pain or premature ventricular contractions in the absence of clinical findings consistent with MVP. Because there is no evidence that such patients have an increased risk of endocarditis beyond the general population which does not have “echo-only” MVP, echocardiography is generally not indicated in this situation.

An echocardiographic study is not indicated when the pathology and/or systolic ventricular function have been adequately defined by other techniques, making the echocardiographic study redundant. Furthermore, echocardiography

should be performed by laboratories with adequately trained physicians and cardiac sonographers where patient volume recommendations are met as previously described (3).

These guidelines also address recommendations about the frequency with which an echocardiographic study is repeated. If the frequency with which studies are repeated could be decreased without adversely affecting the quality of care, the economic savings realized would likely be significant. With a noninvasive diagnostic study and no known complications, the potential for repeating the study unnecessarily exists. It is easier to state when a repeat echocardiogram is not needed than when and how often it should be repeated, since no studies in the literature address this question. An adult patient with hemodynamically insignificant aortic regurgitation almost certainly does not need a repeat echocardiogram unless there is a change in the clinical picture. The asymptomatic patient with hemodynamically severe aortic regurgitation probably needs repeat echocardiography to monitor left ventricular (LV) function. How often this should be done depends on the individual patient and must be left to the judgment of the physician until evidence-based data address-

ing this issue are available.

The use of echocardiography in establishing cardiac diagnoses and making therapeutic decisions, at times without further diagnostic studies, is well established. Examples include the demonstration of an acquired ventricular septal defect in a patient with an acute myocardial infarction. In the past this diagnosis required catheterization; now the definitive diagnosis can be made in most cases with Doppler echocardiography. At times the echocardiogram can enable cardiac surgery to proceed without a comprehensive catheterization. Examples of this are the finding of severe aortic stenosis or mitral or aortic regurgitation in the symptomatic young patient or the finding of a left atrial myxoma.

The use of repeated echocardiographic studies in monitoring patients is illustrated in adult patients with moderate aortic stenosis who have a change in symptoms. Similarly, the follow-up evaluation of ventricular function in the patient with chronic aortic or mitral valvular insufficiency lesions can help determine the timing of valvular surgery.

The American Heart Association has published a Scientific Statement on standardizing myocardial segmentation nomenclature for tomographic imaging of the heart, whether by magnetic resonance imaging, echocardiography PET scanning, or computed tomography (832). These recommendations may become standard terminology for all imaging techniques.

This document assumes that echocardiographic studies are performed and interpreted in accordance with the statements for clinical competence in echocardiography set forth by the Joint Task Force of the American College of Physicians/American College of Cardiology/American Heart Association. Optimal training for such studies is set forth by the ASE, the ACC, and the Society of Pediatric Echocardiography.

II. MURMURS AND VALVULAR HEART DISEASE

Echocardiography is extremely useful in the assessment of cardiac murmurs, stenosis and regurgitation of all four cardiac valves, prosthetic valve function, and patients with infective endocarditis. Echocardiography provides valuable information regarding diagnosis, valvular morphology, etiology of valve disease, identification and quantification of lesions, detection and evaluation of associated abnormalities, delineation of cardiac size and function, and assessment of the adequacy of ventricular compensation. Echocardiography readily detects structural abnormalities such as fibrosis, calcification, thrombus, or vegetation and abnormalities of valvular motion such as immobility, flail or prolapsing leaflets, or prosthetic valve dehiscence. A full echocardiographic evaluation should provide prognostic as well as diagnostic information, allow for risk stratification, establish baseline data for subsequent examinations, and help guide and evaluate the therapeutic approach.

Echocardiography often provides a definitive diagnosis

and may obviate the need for catheterization in selected patients. Patients' acceptance of this noninvasive technique for initial and re-evaluation observation is high (6-8). MRI has the capability to detect the presence of stenotic and regurgitant lesions (9,10) and has several advantages. However, MRI instrumentation is substantially more expensive and not as widely available.

A. Murmurs

Cardiac auscultation remains the most widely used method of screening for heart disease. Heart murmurs are produced by turbulent blood flow and are often signs of stenotic or regurgitant valve disease or acquired or congenital cardiovascular defects. In valvular and congenital forms of heart disease, a murmur is usually the major evidence of the abnormality, although some hemodynamically significant regurgitant lesions may be silent (11,12). However, many murmurs in asymptomatic people are innocent and of no functional significance. Such murmurs are defined as having the following characteristics: a systolic murmur of short duration, grade 1 or 2 intensity at the left sternal border, a systolic ejection pattern, a normal S₂, no other abnormal sounds or murmurs, no evidence of ventricular hypertrophy or dilation, no thrills, and the absence of an increase in intensity with the Valsalva maneuver. Such murmurs are especially common in high-output states such as pregnancy (13,14). When the characteristic findings of an individual murmur are considered together with other patient information and clinical data from the physical examination, the correct diagnosis can usually be established (15). In patients with ambiguous clinical findings, the echocardiogram may be the preferred test because it can provide a definitive diagnosis, rendering a chest radiograph and/or ECG unnecessary. In some patients the Doppler echocardiogram is the only noninvasive method capable of identifying the cause of a heart murmur (12,520).

In the evaluation of heart murmurs, the purposes of performing a Doppler echocardiogram are to

- Define the primary lesion and its etiology and judge its severity
- Define hemodynamics
- Detect coexisting abnormalities
- Detect lesions secondary to the primary lesion
- Evaluate cardiac size and function
- Establish a reference point for future observations
- Reevaluate the patient after an intervention

As valuable as echocardiography may be, the basic cardiovascular evaluation, including history, physical examination and ECG, is still the most appropriate method to screen for cardiac disease and will establish many clinical diagnoses (17). Echocardiography should not be used to replace the cardiovascular examination but can be helpful in determin-

ing the etiology and judging the severity of lesions, particularly in pediatric and elderly patients (15,17-19).

Recommendations for Echocardiography in the Evaluation of Patients With a Heart Murmur

Class I

1. A patient with a murmur and cardiorespiratory symptoms.
2. An asymptomatic patient with a murmur in whom clinical features indicate at least a moderate probability that the murmur is reflective of structural heart disease.

Class IIa

A murmur in an asymptomatic patient in whom there is a low probability of heart disease but in whom the diagnosis of heart disease cannot be reasonably excluded by the standard cardiovascular clinical evaluation.

Class III

In an asymptomatic adult, a heart murmur that has been identified by an experienced observer as functional or innocent.

B. Native Valvular Stenosis

Two-dimensional and Doppler echocardiography reliably identify and quantitate the severity of stenotic lesions of both native and prosthetic valves. Mitral stenosis is accurately quantified by planimetry of transthoracic or transesophageal two-dimensional images, Doppler measurement of transvalvular gradients, and estimation of valve area by the pressure half-time or continuity methods (20-23). In difficult-to-image patients, contrast may improve signal detection.

When the Doppler flow signal is suboptimal, administration of an echocardiographic contrast agent may improve signal detection. Agitated saline may be adequate for right-sided lesions, but left-sided contrast agents will be required for left-sided lesions.

Prognostic information is obtained from assessment of the hemodynamic response to stress including exercise (24) and dobutamine stress in the case of aortic stenosis (521) and/or by delineation of morphological characteristics (25), which in turn help guide the selection of therapeutic interventions (26).

Recommendations for Echocardiography in Valvular Stenosis

Class I

1. **Diagnosis; assessment of hemodynamic severity.**
2. **Assessment of LV and right ventricular (RV) size, function, and/or hemodynamics.**
3. **Re-evaluation of patients with known valvular stenosis with changing symptoms or signs.**
4. **Assessment of changes in hemodynamic severity and ventricular compensation in patients with known**

valvular stenosis during pregnancy.

5. **Re-evaluation of asymptomatic patients with severe stenosis.**

Class IIa

1. **Assessment of the hemodynamic significance of mild to moderate valvular stenosis by stress Doppler echocardiography.**
2. **Re-evaluation of patients with mild to moderate aortic stenosis with LV dysfunction or hypertrophy even without clinical symptoms.**

Class IIb

1. **Re-evaluation of patients with mild to moderate aortic valvular stenosis with stable signs and symptoms.**
2. **Dobutamine echocardiography for the evaluation of patients with low-gradient aortic stenosis and ventricular dysfunction.**

Class III

1. **Routine re-evaluation of asymptomatic adult patients with mild aortic stenosis having stable physical signs and normal LV size and function.**
2. **Routine re-evaluation of asymptomatic patients with mild to moderate mitral stenosis and stable physical signs.**

(See also “Recommendations for Echocardiography in Interventions for Valvular Heart Disease and Prosthetic Valves.”)

TEE has also been useful in guiding balloon valvuloplasty procedures (27).

Although tricuspid stenosis is readily detected and assessed hemodynamically, the accuracy of Doppler echocardiographic determinations is less well validated but still preferred over other methods (28).

Aortic stenosis is accurately quantified by Doppler measurements of instantaneous and mean transvalvular gradients, estimation of valve area by the continuity method, or determination of aortic valve resistance (29-31,522,523). In patients with reduced LV function, gradient measurements may appear falsely low, while valve area and resistance measurements will more reliably predict the severity of stenosis. Dobutamine perturbation with Doppler assessment of gradients may also be of use (32), particularly in patients with low output and a low gradient. The problem is differentiating the patient with severe aortic stenosis with poor LV function and a small stroke volume from the patient with mild aortic stenosis and poor LV function resulting from another cause such as coronary artery disease or cardiomyopathy. By increasing the cardiac output with dobutamine, the patient with severe aortic stenosis, unable to further open the valve, will have an increase in systolic gradient, whereas the patient with cardiomyopathy will open the valve wider, and the gradient will not increase (524,525). Pulmonic valve gradients are similarly quantified. While still experimental, contrast injection may allow more accurate recording of

stenotic jet velocities and therefore transvalvular gradients (33).

C. Native Valvular Regurgitation

Doppler echocardiography is the most sensitive technique available for detection of native valve regurgitation; care must be taken to distinguish physiological phenomena from pathological lesions. Mild retrograde flow disturbances are frequently detected in normal subjects (34,35) and if trivial should be identified as being within the expected normal range and not suggestive of the presence of valvular heart disease. On the other hand, significant regurgitation may be silent on auscultation, most often, but not always, in unstable symptomatic patients (36). Because the finding of clinically silent valvular regurgitation in an asymptomatic patient carries an unknown significance, performance of Doppler echocardiography to exclude valvular heart disease in an asymptomatic patient with a normal physical examination is not indicated.

Precise assessment of the severity of regurgitant valvular lesions capable of causing significant hemodynamic compromise is difficult using any invasive or noninvasive technique, and no gold standard is available to judge relative accuracy (7). Doppler methods for detection of regurgitation are similar for all four native valves and prosthetic valves. Methods include assessment of regurgitant jet characteristics (length, height, area, and width at the vena contracta), effective regurgitant orifice area, and measurement of regurgitant flow volume using the proximal isovelocity surface area (7,37-45). The severity of semilunar valve regurgitation is also assessed by the rate of decline in regurgitant gradient as measured by the slope of diastolic flow velocity envelope (46,47). The severity of atrioventricular regurgitation is also reflected by reduction or reversal of the systolic components of venous inflow (48). Finally, in isolated valve disease, regurgitant fraction may be assessed by comparison of stroke volumes at the regurgitant valve and an uninvolved valve.

Doppler echocardiography is also the test of choice in the re-evaluation of regurgitant lesions and in determination of the timing of operative intervention (49-51,522,523). Echocardiographically obtainable information about the severity of regurgitation and associated structural and functional changes are all important to this therapeutic decision. The choice between mitral valve repair and replacement is greatly aided by TTE and TEE; intraoperative assessment of valve repair is essential to optimal surgical practice, while intraoperative determination of prosthetic valve seating and function is also useful (52).

Anorectic drug use (fenfluramines) has been reported to result in generally mild valvular thickening and regurgitation in a small number of users, particularly those with long-term drug exposure (526,527). Echocardiography is indicated in those patients with symptoms or murmurs or those who have an inadequate auscultatory examination (528). Repeat studies in individuals without significant disease are not indicated. The American Society of Echocardiography (ASE) has

published recommendations concerning valvular regurgitation consistent with those in this document (833).

Recommendations for Echocardiography in Native Valvular Regurgitation

Class I

- 1. Diagnosis; assessment of hemodynamic severity.**
- 2. Initial assessment and re-evaluation (when indicated) of LV and RV size, function, and/or hemodynamics.**
- 3. Re-evaluation of patients with mild to moderate valvular regurgitation with changing symptoms.**
- 4. Re-evaluation of asymptomatic patients with severe regurgitation.**
- 5. Assessment of changes in hemodynamic severity and ventricular compensation in patients with known valvular regurgitation during pregnancy.**
- 6. Re-evaluation of patients with mild to moderate regurgitation with ventricular dilation without clinical symptoms.**
- 7. Assessment of the effects of medical therapy on the severity of regurgitation and ventricular compensation and function when it might change medical management.**
- 8. Assessment of valvular morphology and regurgitation in patients with a history of anorectic drug use, or the use of any drug or agent known to be associated with valvular heart disease, who are symptomatic, have cardiac murmurs, or have a technically inadequate auscultatory examination.**

Class IIb

- 1. Re-evaluation of patients with mild to moderate mitral regurgitation without chamber dilation and without clinical symptoms.**
- 2. Re-evaluation of patients with moderate aortic regurgitation without chamber dilation and without clinical symptoms.**

Class III

- 1. Routine re-evaluation in asymptomatic patients with mild valvular regurgitation having stable physical signs and normal LV size and function.**
- 2. Routine repetition of echocardiography in past users of anorectic drugs with normal studies or known trivial valvular abnormalities.**

(See also "Recommendations for Echocardiography in Interventions for Valvular Heart Disease and Prosthetic Valves.")

D. Repeated Studies in Valvular Heart Disease

A routine follow-up echocardiographic examination is not indicated after an initial finding of minimal or mild abnormalities in the absence of a change in clinical signs or symptoms. Patients with more significant abnormalities on the initial study may be followed echocardiographically even in the absence of such changes, with the frequency determined by

the hemodynamic severity of the lesion and the extent of ventricular compensation noted on initial and subsequent studies. Marked changes in the echocardiographic findings, which may indicate an alteration in management even in the absence of changes in clinical signs and symptoms, should be confirmed by re-evaluation at a shorter interval. (See “Recommendations for Echocardiography in Valvular Stenosis,” “Recommendations for Echocardiography in Native Valvular Regurgitation,” and “Recommendations for Echocardiography in Interventions for Valvular Heart Disease and Prosthetic Valves.”)

E. Mitral Valve Prolapse

The physical examination remains the optimal method of diagnosing MVP, because echocardiography may detect systolic billowing of the leaflets not representing clinically relevant disease. There are changing criteria for diagnosing MVP since the first echocardiographic description, and in some studies, valve prolapse of 2 mm or more above the mitral annulus in the long-axis parasternal view and other views is

required (529). The presence of thickening and redundancy of the valve may predict complications. Because of the change in definition, the prevalence of MVP in the population is now believed to be 2% to 3% (530,531). The etiology of the auscultatory finding of systolic clicks may be defined (as valvular or chordal), valvular thickening assessed, and the presence, timing, and severity of regurgitation determined (49,53). In patients with a nonejection click and/or murmur, an echocardiogram is useful for diagnosis and risk stratification, particularly by identifying leaflet thickening and LV dilation (Table 2) (54-59). Routine repeated studies are of little value unless there is significant (nontrivial) mitral regurgitation or a change in symptoms or physical findings.

Echocardiography to diagnose MVP is of little use in the absence of physical findings unless there is supportive clinical evidence of structural heart disease or a family history of myxomatous valve disease.

Table 2. Use of Echocardiography for Risk Stratification in Mitral Valve Prolapse

Study (Ref)	n	Features Examined	Outcome	P Value
Nishimura (54)	237	MV leaflet \geq 5 mm	Increased sum of sudden death, endocarditis, and cerebral embolus	<.02
		LVID \geq 60 mm	Increased MVR (26% vs 3.1%)	<.001
Zuppiroli (55)	119	MV leaflet > 5 mm	Increased complex ventricular arrhythmiano relation to complex ventricular arrhythmias	<.001
Babuty (56)	58	Undefined MV thickening		NS
Takamoto (57)	142	MV leaflet \geq 3 mm, redundant, low echocardiographic density	Increased ruptured chordae (48% vs 5%)	
Marks (58)	456	MV leaflet \geq 5 mm	Increased endocarditis (3.5% vs 0%)	<.02
			Increased moderate-severe MR (11.9% vs 0%)	<.001
			Increased MVR (6.6% vs 0.7%) Increased stroke (7.5% vs 5.8%)	<.02 NS
Chandraratna (59)	86	MV leaflets > 5.1 mm	Increased cardiovascular abnormalities (60% vs 6%)(Marfan syndrome, TVP, MR, dilated ascending aorta)	<.001
Zuppiroli* (843)	316	Overall risk of fatal and nonfatal complications 1/100 subject-years		
		LA diameter \geq 4.0 cm	OR 15.1	
		LV diameter \geq 6.0 cm	OR 16.7	
		Men higher complications than women	OR 3.2	
		Age \geq 45 years	OR 3.4	
		(71% clinically recognized MVP; 29% found in family study) Clinically recognized patients than affected family member	OR 3.8	
Follow-up mean 102 months	Presence of holosystolic murmur	26.9		

LA indicates left atrial; LV, left ventricular; LVID, left ventricular internal diameter; MR, mitral regurgitation; MV indicates mitral valve; MVP, mitral valve prolapse; MVR, mitral valve replacement; OR, odds ratio; NS, not significant; TVP, tricuspid valve prolapse.

Recommendations for Echocardiography in Mitral Valve Prolapse

Class I

Diagnosis; assessment of hemodynamic severity, leaflet morphology, and/or ventricular compensation in patients with physical signs of MVP.

Class IIa

1. **To exclude MVP in patients who have been diagnosed but without clinical evidence to support the diagnosis.**
2. **To exclude MVP in patients with first-degree relatives with known myxomatous valve disease.**
3. **Risk stratification in patients with physical signs of MVP or known MVP.**

Class III

1. **Exclusion of MVP in patients with ill-defined symptoms in the absence of a constellation of clinical symptoms or physical findings suggestive of MVP or a positive family history.**
2. **Routine repetition of echocardiography in patients with MVP with no or mild regurgitation and no changes in clinical signs or symptoms.**

F. Infective Endocarditis: Native Valves

The Duke criteria have improved the specificity and sensitivity of the diagnosis of infective endocarditis by assigning major and minor pathological and clinical criteria. Included as major criteria are the echocardiographic findings of an oscillating intracardiac mass or vegetation, an annular abscess or new valvular regurgitation, or prosthetic valve partial dehiscence (532).

Echocardiography is useful for the detection and characterization of the hemodynamic and pathological consequences of infection, including valvular vegetations, regurgitant lesions, ventricular function, and associated abnormalities such as abscesses, shunts, and ruptured chordae (60). TTE is less sensitive in detecting vegetations than TEE (61,62). Because of the possibility of a false-negative examination (or the absence of a vegetation) or a false-positive study (Lambl's excrescences, noninfective vegetations, thrombi), echocardiography should not supplant clinical and microbiological diagnosis. Echocardiography may be useful in the case of culture-negative endocarditis (63) or in the diagnosis of a persistent bacteremia whose source remains unidentified after appropriate evaluation.

Controversy remains as to whether the echocardiographic characteristics of vegetations are of use in predicting embolization (64,65), although vegetation size and mobility, identification of the involved valve(s), and especially diagnosis of extravalvular extension are important for risk stratification and prognosis (Table 3) (66-68). These features, along with clinical characteristics such as persistent fever and infecting organism, may help guide decision making regarding repeated studies and even valve replacement.

In most cases TEE is not indicated as the initial examination in the diagnosis of native valve endocarditis. When the

valvular structure or pathology is well visualized by TTE, there is no recommendation to perform TEE. Recommendations for routine TEE in established endocarditis are unclear because the clinical importance of the possible additional information obtained is unproved (69). However, TEE should be performed when specific questions are not adequately addressed by the initial TTE examination or in cases where TEE is clearly superior to TTE. Clinical situations in which TEE is indicated include instances when the TTE is diagnostically inadequate because of poor quality or limited echocardiographic windows, when the TTE is negative despite high clinical suspicion, when a prosthetic valve is involved, when there is high suspicion such as staphylococcus bacteremia, or in an elderly patient with valvular abnormalities that make diagnosis difficult (70,533).

Recommendations for Echocardiography in Infective Endocarditis: Native Valves

Class I

1. **Detection and characterization of valvular lesions, their hemodynamic severity, and/or ventricular compensation.***
2. **Detection of vegetations and characterizations of lesions in patients with congenital heart disease suspected of having infective endocarditis.**
3. **Detection of associated abnormalities (eg, abscesses, shunts).***
4. **Re-evaluation studies in complex endocarditis (eg, virulent organism, severe hemodynamic lesion, aortic valve involvement, persistent fever or bacteremia, clinical change, or symptomatic deterioration).**
5. **Evaluation of patients with high clinical suspicion of culture-negative endocarditis.***
6. **If TTE is equivocal, TEE evaluation of bacteremia, especially staphylococcus bacteremia and fungemia without a known source.**

Class IIa

1. **Evaluation of persistent nonstaphylococcus bacteremia without a known source.***
2. **Risk stratification in established endocarditis.***

Class IIb

Routine re-evaluation in uncomplicated endocarditis during antibiotic therapy.

Class III

Evaluation of transient fever without evidence of bacteremia or new murmur.

*TEE may frequently provide incremental value in addition to information obtained by TTE. The role of TEE in first-line examination awaits further study.

G. Prosthetic Valves

Valve replacement is a palliative procedure that carries a subsequent risk of valve degeneration, development of regurgitation or stenotic lesions, thrombosis, and endocarditis.

Table 3. Sensitivity, Specificity, and Predictive Value of Echocardiography in Diagnosis of Infective Endocarditis and Detection of Complications

Type of TEE	Monoplane	Biplane	Multiplane	Note	
Job <i>et al.</i> (71)	41 patients 83 veg with IE 6 abscesses	4 (10%) false-negative 3 missed abscesses	No false-negative 1 missed abscess	Additional veg ● 23% vs monoplane ● 9% vs biplane	Area underestimated in 60% of all veg
Negative Predictive Value of TEE		Negative Predictive Value			
Lowry <i>et al.</i> (72)	93 patients undergoing TEE for suspected IE	NV IE, 100% PV IE, 90%		With suspected PV IE, negative TEE does not rule out	
Sensitivity of TTE vs TEE					
Shapiro <i>et al.</i> (61)	64 patients with suspected IE, prospective study	34 with veg on either TTE or TEE TTE 24 (70.6%) TEE 33 (97.1%) 12 with veg <1 cm TTE 5 (41.7%) TEE 12 (100%) 9 with periannular complications TTE 2 (22.2%) TEE 9 (100%)	$P = .004$ $P = .02$ $P = .001$		
Birmingham <i>et al.</i> (73)	61 patients with suspected IE 31 (51%) had IE	Sensitivity for veg TTE 30% $P < .01$ TEE 88%	For aortic veg TTE 25% $P < .01$ TEE 88%	For mitral veg TTE 50% $P < .01$ TEE 100%	
Sensitivity of TTE					
Watanakunakorn, Burkert (74)	204 patients 219 episodes of IE 148 host valves 33 IVDU 2 early PV IE 27 late PV IE	2D TTE in 164 episodes 67 (40.9%) positive for veg			
Sensitivity and Specificity TTE vs TEE					
Shively <i>et al.</i> (75)	66 episodes of suspected IE in 62 patients Diagnosis of IE made by clinical picture and lab studies in 16 of 66 episodes	TTE 7 of 16 TEE 15 of 16	sensitivity specificity	TTE 44% $P < .01$ TEE 94% TTE 98% TEE 100%	

Continued on next page

Table 3. (Continued)

Sensitivity, Specificity, and Predictive Accuracy of TTE			TTE for Detecting IE	
Burger <i>et al.</i> (76)	106 patients with suspected IE	Group 1, 36 definite veg	Sensitivity 90%	
		Group 2, 65 no veg	Specificity 98%	
	Diagnosis made by clinical picture and lab studies	IE found in 35 patients in Group 1 and 4 patients in Group 2	Predictive accuracy positive test results, 97%	
	5 technically poor images		Predictive accuracy negative test results, 94%	
	101 had TTE			
Complications With IE Sensitivity and Specificity of TTE			Predicting Complications	
Sanfilippo <i>et al.</i> (65)	204 patients with IE had TTE	Complication rate: similar for all valves with veg	Complication rate significantly lower for patients without valve abnormalities on TTE, 27%	MV IE
		MV 53%		70% sensitivity
		AV 62%		92% specificity
		TV 77%		
		PV 61%		AV IE
				76% sensitivity
				62% specificity
		Values with nonspecific abnormalities but no veg, 57%	In left-sided NV, IE, veg size, extent, and mobility are all significant multivariant predictors of complications	

AV indicates aortic valve; IE, infective endocarditis; IVDU, intravenous drug users; MV, mitral valve; NV, native valve; PV, prosthetic valve; TEE, transesophageal echocardiogram; TTE, transthoracic echocardiogram; TV, tricuspid valve; 2D, two-dimensional; veg, vegetations.

Different prostheses carry different risks for these events so that subsequent evaluations must be tailored to the patient's clinical situation and type of prosthesis.

Because the evaluation of prosthetic valves is difficult even in the best of circumstances, it can be useful to obtain baseline postoperative studies for comparison with future evaluations and assessment of changes in ventricular function and hemodynamics in response to surgery. However, the need for routine follow-up echocardiography in the patient with unchanged clinical signs and symptoms is controversial. In some patients with known prosthetic valve dysfunction, re-evaluation is indicated even in the absence of a changing clinical situation, because in some cases reoperation may be dictated by echocardiographic findings alone.

Recommendations for Echocardiography in Interventions for Valvular Heart Disease and Prosthetic Valves

Class I

1. Assessment of the timing of valvular intervention based on ventricular compensation, function, and/or

severity of primary and secondary lesions.

2. Selection of alternative therapies for mitral valve disease (such as balloon valvuloplasty, operative valve repair, valve replacement).*
3. Use of echocardiography (especially TEE) in guiding the performance of interventional techniques and surgery (eg, balloon valvotomy and valve repair) for valvular disease.
4. Postintervention baseline studies for valve function (early) and ventricular remodeling (late).
5. Re-evaluation of patients with valve replacement with changing clinical signs and symptoms; suspected prosthetic dysfunction (stenosis, regurgitation) or thrombosis.*

Class IIa

Routine re-evaluation study after baseline studies of patients with valve replacements with mild to moderate ventricular dysfunction without changing clinical signs or symptoms.

Class IIb

Routine re-evaluation at the time of increased failure rate of a bioprosthesis without clinical evidence of prosthetic dysfunction.

Class III

1. **Routine re-evaluation of patients with valve replacements without suspicion of valvular dysfunction and with unchanged clinical signs and symptoms.**
2. **Patients whose clinical status precludes therapeutic interventions.**

*TEE may provide incremental value in addition to information obtained by TTE.

H. Prosthetic Valve Dysfunction and Endocarditis

Echocardiography is the preferred modality for definition of abnormalities of poppet motion, annular motion, the presence of thrombus or fibrin, or prosthetic leaks or stenoses. Because TEE is often necessary to provide adequate visualization (77), the necessity for previous performance of a transthoracic study has been questioned. However, because a great deal of additional information can be obtained regarding cardiac function and hemodynamics by TTE that may not be otherwise available and/or that may help guide the transesophageal examination, sequential examinations, starting with TTE, are the preferred approach.

Assessment of prosthetic valve stenosis is best performed by a combined echocardiography-Doppler technique. However, the Doppler examination may be problematic because eccentric jets may cause recording of falsely low velocities, especially in valves with central occluders. On the other hand, elevated transvalvular velocities may be recorded with some prosthetic valves and prosthetic valvular lesions due to pressure recovery and which may not accurately represent the true hemodynamic gradient due to pressure recovery. Transvalvular gradients will vary with valve type and size even in the normally functioning prosthesis; individual valve flow characteristics must be considered in the diagnosis of obstruction (78). Re-evaluation may be particularly useful in the individual patient.

Determination of prosthetic valve regurgitation is often hampered by prosthetic shadowing, particularly in the mitral position. The transesophageal approach may be particularly useful in this case. Care must be taken to differentiate between the normal, central regurgitation of many mechanical prostheses and pathological paravalvular leaks (79,80). Contrast injection may enhance the spectral recording of both right-sided regurgitant velocities as well as the extent of the regurgitant jet (81,82).

Diagnosis of prosthetic valve endocarditis by the transthoracic technique is more difficult than diagnosis of endocarditis of native valves because of the reverberations, attenuation, and other image artifacts related to both mechanical valves and bioprosthesis. Particularly in the case of a mechanical valve, TTE may be helpful only when there is a large or mobile vegetation or significant regurgitation. Thus, the technique cannot be used to exclude the presence of small vege-

tations. These limitations are diminished with the use of transesophageal recording techniques because of the superior imaging quality and posterior transducer position. Thus, transesophageal techniques have enhanced echocardiographic assessment of prosthetic valve infective endocarditis, especially of the mitral valve and of both mitral and aortic annular areas for abscesses.

Doppler techniques offer important information about the functional consequences of endocarditis of prosthetic valves, such as the existence of paravalvular leaks. It should be noted, however, that paravalvular leaks are not specific for endocarditis. Importantly, echocardiography may identify vegetations on native valves in patients with suspected prosthetic endocarditis.

Recommendations for Echocardiography in Infective Endocarditis: Prosthetic Valves

Class I

1. **Detection and characterization of valvular lesions, their hemodynamic severity, and/or ventricular compensation.***
2. **Detection of associated abnormalities (eg, abscesses, shunts).***
3. **Re-evaluation in complex endocarditis (eg, virulent organism, severe hemodynamic lesion, aortic valve involvement, persistent fever or bacteremia, clinical change, or symptomatic deterioration).***
4. **Evaluation of suspected endocarditis and negative cultures.***
5. **Evaluation of bacteremia without known source.***

Class IIa

Evaluation of persistent fever without evidence of bacteremia or new murmur.*

Class IIb

Routine re-evaluation in uncomplicated endocarditis during antibiotic therapy.*

Class III

Evaluation of transient fever without evidence of bacteremia or new murmur.

*TEE may provide incremental value in addition to that obtained by TTE.

III. CHEST PAIN

Chest pain can result from many cardiac and noncardiac causes. In mature adults the most common clinical cardiac disorder presenting as chest pain is coronary artery disease (CAD) (see section IV, "Ischemic Heart Disease"). Nonetheless, some patients with chest pain and suspected CAD have other relevant cardiovascular abnormalities that can cause chest pain (182). These disorders, including hypertrophic cardiomyopathy, valvular aortic stenosis, aortic dissection, pericarditis, MVP, and acute pulmonary embolism, produce distinctive and diagnostic echocardiographic findings (see sections II, IV through VI, VIII, and IX).

In patients with chest pain known to be of noncardiac origin, further cardiac testing is usually unnecessary. In patients for whom the character of chest pain or the presence of risk factors raises concern about possible CAD, the role of echocardiography has grown over the last 10 years. Echocardiography can be performed when possible during chest pain in the emergency room; the presence of regional systolic wall motion abnormalities in a patient without known CAD is a moderately accurate indicator of an increased likelihood of acute myocardial ischemia or infarction by pooled data with a positive predictive accuracy of about 50%. The absence of regional wall motion abnormalities identifies a subset of patients unlikely to have had either an acute infarction (83-85,101,102,534) or ischemia, with a weighted mean ("weighted mean" is the mean value after adjustment for the size of each study) negative predictive accuracy of approximately 98%. In a patient with previous myocardial infarction (either clinically evident or silent), the resting echocardiogram can confirm that event and evaluate its functional significance.

Recommendations for Echocardiography in Patients With Chest Pain

Class I

- 1. Diagnosis of underlying cardiac disease in patients with chest pain and clinical evidence of valvular, pericardial, or primary myocardial disease (see sections II, IV through VI, VIII, and IX).**
- 2. Evaluation of chest pain in patients with suspected acute myocardial ischemia, when baseline ECG and other laboratory markers are nondiagnostic and when study can be obtained during pain or within minutes after its abatement (see section IV).**
- 3. Evaluation of chest pain in patients with suspected aortic dissection (see section VIII).**
- 4. Evaluation of patients with chest pain and hemodynamic instability unresponsive to simple therapeutic measures (see section XIII).**

Class III

- 1. Evaluation of chest pain for which a noncardiac etiology is apparent.**
- 2. Diagnosis of chest pain in a patient with electrocardiographic changes diagnostic of myocardial ischemia/infarction (see section IV).**

IV. ISCHEMIC HEART DISEASE

Echocardiography has become an established and powerful tool for diagnosing the presence of CAD and defining its consequences in patients with acute ischemic syndromes and those with chronic coronary atherosclerosis. Transthoracic imaging and Doppler techniques are generally sufficient for evaluating patients with suspected or documented ischemic heart disease. However, TEE may be needed in some patients, particularly those with serious hemodynamic com-

promise but nondiagnostic TTE studies. In these circumstances TEE can distinguish among extensive infarction with pump failure, mechanical complications of infarction, or hypovolemia and can guide prompt therapy (86-89). Stress echocardiography is useful for evaluating the presence, location, and severity of inducible myocardial ischemia, as well as for risk stratification and prognostication.

A. Acute Ischemic Syndromes (Acute Myocardial Infarction and Unstable Angina)

Echocardiography can be used to rapidly diagnose the presence of regional contraction abnormality resulting from acute myocardial infarction, evaluate the extent of associated regional dysfunction, stratify patients into high- or low-risk categories, document serial changes in ventricular function, and diagnose important complications. Some patients with acute chest pain have unstable angina; in these individuals, echocardiography can also be helpful in diagnosis and risk assessment.

1. Diagnosis

The use of echocardiography for diagnosis of acute myocardial infarction provides the greatest amount of incremental information when the clinical history and ECG findings are nondiagnostic.

Segmental LV wall motion abnormalities are characteristic of myocardial infarction. Their location correlates well with the distribution of CAD and pathological evidence of infarction (83,90-100,535-537). However, regional wall motion abnormalities also can be seen in patients with transient myocardial ischemia, chronic ischemia (hibernating myocardium), or myocardial scar. Segmental wall motion abnormalities can also occur in some patients with myocarditis, nonischemic cardiomyopathy or other conditions not associated with coronary occlusion. Table 4 summarizes the utility of TTE in the diagnosis of acute myocardial infarction. In patients presenting with chest pain, segmental LV wall motion abnormalities predict the presence of CAD but can diagnose an acute myocardial infarction with only moderate certainty, because acute ischemia may not be separable from myocardial infarction or even old scar (83-85,90,98-102,535,536). However, the absence of segmental abnormalities (ie, the presence of either normal wall motion or diffuse abnormalities) has a high negative predictive value (weighted mean negative predictive value as high as 98% in suspected myocardial infarction, Table 4). Although it may not be easy to distinguish acute ischemia or necrosis from previous myocardial infarction, preservation of normal wall thickness and normal reflectivity suggest an acute event. Prompt initiation of treatment to achieve reperfusion can reduce mortality, morbidity, and patient care costs (103-106). Hence, early echocardiography is particularly useful in patients with a high clinical suspicion of acute myocardial infarction but a nondiagnostic ECG.

Significant obstructive CAD is usually present in patients with unstable angina. These patients generally are identified

Table 4. Diagnosis of Acute Myocardial Infarction in Patients With Chest Pain

Author (Ref)	Year	Population	Total No. of Pts.	Abn Test	Sens, %	Spec, %	PPV, %	NPV, %	Overall Accuracy, %
Patients With Documented AMI									
Heger (90)	1980	Consec AMI	44	Seg WMA	100	—	—	—	—
Parisi (535)	1981	Prior AMI	20	Seg WMA	95	—	—	—	—
Visser (100)	1981	Consec AMI	66	Seg WMA	98	—	—	—	—
Stamm (536)	1983	Prior AMI	51	Seg WMA	100	—	—	—	—
Nishimura (99)	1984	Consec AMI	61		93	—	—	—	—
Lundgren (537)	1990	Consec AMI	20	Seg WMA	83	—	—	—	—
Patients With Chest Pain, Suspected AMI									
Horowitz (83)	1982	No prior MI	65	Seg WMA	94	84	86	93	89
Sasaki (534)	1986	No prior MI	18	Seg WMA	86	82	75	90	83
Sasaki (534)	1986	No prior MI	28	Seg WMA	100	90	80	100	93
Peels (85)	1990	No prior MI	43	Seg WMA	92	53	46	94	65
Sabia (84)	1991	Consec	169	Seg WMA	93	57	31	98	63
Saeian (101)	1994	No prior MI	60	Seg WMA	88	94	91	92	92
Gibler (102)	1995	Consec	901	Any WMA	47	99	50	99	98

Diagnostic accuracy of echocardiographic wall motion abnormalities (WMA) in detecting acute myocardial infarction (AMI) in patients with previously documented AMI (top) and in patients presenting with chest pain and suspected AMI (bottom).

In each referenced publication included in these data tables, the number of patients appropriate for inclusion in the table was verified carefully (for example, in Table 4, Horowitz [1982] reported 80 patients with suspected AMI, but echocardiographic analysis was performed in only 65 of these patients). The number of true-positive, false-positive, true-negative, and false-negative results was noted. From these data, the sensitivity, specificity, predictive value of positive (PPV) and negative results (NPV), and overall accuracy were calculated. Calculated results were rounded to the nearest full percentage: values ending in xx.1–xx.4 were rounded down, while xx.5–xx.9 were rounded up. Occasional discrepancies between tabulated values in these revised tables and those reported in the original versions of these tables were largely related to the criteria for tabulation, different conventions for rounding, and mathematical errors. All calculations in the current tables were verified carefully. All means given for Sensitivity, Specificity, PPV, NPV, and Accuracy are weighted means, which indicates that they can be heavily influenced by one large study.

Abn Test indicates Abnormal Test, criteria for “positive” test results; AMI, acute myocardial infarction; consec, consecutive; CP, chest pain; MI, myocardial infarction; NPV, negative predictive value; PPV, positive predictive value; Seg, segmental; Sens, Sensitivity; Spec, Specificity; Total number of pts, number of patients in whom two-dimensional transthoracic echocardiographic wall motion analysis was carried out; WMA, wall motion abnormality.

by clinical history, and reversible ECG abnormalities may be recorded during episodes of chest pain. When the clinical history and ECG are unavailable or not reliable and an adequate echocardiographic study can be performed during an episode of chest pain, documentation of transient segmental wall motion abnormalities that normalize with treatment supports the diagnosis of unstable angina.

2. Severity of Disease/Risk Assessment/Prognosis

In patients with acute myocardial infarction, segmental wall motion abnormalities can be seen not only in the zone of acute infarction but also in regions of prior infarction and areas with ischemic “stunning” or “hibernation” of myocardium that is nonfunctional but still viable (90,91,94,

107-109,136-138,538-544). The sum of these segmental abnormalities reflects total ventricular functional impairment, which may overestimate true anatomic infarct size or perfusion defect (109). Thus, echocardiographically derived infarct size (90) correlates modestly with thallium-201 perfusion defects (94), peak creatine kinase levels (91,100), hemodynamic changes (90), findings on ventriculography (95) and coronary angiography (96), and pathological findings (108). However, it does predict the development of early (98-99,110,113) and late (111,113) complications and mortality (90,91,99,110,112,113). In a given patient with acute myocardial infarction, global and regional ventricular function as well as clinical status may improve (especially after reperfusion therapy) or can occasionally deteriorate. As a

noninvasive technique that can be performed at the patient's bedside, initial and late follow-up echocardiography is an excellent test for evaluating changes in LV function in patients with a large myocardial infarction.

Table 5 summarizes the prognostic value of segmental wall motion abnormalities detected early in the course of acute myocardial infarction. In general, more extensive abnormalities denote an increased risk of complications, including death, recurrent infarction, pump failure, and serious ventricular dysrhythmias or heart block, even in patients who appear well clinically (83,84,91,98,99,110,113). Patients with more extensive wall motion abnormalities do not invariably develop complications (weighted mean positive predictive value of about 40%; Table 5) but do merit careful observation. Relatively mild and localized wall motion abnormalities indicate a low risk of complications (weighted mean negative predictive value 92%; Table 5).

3. Assessment of Complications

Echocardiography can be used to evaluate, at the bedside when needed, virtually any complication of acute myocardial infarction.

a. Acute Mitral Regurgitation

Development of acute mitral regurgitation following acute myocardial infarction denotes a significantly worsened prognosis (114). Significant regurgitation can result from acute rupture of a papillary muscle head (115), acute ischemic dysfunction of the papillary muscle and associated free wall (116), late fibrosis and shortening of the papillary muscle apparatus (117), altered mitral closure dynamics due to systolic ventricular impairment (118), or annular dilation. All of these different mechanisms can be identified and regurgitant severity evaluated using echocardiographic imaging and Doppler flow studies.

b. Infarct Expansion and LV Remodeling

Following acute myocardial infarction, development of infarct expansion commonly precedes myocardial rupture (including ventricular septal defect) and denotes a worsened prognosis (119). A follow-up echocardiogram is excellent for identifying infarct expansion (120) in patients with a large myocardial infarction and differentiating it from infarct extension as well as subsequent adverse LV remodeling characterized by progressive chamber dilation and further deterioration in global systolic function.

c. Ventricular Septal Rupture

Both two-dimensional and color Doppler echocardiography can be used to locate and visualize postinfarction ventricular septal defects (121-123) and to demonstrate left-to-right shunting. Doppler techniques in particular provide an accurate means of distinguishing a ventricular septal defect from mitral regurgitation (121) or tricuspid regurgitation that is (either pre-existing or the result of RV infarction).

d. Free Wall Rupture

Antemortem diagnosis of free wall rupture in patients with acute myocardial infarction is relatively infrequent. However, free wall rupture is not inevitably fatal (124), and the diagnosis can be made using echocardiographic imaging and Doppler flow studies. Echocardiographic contrast agents may improve diagnosis in free wall rupture and in identifying intracardiac thrombus.

Patients who survive free wall rupture often develop a pseudoaneurysm that has a characteristic echocardiographic appearance (125,126). Echocardiography also can help define the presence or absence of associated tamponade physiology and determine the timing of surgical intervention.

e. Intracardiac Thrombus

Echocardiography is the definitive test for detecting intracardiac thrombi (127-133). LV thrombi are most often detected in patients with anterior and apical infarctions (127,131-133); their presence denotes an increased risk of both embolism (128) and death (130). The need for serial echocardiography in patients with ventricular thrombi remains controversial.

f. RV Infarction

In approximately one third of patients with inferior myocardial infarction, associated RV infarction also occurs (134). This can have significant hemodynamic consequences and implications for patient treatment. Characteristic echocardiographic features of RV infarction have been described (135). In addition, anterior wall infarctions can involve small portions of the right ventricle, but rarely enough to cause hemodynamically evident RV infarction.

g. Pericardial Effusion

Pericardial effusion may accompany transmural infarction; its presence does not necessarily imply free wall rupture. The role of echocardiography in evaluating pericardial effusion is discussed in section VI, "Pericardial Disease."

4. Assessment of Therapy

Given the frequent use of reperfusion therapy (involving either thrombolytic agents or primary angioplasty) in patients with acute myocardial infarction, assessment of myocardial salvage is an important clinical issue. Serial echocardiographic studies can be used to assess recovery of regional myocardial function from initial stunning.

In patients with unstable angina who undergo revascularization (by angioplasty or surgery), the completeness of revascularization and the functional significance of residual lesions can be determined using exercise or pharmacological stress echocardiography techniques. These applications in unstable angina patients are similar to those in patients with chronic ischemic heart disease, discussed below

Table 5. Prognostic Value of Wall Motion Abnormalities in Patients With Acute Myocardial Infarction

Author (Ref)	Year	Population	Total No. of Pts.	Adverse Outcomes	Criteria	Prediction of Adverse Outcomes %				
						Sensitivity, %	Specificity, %	PPV, %	NPV, %	Overall Accuracy, %
Horowitz (83)	1982	No prior AMI	65	D, PumF, MalignAr, RecAP	SWMA	100	53	28	100	60
Gibson (91)	1982	Consec AMI	68	D, PumF, MI	Remote WMA	81	81	78	83	81
Horowitz (110)	1982	Proved AMI	43	D, PumF, MalignAr	WMS greater than 7	85	83	69	93	84
Nishimura (99)	1984	Consec AMI	61	D, PumF, MalignAr	WMS index greater than 2	80	90	89	82	85
Jaarsma (98)	1988	AMI; Killip 1 or 2	77	Progression to PumF	WMS greater than 7	88	57	35	95	64
Sabia (84)	1991	Consec AMI	29	PumF, MalignAr, RecAP	SWMA	100	13	48	100	52
Sabia (113)	1991	Consec CP (ER)	171	D, MI, MalignAr, RecAP less than 48 h	LV dysfx	94	48	28	97	54
Sabia (113)	1991	Consec CP (ER)	139	D, MI, MalignAr, RecAP greater than 48 h	LV dysfx	83	50	25	94	55

Prognostic value of echocardiographic wall motion abnormalities (WMA) in predicting adverse outcomes in patients studied early in the course of an acute myocardial infarction (AMI).

Adverse Outcomes indicates subsequent adverse clinical events; AMI, acute myocardial infarction; consec, consecutive; CP, chest pain; Criteria, echocardiographic features considered as a "positive" indicator of increased risk; D, death; dysfx, dysfunction; ER, evaluated in emergency room; MalignAr, malignant arrhythmias; MI, recurrent myocardial infarction; NPV, negative predictive value; PPV, positive predictive value; PumF, pump failure; RecAP, recurrent severe angina pectoris; remote WMA, wall motion abnormalities in regions remote from the area of infarction, implying multivessel disease; SWMA, segmental wall motion abnormalities; Total No. of Pts, number of patients with AMI in whom echocardiographic wall motion analysis was performed; WMS, wall motion score [higher=worse].

5. PredischARGE Evaluation Using Stress Echocardiography

Graded stress echocardiography using intravenous dobutamine can help in assessing myocardial viability early after myocardial infarction (136-138,538-544). When acute ischemia is followed by restoration of adequate blood flow, myocardial stunning may occur and may last for days to months. Although serious complications have been reported (545), general experience suggests that carefully performed pharmacological stress echocardiography using a gradual protocol and beginning at low doses of dobutamine appears to be feasible and reasonably safe when performed 4 to 10 days after acute myocardial infarction. Although publications do not indicate a major risk to testing in this time frame, the number of patients studied less than 5 days after infarction is not extensive, and the safety of testing within the first few days after infarction is not fully established. Reperfusion-salvaged, stunned myocardium (with depressed function at rest) can respond to inotropic stimulation (139,140). As summarized in Table 6, wall segments that show hypokinesia or akinesia at rest but improved function during low-dose dobutamine infusion often recover function (136-138,538-544) (weighted mean positive predictive value 71%; Table 6), which suggests that these segments are “stunned.” However, when segments with hypokinesia or akinesia at rest show no improvement during dobutamine infusion, functional recovery is uncommon (weighted mean negative predictive value 88%; Table 6), which suggests that most of these segments are infarcted. Segments with initial improvement during low-dose dobutamine infusion but deterioration of function with higher doses (showing a “biphasic response”) frequently are supplied by arteries with significant residual stenoses. Continuing augmentation of systolic wall thickening with higher doses of dobutamine denotes preserved viability and implies the lack of critical stenosis in the infarct-related artery.

Because echocardiographic images obtained during graded exercise demonstrate the location and approximate size of the ischemic territory, they will provide useful information in identifying high-risk patients after acute myocardial infarction (141-146,543,546-552). Population-based studies have demonstrated a significant decline in postinfarction mortality in patients treated with thrombolytic therapy compared to earlier experience in the prethrombolytic era (553,554). However, in patients studied by predischARGE stress echocardiography after an acute myocardial infarction (both those who have and those who have not undergone thrombolytic or other reperfusion therapy), an ischemic response generally predicts a higher rate of adverse events such as death and reinfarction (Table 6a). Prospective natural history studies are difficult to accomplish because many clinicians now perform angiography and recommend revascularization in patients with an ischemic response. Nonetheless, when coronary anatomy is unknown, patients who have had an acute myocardial infarction should undergo predischARGE functional testing for risk assessment. In those patients unable to

exercise because of deconditioning, neurological, or orthopedic limitations, pharmacological stress echocardiography is a valuable alternative for graded stress testing.

In patients with unstable angina but no myocardial infarction, echocardiography is most helpful for answering specific unresolved clinical questions. When ECG changes of ischemia are obscured by baseline abnormalities (such as chronic left bundle branch block, ventricular pacing, or chronic repolarization changes), reversible segmental wall motion abnormalities during pain can document not only the presence of transient ischemia but also the coronary territory involved and the size of the area at risk. The sensitivity of echocardiography for detecting transient wall motion abnormalities resulting from acute ischemia diminishes as the time between resolution of chest pain and acquisition of echocardiographic images increases. When myocardial viability is uncertain because of persistent impairment of ventricular function in the absence of chest pain (which could be due to “silent” ischemia, myocardial stunning, prior infarction, or cardiomyopathy), the response to carefully graded dobutamine infusion can be clinically useful. However, large-scale studies of this latter question have not been reported.

The recommendations for echocardiography in acute myocardial ischemic syndromes are summarized below.

Recommendations for Echocardiography in the Diagnosis of Acute Myocardial Ischemic Syndromes

Class I

1. **Diagnosis of suspected acute ischemia or infarction not evident by standard means.**
2. **Measurement of baseline LV function.**
3. **Evaluation of patients with inferior myocardial infarction and clinical evidence suggesting possible RV infarction.**
4. **Assessment of mechanical complications and mural thrombus.***

Class IIa

Identification of location/severity of disease in patients with ongoing ischemia.

Class III

Diagnosis of acute myocardial infarction already evident by standard means.

*TEE is indicated when TTE studies are not diagnostic.

Recommendations for Echocardiography in Risk Assessment, Prognosis, and Assessment of Therapy in Acute Myocardial Ischemic Syndromes

Class I

1. **Assessment of infarct size and/or extent of jeopardized myocardium.**
2. **In-hospital assessment of ventricular function when the results are used to guide therapy.**
3. **In-hospital or early postdischarge assessment of the presence/extent of inducible ischemia whenever base-**

Table 6. Myocardial Viability: Detection of Stunned Myocardium by DSE Early After Acute Myocardial Infarction

Author (Ref)	Year	Time p MI, d	Stress	Total No. of Pts	Criteria	Sensitivity, %	Specificity, %	PPV, %	NPV, %	Overall Accuracy, %
Pierard (137)	1990	Avg 7 ± 4	LD-DSE	17	ImpWM*	100	70	70	100	82
Barilla (138)	1991	Avg 4 ± 2	LD-DSE	21	ImpWM*	95	—	—	—	95
Smart (136)	1993	Range 2-7	LD-DSE	51	ImpWM*	86	90	86	90	88
Previtali (538)	1993	Avg 8 ± 4	LD-DSE	42	ImpWM†	79	68	50	89	71
Watada (539)	1994	Avg 3, Range 2-5	LD-DSE	21	ImpWM†	83	86	89	80	84
Salustri (540)	1994	Within 7	LD-DSE	57	ImpWM†	66	94	79	88	86
Poli (541)	1996	Within 10	LD-DSE	51	ImpWM†	72	68	50	85	69
Bolognese (542)	1996	3	LD-DSE	30	ImpWM†	89	91	86	93	90
Minardi (543)	1997	Range 3-5	LD-DSE	50	ImpWM†	86	100	100	94	96
Smart (544)	1997	Range 2-7	LD-DSE	115	ImpWM†	86	83	80	88	84

Evaluation of myocardial viability, using dobutamine stress echocardiography (DSE) early following acute myocardial infarction to detect stunned myocardium. The presence or absence of viability was established by follow-up resting transthoracic echocardiography.

In the studies of Watada et al. and Bolognese et al. patients were treated at admission with primary angioplasty; in the remaining studies, patients were treated at admission with thrombolytic therapy.

*Wall motion analyzed by patient.

†Wall motion analyzed by segment.

Criteria indicates findings on DSE considered as a "positive" indicator of viability; d, days; impWM, improved segmental wall motion seen on follow-up echocardiogram; LD-DSE, low-dose dobutamine stress echocardiography; MI, myocardial infarction; NPV, negative predictive value (likelihood of lack of subsequent improvement in patients without viability); PPV, positive predictive value (likelihood of subsequent improvement in patients with evidence of viability); Sensitivity, sensitivity for detecting viable myocardium; Specificity, specificity for detecting viable myocardium; Stress, DSE protocol used for pharmacological stress; Time p MI, time between myocardial infarction and stress testing; Total No. of Pts, number of patients with AMI in whom DSE studies were analyzed.

Table 6a. Prognostic Value of Stress Echocardiography Early After Acute Myocardial Infarction

Author (Ref)	Year	Time After MI, d	Stress	Total No. of Pts	Average Follow-up, mo	Events	Annualized Event Rate, %	
							Ischemia	No Ischemia
Applegate (144)	1987	13	TME	67	11	D, MI, Re	55	14
Ryan (142)	1987	11-21	TME	40	7.2	D, MI	59	0
Bolognese (145)	1992	8-10	DIP	217	24.3	D, MI	3.9	1.1
Sclavo* (548)	1992	5-8	DIP	107	14.5	D, MI	0	3.5
Picano* (549)	1993	10	DIP	925	14	D	4.6	2
van Daele* (550)	1994	9	DIP	89	24	D	9.7	2.7
Picano* (141)	1995	10	DIP	1080	14	D, MI	5.4	2.8
Quintana* (146)	1995	7	BE	70	36	D, MI	22.2	4.7
Sicari* (551)	1997	12	DASE	778	9	D, MI	6.4	6.6
Greco* (547)	1997	12	DASE	178	17	D, MI	7.7	1.5
Carlos* (546)	1997	2-7	DASE	214	16.2	D, MI, VT	26.6	4.3
Mimardi* (543)	1997	3-5	DSE	50	28	D, MI	4.3	0
Previtalli* (552)	1998	9	DASE	152	15	D, MI	8.4	0

Prognostic value of inducible ischemia, detected using different forms of stress echocardiography, early after acute myocardial infarction. Of the 3967 patients included in these publications, 2137 (53.9%) received thrombolytic treatment.

*Patients in this study were treated with thrombolytic therapy.

Annualized Event Rate indicates percentage of patients, per year, who developed at least one adverse event during the period of follow-up, depending on whether inducible ischemia was, or was not demonstrated by stress echocardiography; Average Follow-Up (mo), average period of follow-up after stress echocardiography; BE, bicycle stress echocardiography; D, cardiac death; DASE, dobutamine/atropine stress echocardiography; DIP, dipyridamol stress echocardiography; DSE, dobutamine stress echocardiography; Events, adverse events; MI, recurrent nonfatal myocardial infarction; Re, revascularization necessary; Stress, stress echocardiography protocol; Time After MI, number of days between presenting MI and stress echocardiography; TME, treadmill stress echocardiography; Total No. of Pts, number of patients studied using stress echocardiography and subsequently followed up for the development of adverse events (including death, nonfatal myocardial infarction, revascularization, or sustained ventricular tachycardia); VT, sustained ventricular tachycardia.

line abnormalities are expected to compromise electrocardiographic interpretation.*

4. Assessment of myocardial viability when required to define potential efficacy of revascularization.†

Class IIa

1. In-hospital or early postdischarge assessment of the presence/extent of inducible ischemia in the absence of baseline abnormalities expected to compromise ECG interpretation.*
2. Re-evaluation of ventricular function during recovery when results are used to guide therapy.
3. Assessment of ventricular function after revascularization.

Class IIb

Assessment of late prognosis (greater than or equal to 2 years after acute myocardial infarction).

Class III

Routine re-evaluation in the absence of any change in clinical status.

*Exercise or pharmacological stress echocardiogram.

†Dobutamine stress echocardiogram.

B. Chronic Ischemic Heart Disease

In patients with chronic ischemic heart disease, echocardiography is useful for a range of recommendations, including diagnosis, risk stratification, and clinical management decisions. Quantitative indices of global and regional systolic function (including fractional shortening, fractional area change, ejection fraction, and wall motion score) are valuable in describing LV function, determining prognosis, and evaluating the results of therapy. Doppler techniques are also extremely valuable for evaluating both systolic and diastolic ventricular function in patients with chronic ischemic heart disease (see section V, “Cardiomyopathy, Congestive Heart Failure, and Assessment of Left Ventricular Function: Echocardiographic Parameters”).

1. Diagnostic Accuracy of Echocardiographic Techniques in Chronic CAD

a. TTE (at Rest)

Chronic ischemic heart disease often results in impaired systolic LV function. The extent and severity of regional and global abnormalities are important considerations in choosing appropriate medical or surgical therapy. Abnormal diastolic ventricular function, which frequently accompanies impaired systolic function but may also occur when global systolic function is preserved, also can be evaluated (see section V, “Cardiomyopathy, Congestive Heart Failure, and Assessment of Left Ventricular Function: Echocardiographic Parameters”).

Other structural and functional alterations can complicate chronic ischemic heart disease. Mitral regurgitation may result from global LV systolic dysfunction (118), regional

papillary muscle dysfunction (116), scarring and shortening of the submitral chords (117), papillary muscle rupture (115), or other causes. The presence, severity, and mechanism of mitral regurgitation can be detected reliably using transthoracic imaging and Doppler echocardiographic techniques. Potential surgical approaches also can be defined. In patients with heart failure or significant ventricular arrhythmias, the presence or absence of ventricular aneurysm can be established (147,148). When an aneurysm is demonstrated, the function of the nonaneurysmal portion of the left ventricle is an important consideration in choosing medical or surgical therapy (149).

b. Stress Echocardiography

As currently practiced (with the aid of digital acquisition and storage of relevant images), stress echocardiography is both sensitive and specific for detecting inducible myocardial ischemia in patients with intermediate to high pretest probability of CAD. A variety of methods can be used to induce stress; exercise (treadmill, upright or supine bicycle) and pharmacological techniques (using either adrenergic stimulating or vasodilator agents) are most often used. In patients studied with exercise echocardiography, weighted mean sensitivity is 86%, specificity 81%, and overall accuracy 85%. With dobutamine stress echocardiography, corresponding values are 82%, 84%, and 83%. The accuracy of stress echocardiography is summarized in Tables 7 and 8. As with other noninvasive methods, sensitivity is higher in patients with multivessel disease than in those with one-vessel disease, in those with prior infarction, and in those with greater than 70% stenosis compared with those with more moderate lesions (150-184,543,555-582). Compared with standard treadmill exercise testing, stress echocardiography is of significant additive clinical value for detecting and localizing inducible myocardial ischemia. Moreover, when the pretest probability is in the intermediate range, stress echocardiography may be more cost-effective for identifying the presence or absence of CAD than conventional exercise testing (583-585). However, some of these studies do not assume sequential testing, just exercise testing and coronary arteriography, without stress imaging as an intermediate step. These studies also do not take into account posttest referral bias, which always favors the new test (stress imaging) over the old test (exercise treadmill). Because of the increased incidence of false-positive exercise ECG tests in women, stress imaging has been recommended as the initial test. The optimal strategy for detecting CAD in women remains to be defined. The ACC/AHA/ACP Committee to Update Guidelines for the Management of Chronic Stable Angina believes that the data available at present are insufficient to justify replacing standard exercise testing with stress imaging when evaluating women for CAD. In women with a low pretest likelihood of disease, a negative exercise stress test will be sufficient, and further stress imaging will not be necessary (586).

Table 7. Diagnostic Accuracy of Exercise Echocardiography in Detecting Angiographically Proved CAD (Without Correction for Referral Bias)

Author/Ref	Year	Exercise	Significant CAD	Total No. of Pts	Sens, %	Sens 1-VD	Sens MVD	Specificity %	PPV, %	NPV, %	Overall Accuracy, %
Limacher (555)	1983	TME	> 50%	73	91	64	98	88	96	75	90
Armstrong (556)	1986	TME	≥ 50%	95	88	—	—	87	97	57	87
Armstrong (154)	1987	TME	≥ 50%	123	88	81	93	86	97	61	88
Ryan (155)	1988	TME	≥ 50%	64	78	76	80	100	100	73	86
Labovitz (156)	1989	TME	≥ 70%	56	76	—	—	100	100	74	86
Sawada (152)	1989	TME or UBE	≥ 50%	57	86	88	82	86	86	86	86
Sheikh (557)	1990	TME	≥ 50%	34	74	74	—	91	94	63	79
Pozzoli (158)	1991	UBE	≥ 50%	75	71	61	94	96	97	64	80
Crouse (157)	1991	TME	≥ 50%	228	97	92	100	64	90	87	89
Galanti (159)	1991	UBE	≥ 70%	53	93	93	92	96	96	93	94
Marwick (160)	1992	TME	≥ 50%	150	84	79	96	86	95	63	85
Quinones (161)	1992	TME	≥ 50%	112	74	59	89	88	96	51	78
Salustri (162)	1992	BE	≥ 50%	44	87	87	—	85	93	75	86
Amanullah (163)	1992	UBE	≥ 50%	27	82	—	—	80	95	50	81
Hecht (168)	1993	SBE	≥ 50%	180	93	84	100	86	95	79	91
Ryan (164)	1993	UBE	≥ 50%	309	91	86	95	78	90	81	87
Mertes (165)	1993	SBE	≥ 50%	79	84	87	89	85	91	75	85
Hoffmann (166)	1993	SBE	> 70%	66	80	79	81	88	95	58	82
Cohen (167)	1993	SBE	> 70%	52	78	63	90	87	94	62	81

Continued on next page

Diagnostic accuracy of exercise echocardiography in detecting coronary artery disease (CAD) proved by angiography. A new or worsening regional wall motion abnormality induced by stress generally was considered a "positive" result.

BE indicates bicycle ergometry; Exercise, type of exercise testing, used in conjunction with transthoracic echocardiographic imaging; CAD, coronary artery disease; MVD, test results positive in patients with multivessel disease; NPV, negative predictive value (likelihood of absence of angiographically significant CAD in patients without inducible wall motion abnormalities by exercise echocardiography); 1-VD test results positive in patients with single-vessel CAD; PPV, positive predictive value (likelihood of angiographically significant CAD in patients with inducible wall motion abnormalities by exercise echocardiography); Significant CAD, % coronary luminal diameter narrowing, demonstrated by selective coronary angiography, considered to represent significant CAD; SBE, supine bicycle ergometry; Sens, sensitivity; Spec, specificity; TME, treadmill exercise; Total No. of Pts, number of patients in each series undergoing selective coronary angiography in whom exercise echocardiographic studies were also performed and wall motion analysis was performed; UBE, upright bicycle ergometry.

Table 7. (Continued)

Author/Ref	Year	Exercise	Significant CAD	Total No. of Pts	Sens, %	Sens I-VD	Sens MVD	Specificity %	PPV, %	NPV, %	Overall Accuracy, %
Marwick (558)	1994	BE	> 50%	86	88	82	91	80	89	77	85
Roger (169)	1994	TME	≥ 50%	150	91	—	—	—	—	—	—
Marangelli (153)	1994	TME	≥ 75%	80	89	76	97	91	93	86	90
Beleslin (183)	1994	TME	≥ 50%	136	88	88	91	82	97	50	88
Williams (559)	1994	UBE	> 50%	70	88	89	86	84	83	89	86
Roger (170)	1995	TME	≥ 50%	127	88	—	—	72	93	60	—
Dagianti (184)	1995	SBE	> 70%	60	76	70	80	94	90	85	87
Marwick (560)	1995	TME	≥ 50%	161	80	75	85	81	71	91	81
		or UBE									
Bjornstad (561)	1995	UBE	≥ 50%	37	84	78	86	67	93	44	81
Marwick (562)	1995	TME	> 50%	147	71	63	80	91	85	81	82
Tawa (563)	1996	TME	> 70%	45	94	—	—	83	94	83	91
Luotolahti (564)	1996	UBE	≥ 50%	118	94	94	93	70	97	50	92
Tian (565)	1996	TME	> 50%	46	88	91	86	93	97	76	89
Roger (580)	1997	TME	≥ 50%	340	78	—	—	41	79	40	69

Diagnostic accuracy of exercise echocardiography in detecting coronary artery disease (CAD) proved by angiography. A new or worsening regional wall motion abnormality induced by stress generally was considered a "positive" result.

BE indicates bicycle ergometry; Exercise, type of exercise testing, used in conjunction with transthoracic echocardiographic imaging; CAD, coronary artery disease; MVD, test results positive in patients with multivessel disease; NPV, negative predictive value (likelihood of absence of angiographically significant CAD in patients without inducible wall motion abnormalities by exercise echocardiography); I-VD test results positive in patients with single-vessel CAD; PPV, positive predictive value (likelihood of angiographically significant CAD in patients with inducible wall motion abnormalities by exercise echocardiography); Significant CAD, % coronary luminal diameter narrowing, demonstrated by selective coronary angiography, considered to represent significant CAD; SBE, supine bicycle ergometry; Sens, sensitivity; Spec, specificity; TME, treadmill exercise; Total No. of Pts, number of patients in each series undergoing selective coronary angiography in whom exercise echocardiographic studies were also performed and wall motion analysis was performed; UBE, upright bicycle ergometry.

Table 8. Diagnostic Accuracy of Dobutamine Stress Echocardiography in Detecting Angiographically Proved CAD (Without Correction for Referral Bias)

Author (Ref)	Year	Protocol	Significant CAD	Total No. of Pts	Sens, %	Sens, 1-YD	Sens, MVD	Specificity %	PPV, %	NPV, %	Overall Accuracy, %
Berthe (171)	1986	DSE 5-40	≥ 50%	30	85	—	85	88	85	88	87
Sawada (174)	1991	DSE 2.5-30	≥ 50%	55	89	81	100	85	91	81	74
Sawada (174)	1991	DSE 2.5-30	≥ 50%	41	81	—	81	87	91	72	87
Previtalli (173)	1991	DSE 5-40	≥ 70%	35	68	50	92	100	100	44	83
Cohen (175)	1991	DSE 2.5-40	> 70%	70	86	69	94	95	98	72	89
Martin (566)	1992	DSE 10-40	> 50%	34	76	—	—	44	79	40	68
McNeill (151)	1992	DASE 10-40	≥ 50%	28	71	—	—	—	—	—	71
Segar (178)	1992	DSE 5-30	≥ 50%	88	95	—	—	82	94	86	92
Mazeika (176)	1992	DSE 5-20	≥ 70%	50	78	50	92	93	97	62	82
Marcovitz (177)	1992	DSE 5-30	≥ 50%	141	96	95	98	66	91	84	89
McNeill (567)	1992	DASE 10-40	≥ 50%	80	70	—	—	88	89	67	78
Salustri (172)	1992	DSE 5-40	≥ 50%	46	79	—	—	78	85	70	78
Marwick (150)	1993	DSE 5-40	≥ 50%	97	85	84	86	82	88	78	84
Forster (180)	1993	DASE 10-40	> 50%	21	75	—	—	89	90	73	81
Günalp (181)	1993	DSE 5-30	> 50%	27	83	78	89	89	94	73	85
Marwick (179)	1993	DSE 5-40	≥ 50%	217	72	66	77	83	89	61	76
Hoffmann (166)	1993	DASE 5-40	> 70%	64	79	78	81	81	93	57	80
Previtalli (568)	1993	DSE 5-40	> 50%	80	79	63	91	83	92	61	80
Takeuchi (569)	1993	DSE 5-30	≥ 50%	120	85	73	97	93	95	80	88

Continued on next page

Diagnostic accuracy of dobutamine stress echocardiography in detecting angiographically proved coronary artery disease (CAD). A new or worsening regional wall motion abnormality induced by stress generally was considered a "positive" result.

CAD, coronary artery disease; DASE, dobutamine/atropine stress echocardiography; DSE, dobutamine stress echocardiography; MVD, test results positive in patients with multivessel CAD; NPV, negative predictive value (likelihood of absence of angiographically significant CAD in patients without inducible wall motion abnormalities by pharmacological stress echocardiography); 1-YD, test results positive in patients with single-vessel CAD; PPV, positive predictive value (likelihood of angiographically significant CAD in patients with inducible wall motion abnormalities by pharmacological stress echocardiography); Protocol, dobutamine stress protocol, including initial and peak infusion rates (expressed in mg per kg per min); Sens, sensitivity; Significant CAD, % coronary luminal diameter narrowing demonstrated by selective coronary angiography, considered to represent "significant" CAD; Total No. of Pts, number of patients in each series undergoing selective coronary angiography in whom dobutamine stress echocardiographic studies were also performed and wall motion analysis was performed.

Table 8. (Continued)

Author (Ref)	Year	Protocol	Significant CAD	Total No. of Pts	Sens, %	Sens I-VD	Sens MVD	Specificity %	PPV, %	NPV, %	Overall Accuracy, %
Cohen (167)	1993	DSE 2.5-40	> 70%	52	86	75	95	87	94	72	87
Ostojic (570)	1994	DSE 5-40	≥ 50%	150	75	74	81	79	96	31	75
Marwick (558)	1994	DSE 5-40	> 50%	86	54	36	65	83	86	49	64
Beleslin (183)	1994	DSE 5-40	≥ 50%	136	82	82	82	76	96	38	82
Sharp (582)	1994	DSE 5-50	≥ 50%	54	83	69	89	71	89	59	80
Pellikka (182)	1995	DSE 5-40	≥ 50%	67	98	—	—	65	84	94	87
Ho (571)	1995	DSE 5-40	≥ 50%	54	93	100	92	73	93	73	89
Daoud (572)	1995	DSE 5-30	≥ 50%	76	92	91	93	73	95	62	89
Dagianti (184)	1995	DSE 5-40	≥ 70%	60	72	60	80	97	95	83	87
Pingitore (573)	1996	DASE 5-40	≥ 50%	110	84	78	88	89	97	52	85
Schroder (574)	1996	DASE 10-40	≥ 50%	46	76	71	90	88	97	44	78
Anthopoulos (575)	1996	DASE 5-40	≥ 50%	120	87	74	90	84	94	68	86
Ling (576)	1996	DASE 5-40	≥ 50%	183	93	—	—	62	95	54	90
Takeuchi (579)	1996	DASE 5-40	≥ 50%	70	75	78	73	92	79	90	87
Minardi (543)	1997	DASE 5-40	≥ 50%	47	75	81	67	67	97	15	74
Dionisopoulos (577)	1997	DASE 5-40	≥ 50%	288	87	80	91	89	95	71	87
Elhendy (581)	1997	DASE 5-40	≥ 50%	306	74	59	83	85	94	50	76
Ho (578)	1998	DSE 5-40	≥ 50%	51	93	89	95	82	87	90	88

Diagnostic accuracy of dobutamine stress echocardiography in detecting angiographically proved coronary artery disease (CAD). A new or worsening regional wall motion abnormality induced by stress generally was considered a "positive" result.

CAD indicates coronary artery disease; DASE, dobutamine/atropine stress echocardiography; DSE, dobutamine stress echocardiography; MVD, test results positive in patients with multivessel CAD; NPV, negative predictive value (likelihood of absence of angiographically significant CAD in patients without inducible wall motion abnormalities by pharmacological stress echocardiography); I-VD, test results positive in patients with single-vessel CAD; PPV, positive predictive value (likelihood of angiographically significant CAD in patients with inducible wall motion abnormalities by pharmacological stress echocardiography); Protocol, dobutamine stress protocol, including initial and peak infusion rates (expressed in mcg per kg per min); Sens, sensitivity; Significant CAD, % coronary luminal diameter narrowing demonstrated by selective coronary angiography, considered to represent "significant" CAD; Total No. of Pts, number of patients in each series undergoing selective coronary angiography in whom dobutamine stress echocardiographic studies were also performed and wall motion analysis was performed.

In patients with a significant clinical suspicion of CAD, stress echocardiography is appropriate when standard exercise testing is likely to be nondiagnostic for identifying the presence or absence of CAD. Examples include conditions likely to reduce the validity of ST-segment analysis, such as the presence of resting ST-T wave abnormalities, left bundle branch block, ventricular paced rhythms, LV hypertrophy/strain, or digitalis treatment. When a noncardiac limitation precludes adequate exercise testing, pharmacological stress echocardiography is an appropriate alternative. Dobutamine stress echocardiography has substantially higher sensitivity than vasodilator stress echocardiography for detecting coronary stenoses in most (150,173,183,184,566,568,575) but not all (172,543,574) studies. Treadmill stress echocardiography may have lowered sensitivity if there is a significant delay from the end of exercise to the acquisition of postexercise images (152,164). Sensitivity can also be diminished if all myocardial segments are not adequately visualized (160). This shortcoming occurs quite variably but is not insignificant. When endocardial visualization is inadequate, contrast echocardiography (587) usually permits meaningful evaluation of LV wall motion with TTE and harmonic imaging. Alternatively, dobutamine stress echocardiography can be used in conjunction with transesophageal echocardiographic imaging (588-591). In an asymptomatic patient with prior infarction, stress echocardiography may be helpful in assessing the presence, distribution, and severity of inducible myocardial ischemia and thereby determining the need for cardiac catheterization. However, in certain circumstances it may be difficult to detect residual ischemia within a zone of infarction that exhibits akinetic wall motion (161).

2. Special Issues With Regard to Stress Echocardiography for the Diagnosis of CAD

a. *The Influence of Bayes' Theorem*

In using any testing method, it is important to consider the pretest likelihood of the disorder being sought. With specific regard to stress echocardiography, the diagnostic value is greatest in patients in whom the pretest probability of clinical CAD is intermediate (roughly 20% to 80%). Subsets of patients with an intermediate pretest likelihood would include symptomatic middle-aged women with typical angina, patients with coronary risk factors and abnormal ECG findings at baseline, and patients with risk factors and atypical angina pectoris. In such patients, stress echocardiography would be expected to have the greatest value in increasing (based on a positive result) or lowering (based on a negative result) the likelihood of CAD. In patients with a very low pretest likelihood for CAD (such as patients with no risk factors or those with highly atypical or nonanginal chest pain), positive stress echocardiography results may often be false-positive. In patients with a very high pretest likelihood of CAD (such as middle-aged or elderly men with multiple coronary risk factors and classic angina pectoris), negative stress echocardiography results are often false-negative.

Notwithstanding these concerns, the results of stress echocardiography may have important prognostic value (even if the test is less valuable for diagnostic purposes). A positive stress echocardiographic study can be helpful in determining the location and severity of inducible myocardial ischemia, even in a patient with a high pretest likelihood that disease is present. A negative stress echocardiographic evaluation may also be prognostically helpful because it predicts a low risk for future cardiovascular events such as death and nonfatal myocardial infarction (185-193,575,592-598). Table 9 summarizes the prognostic value of stress echocardiography in various patient populations with chronic CAD.

b. *Influence of Posttest Referral Bias*

As discussed in more detail in the recent Stable Angina Guidelines (599), the issue of "posttest referral bias" (sometimes referred to as "workup bias" or "verification bias") is important in understanding the clinical usefulness of a diagnostic test. Once a test becomes used to guide patient management, it becomes more difficult to establish its true sensitivity and specificity. Because coronary angiography is far more likely to be recommended when the results of stress testing for CAD are positive and far less likely when stress testing is negative, this leads to a lower number of false-negative results and therefore tends to raise the measured sensitivity and lower the measured specificity of the test. This consideration pertains not only to stress echocardiography but also to other noninvasive diagnostic approaches such as conventional stress testing (without imaging) and stress myocardial perfusion imaging.

c. *Pharmacological Stress Echocardiography*

Pharmacological agents can be used to increase cardiac workload in lieu of treadmill or bicycle exercise, or to cause coronary arteriolar dilation or vasodilation and increased coronary blood flow; these are generally adrenergic-stimulating (such as dobutamine or arbutamine) or vasodilating agents (such as dipyridamole or adenosine). Adrenergic-stimulating agents increase myocardial oxygen demand by increasing contractility, blood pressure, and heart rate. They can be given in graded doses to titrate myocardial workload in a manner akin to standard exercise testing. Vasodilator agents, in contrast, cause heterogeneous myocardial perfusion, which in some patients is sufficient to cause functional myocardial ischemia.

These considerations suggest that pharmacological stress echocardiography might best be accomplished using adrenergic stimulants, since they enhance myocardial contractile performance, which can be evaluated directly by echocardiography. Vasodilator agents could cause heterogeneity of myocardial perfusion without actually altering workload (or wall motion) directly. Indeed, comparative studies have suggested a somewhat lower sensitivity for stress echocardiography using vasodilators compared with dobutamine (150,173,183,184,566,568,575). However, pharmacological stress echocardiography using vasodilator agents does appear to be useful in detecting inducible myocardial

Table 9. Prognostic Value of Stress Echocardiography in Various Patient Populations*

Author (Ref)	Year	Stress	Total No. of Pts	Average Follow-up, mo	Events	Ischemia	No Ischemia	Annualized Event Rate, %	Normal
Chronic Ischemic Heart Disease									
Picano (598)	1989	DIP†	539	36	D, MI	2.3	0.7	—	—
Sawada (185)	1990	NL TME	148	28.4	D, MI	—	—	—	0.6
Mazeika (187)	1993	DSE†	51	24	D, MI, UA	16	3.8	—	—
Krivokapich (186)	1993	TME†	360	~12	D, MI	10.8	3.1	—	—
Afridi (191)	1994	DSE†	77	10	D, MI	48	8.9	—	3
Poldermans (593)	1994	DSE†	430	17	D, MI	6.6	3.4	—	—
Coletta (189)	1995	DIP†	268	16	D, MI	17.9	1.4	—	—
Kamaran (192)	1995	DSE†	210	8	D, MI	69	1	—	—
Williams (190)	1996	DSE†	108	16	D, MI, Re	32.6	7.3	—	—
Anthopoulos (575)	1996	DSE†	120	14	D, MI	13.6	0	—	—
Marcovitz (193)	1996	DSE†	291	15	D, MI	12.8	8.2	—	1.1
Heupler (594)	1997	TME†	508w	41	D, MI, Re	9.2	1.3	—	—
McCully (595)	1998	NL TME	1325	23	D, MI	—	—	—	0.5
Chuah (596)	1998	DSE‡	860	24	D, MI	6.9	6.3	—	1.9
Cortigiani (592)	1998	DSE or DIP†	456w	32	D, MI	2.9	0.3	—	—
Davar (597)	1999	NL DSE	72w	13	D, MI	—	—	—	0
After Cardiac Transplantation									
Ciliberto (606)	1993	DIP‡	80	9.8	D, MI, CHF	26.2	0	—	—
Lewis (607)	1997	DSE‡	63	8	D, MI, CHF	28.6	3.6	—	—

*Prognostic value of inducible ischemia, detected using different forms of stress echocardiography, in patients with chronic ischemic heart disease and patients after cardiac transplantation. The "Early After Acute Myocardial Infarction" section of Table 9 as published in 1997 appears in Table 6a.

†New wall motion abnormality considered "positive" for inducible ischemia.

‡Any wall motion abnormality (at rest or with stress) considered "positive."

Annualized Event Rate indicates percentage of patients, per year, who developed at least one adverse event during follow-up, depending on whether inducible ischemia was or was not demonstrated by stress echocardiography. The annualized event rate is also tabulated for those series describing patients who had normal resting as well as normal stress results (NL). Average Follow-up (mo) indicates average period of follow-up after stress echocardiography; CHF, development of severe congestive heart failure; D, death; DIP, dipyridamole stress echocardiography; DSE, dobutamine stress echocardiography; LD-DSE, low-dose dobutamine stress echocardiography; MI, nonfatal myocardial infarction; NL, series describing follow-up only in subjects with normal stress echocardiography test results; Re, revascularization necessary; Stress, stress echocardiography protocol; TME, treadmill stress echocardiography; Total No. of Pts, number of patients studied using stress echocardiography and subsequently followed for the development of adverse events (including death, nonfatal myocardial infarction, revascularization, or unstable angina; in posttransplant patients, development of severe congestive heart failure was also considered an adverse event); UA, unstable angina; w, patients in these series were all women.

ischemia (172,543,574) and particularly valuable in determining prognosis (141,145,153,188,189,548-550,592,598).

d. Stress Echocardiography for Diagnosis of CAD in Women

The majority of studies reporting noninvasive diagnostic testing for the detection of CAD have described predominantly male patient populations. In part because men typically have a higher prevalence of angiographically proved CAD than women, the accuracy of exercise testing is lower in women than in men (600,601). In studies of nearly 1000 women with suspected CAD (most with chest pain), stress echocardiography has demonstrated good diagnostic accuracy for detecting or excluding significant CAD proven by subsequent angiography, with a weighted mean sensitivity of 81% (89% in women with multivessel disease), specificity of 86%, and overall accuracy of 84%. In women, stress echocardiography clearly has a higher diagnostic accuracy than conventional treadmill testing (600). Several studies, although uncorrected for referral bias, do suggest that stress echocardiography may be a cost-effective diagnostic strategy in women with an intermediate pretest probability of CAD (560,584,585), because it allows avoidance of inappropriate angiography. Table 9a summarizes the diagnostic accuracy of stress echocardiography in women.

e. Stress Echocardiography for Diagnosis of CAD in Patients After Cardiac Transplantation

Coronary arteriopathy is common in patients who have undergone cardiac transplantation and is a significant cause of morbidity and mortality (602). Angiographic assessment of transplant-associated CAD is difficult because of the diffuse nature of this disease, and some centers use intracoronary ultrasound to evaluate intimal thickening as part of post-transplant surveillance (602-605). In an effort to avoid repeated invasive evaluation, transplant cardiologists have used noninvasive testing methods to detect or exclude transplant coronary arteriopathy. Conventional treadmill exercise is often unsuccessful because of chronotropic incompetence in many patients after cardiac transplantation. Table 9b summarizes the diagnostic accuracy of stress echocardiographic testing in cardiac transplant patients. Although the number of patients studied was modest, dobutamine stress echocardiography appeared to offer a higher sensitivity (weighted mean 76%) compared with other stress echocardiographic methods. In addition, in several series (606,607), the presence or absence of ischemic abnormalities on stress echocardiographic studies has been reported to identify, respectively, heart transplant patients at high and low risk of adverse cardiac events during 8 to 10 months of follow-up (Table 9).

f. Detection of CAD in Asymptomatic Patients

Stress echocardiography is not recommended for screening in asymptomatic patients without known CAD because of the low pretest likelihood of disease. However, if a false-positive result is suspected in an asymptomatic patient with a positive

exercise treadmill test, a negative stress echocardiographic study may be helpful by lowering the likelihood of CAD and indicating a low likelihood of cardiac death or nonfatal myocardial infarction (185,191,193,595-597).

g. Stress Echocardiography for Preoperative Evaluation

This topic is discussed in the ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery available at http://www.acc.org/clinical/guidelines/periop/update/periupdate_index.htm (517).

3. Diagnosis of Myocardial Viability in Chronic CAD

In patients with chronic stable CAD, myocardial contractile function can be impaired because of irreversible myocardial necrosis or as a result of hibernating myocardium (194,608). Myocardial hibernation is thought to be due to chronic reduction in myocardial perfusion to levels inadequate to support normal myocardial contractile performance but sufficient to preserve viability (609). Since this condition is potentially reversible, identifying it accurately has important clinical value; revascularization of hibernating myocardium can lead to functional recovery and clearly improves long-range outlook (610-612). In patients with multivessel CAD and depressed LV function, improvement in regional LV function during dobutamine stress echocardiography indicates contractile reserve and is predictive of improved ventricular function after revascularization (195-200,613-628). The lack of contractile reserve during low-dose dobutamine infusion denotes a low likelihood of improvement after bypass surgery. The presence or absence of contractile reserve by low-dose dobutamine stress echocardiography has both weighted mean positive and negative predictive values of 83%. Table 9c summarizes the role of dobutamine stress echocardiography for evaluating hibernating myocardium.

4. Assessment of Disease Severity/Risk Stratification/Prognosis in Chronic CAD

Echocardiographic techniques, at rest and particularly coupled with stress, can be helpful in clinical decision making regarding medical therapies and clinical interventional therapies, in evaluating the results of therapy, in prognostication, and clinical follow-up of patients with known CAD and new or changing symptoms. There is evidence that patients with a recent myocardial infarction and an ejection fraction less than or equal to 30% randomized to an implantable cardioverter-defibrillator (ICD) had a 31% relative risk reduction in mortality compared to those in the conventionally treated group after the mean follow up of 20 months (834, 835).

In patients with chronic ischemic heart disease, LV ejection fraction measured at rest has an important influence on long-term prognosis (201); as LV ejection fraction declines, mortality increases. Ejection fraction is an important consideration in choosing appropriate medical or surgical therapies

Table 9a. Diagnostic Accuracy of Stress Echocardiography in Detecting Angiographically Proved CAD in Women (Generally Without Correction for Referral Bias)

Author (Ref)	Year	Protocol	Significant CAD	Total		Sens, %	Sens I-VD	Sens MVD	Specificity %	PPV, %	NPV, %	Overall Accuracy, %
				No. of Pts	%							
Masini (844)	1988	DIP	≥ 70%	83	79	—	—	—	93	91	84	87
Sawada (152)	1989	TME or UBE	≥ 50%	57	86	88	82	—	86	86	86	86
Severi (188)	1994	DIP	≥ 75%	122	68	—	—	—	96	90	86	87
Williams (559)	1994	UBE	> 50%	70	88	89	86	86	84	83	89	86
Marwick (560)	1995	TME or UBE	≥ 50%	161	80	75	85	85	81	71	87	81
Takeuchi (579)	1996	DASE	≥ 50%	70	75	78	73	73	92	79	90	87
Roger (580)	1997	TME or UBE	≥ 50%	96	79	—	—	—	37	66	54	63
Dionisopoulos (577)	1997	DASE	≥ 50%	101	90	79	94	94	79	90	79	86
Lauritzen (590)	1997	DS-TEE	≥ 70%	84	82	—	—	—	100	100	94	95
Elhendy (581)	1997	DASE	≥ 50%	96	76	64	92	92	94	96	68	82
Ho (578)	1998	DSE	≥ 50%	51	93	89	95	95	82	87	90	88
Studies Accounting for Referral Bias												
Lewis (845)	1999	DSE	≥ 50%	92	40	40	60*	60*	81	71	84	70
(by design)								82†				
Roger (580)	1997	TME	≥ 50%	1714	32	24	31 (2V)	43 (3V)	86	66		
(by adjustment)												

Diagnostic accuracy of stress echocardiography, using either exercise or pharmacological stress, in detecting angiographically proved coronary artery disease (CAD) in women. A new or worsening regional wall motion abnormality induced by stress generally was considered a "positive" result.

CAD indicates coronary artery disease; DASE, dobutamine/atropine stress echocardiography; DIP, dipyridamol stress echocardiography; DSE, dobutamine stress echocardiography; DS-TEE, dobutamine stress transesophageal echocardiography; MVD, test results positive in patients with multivessel CAD; NPV, negative predictive value (likelihood of absence of angiographically significant CAD in patients without inducible wall motion abnormalities by stress echocardiography); I-VD, test results positive in patients with single-vessel CAD; PPV, positive predictive value (likelihood of angiographically significant CAD in patients with inducible wall motion abnormalities by stress echocardiography); Protocol, exercise or pharmacologic protocol, used in conjunction with trans thoracic echo imaging; Pts, patients; Sens, sensitivity; Significant CAD, % coronary luminal diameter narrowing, documented by selective coronary angiography, considered to represent "significant" CAD; Spec, specificity; TME, treadmill stress echocardiography; Total Pts, number of women in each series undergoing selective coronary angiography, in whom stress echocardiography studies were also performed and wall motion analysis performed; UBE, upright bicycle stress echocardiography.

*including all patients.
†excluding patients with indeterminate studies.

Table 9b. Diagnostic Accuracy of Stress Echocardiography in Detecting CAD After Cardiac Transplantation (Without Correction for Referral Bias)

Author (Ref)	Year	Protocol	Total No. of Pts	Ref Std	Sensitivity, (%)	Specificity, %	PPV %	NPV %	Overall Accuracy, %
Exercise Protocol									
Collings (603)	1994	TME*	51	CAD angio	25	86	25	86	69
Cohn (604)	1996	UBE*	51	CAD angio	26	95	85	54	73
Cohn (604)	1996	UBE*	51	ICUS \geq III	15	84	67	32	57
Vasodilator Protocol									
Ciliberto (606)	1993	DIP*	80	CAD angio	32	100	100	76	79
Ciliberto (606)	1993	DIP†	80	CAD angio	76	85	70	89	83
Dobutamine Protocol									
Akosah (846)	1994	DSE*	41	CAD angio	95	55	69	92	76
Herregods (847)	1994	DSE*	28	CAD angio	0	100	0	100	50
Derumeaux (848)	1995	DSE*	37	CAD angio	86	91	86	91	89
Spes (605)	1996	DSE*	46	CAD angio	83	56	40	90	63
Spes (605)	1996	DSE*	46	ICUS \geq III	79	83	88	71	80
Akosah (849)	1996	DSE*	41	CAD angio	95	55	69	92	76
Derumeaux (850)	1998	DSE†	37	CAD angio	71	95	92	79	84
Akosah (851)	1998	DSE†	22	CAD angio	100	73	64	100	82
Larsen (773)	1998	DSE*	70	CAD angio	72	80	48	90	79
Spes (852)	1999	DSE†	98	ICUS and angio	72	88	92	62	NA

Diagnostic accuracy of stress echocardiography, using either exercise or pharmacological stress, in detecting documented coronary arteriopathy in patients evaluated after cardiac transplantation. A new or worsening regional wall motion abnormality induced by stress generally was considered a "positive" result.

*New wall motion abnormality during stress considered "positive."

†Resting wall motion abnormalities considered "positive."

Angio indicates angiography; CAD angio, coronary artery disease documented by angiography; DIP, dipyridamole stress echocardiography; DSE, dobutamine stress echocardiography; ICUS \geq III, coronary intimal thickening grade III or more by intracoronary ultrasound; NPV, negative predictive value (likelihood of absence of significant CAD in patients without wall motion abnormalities by stress echocardiography); PPV, positive predictive value (likelihood of significant CAD in patients with wall motion abnormalities by stress echocardiography); Protocol, exercise or pharmacological modality used to induce stress; Ref Std, reference standard used to establish the presence or absence of coronary arteriopathy; TME, treadmill stress echocardiography; Total No. of Pts, number of patients in each series undergoing either selective coronary angiography or intracoronary ultrasound studies in whom stress echocardiography procedures were also performed and analyzed for wall motion abnormalities; UBE, upright bicycle stress echocardiography.

Table 9c. Myocardial Viability: Detection of Hibernating Myocardium by DSE in Patients With Chronic CAD and LV Dysfunction

Author (Ref)	Year	Stress	Total				Sensitivity, %	Specificity, %	PPV, %	NPV, %	Overall Accuracy, %
			No. of Pts	Criteria							
Marzullo (614)	1993	LD-DSE	14	ImpWM*	82	92	95	73	85		
Cigarroa (195)	1993	LD-DSE	25	ImpWM†	82	86	82	86	84		
Alfieri (615)	1993	LD-DSE	14	ImpWM*	91	78	92	76	88		
LaCanna (197)	1994	LD-DSE	33	ImpWM*	87	82	90	77	85		
Charney (616)	1994		17	ImpWM*	71	93	92	74	81		
Afridi (196)	1995	DSE	20	ImpWM†	80	90	89	82	85		
Perrone-Filardi (199)	1995	LD-DSE	18	ImpWM*	88	87	91	82	87		
Senior (617)	1995	LD-DSE	22	ImpWM*	87	82	92	73	86		
Haque (618)	1995	LD-DSE	26	ImpWM*	94	80	94	80	91		
Arnese (198)	1995	LD-DSE	38	ImpWM*	74	96	85	93	91		
deFilippi (200)	1995	LD-DSE	23	ImpWM*	97	75	87	93	89		
Iliceto (619)	1996	LD-DSE	16	ImpWM*	71	88	73	87	83		
Varga (620)	1996	LD-DSE	19	ImpWM*	74	94	93	78	84		
Baer (621)	1996	LD-DSE	42	ImpWM†	92	88	92	88	90		
Vanovershelde (622)	1996	LD-DSE	73	impWM†	88	77	84	82	84		
Gerber (623)	1996	LD-DSE	39	ImpWM*	71	87	89	65	77		
Bax (624)	1996	LD-DSE	17	ImpWM*	85	63	49	91	70		
Perrone-Filardi (625)	1996	LD-DSE	18	ImpWM*	79	83	92	65	81		
Qureshi (626)	1997	LD-DSE	34	ImpWM*	86	68	51	92	73		
Qureshi (626)	1997	DSE	34	Biphasic resp*	74	89	72	89	85		
Nagueh (853)	1997	LD-DSE	18	ImpWM*	91	66	61	93	75		
Nagueh (853)	1997	DSE	18	Biphasic resp*	68	83	70	82	77		
Furukawa (627)	1997	LD-DSE	53	ImpWM*	79	72	76	75	76		
Cornel (628)	1997	LD-DSE	30	ImpWM*	89	82	74	93	85		

Evaluation of myocardial viability, using dobutamine stress echocardiography (DSE), in patients with chronic coronary artery disease (CAD) and impaired systolic left ventricular (LV) function to detect hibernating myocardium. In these patients, percutaneous or surgical revascularization was performed after DSE testing. Those patients demonstrating improved wall motion on follow-up resting transthoracic echocardiography were considered to have had impaired LV function due to hibernating myocardium, whereas those demonstrating no improvement despite revascularization were considered to have had impaired LV function due to necrotic myocardium.

†wall motion analyzed by patient; *wall motion analyzed by segment.

Biphasic resp indicates biphasic response, defined as improvement in wall motion during low-dose dobutamine stress followed by worsening at high-dose; Criteria, findings on DSE considered as a "positive" indicator of viability; DSE, dobutamine stress echocardiography (dobutamine infused at both low and high doses); ImpWM, improved wall motion during dobutamine stress in a previously asymptomatic segment; LD-DSE, low-dose dobutamine stress echocardiography; NPV, negative predictive value (likelihood that absence of viability by DSE is indicative of lack of functional recovery following revascularization); PPV, positive predictive value (likelihood that presence of viability by DSE is indicative of subsequent functional recovery after revascularization); Stress, DSE protocol used for pharmacological stress; Total No. of Pts, number of patients with chronic CAD and LV dysfunction in whom DSE studies were analyzed.

and in making recommendations about activity levels. In patients with clinical signs and symptoms of congestive heart failure, echocardiography is also helpful in establishing pathophysiological mechanisms and guiding therapy. For example, after a myocardial infarction, a patient with congestive heart failure might have systolic LV dysfunction, predominant diastolic dysfunction, mitral regurgitation, some combination of these abnormalities, or a noncardiac cause for heart failure symptoms. How best to treat the patient can be planned more rationally when one knows the state of LV systolic and diastolic function, valvular function, and right-heart hemodynamics. These recommendations are discussed in section II, "Murmurs and Valvular Heart Disease," and section V, "Cardiomyopathy, Congestive Heart Failure, and Assessment of Left Ventricular Function: Echocardiographic Parameters."

As summarized in Table 9, the presence or absence of inducible myocardial ischemia has useful prognostic value in patients undergoing exercise or pharmacological stress echocardiography. A negative stress echocardiographic study generally denotes a low rate of adverse cardiovascular events during follow-up (185-193,575,592-598). Compared with standard treadmill testing, stress echocardiography is more specific for identifying patients with inducible myocardial ischemia. In general, patients with a positive electrocardiographic response to treadmill stress test but no inducible wall motion abnormality on stress echocardiogram have a very low rate of adverse cardiovascular events during follow-up (185,186,594), albeit higher than patients with a completely negative test result.

The prognosis is not benign in patients with a positive stress echocardiographic study. In this subset, morbid or fatal cardiovascular events are more likely, but the overall event rates are rather variable. Hence, the cost-effectiveness of using routine stress echocardiography testing to establish prognosis is uncertain. Nonetheless, a number of studies involving nearly 6000 patients with chronic CAD do indicate that the risk of future cardiac events can be stratified based on the presence or absence of inducible ischemia on stress echocardiography testing (Table 9).

5. Echocardiographic Assessment Before and After Revascularization

Echocardiographic studies may help in planning revascularization procedures by demonstrating the functional significance of a given coronary stenosis. This may be of particular value in determining the need for percutaneous transluminal coronary angioplasty, particularly when the degree of angiographic stenosis is of uncertain physiological significance or when multiple lesions are present. Moreover, because restenosis is a common complication, stress echocardiography is useful in evaluating patients after coronary angioplasty (151). Reassessment roughly 1 month after angioplasty is a reasonable time frame within which to assess the functional results of angioplasty. However, in an asymptomatic stable patient, routine stress testing (with or without an imaging modality) does not appear to be cost-effective. When a patient is symptomatic or when there are other clinical recommendations, an evaluation can be performed using either treadmill, bicycle, or pharmacological methods to induce stress, depending on the patient's physical capabilities. Compared with the preangioplasty evaluation, improvement in wall motion on stress echocardiography evaluation after angioplasty confirms a successful result; persistent evidence of inducible ischemia after angioplasty indicates an inadequate result or restenosis. More extensive studies are needed to document the value of stress echocardiography in assessing the results of percutaneous revascularization.

In patients with heart failure due to ischemic LV dysfunction, evaluation of myocardial viability by dobutamine stress echocardiography can help determine the potential benefit of revascularization. The demonstration of significant hibernating myocardium, suggesting a high likelihood of improved function after revascularization (195-200,613-628), can help in choosing revascularization rather than heart transplantation. The prognostic value of contractile reserve, demonstrated with using low dose dobutamine stress echocardiography in patients with CAD and chronic impairment of LV systolic function, and the influence of revascularization on subsequent adverse events are summarized in Table 9d.

After successful bypass surgery, routine follow-up testing generally is not necessary in the asymptomatic individual. Improvement in patient outcomes by identifying asympto-

Table 9d. Prognostic Value of Viable (Hibernating) Myocardium by LD-DSE, and Influence of Revascularization

Author (Ref)	Year	Stress	Total No. of Pts	Average Follow-up, mo	Annualized Event Rate, %			
					Events	Viable, +Re	Viable, -Re	Not Viable
Meluzin (854)	1998	LD-DSE	133	20	D, MI	4.1	—	9.5
Afridi (855)	1998	LD-DSE	353	18	D	4	20	19

Prognostic value of contractile reserve, detected using low-dose dobutamine stress echocardiography (LD-DSE), in patients with chronic ischemic heart disease and impaired left ventricular systolic function. The annualized rate of death or nonfatal myocardial infarction is tabulated in patients with viable myocardium by LD-DSE depending on whether they did or did not undergo revascularization, and also in those patients without viable myocardium.

Annualized Event Rate indicates percentage of patients, per year, who developed an adverse event during follow-up after LD-DSE; Average Follow Up (mo), average period of follow-up, after LD-DSE; D, death; Events, adverse events; LD-DSE, low-dose dobutamine stress echocardiography; MI, nonfatal myocardial infarction; Not Viable, patients without contractile reserve by LD-DSE, who were followed up for adverse events; Stress, stress echocardiography protocol; Total No. of Pts, number of patients with chronic ischemic heart disease and impaired left ventricular systolic function studied using low-dose dobutamine stress echocardiography and subsequently followed up for the development of an adverse event (death or nonfatal myocardial infarction); Viable,+Re, patients with viability (contractile reserve) demonstrated by LD-DSE who underwent revascularization and were then followed; Viable,-Re, patients with viability (contractile reserve) demonstrated by LD-DSE who did not undergo revascularization and were then followed up.

matic residual inducible ischemia has not been demonstrated, hence routine testing cannot be recommended. However, when symptoms persist or recur after coronary bypass surgery, stress echocardiography testing can be helpful. After cardiac surgery many patients have abnormal baseline ECG findings, and early after bypass surgery some demonstrate abnormal ECG responses on standard treadmill testing (202). When the possibility of incomplete revascularization is of clinical concern, stress echocardiography studies may be helpful in evaluating the location and severity of residual ischemia. When an initial postoperative stress echocardiographic study is negative for inducible ischemia but a subsequent test is positive, the likelihood of graft closure or development of new obstructive lesions can be inferred.

The recommendations for echocardiography in chronic ischemic heart disease are summarized below.

Recommendations for Echocardiography in Diagnosis and Prognosis of Chronic Ischemic Heart Disease

Class I

1. **Diagnosis of myocardial ischemia in symptomatic individuals.***
2. **Exercise echocardiography for diagnosis of myocardial ischemia in selected patients (those where ECG assessment is less reliable because of digoxin use, LVH or with more than 1 mm ST depression at rest on the baseline ECG, those with pre-excitation [Wolff-Parkinson-White] syndrome, complete left bundle-branch block) with an intermediate pretest likelihood of CAD.**
3. **Assessment of global ventricular function at rest.**
4. **Assessment of myocardial viability (hibernating myocardium) for planning revascularization.†**
5. **Assessment of functional significance of coronary lesions (if not already known) in planning percutaneous transluminal coronary angioplasty.***

Class IIa

1. **Prognosis of myocardial ischemia in selected patients (those in whom ECG assessment is less reliable) with the following ECG abnormalities: pre-excitation (Wolff-Parkinson-White) syndrome, electronically-paced ventricular rhythm, more than 1 mm of ST depression at rest, complete left bundle-branch block.***
2. **Detection of coronary arteriopathy in patients who have undergone cardiac transplantation.†**
3. **Detection of myocardial ischemia in women with an intermediate pretest likelihood of CAD.***

Class IIb

1. **Assessment of an asymptomatic patient with positive results from a screening treadmill test.***
2. **Assessment of global ventricular function with exercise.***

Class III

1. **Screening of asymptomatic persons with a low likelihood of CAD.**

2. **Routine periodic reassessment of stable patients for whom no change in therapy is contemplated.**
3. **Routine substitution for treadmill exercise testing in patients for whom ECG analysis is expected to suffice.***

*Exercise or pharmacological stress echocardiogram.

†Dobutamine stress echocardiogram.

The use of echocardiography in the evaluation of patients undergoing noncardiac surgery is covered in the ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery (517).

Recommendations for Echocardiography in Assessment of Interventions in Chronic Ischemic Heart Disease

Class I

1. **Assessment of LV function when needed to guide institution and modification of drug therapy in patients with known or suspected LV dysfunction.**
2. **Assessment for restenosis after revascularization in patients with atypical recurrent symptoms.***

Class IIa

1. **Assessment for restenosis after revascularization in patients with typical recurrent symptoms.***
2. **Assessment of LV function in patients with previous myocardial infarction when needed to guide possible implantation of implantable cardioverter-defibrillator (ICD) in patients with known or suspected LV dysfunction.**

Class III

Routine assessment of asymptomatic patients after revascularization.

*Exercise or pharmacological stress echocardiography.

V. CARDIOMYOPATHY, CONGESTIVE HEART FAILURE, AND ASSESSMENT OF LEFT VENTRICULAR FUNCTION: ECHOCARDIOGRAPHIC PARAMETERS

The evaluation of ventricular systolic function is the most common recommendation for echocardiography. Current techniques permit a comprehensive assessment of LV size and function. LV cavity measurements and wall thickness at end diastole and end systole and shortening fraction may be obtained with precision by M-mode echocardiography; conventions for obtaining these measurements (204,205) and reference normal values have been published. Two-dimensional echocardiography, because of its superior spatial resolution, is used to guide appropriate positioning of the M-mode beam and is used for direct measurements of ventricular dimensions (204) as well as for calculation of LV volumes and ejection fraction. An advantage of two-dimensional (compared with M-mode) echocardiography is that the

chamber volumes, ejection fraction, and the LV mass of an abnormally shaped ventricle can be determined. Therefore, in most laboratories two-dimensional echocardiography is the principal noninvasive method used for quantitating LV volumes and assessing global and regional systolic function. LV mass and volume quantitation by echocardiography requires high-quality images, meticulous attention to proper beam orientation, and the use of geometric models to approximate LV shape (206).

A. Assessment of Ejection Fraction

M-mode echocardiographic methods can be used to define many indices of global LV function; the most widely used parameters are ejection phase indices, including fractional shortening of the minor axis and velocity of circumferential fiber shortening. However, ejection fraction, a more widely used index, must either be derived using algorithms developed for volume determination from M-mode linear dimensions (206,207), visually estimated from two-dimensional echocardiographic images (208), or obtained by quantitative analysis of two-dimensional echocardiographic images (209,210). The algorithms vary considerably in complexity. In general, although all are suitable for assessing performance of a normally shaped, normally contracting left ventricle, more complex approaches are required to assess deformed ventricles with regional wall motion abnormalities. Simplified approaches combining measurements and visual assessment of the function of the apex have been proposed (209), but these also have limited applicability in distorted ventricles.

In clinical practice the visual estimation of ejection fraction from two-dimensional echocardiography is common (208,211). Ejection fraction may be reported quantitatively or qualitatively as increased, normal, or mildly, moderately, or severely reduced; or it may be quantitated. When performed by skilled observers, ejection fraction by visual estimation corresponds closely to that obtained by angiography (207) or gated blood pool scanning (208). However, because of its subjective nature, a visual estimate of ejection fraction may be less reproducible than quantitative methods. Optimally, its use should be restricted to those practitioners with considerable experience in echocardiography who can periodically compare their visual estimates to those obtained with a nonechocardiographic method. Alternative approaches such as LV angiography and nuclear methods are often used to obtain a quantitative ejection fraction. However, in the absence of an interval change in patient status or another recommendation for testing, duplicate estimates of ejection fraction with multiple modalities should be discouraged. The administration of echocardiographic contrast agents improves the delineation of the endocardial/LV cavity interface and improves the accuracy of two-dimensional echocardiographic estimates of ejection fraction (629).

All ejection phase indices of myocardial contractile performance are limited by their load dependence. Indices that are less sensitive to hemodynamic loads, including end-systolic pressure-volume relations (210), preload recruitable

stroke work, and end-systolic stress-length relations (212), can be derived. Although these indices may be used in the clinical setting, practically speaking, their use is limited by the need for simultaneous LV pressure and difficult complex mathematical calculations.

Although Doppler analysis of aortic outflow spectra may be used to derive systolic time intervals, peak aortic flow velocity, and acceleration, these measurements are not generally used in clinical practice. The determination of cardiac output is a potentially more useful Doppler application (213,214) and can be performed in outpatients, but for serial studies in critically ill patients thermodilution methods are usually used for this purpose.

B. Regional LV Function

Echocardiography is well suited for the assessment of regional LV contractile function in view of its high spatial and temporal resolution and its ability to define regional wall thickening and endocardial excursion. Controversy still surrounds the optimal method for assessing regional LV function; however, virtually all carefully tested methods have yielded useful data (210).

1. Clinical Syndromes

a. Edema and Dyspnea

The causes of peripheral edema, both cardiac and noncardiac, are numerous. Cardiac causes include any abnormality that results in elevated central venous pressure and thus encompasses the full spectrum of myocardial, valvular, and pericardial disease. Echocardiography provides the diagnosis in many, if not most, instances. In some instances, however, the overlapping features of constrictive pericarditis and restrictive cardiomyopathy make definitive diagnosis by ultrasound difficult (see section VI, "Pericardial Disease"). Echocardiography is not recommended in patients with edema when the jugular venous pressure does not appear to be elevated.

Dyspnea, either at rest or with exertion, is one of the cardinal symptoms of heart disease. When present in patients with heart failure, dyspnea may denote pulmonary venous hypertension. It can be difficult to distinguish among the various etiologies of dyspnea, which include primary cardiac or respiratory abnormalities, deconditioning, anemia, difficulties with peripheral circulation, or anxiety. Certain features of the history help establish that dyspnea is of cardiac origin, such as a progressive decrease in the intensity of exertion necessary to produce symptoms. Certainly dyspnea accompanying obvious signs of heart disease strongly suggests a cardiac etiology. When the etiology is in doubt, echocardiography can document or rule out the common cardiac causes of pulmonary congestion: left-sided valvular disease, depressed systolic or diastolic function, and cardiomyopathy. In this regard, echocardiography is the preferred initial diagnostic test when the history, physical examination, and routine laboratory tests suggest (or cannot eliminate) cardiac disease.

Table 9e. Doppler Echocardiographic Indices of Diastolic Function

- Mitral inflow velocities (E wave, A wave, E/A ratio)
- Mitral E-wave deceleration time
- Isovolumic relaxation time
- Pulmonary vein systolic and diastolic velocities (S, D, S/D ratio)
- Pulmonary vein atrial systolic reversal (PVa)
- Difference between PVa and mitral A-wave duration
- Mitral annular velocities as measured by tissue Doppler imaging: E' (early), A' (late), and ratio of mitral E to Doppler tissue E'
- Color M-mode flow propagation

b. Heart Failure

Most instances of systolic dysfunction are due to ischemic heart disease, hypertensive disease, or valvular heart disease. However, primary disorders of the heart muscle may also be often encountered and are usually of unknown etiology. These disorders are often categorized as dilated/congestive, hypertrophic, and restrictive (215). Ultrasound techniques permit a comprehensive assessment of morphology and function and often allow assessment of hemodynamic status regardless of etiology. Left-sided contrast agents and TEE extends the capability of ultrasound techniques to the acutely ill patient in the intensive care unit, a setting where routine transthoracic imaging may be of limited value (see section XIII, "Echocardiography in the Critically Ill"). For these reasons, echocardiography often provides important insight into the etiology of congestive heart failure signs and symptoms. In a retrospective analysis of 50 consecutive patients with the principal diagnosis of congestive heart failure who underwent M-mode and two-dimensional echocardiography, Echeverria et al. (216) reported that echocardiography often furnished unexpected information; in 40% of patients with reduced systolic function, the ejection fraction was worse than expected, and 20 of the 50 patients (unexpectedly) had normal systolic function. In the study population as a whole, echocardiography was associated with a change in management in 29 of 50 (or 58%) of patients. The utility of echocardiography was greatest in the subgroup of 20 patients in whom echocardiography revealed normal systolic function; this information led to a change in diagnosis and management in 18 (90%) of these patients.

These observations concerning the utility of echocardiography in patients with congestive heart failure were extended by Aguirre et al. (217), who prospectively studied 151 consecutive patients undergoing Doppler echocardiography who had a clinical diagnosis of congestive heart failure; a normal ejection fraction (greater than 55%) was observed in 34% of patients. More recent data from population studies confirm the high prevalence of normal ejection fraction in older patients hospitalized for congestive heart failure (630,631).

c. Heart Failure With Normal Ejection Fraction (Diastolic Dysfunction)

Diastolic dysfunction, defined as heart failure in the presence of an ejection fraction greater than 40% (80,217,218,235), as

mentioned above, is common. This syndrome is related to the inability of the left ventricle to fill adequately at normal pressure. There are other, subtler manifestations of diastolic dysfunction, including failure to augment cardiac output with exercise (236). Given that the optimal management for the patient with heart failure with normal ejection fraction (and probably the patient's prognosis) is likely to be quite different from the heart failure patient with reduced ejection fraction, it is important that the proper diagnosis be made. A large number of indices of diastolic function based on information from M-mode and two-dimensional echocardiography Doppler mitral and pulmonary flow profiles (see below) have been investigated. The most commonly used Doppler indices are the early E wave and late A wave and their ratio, the deceleration time of the E wave, and the isovolumic relaxation time (see Table 9e). When these variables are used for the evaluation of impaired relaxation and the semiquantification of filling pressures, care must be taken to understand their limitations. Impaired relaxation may be overdiagnosed in patients with decreased preload and tachycardia. Normal values also need to be adjusted for age. Validation of filling pressures has been performed predominantly in patients with a decreased LV ejection fraction and sinus rhythm.

Nonetheless, when these indices are interpreted in the context of clinical and other echocardiographic variables, such as left atrial size, and with the recognition of all the potentially confounding influences, they may provide valuable information in individual subjects. As diastolic dysfunction progresses, there may be a period of pseudonormalization during which there is a combination of impaired relaxation and elevation of LV filling pressures. The use of pulmonary vein flow velocities, as well as newer techniques such as tissue Doppler imaging and analysis of flow propagation by color M-mode, may provide additional information to enable the clinician to detect filling abnormalities when standard mitral flow velocities appear normal. The duration of pulmonary vein diastolic flow reversal may indicate increased LV filling pressure, particularly when it exceeds the duration of the mitral A wave (632). In patients with heart disease, analysis of the early velocity of mitral annular motion, coupled with the transmitral E wave, has been shown to correlate with LV filling pressures (633,634) regardless of the LV ejection fraction and rhythm. Doppler-derived parameters of diastolic filling can be useful in assessing prognosis.

d. Hypertrophic Cardiomyopathy

Echocardiography provides a definitive diagnosis of hypertrophic cardiomyopathy, revealing ventricular hypertrophy in patients without other primary causes. Echocardiographic imaging also permits a comprehensive assessment of the degree and distribution of hypertrophy (228), which may affect prognosis. Doppler techniques may be used to localize and quantify intraventricular gradients at rest and with provocative maneuvers, evaluate diastolic filling, and quantify associated mitral regurgitation (229). Several investigators have shown that tissue Doppler imaging can detect abnormal diastolic function in patients with hypertrophic cardiomy-

opathy even prior to the development of ventricular hypertrophy (836,837). When coronary disease is not strongly suspected, comprehensive Doppler echocardiographic examination may obviate the need for catheterization.

Echocardiography may also be used to evaluate the response to therapeutic interventions, such as alterations in medical therapy, dual-chamber pacing, septal ethanol ablation, and surgical myectomy (230). Echocardiographic contrast assessment of the myocardial infarct zone during interventional septal alcohol ablation is very useful (838,839).

e. Restrictive Cardiomyopathy

Echocardiography generally demonstrates normal ventricular size and systolic function but atrial enlargement in patients with restrictive cardiomyopathy. Doppler studies have demonstrated characteristic ventricular inflow velocity profiles that consist of elevated peak early flow velocity, rapid deceleration, and reduced flow velocity associated with atrial contraction (231,232). Frequently, isovolumic relaxation time is shortened, and pulmonary venous flow velocities demonstrate significant diastolic flow reversal (233,234). The subject of myocardial tissue characterization by echocardiography is still being investigated. However, the intense echocardiography reflectance that gives the myocardium a characteristic stippled appearance in some cases of amyloidosis is clinically useful in identifying this cause of restrictive cardiomyopathy (635,636).

f. Heart Failure With Reduced Ejection Fraction and LV Dilation

Echocardiography demonstrates dilation of ventricular chambers, usually without increased wall thickness, as well as valvular function, pericardial abnormality and size and function of the RV. Systolic function is depressed to varying degrees, with diffusely abnormal wall motion. Doppler techniques are used to evaluate the presence and extent of associated valvular regurgitation, to estimate pulmonary pressures, and to gain insight into diastolic function of the left ventricle (218). Echocardiography also permits re-evaluation of ventricular size and function so that disease progression and response to therapy may be monitored noninvasively. Doppler mitral inflow abnormalities ("restrictive" pattern) correlate strongly with congestive symptoms (219). A short deceleration time (less than 115 ms) is an independent predictor of poor outcome or need for transplantation (220). In view of the established benefits of angiotensin converting enzyme inhibitors (221,222) in patients with ventricular dysfunction, echocardiography is also used to evaluate the appropriateness of such therapy.

Chemotherapy with doxorubicin produces a dose-dependent degenerative cardiomyopathy (223). It is therefore recommended that cumulative doses of doxorubicin be kept to less than 450 to 500 mg/m² (224). In fact, subtle abnormalities of systolic function (increases in wall stress) are evident in 17% of patients receiving only one dose of doxorubicin; most patients who receive at least 228 mg/m² show either increased wall stress or evidence of reduced contractility by

stress-shortening analysis (223). For this reason, re-evaluation monitoring of ejection fraction throughout the course of chemotherapy appears to be important in that further administration of doxorubicin appears safe if resting ejection fraction remains in the normal range and, conversely, further treatment may be dangerous if ejection fraction is abnormally low. It has been hypothesized that Doppler mitral inflow abnormalities suggestive of impaired relaxation might precede reductions in ejection fraction in patients receiving serial doses of doxorubicin (225). Abnormalities in diastolic filling, either by Doppler or radionuclide techniques, in patients with normal systolic function have been demonstrated in patients receiving 200 to 300 mg/m² of doxorubicin (226,227).

g. Evaluation of the Right Ventricle

Approaches to obtaining quantitative determinations of RV size (204) and volume (239) have been proposed but are more problematic than comparable measurements of the left ventricle. This is due both to the complex shape of this chamber with its heavy trabecular pattern and to the difficulty in obtaining standardized imaging planes. The myocardial performance index (or Tei index) has been proposed as a way of quantitating RV performance, but experience with this index is limited (840,841). Thus, the assessment of RV size is often performed in a qualitative fashion. Similarly, although global RV systolic function in adults is difficult to quantitate by echocardiography, useful qualitative information may be obtained. In children, useful quantitative measures of RV systolic function may be made.

Recommendations for Echocardiography in Patients With Dyspnea, Edema, or Cardiomyopathy

Class I

- 1. Assessment of LV size and function in patients with suspected cardiomyopathy or clinical diagnosis of heart failure.***
- 2. Edema with clinical signs of elevated central venous pressure when a potential cardiac etiology is suspected or when central venous pressure cannot be estimated with confidence and clinical suspicion of heart disease is high.***
- 3. Dyspnea with clinical signs of heart disease.**
- 4. Patients with unexplained hypotension, especially in the intensive care unit.***
- 5. Patients exposed to cardiotoxic agents, to determine the advisability of additional or increased dosages.**
- 6. Re-evaluation of LV function in patients with established cardiomyopathy when there has been a documented change in clinical status or to guide medical therapy.**
- 7. Suspicion of hypertrophic cardiomyopathy based on abnormal physical examination, ECG, or family history.**
- 8. Contrast echocardiographic assessment of myocardial infarct zone during interventional septal alcohol ablation studies.**

Class IIb

1. **Re-evaluation of patients with established cardiomyopathy when there is no change in clinical status but where the results might change management.**
2. **Re-evaluation of patients with edema when a potential cardiac cause has already been demonstrated.**

Class III

1. **Evaluation of LV ejection fraction in patients with recent (contrast or radionuclide) angiographic determination of ejection fraction.**
2. **Routine re-evaluation in clinically stable patients in whom no change in management is contemplated and for whom the results would not change management.**
3. **In patients with edema, normal venous pressure, and no evidence of heart disease.**

*TEE is indicated when TTE studies are not diagnostic.

VI. PERICARDIAL DISEASE

One of the earliest clinical applications of echocardiography was in the detection of pericardial effusion (240,637), and it remains the procedure of choice for evaluating this condition. The pericardium usually responds to disease or injury by inflammation, which may result in pericardial thickening, the formation of an exudate, or both, which in turn is manifested in the clinical picture of pericardial effusion with or without tamponade or constriction physiology. Pericarditis can occur after cardiac surgery (postpericardiotomy syndrome). The anatomic evidence of pericardial disease and its functional effects on cardiovascular physiology can often be seen on M-mode, two-dimensional, and Doppler echocardiograms.

A. Pericardial Effusion

Echocardiography provides a semiquantitative assessment of pericardial effusion and a qualitative description of its distribution. Differentiation among types of pericardial fluid (blood, exudate, transudate, and others) cannot be made, but fibrous strands, pericardial adhesions, tumor masses, and blood clots can often be distinguished. It should be remembered that all “echo-free” spaces adjacent to the heart are not the result of pericardial effusion (241). Focal epicardial fat must be distinguished from small to medium localized effusions.

Most pericardial effusions that require pericardiocentesis are located both anteriorly and posteriorly, but loculated effusions may occur, particularly after cardiac surgery. In such cases, echocardiography can define the distribution of the fluid so that the safest and most effective approach can be planned for the pericardiocentesis. TEE may be used in those with technically unsuitable surface studies and in early postoperative cases, in whom it is often difficult to obtain a surface echocardiogram of diagnostic quality. Postoperative loculated effusions may be difficult to detect, and typical echocardiographic signs of tamponade may not be present, but small chamber size and elevated filling pressures should

suggest the correct diagnosis. The risk of pericardiocentesis may be reduced by the use of echocardiographic guidance and monitoring of needle aspiration, particularly for loculated or small effusions (637,638).

B. Cardiac Tamponade

Enlarging pericardial effusions may cause cardiac tamponade. Although the diagnosis of cardiac tamponade is based on established clinical criteria, an accurate and early diagnosis of tamponade can often be made using echocardiography. The elevated intrapericardial pressure in tamponade decreases the transmural pressure gradient between the pericardium and right atrium and ventricle and increases the distending force necessary for ventricular filling. Echocardiographic evidence of right atrial invagination (collapse) at onset of systole with the X descent and RV collapse in diastole are signs of hemodynamic compromise (242-245). Right atrial collapse is a sensitive sign of increased intrapericardial pressure; however, diastolic RV collapse is more specific for tamponade. Distension of the inferior vena cava that does not diminish on deep inspiration may also be seen and indicates an elevation of central venous pressure (246). The respiratory changes in mitral valve motion and ventricular dimensions were correlated with paradoxical pulse (247). Doppler flow studies have shown marked respiratory variation in transvalvular flow velocities, LV ejection, and LV isovolumetric times in patients with pericardial tamponade (248,249). These echocardiographic findings often precede the clinical signs of tamponade and may provide an opportunity for early therapeutic intervention.

C. Increased Pericardial Thickness

Increased echocardiographic density behind the posterior wall suggests pericardial thickening, but echocardiographic measurement of the precise pericardial thickness may be inaccurate (250). The causes of such thickening include fibrosis, calcification, and neoplasms, although it is usually not possible to differentiate the specific cause by echocardiography. Improved resolution by TEE provides a more accurate assessment of pericardial thickness, especially if fluid accumulation is also present (251).

D. Pericardial Tumors and Cysts

Tumor in the pericardium is usually metastatic from the breast or lung, but other types occasionally occur (252). The clinical findings are typically a sizable pericardial effusion, at times leading to tamponade, but tumor may also present as single or multiple epicardial tumor nodules, as effusive-constrictive pericarditis, or even as constrictive pericarditis (253). The effects of radiation therapy on the tumor may further affect the pericardium, resulting in inflammation, effusion, or fibrosis.

Pericardial cysts are rare and are usually located at the right costophrenic angle. They are readily visualized by echocardiography, and their cystic nature can be differentiated from that of a solid mass (254).

E. Constrictive Pericarditis

In constrictive pericarditis there are such prominent pathological and physiological changes that echocardiographic abnormalities are always present, and in most cases there are multiple abnormalities. However, no single echocardiographic sign is diagnostic of constrictive pericarditis. Some frequently seen findings are pericardial thickening, mild atrial enlargement with a normal-sized left ventricle, dilation of the vena cava, flattening of LV endocardial motion in mid and late diastole, various abnormalities of septal motion, and premature opening of the pulmonary valve.

The Doppler findings of respiratory variations in flow velocities across the atrioventricular valves as well as across the LV outflow and pulmonary venous flow are often highly characteristic and provide additional supportive evidence favoring constriction. A combination of echocardiographic (255-258) and Doppler flow data (232,259,639) in an appropriate clinical context usually indicates the diagnosis of pericardial constriction.

The pericardial thickness may also be assessed, often more accurately by computed tomography or MRI.

F. Congenital Absence of the Pericardium and Pericardial Disease After Open-Heart Surgery

In both total and partial absence of the pericardium, there are echocardiographic findings that are helpful in establishing the diagnosis (260,261). Pericardial disease occurs in patients after open-heart surgery; early postoperative bleeding may result in localized accumulation of blood clots, especially posteriorly. This situation is often difficult to diagnose except by TEE.

Recommendations for Echocardiography in Pericardial Disease

Class I

- 1. Patients with suspected pericardial disease, including effusion, constriction, or effusive-constrictive process.**
- 2. Patients with suspected bleeding in the pericardial space (eg, trauma, perforation).**
- 3. Follow-up study to evaluate recurrence of effusion or to diagnose early constriction. Repeat studies may be goal directed to answer a specific clinical question.**
- 4. Pericardial friction rub developing in acute myocardial infarction accompanied by symptoms such as persistent pain, hypotension, and nausea.**

Class IIa

- 1. Follow-up studies to detect early signs of tamponade in the presence of large or rapidly accumulating effusions. A goal-directed study may be appropriate.**
- 2. Echocardiographic guidance and monitoring of pericardiocentesis.**

Class IIb

- 1. Postsurgical pericardial disease, including postpericardiotomy syndrome, with potential for hemody-**

amic impairment.

- 2. In the presence of a strong clinical suspicion and non-diagnostic TTE, TEE assessment of pericardial thickness to support a diagnosis of constrictive pericarditis.**

Class III

- 1. Routine follow-up of small pericardial effusion in clinically stable patients.**
- 2. Follow-up studies in patients with cancer or other terminal illness for whom management would not be influenced by echocardiographic findings.**
- 3. Assessment of pericardial thickness in patients without clinical evidence of constrictive pericarditis.**
- 4. Pericardial friction rub in early uncomplicated myocardial infarction or in the early postoperative period after cardiac surgery.**

VII. CARDIAC MASSES AND TUMORS

TTE and TEE are accurate, high-resolution techniques for identifying masses within any of the four cardiac chambers. Masses that can be identified by echocardiographic techniques include primary tumors of the heart, such as atrial myxoma, metastatic disease from extracardiac primary sites, thrombi within any of the four chambers, and vegetations (infectious or noninfectious) on any of the four cardiac valves. Atrial myxoma is the most common primary tumor of the heart, and two-dimensional echocardiography is the primary method for its diagnosis.

Intracardiac masses should be suspected in the context of the clinical presentation. Examples of this include suspicion of vegetative lesions in patients with signs and symptoms that suggest infective endocarditis, as well as those with underlying connective tissue diseases. Intracardiac thrombi should be suspected in several clinical situations. These include patients who have sustained extensive anterior myocardial infarction and patients with atrial fibrillation in whom left atrial thrombi should be considered. The latter is especially pertinent if atrial fibrillation is present in association with rheumatic heart disease. Patients with signs and symptoms of peripheral embolic phenomenon (neurological events as well as non-neurological) should be suspected of having intracardiac masses if another embolic source is not identified.

In addition to detection and localization, echocardiographic techniques can play a role in stratifying the embolic risk of a cardiac mass. When seen in the left ventricle, sessile, laminar thrombi represent less of a potential embolic risk than do pedunculated and mobile thrombi.

Identifying patients who are appropriate candidates for echocardiographic screening for intracardiac masses represents a greater clinical dilemma than does the actual detection of a mass. An intracardiac mass should be suspected in patients with one or more embolic peripheral or neurological events or in those who have hemodynamic or auscultatory findings suggesting intermittent obstruction to intracardiac flow. Patients in whom a mass may be suspected because of

a predisposing condition include those with rheumatic heart disease, dilated cardiomyopathy, and atrial fibrillation, as well as following anteroapical myocardial infarction. Patients with malignancies known to have a high incidence of cardiovascular involvement may also be appropriate targets for screening. This includes individuals with hypernephroma as well as those with metastatic melanoma or metastatic disease with known primary tumors of intrathoracic organs. Clinical suspicion of disease entities such as endocarditis in which a mass is known to develop would also fall into the latter category. Special considerations referable to pediatric populations are found in the section on congenital heart disease.

Recommendations for Echocardiography in Patients With Cardiac Masses and Tumors

Class I

1. Evaluation of patients with clinical syndromes and events that suggest an underlying cardiac mass.
2. Evaluation of patients with underlying cardiac disease known to predispose to mass formation for whom a therapeutic decision regarding surgery or anticoagulation will depend on the results of echocardiography.
3. Follow-up or surveillance studies after surgical removal of masses known to have a high likelihood of recurrence (ie, myxoma).
4. Patients with known primary malignancies when echocardiographic surveillance for cardiac involvement is part of the disease staging process.

Class IIb

Screening persons with disease states likely to result in mass formation but for whom no clinical evidence for the mass exists.

Class III

Patients for whom the results of echocardiography will have no impact on diagnosis or clinical decision making.

VIII. DISEASES OF THE GREAT VESSELS

Echocardiography can be effectively used to visualize the entire thoracic aorta in most adults. Complete aortic visualization by combined transthoracic imaging (left and right parasternal, suprasternal, supraclavicular, and subcostal windows) frequently can be achieved. Biplane or multiplane TEE provides high-resolution images of the aortic root, the ascending aorta, and the descending thoracic and upper abdominal aorta. The only portion of the aorta that cannot be visualized is a small segment of the upper ascending portion adjacent to the tracheobronchial tree. Roman *et al.* have published nomograms for aortic root, diameters at the annulus, sinuses of Valsalva, sino-tubular junction and proximal ascending aorta for children and adults (842). Using transthoracic imaging, good visualization of the main pulmonary artery segment and the proximal right and left pul-

monary arteries can also be achieved in most children and the majority of adults. Visualization of the proximal portion of the innominate veins along with the superior vena cava can be achieved in nearly all patients with the use of the right supraclavicular fossa and suprasternal notch approaches. Similarly the proximal inferior vena cava and hepatic (subcostal) and pulmonary veins (apical and transesophageal) can be visualized in many patients.

A. Aortic Aneurysm

Aneurysms of the ascending aorta can be characterized by TTE. The aneurysm may be localized to one of the sinuses of Valsalva. With Doppler color flow imaging, rupture of an aneurysm in the sinus of Valsalva can be diagnosed, and its communication with the receiving cardiac chamber can be documented. Annuloaortic ectasia as well as localized atherosclerotic aneurysms of the ascending aorta can be well visualized with the use of the left as well as the right parasternal windows. Echocardiography is particularly well suited for the re-evaluation of patients with ascending aortic aneurysms (especially in patients with Marfan syndrome) to determine the increase in the size of the aneurysm. Descending thoracic aortic aneurysms are difficult to visualize with the transthoracic approach. TEE is particularly suited for complete characterization of these aneurysms (265).

B. Aortic Dissection

Acute aortic dissection is a life-threatening emergency, and an early and prompt diagnosis is mandatory for appropriate patient care (640). Although TTE may visualize the intimal flap in patients with aortic dissection, TEE has proved to be a far more sensitive diagnostic procedure (262,263). Since time is of the essence in the prompt diagnosis of dissection, only a brief transthoracic study should precede TEE. Multiplane TEE should be used for a comprehensive and accurate visualization of the thoracic aorta. In addition to establishing the diagnosis and extent of aortic dissection, echocardiography is useful in delineating any associated complications, such as pericardial effusion with or without tamponade and the degree and mechanism of aortic regurgitation and pleural effusion, as well as evaluating proximal coronary artery involvement and LV size and function. The ability to detect branch vessel involvement may be incomplete and on occasion require other imaging techniques. TEE studies can also determine the potential for aortic valve-sparing operations (641). TEE is also suited for postoperative evaluation of patients with aortic dissection (264).

C. Aortic Intramural Hematoma

Aortic intramural hematoma may be difficult to distinguish clinically from aortic dissection. The etiology may be spontaneous, often related to hypertension or iatrogenic trauma. All imaging techniques have lower sensitivity and specificity in detecting intramural hematoma compared with aortic dissection (642,643). The combined use of multiple imaging techniques may be required to establish the diagnosis. TEE

studies can demonstrate a localized or extensive abnormal degree of aortic wall thickness. Care must be taken to not confuse hematoma with areas of calcification or atheroma. Serial studies may demonstrate healing or progression to aortic dissection.

D. Aortic Rupture and Thoracic Aortic Degenerative Disease

TEE has provided diagnostic information in traumatic and other causes of aortic rupture. Although large tears are easily visualized, it is possible to overlook small tears, which may also have dire prognostic implications (266).

The use of TEE has made it possible to detect atheromatous debris, clots, and other lesions capable of producing embolic occlusions downstream. A grading system has been established to determine the amount of aortic atheroma seen on TEE studies. This correlates with the risk of embolic events, especially if surgery requiring aortic manipulation is being considered. TEE can also detect complications of atheromatous lesions (eg, ulceration or contained rupture) (308-313).

Recommendations for Echocardiography in Suspected Thoracic Aortic Disease

Class I

- 1. Aortic dissection, diagnosis, location, and extent.**
- 2. Aortic aneurysm.***
- 3. Aortic intramural hematoma.**
- 4. Aortic rupture.**
- 5. Aortic root dilation in Marfan syndrome or other connective tissue syndromes.***
- 6. Degenerative or traumatic aortic disease with clinical atheroembolism.**
- 7. Follow-up of aortic dissection, especially when complication or progression is suspected.**
- 8. First-degree relative of a patient with Marfan syndrome or other connective tissue disorder for which TTE is recommended* (see section XIIa).**

Class IIa

Follow-up of a patient with surgically repaired aortic dissection.*

*TTE should be the first choice in these situations, and TEE should only be used if the examination is incomplete or additional information is needed.

Note: TEE is the technique that is indicated in examination of the entire aorta, especially in emergency situations.

E. The Great Veins

Echocardiography is a useful technique for visualizing the superior vena cava and diagnosing various congenital and acquired abnormalities. A persistent left superior vena cava often can be imaged directly from the left supraclavicular fossa. Its connection, which is frequently to the coronary sinus, can be seen from a parasternal window as dilation of that structure. In some cases the connection to the coronary sinus can be better delineated with contrast echocardiography with injection of a contrast into a left arm vein. Other

abnormalities, such as vena caval thrombosis, can also be diagnosed with combined use of echocardiographic and Doppler techniques. The proximal inferior vena cava can be readily visualized in nearly all patients, and vena caval dilation and thrombosis or extension of tumors from the inferior vena cava to the right-heart chambers have been visualized. The hepatic veins, their size, connection, and flow dynamics can be characterized with combined use of two-dimensional and Doppler echocardiography. Although visualization of all four pulmonary veins is not feasible with the transthoracic approach in the majority of adult patients, TEE frequently permits clear visualization of the pulmonary vein connections although four pulmonary veins may not be visualized even with TEE. However, some pulmonary veins can usually be visualized by TTE and interrogated with Doppler as part of routine examination which will often provide additional hemodynamic information. Anomalous pulmonary veins can be missed.

IX. PULMONARY AND PULMONARY VASCULAR DISEASE

As a general rule, patients who have primary pulmonary disease are not ideal subjects for echocardiographic examinations because the hyperinflated lung is a poor conductor of ultrasound. Despite these technical limitations, TTE can still be very informative in some patients with primary lung disease. The usual precordial or parasternal windows are frequently unavailable in patients with hyperinflated lungs. However, in these same patients the diaphragms are frequently lower than normal. Thus, the subcostal or subxiphoid transducer position can offer a useful window for echocardiographic examinations. For those few patients in whom transthoracic and subcostal echocardiographic windows are totally unavailable, the transesophageal approach provides an unobstructed view of the heart in patients with lung disease. As a result, with use of one examining technique or another, almost all patients with primary lung disease can be studied echocardiographically.

If lung disease does not result in anatomic or physiological alteration of cardiac structure or function, the findings on the echocardiogram will be normal. Although a normal result on echocardiography does not indicate a diagnosis of lung disease, the differential diagnosis of cardiac versus pulmonary symptoms can often be made on the basis of the echocardiogram. When shortness of breath could be due to either a lung or heart condition, normal findings on the echocardiogram can be extremely helpful in such a differential diagnosis.

In those patients whose lung disease affects cardiac function, the echocardiogram can be of significant value. Pulmonary hypertension is one of the most common complications of primary lung disease, and echocardiography is helpful in evaluating its presence and severity. The right ventricle commonly dilates, which can be detected on both the M-mode and two-dimensional echocardiogram. With marked systolic or diastolic overload of the right ventricle, the shape or motion, or both, of the interventricular septum is distort-

ed, with abnormal diastolic bulging toward the left ventricle. In patients with increased pulmonary vascular resistance, the M-mode recording of the pulmonary valve shows a distinctive early to mid-systolic notch with loss of its A wave. A somewhat similar pulmonary artery velocity flow pattern is seen on the Doppler recording in such patients.

Any valvular regurgitation resulting from pulmonary hypertension can be detected with Doppler techniques. If adequate tricuspid and pulmonary valve regurgitation signals are obtained (as is the case in nearly 70% of all subjects), Doppler techniques can be used to accurately calculate RV systolic pressure (267,268). The tricuspid regurgitation signal is especially suited for saline contrast enhancement. The pulmonary artery diastolic pressure may also be estimated. This type of determination can be made in a high percentage of patients with significant pulmonary hypertension.

A. Pulmonary Thromboembolism

Echocardiography has aided a diagnosis of central pulmonary artery thromboembolic disorders, especially in patients with severe or massive pulmonary embolism. Echocardiography has a low sensitivity and specificity in diagnosing pulmonary emboli. In patients with larger pulmonary emboli, TEE may detect thrombus in the main portion and proximal branches of the pulmonary artery (644). The effects of severe embolization may be detected by the presence of pulmonary hypertension and RV dilatation and dysfunction.

Recommendations for Echocardiography in Pulmonary and Pulmonary Vascular Disease

Class I

1. Suspected pulmonary hypertension.
2. For distinguishing cardiac versus noncardiac etiology of dyspnea in patients in whom all clinical and laboratory clues are ambiguous.*
3. Follow-up of pulmonary artery pressures in patients with pulmonary hypertension to evaluate response to treatment.
4. Lung disease with clinical suspicion of cardiac involvement (suspected *cor pulmonale*).

Class IIa

1. Pulmonary emboli and suspected clots in the right atrium or ventricle or main pulmonary artery branches.*
2. Measurement of exercise pulmonary artery pressure.
3. Patients being considered for lung transplantation or other surgical procedure for advanced lung disease.*

Class III

1. Lung disease without any clinical suspicion of cardiac involvement.
2. Re-evaluation studies of RV function in patients with chronic obstructive lung disease without a change in clinical status.

*TEE is indicated when TTE studies are not diagnostic.

X. SYSTEMIC HYPERTENSION

Echocardiography is the noninvasive procedure of choice in evaluating the cardiac effects of systemic hypertension, the most common cause of LV hypertrophy and congestive heart failure in adults (270). M-mode and two-dimensional echocardiographic estimates of LV mass are more sensitive and specific than either the ECG or chest radiograph in diagnosing LV hypertrophy or concentric remodeling (271-274), and these estimates have been shown to correlate accurately with LV mass at necropsy (275,276). These techniques have been used to evaluate the relation of LV mass to rest and exercise blood pressure as well as multiple other physiological variables (277). Newer diagnostic techniques such as MRI are arguably more accurate but are often more expensive and less readily available (278). Assessment of hypertrophy is relevant because several cohorts have shown that the risks of cardiac morbidity and mortality are increased in hypertensive patients with electrocardiographic or echocardiographic criteria of LV hypertrophy and are independent of traditional coronary risk factors (272,279-281). Moreover, even in those patients without increased LV mass, concentric remodeling or an increased wall thickness relative to cavity size carries a poor prognosis (272). For these reasons, in patients with borderline hypertension a decision to initiate therapy may be based on the presence of hypertrophy or concentric remodeling. In borderline hypertensive patients without evidence of LV hypertrophy by ECG, a goal-directed echocardiogram to evaluate LV hypertrophy may be indicated.

Echocardiography can also be used to evaluate systolic and diastolic properties of the left ventricle, such as the speed and extent of contraction, end-systolic wall stress, and the rate of ventricular filling throughout diastole (275), and to evaluate related CAD and degenerative valve disease, especially in the elderly. Stress echocardiography is indicated in the diagnosis and assessment of the functional severity of concomitant CAD. The usefulness of echocardiography in an individual patient with hypertension without suspected concomitant heart disease depends on the clinical relevance of the assessment of LV mass or function in that patient. Thus, not every patient with hypertension should have resting LV function assessed (Class I), but if such an assessment is relevant, echocardiography is a well-documented and accepted method by which to achieve it.

The value of repeated studies in asymptomatic hypertensive patients with normal LV function is not clearly established. A decrease in LV mass in hypertensive patients through control of blood pressure or weight loss has been demonstrated by many regimens in several studies (282-285). While data suggest that LV hypertrophy regression improves LV filling (282), data linking treatment-associated reduction in echocardiographic LV mass and improved outcome have appeared (645). In view of the limited test-retest reliability of echocardiography in an individual patient, a relatively large reduction in LV mass appears to be necessary to unequivocally prove that true mass regression has taken place (272,286,646). Despite this, there may be a role for quantitation of LV mass and assessment of regression of LV

hypertrophy after adequate blood pressure control with anti-hypertensive therapy (282). More study is required to prove that regression of LV hypertrophy alters cardiac morbidity and mortality and that echocardiography is a cost-effective method for both detection of hypertrophy and follow-up evaluation of the large number of patients with hypertension. Until these studies are available, the monitoring of LV hypertrophy by echocardiography cannot be supported.

Recommendations for Echocardiography in Hypertension

Class I

1. When assessment of resting LV function, hypertrophy, or concentric remodeling is important in clinical decision making (see LV function).
2. Detection and assessment of functional significance of concomitant CAD by stress echocardiography (see coronary disease).
3. Follow-up assessment of LV size and function in patients with LV dysfunction when there has been a documented change in clinical status or to guide medical therapy.

Class IIa

1. Identification of LV diastolic filling abnormalities with or without systolic abnormalities.
2. Assessment of LV hypertrophy in a patient with borderline hypertension without LV hypertrophy on ECG to guide decision making regarding initiation of

therapy. A limited goal-directed echocardiogram may be indicated for this purpose.

Class IIb

Risk stratification for prognosis by determination of LV performance.

Class III

1. Re-evaluation to guide antihypertensive therapy based on LV mass regression.
2. Re-evaluation in asymptomatic patients to assess LV function.

XI. NEUROLOGICAL DISEASE AND OTHER CARDIOEMBOLIC DISEASE

Acute interruption of blood flow to the cerebral vasculature or a peripheral artery results in an identifiable clinical syndrome such as transient ischemic attack, cerebrovascular accident, acute limb ischemia, or mesenteric or renal artery insufficiency. The above clinical scenarios can be the result of intrinsic local vascular disease, atheromatous emboli from proximal vessels, or emboli of cardiac origin. Depending on the target organ, the age of the patient, and the likelihood of underlying primary vascular disease, the prevalence of a cardioembolic etiology is highly variable. Most studies have documented that a substantial proportion of patients with embolic events, even those with vascular disease, also have a potential cardiac source of embolus (Table 10). Proving

Table 10. Prevalence of Cardiac Abnormalities in Patients With and Without Presumed Embolic Events Derived From General Surveillance Studies

Author (Ref)	Event Patients*			Range (%)†	Control Patients*			Range (%)†
	n=Population	(n)	%		n=Population	(n)	%	
No potential source of embolus	1530	772	50.5	32-85	—	—	—	—
Any potential source of embolus	1530	758	49.5	15-68	—	—	—	—
Left atrial thrombus	1153	98	8.5	3-17	877	28	3.2	2-81
Spontaneous contrast	1081	187	17.3	11-23	1105	63	5.7	5-6
Patent foramen ovale	1292	247	19.1	8-45	1043	87	8.3	2-23
Atrial septal aneurysm	1131	150	13.3	3-28	1204	85	7.1	3-12
Aortic atheroma	348	49	14.1	4-44	n/a	—	—	—
Mitral valve prolapse	1131	57	5.0	2-9	927	83	8.9	5-9

n/a indicates reliable extraction data not available.

*Control patient population derived from studies in which lesion was specifically sought. The control subjects were not necessarily age and risk factor matched.

†Range refers to minimum and maximum prevalence of abnormalities reported for the cited references.

Lesions not tabulated above, such as vegetations, myxoma, other tumors, mitral valve strands, etc, were too few in number and in too few studies to derive meaningful conclusions.

Table 11. Prevalence of Echocardiographic Abnormalities Based on Cryptogenic Versus Noncryptogenic Embolic Event

	Cryptogenic (n=308)			Noncryptogenic (n=263)		
	Total*	Echo+	Echo %	Total*	Echo+	Echo %
PFO	308	100	32.5	263	64	24.3
SC	104	17	16.3	74	10	13.5
ASA	168	38	22.6	110	14	12.7

ASA indicates atrial septal aneurysm; PFO, patent foramen ovale; SC, spontaneous contrast; .

*Total indicates number of patients in each subgroup for whom each entity was specifically tabulated in the referenced studies. Not all studies tabulated data for each entity.

Data from references 296, 303, 307, 314, 317, 318.

cause and effect between the clinical event and a potential embolic source has been more elusive for many entities. Exceptions include the obvious link between embolic events and bacterial endocarditis and embolic phenomena occurring in patients with prosthetic valves.

The level of evidence for proving a relation between potential cardiac sources of embolus and neurological events is relatively low. Virtually all studies published to date rely on data from nonrandomized trials and frequently nonconsecutive patients compared with either historical or concurrent control populations. No large-scale prospective studies are available from which a definite cause and effect between cardiac source of embolus and subsequent neurological events can be proved. The available data are all concordant, however, in suggesting a high prevalence of potential cardiac sources of embolus in subjects with peripheral or neurological embolic events.

Many studies have evaluated the frequency with which a potential cardioembolic source of an acute neurological syndrome is found on echocardiography. The definition of cardioembolic events can be characterized either from the reference of a potential source of embolus or the reference of the end-organ event. Both definitions have been used in the literature, and sufficient exceptions to any given stratification scheme occur. The Cerebral Embolism Task Force defined a cardioembolic neurological event as “presence of a potential cardioembolic source in the absence of cerebrovascular disease in a patient with nonlacunar stroke” (287,647). This definition obviously implies cause and effect when a potential cardiac source of embolus is noted in an individual with a neurological event. Historically several types of neurological events have been thought to be more likely embolic than due to intrinsic cerebrovascular disease. The neurological findings traditionally thought to suggest an embolic source are sudden onset in a previously asymptomatic individual, middle or anterior circulation defects, and multiple events in peripheral territories. Conversely, classic lacunar strokes or hemorrhagic strokes have been thought more likely due to intrinsic cerebrovascular disease. Recent data have called into question the classification of the latter. At this time there are no highly specific types of neurological events that should exclude the possibility of a cardioembolic source. Clinical studies have suggested that up to 20% of acute neurological events may be attributable to a cardioembolic source (288-302), with an additional 40% classified as cryp-

togenic. Recently it has been suggested that a substantial proportion of the latter may also be attributed to a cardiac etiology (Table 11). This prevalence is obviously age dependent, with some studies suggesting a prevalence of cardioembolic disease greater than 50% for younger persons (303,648-650). As such, the use of echocardiographic techniques in patients with acute embolic events should be placed in the context of the clinical presentation and the likelihood of other (ie, intrinsic cerebrovascular) responsible pathology.

Additionally, a decision to use echocardiographic screening for a potential source must take into account the presence of underlying cardiac disease. Clearly the presence of rheumatic heart disease or atrial fibrillation predisposes a patient to atrial thrombus formation and the likelihood of an embolic event. Other cardiac diseases that may predispose to thrombus formation and subsequent embolization include cardiomyopathy and anterior myocardial infarction with aneurysm formation. Data to support a link between cardioembolic disease and entities such as atrial septal aneurysm, valvular strands, and mitral annular calcification are less robust. Several studies have demonstrated that the prevalence of potential embolic sources is greater in persons with clinically apparent organic heart disease than in those without clinically apparent heart disease (Table 12). This relatively high prevalence of clinically unsuspected cardiovascular disease (presumably with embolic potential) suggests that echocardiographic screening may be applicable to patients other than those with clinically suspected disease.

Two-dimensional echocardiography is the only technique that is easily applied and widely available for evaluation of a potential cardioembolic source. Intravenous injection of agitated saline can be used to detect right-to-left shunting across a patent foramen ovale. Examinations can be performed

Table 12. Prevalence of Echocardiographic Abnormalities Based on Clinically Apparent Organic Heart Disease

	Potential Source of Embolus		
	(n)	(n)	%
All patients	370	164	44
Organic heart disease*	85	58	68
No organic heart disease	186	67	36

*Organic heart disease is defined variably as significant valvular or myocardial disease or evidence of reduced left ventricular function.

Data from references 289, 290, 296.

either from a transthoracic or transesophageal approach. Comparative studies between the two approaches have suggested a higher yield for potential cardiac source of embolus when TEE is used (299,300). Table 13 outlines the relation between TEE and TTE for detection of potential cardioembolic sources. Entities such as mitral stenosis, cardiomyopathy, and LV mural thrombus are equally well identified with either technique, and once identified by TTE, the additive cost, inconvenience, and risk of TEE may not be warranted. Conversely, TEE is uniquely suited for detection of left atrial spontaneous contrast (302,304,305), left atrial thrombi, septal aneurysm (306,307), and atheroma of the ascending aorta and aortic arch (308-313) as well as several other more recently described anomalies. Atrial septal aneurysm (306,307) and right-to-left shunting through a patent foramen ovale can generally be detected with either technique.

In a similar fashion, the issues of age and presence or absence of atrial fibrillation have been addressed in several of the published series. While in each series the prevalence of potential cardiovascular abnormalities is greater in older patients and in those with atrial fibrillation, a clinically pertinent proportion of patients without either risk factor (age or atrial fibrillation) will have cardiovascular pathology that places them at risk for arterial embolization. Recent data have suggested a role for TEE in stratifying risk of thrombus formation in patients with atrial fibrillation (651-653).

Traditionally, it has been assumed that there is an inverse relation between age and the prevalence of potential cardiac sources of embolus in patients with neurological events. Several studies, however, have clearly demonstrated an almost equal prevalence of potential cardiac sources of embolus in older patients when compared with younger cohorts. Younger patients typically have a higher likelihood that the potential cardiac source of embolus is the only identifiable abnormality, whereas older patients are more likely to have identifiable concurrent cerebrovascular disease. The definition of “younger” and “older” patients has been variable. It should be recognized that there is a gradation of age and prevalence of potential cardiac source of embolus rather than distinct age cutoffs. From a standpoint of data analysis, most studies have assumed an age break at approximately 45 years. Clearly there is a range of likelihood of finding potential cardioembolic sources, with the likelihood of an exclusive cardioembolic source being greater in younger patients and progressively less in older patients.

Few published series have investigated recurrence rates of neurological events in relation to specific cardiovascular abnormalities. It appears that lesions such as mitral stenosis, left atrial spontaneous contrast, patent foramen ovale with right-to-left shunting, and atrial septal aneurysm represent relatively higher risk entities with respect to recurrent cerebrovascular events (654,655). Presumably more aggressive therapy should be directed at these patients.

In addition to the presence or absence of a specific disease that impacts the likelihood of an embolic event, the nature of the occlusive event also has implications for the necessity of further evaluation. Clearly, younger persons with cerebrovascular events are more likely to have had an event with an

Table 13. Transthoracic Versus Transesophageal Echocardiography for Detection of Potential Cardioembolic Source

Diagnosis by TTE*	Diagnosis by TEE (primarily or alone)
Mitral stenosis	Left atrial thrombus
Dilated cardiomyopathy	Left atrial spontaneous contrast
Left ventricular aneurysm	Atrial septal aneurysm
Left ventricular thrombus	Patent foramen ovale
Mitral valve prolapse	Aortic atheroma
Vegetation	
Atrial septal defect	

TEE indicates transesophageal echocardiography; TTE, transthoracic echocardiography.

*TTE is sufficient; TEE may be additive but not essential. “TTE sufficient” identifies disease entities for which TTE is sufficient to establish a diagnosis and for which TEE is unlikely to provide additional information. When detected with TTE, further evaluation by TEE is not necessary in all patients. “TEE additive” identifies entities for which documented incremental diagnostic yield can be obtained by performing TEE after negative TTE or entities for which the likelihood of unique TEE-identified abnormalities is high enough to warrant TEE even after adequate TTE.

These categories assume that high-quality TTE is feasible and has been conducted to evaluate all potential cardiac sources of embolus. When adequate TTE is not feasible, TEE is essential.

embolic basis than are elderly patients with intrinsic cerebrovascular disease. Likewise, in persons with events in multiple cerebrovascular territories it is more likely for the event to be embolic. Additionally, occlusion of a large peripheral vessel such as a femoral or renal artery is far more likely to represent a cardioembolic event. The heart represents the only source for a mass of sufficient size to cause total occlusion of an otherwise normal large-caliber vessel. In individuals with an abrupt occlusion of a large vessel, cardioembolic disease should be suspected. Several recently published studies have evaluated the link between specific entities of embolic potential and neurological events. These include atrial septal aneurysm (303,306,307), patent foramen ovale (303,314-317), left atrial spontaneous contrast (302,304, 305), and aortic atheroma (308-313). In each case a statistically significant increase in the prevalence of these entities has been demonstrated in individuals with neurological events. Tables 14 through 17 outline results of studies that have evaluated these phenomena. In each case there is a greater likelihood of finding one or more of these entities in patients with neurological events than in control populations without neurological events.

Recommendations for Echocardiography in Patients With Neurological Events or Other Vascular Occlusive Events

Class I

1. Patients of any age with abrupt occlusion of a major peripheral or visceral artery.
2. Younger patients (typically less than 45 years) with cerebrovascular events.
3. Older patients (typically more than 45 years) with neurological events without evidence of cerebrovascular disease or other obvious cause.
4. Patients for whom a clinical therapeutic decision (eg, anticoagulation) will depend on the results of echocardiography.

Table 14. Prevalence of Patent Foramen Ovale in Patients With Embolic Events

	(n)	PFO+	PFO%
Control	543	56	10.3
CVA/TIA	526	163	30.9
Known etiology	153	39	25.5
Cryptogenic	204	97	47.5

CVA/TIA indicates patients with documented cerebrovascular accident or transient ischemic attack; PFO indicates patent foramen ovale.

Known etiology refers to patients for whom an obvious primary neurological, cerebrovascular, or other etiology was present in a location adequate to explain the event. Cryptogenic refers to patients for whom a known etiology was not present.

Data from references 303, 307, 314, 317, 318.

Class IIa

Patients with suspicion of embolic disease and with cerebrovascular disease of questionable significance.

Class IIb

Patients with a neurological event and intrinsic cerebrovascular disease of a nature sufficient to cause the clinical event.

Class III

Patients for whom the results of echocardiography will not impact a decision to institute anticoagulant therapy or otherwise alter the approach to diagnosis or treatment.

XII. ARRHYTHMIAS AND PALPITATIONS

Arrhythmias can occur as primary electrophysiological abnormalities or as a complication of or in association with structural heart disease. The spectrum of heart disease associated with arrhythmias is broad, including congenital abnormalities as well as acquired diseases of the myocardium, valves, pericardium, and coronary arteries. While some arrhythmias may be life-threatening or carry significant morbidity, others are considered benign.

In the setting of arrhythmias, the utility of echocardiography lies primarily in the identification of associated heart disease, the knowledge of which will influence treatment of the arrhythmia or provide prognostic information. In this regard, echocardiographic examination is frequently performed to assess patients with atrial fibrillation or flutter, re-entrant tachycardias, ventricular tachycardia, or ventricular fibrillation. Echocardiography detects an underlying cardiac disorder in approximately 10% of patients with atrial fibrillation who have no other clinically suspected cardiac disease (319,320) and in 60% of those with equivocal indicators of other heart disease (319). Ventricular arrhythmias of RV origin should alert the physician to a diagnosis of RV abnormalities, including RV dysplasia (321-323), while ventricular tachycardias of LV origin are frequently associated with reduced LV function. Evaluation of LV function is important when antiarrhythmic drugs are used, since the proarrhythmic

Table 15. Prevalence of Aortic Atheroma in Patients With Prior Cerebrovascular Accident or Transient Ischemic Attack

	(n)	Atheroma	%
Control	574	23	4
CVA	677	139	20.5
Known etiology	217	31	14.3
Cryptogenic	123	29	23.6
Mobile Atheroma			
	(n)	Mobile	%
Control	324	1	0.3
CVA/TIA	427	29	6.8

CVA/TIA indicates patients with documented cerebrovascular accident or transient ischemic attack.

Known etiology refers to patients for whom an obvious primary neurological, cerebrovascular, or other etiology was present in a location adequate to explain the event. Cryptogenic refers to patients for whom a known etiology was not present.

Data from references 309 through 313.

effect of some antiarrhythmic drugs increases markedly with decreased LV systolic function.

A large group of patients have benign arrhythmias such as atrial or ventricular premature beats. Although, in general, echocardiographic evaluation should be reserved for those for whom there is a clinical suspicion of structural heart disease, there may be a therapeutic role for cardiac ultrasound by reassuring the anxious patient that the heart is structurally normal. Unless there are other recommendations for testing, echocardiography need not be performed in a subject with palpitation for which an arrhythmic basis has been ruled out.

Although echocardiography has provided useful insights into the effects of arrhythmias on cardiac function (324), there is no recommendation for repeated clinical testing for this purpose unless there has been a change in clinical status or the result might impact a therapeutic decision. One situation where treatment might be impacted is in the selection of appropriate settings for DDD pacing where Doppler studies can be used to determine stroke volume at various settings to provide optimum cardiac output (325). However, it appears that for most patients, similar settings provide optimum output. Thus, this application of echocardiography might be limited to those in whom the usual settings do not appear to convey favorable hemodynamics. Similarly, while there have been reports that echocardiography may assist in identification of an arrhythmia when a surface ECG is nondiagnostic (326-328) or allow accurate localization of the bypass tract in

Table 16. Prevalence of Atrial Septal Aneurysm in Patients With Prior Embolic Events

	Total (n)	ASA Present	ASA (%)
Control	1213	53	4.3
All events	1635	213	13
Cryptogenic	168	38	22.6

ASA indicates atrial septal aneurysm.

Events refers to cerebrovascular accidents or transient ischemic attacks plus peripheral embolization. Cryptogenic refers to patients for whom a known etiology was not present.

Table 17. Prevalence of Neurological Events in Patients With and Without Spontaneous Contrast

	Left Atrial Clot			
	(n)	(%)	Event	%
All patients	713	90 (12.6)	87	12.2
Spontaneous contrast present	311	79 (25)	64	20.5
No spontaneous contrast	402	11 (3)	23	5.7

Data from references 302, 304, 305.

patients with Wolff-Parkinson-White syndrome (329), cardiac ultrasound is rarely used for these purposes.

In this era of interventional electrophysiology, an expanded role for echocardiography has developed. Thus, TEE (655,657) and intracardiac ultrasound (658,659) have been reported to be helpful during radiofrequency ablative procedures, particularly when transseptal catheterization is required. Early studies also proposed routine postprocedural evaluation of patients undergoing ablation. However, the yield has been low enough in laboratories with established ablative programs that the test is no longer recommended in uncomplicated cases (660).

The Maze procedure for atrial fibrillation is generally performed with intraoperative transesophageal monitoring (661) and postoperative TTE and may be used to monitor the return of atrial mechanical function in this setting (662).

For a discussion of the role of echocardiography in the assessment of children with arrhythmias, see the corresponding section in section XV, "Echocardiography in the Pediatric Patient."

Recommendations for Echocardiography in Patients With Arrhythmias and Palpitations

Class I

1. Arrhythmias with clinical suspicion of structural heart disease.
2. Arrhythmia in a patient with a family history of a genetically transmitted cardiac lesion associated with arrhythmia such as tuberous sclerosis, rhabdomyoma, or hypertrophic cardiomyopathy.
3. Evaluation of patients as a component of the workup before electrophysiological ablative procedures.

Class IIa

1. Arrhythmia requiring treatment.
2. TEE or intracardiac ultrasound guidance of radiofrequency ablative procedures.

Class IIb

1. Arrhythmias commonly associated with, but without clinical evidence of, heart disease.
2. Evaluation of patients who have undergone radiofrequency ablation in the absence of complications. (In centers with established ablation programs, a postprocedural echocardiogram may not be necessary.)
3. Postoperative evaluation of patients undergoing the Maze procedure to monitor atrial function.

Class III

1. Palpitation without corresponding arrhythmia or other cardiac signs or symptoms.
2. Isolated premature ventricular contractions for which there is no clinical suspicion of heart disease.

A. Cardioversion of Patients With Atrial Fibrillation

Studies have supported a role for echocardiography in patients with atrial fibrillation undergoing cardioversion. Echocardiography may help identify subjects who are most likely to undergo cardioversion successfully and maintain sinus rhythm after conversion. LV dysfunction argues against long-term success. The relation between atrial size and successful conversion is more controversial (330-336,663) (Table 18). Doppler indices of atrial appendage function measured by TEE have been reported to predict both restoration and long-term maintenance of sinus rhythm after cardioversion (664-667). However, study results are somewhat inconsistent, and TEE assessment before cardioversion is not indicated for this purpose.

The issue of performing TEE before elective cardioversion from atrial fibrillation has recently been addressed in patients with atrial fibrillation of more than 48 hours' duration (337,664,668-670). Historical data suggest a 5% to 7% incidence rate of cardioembolic events associated with electrical cardioversion from atrial fibrillation in patients who have not undergone anticoagulation. The presumed mechanism is dislodgment of previously existing atrial thrombi after cardioversion to atrial fibrillation. It has been demonstrated that transient left atrial mechanical dysfunction and spontaneous echocardiography contrast may occur after cardioversion to sinus rhythm, potentially explaining the mechanism of delayed cardioembolic events (339).

In subjects undergoing cardioversion it has been reported that exclusion of intra-atrial thrombus with TEE can obviate the need for extended precardioversion anticoagulation (337,670-672). In light of encouraging results in small series, a large multicenter randomized trial (ACUTE) was initiated in which TEE-guided cardioversion was compared to cardioversion after 4 weeks of anticoagulation. The rate of embolism was low and similar between both groups, 5 (0.8%) of 619 in the TEE group and 3 (0.5%) of 603 in the conventional therapy group after follow-up of 8 weeks. Although there was a significant reduction in composite major and minor bleeding events in the TEE-guided arm, the hypothesized reduction in embolic events in the TEE arm did not occur, and the study was terminated prematurely (673).

Table 18. Echocardiographic Predictors for Outcome of Elective Cardioversion

Author, y (Ref)	n	Study Design	Patient Group	Method	Parameters Evaluated	Results	Comments
Danias, 1998 (663)	356	Retrospective	AF more than 72 h outcome equals spontaneous conversion	2-D, M-mode	LA diam, LV Fxn	LA size not predictive, NILV fxn more common in spontaneous converters but not independently predictive	
Dittrich, 1989 (330)	85	Retrospective	Variety of etiologies and coexisting cardiac problems Most pts in AF greater than 5 y	M-mode and 2D	<i>M-mode</i> LV diam, LVH (greater than 11 mm) LVEDD, LVFS% <i>2D</i> LA long axis, RA long axis, LA area (E _s), RA area (E _s)	<i>Initial success</i> No echo parameter predictive <i>1-mo maintenance of NSR</i> LA diam (M-mode) and RA long axis not predictive LA long axis and LAA larger in pts staying in NSR (but great overlap) <i>6-mo maintenance of NSR</i> No echo parameter predictive	
Flugelman, 1984 (336)	40	Retrospective	All had chronic AF, postoperative mitral valvotomy or MVR for rheumatic MS	2D	LA diam LVFS% LV diam RV diam	<i>Success</i> NSR at 3 mo LA size larger in those with failure (<i>P</i> = 0.03)	No breakdown separating pts who initially failed conversion vs those who reverted within 3 mo. No useful cutoff was suggested or evaluated.
Dethy, 1988 (331)	50	Prospective	All maintained NSR greater than 24 h	2D, M-mode	<i>M-mode</i> LA diam <i>Doppler parameters</i> based on E and A waves	<i>Maintenance of NSR at 6 mo</i> LA diam greater than 45 Sens = 59% Spec = 44% PPV 66 LA diam greater than 50 Sens = 38% Spec = 6% PPV = 63% <i>Success vs failure</i> LA size ns A wave at 24 h <i>P</i> = 0.12 Increase in A wave from 4-24 h, <i>P</i> = 0.003	
Henry, 1976 (333)	37	Retrospective	Pts had MV diagnosis or ASH. Many had multiple attempts at cardioversion. No pt had LA less than 45.	M-mode	LA diam	Only 25% maintained NSR to 6 mo 10% of pts with LA diam greater than 5 had NSR at 6 mo	This study has been widely cited to support the concept that LA size of 45 argues against successful cardioversion. However, no pt had LA less than 45. No information on initial success. Group not typical of usual mix of pts with AF.
Ewy, 1980 (334)	74	Retrospective	Rheumatic heart disease, idiopathic AF	M-mode	LA diam	No pt with LA greater than 6 cm and rheumatic heart disease of greater than 5 in pts with idiopathic AF successfully converted, but there was no significant difference in LA size when entire group of successful vs failed cardioversion was compared.	Considerable overlap between groups and small number in successfully cardioverted group.
Hoglund, 1985 (332)	26	Prospective	Rheumatic heart disease, idiopathic AF	M-mode	LA diam	<i>Success</i> Maintenance of NSR for 1 mo LA diam smaller in pts with success vs failure <i>P</i> = 0.001. No pt with recurrence had LA less than 4.5. Success in 1 pt with LA greater than 4.5	Small number, but 4.5 appears to be a good cutoff in this pt group.
Halpern, 1980 (335)	21	Prospective	Mixed group re coexisting heart disease. Cardioversion attempted only with procainamide.	M-mode	LA diam	<i>Initial success</i> LA smaller in converters <i>P</i> less than 0.005 All converters had LA less than 4 Only 1 nonconverter had LA less than 4	Small number

AF indicates atrial fibrillation; ASH, asymmetric septal hypertrophy; E_s, end systole; LA, left atrium; LAA, left atrial appendage; LV, left ventricular; diam, diameter; LVEDD, left ventricular end-diastolic diameter; LVFS, left ventricular fraction shortening; LVH, left ventricular hypertrophy; RA, right atrium; NSR, normal sinus rhythm; MVR, mitral valve replacement, MS, mitral stenosis; Pts, patients; RV, right ventricular; Sens, sensitivity; Spec, specificity; PPV, positive predictive value; 2D, two-dimensional.

Because there were comparable embolic event rates in both arms, it appears that TEE is an alternative to but is not superior to conventional extended anticoagulation. Should a TEE-guided approach be used, it is essential that patients be given anticoagulation from the time of study until the time of cardioversion and subsequently until atrial mechanical function has returned after conversion. In a group of patients not routinely given anticoagulation in the pericardioversion period, there was a 2.4% incidence of embolic events despite the absence of thrombus at the time of precardioversion TEE (338). For individuals in whom anticoagulation confers more than a minimal risk, further stratification into subgroups at high and low risk for embolic events with TEE may be warranted.

There is less information available about the risk of thrombus and pericardioversion embolism in patients with atrial fibrillation of recent onset. The most recent American College of Chest Physicians Consensus Conference on Antithrombotic Therapy (674) makes no specific recommendation regarding anticoagulation in this setting, citing the lack of sufficient data. However, the report acknowledges that it is common practice not to provide anticoagulation to patients with atrial fibrillation of less than 48 hours' duration before cardioversion. The ACC/AHA/ESC guidelines (675) have listed cardioversion without TEE guidance during the first 48 hours of atrial fibrillation as a class IIb recommendation and stated that anticoagulation before and after cardioversion is optional, depending on assessment of risk. This assumes that thrombus formation does not occur in this time interval. However, a recent TEE study has reported left atrial appendage thrombus in 14% of patients with acute-onset atrial fibrillation (341). These results suggest that anticoagulation and TEE in patients undergoing cardioversion of atrial fibrillation should not differentiate between those with recent versus chronic fibrillation. However, until studies show that there is an increased danger of systemic embolization, recommendations for cardioversion without anticoagulation when atrial fibrillation occurs within 48 hours in otherwise low-risk patients will remain unchanged.

The prevalence of thrombus in patients with atrial flutter appears to be lower than that for those with atrial fibrillation or fibrillation/flutter (342). However, no studies have addressed the role of pericardioversion anticoagulation and TEE in these patients.

Recommendations for Echocardiography Before Cardioversion

Class I

1. Patients requiring urgent (not emergent) cardioversion for whom extended precardioversion anticoagulation is not desirable.*
2. Patients who have had prior cardioembolic events thought to be related to intra-atrial thrombus.*
3. Patients for whom anticoagulation is contraindicated and for whom a decision about cardioversion will be influenced by TEE results.*
4. Patients for whom intra-atrial thrombus has been

demonstrated in previous TEE.*

5. Evaluation of patients for whom a decision concerning cardioversion will be impacted by knowledge of prognostic factors (such as LV function or coexistent mitral valve disease).

Class IIa

Patients with atrial fibrillation of less than 48 hours' duration and other heart disease.*

Class IIb

1. Patients with atrial fibrillation of less than 48 hours' duration and no other heart disease.*
2. Patients with mitral valve disease or hypertrophic cardiomyopathy who have been on long-term anticoagulation at therapeutic levels before cardioversion unless there are other reasons for anticoagulation (eg, prior embolus or known thrombus on previous TEE).*
3. Patients undergoing cardioversion from atrial flutter.*

Class III

1. Patients requiring emergent cardioversion.
2. Patients who have been on long-term anticoagulation at therapeutic levels and who do not have mitral valve disease or hypertrophic cardiomyopathy before cardioversion unless there are other reasons for anticoagulation (eg, prior embolus or known thrombus on previous TEE).*
3. Precardioversion evaluation of patients who have undergone previous TEE and with no clinical suspicion of a significant interval change.

*TEE only.

B. Syncope

Syncope is a common clinical problem with multiple causes. The role of echocardiography in the diagnostic evaluation of patients with syncope relates to its ability to diagnose and quantitate obstructive lesions and identify abnormalities such as LV dysfunction that provide a substrate for malignant arrhythmias. The abnormality identified may be solely causative or one of several combining to cause syncope.

Whether the use of echocardiography can be justified as a routine component of a syncopal workup is controversial. One retrospective study reported that echocardiography did not identify an unsuspected cause in patients in whom history, physical examination, and ECG failed to indicate a cause (676). However, in a prospective study of 155 patients with syncope unexplained by history, physical examination, or ECG, routine echocardiography found no abnormalities that established the cause of the syncope (677).

Recommendations for Echocardiography in the Patient With Syncope

Class I

1. Syncope in a patient with clinically suspected heart disease.

2. Periexertional syncope.

Class IIa

Syncope in a patient in a high-risk occupation (eg, pilot).

Class IIb

Syncope of occult etiology with no findings of heart disease on history or physical examination.

Class III

1. **Recurrent syncope in a patient in whom previous echocardiographic or other testing demonstrated a cause of syncope.**
2. **Syncope in a patient for whom there is no clinical suspicion of heart disease.**
3. **Classic neurogenic syncope.**

XIIa. SCREENING

If screening asymptomatic individuals for cardiac abnormalities is to be recommended, several criteria must be met. First, the test used must be accurate, free of complications, widely available, and inexpensive. Second, the abnormalities sought should occur with reasonable frequency in the population to be screened and, if present, should convey risk to the affected individual. Third, recognition of the abnormality should ideally lead to initiation of a management plan that will favorably affect long-term outcome or prevent initiation of a potentially detrimental plan. At a minimum, identification of the disease should provide prognostic information that will influence the patient's life decisions.

As a testing modality, echocardiography is a safe, widely available, and accurate method for identifying most structural heart disease. Its cost varies, depending in part on which components are included in the examination. In general, its cost is higher than that of a physical examination, ECG, or a conventional stress test but lower than that of cardiac imaging with computerized axial tomography, MRI, or nuclear methods. Thus, echocardiography has several properties that promote its use as a screening tool. However, of the many conditions that echocardiography is capable of identifying, few meet the criteria enumerated above.

Among those that meet these criteria are heritable diseases of the heart and great vessels when the target group for screening is the family of an affected individual. The most common diseases that fall into this category are cardiomyopathy and Marfan syndrome.

Recent advances in molecular genetics have identified a familial basis for many forms of cardiomyopathy. Although genetic testing will likely become more widely available as a screening tool in the future, echocardiography currently plays a pivotal role in the process. Genetic testing and echocardiography will likely always play complementary roles in screening, the former documenting the genetic substrate for the disease and the latter defining its manifestations and progression. Three forms of myopathy in which there is a defined role for echocardiographic screening are hyper-

trophic cardiomyopathy, dilated cardiomyopathy, and arrhythmogenic RV dysplasia.

The inheritance pattern of hypertrophic cardiomyopathy is variable, with familial occurrence reported in 56% and sporadic occurrence in 44% (346). In a large-scale screening study (346), the proportion of first-degree relatives of probands with hypertrophic cardiomyopathy also found to have the disease was 22%. Of relevance to the screening process is the fact that in an affected individual, the hypertrophy may develop *de novo* or increase dramatically during childhood and adolescence (347). These observations provide justification for more than one screening examination of subjects in this age group. In contrast, one study has suggested that hypertrophy does not progress in adulthood (348). However, emerging data demonstrating the genetic heterogeneity and variable expression of hypertrophic cardiomyopathy (678) raise the possibility that patterns of disease progression may be similarly variable. Thus, repeat screening of adults may also be indicated in some cases.

Although published series are still small, it has been reported that up to two thirds of patients initially diagnosed as having idiopathic dilated cardiomyopathy have familial forms of the disease, which are typically inherited in an autosomal dominant pattern (351,679,680). Clinical profiles of affected families suggest that 29% of asymptomatic relatives have echocardiographic abnormalities and roughly one third of these will go on to develop full-blown cardiomyopathy (681). These observations support echocardiographic screening of first-degree relatives of patients with apparent idiopathic dilated cardiomyopathy and more extended screening of kindreds with established familial cardiomyopathy. Because the age of onset varies considerably within and between families, more than a single screening echocardiogram may be appropriate (682).

A familial basis has been reported in 30% to 50% of patients with arrhythmogenic RV dysplasia (683). Although genes responsible for the disease have been mapped to several chromosomal loci, no genetic test for the disease is currently available. Arrhythmogenic RV dysplasia is most commonly transmitted as an autosomal dominant with variable expression and penetrance. A recessive form associated with epidermal abnormalities has also been reported (684). These observations support electrocardiographic and echocardiographic screening of first-degree relatives of those with the disease. Because the disease is relatively uncommon and the echocardiographic manifestations may be subtle, it is important that screening be performed by those with expertise in the assessment of the right ventricle.

Marfan syndrome is transmitted as an autosomal dominant with spontaneous mutation occurring in up to 30% of subjects. Despite advances in genetic testing for the disease (685), the diagnosis is still made using a multidisciplinary set of major and minor diagnostic criteria that include abnormalities of the skeleton, eye, cardiovascular system, pulmonary system, skin, and central nervous system and that take into consideration the family history (Table 19). Because the primary method of diagnosing cardiovascular

Table 19. Diagnostic Criteria for Marfan Syndrome: 1995 Ghent Nosology*

To make an initial diagnosis, at least two of the following major criteria must be met:

1. Aortic dilation (by comparison with nomograms accounting for age and body size).
2. Ectopia lentis (detected by slit lamp exam with dilated pupils).
3. Skeletal abnormalities, four of the following:
 - Positive thumb and wrist signs
 - $>20^\circ$ scoliosis
 - Pectus carinatum or pectus excavatum requiring surgery
 - Pes planus (demand displacement of medial malleolus)
 - Abnormal upper/lower segment ratio
 - Arm span greater than 105% of height
 - Typical facies (malar hypoplasia, deep-set eyes, retrognathia)
4. Dural ectasia
5. Positive diagnosis of Marfan syndrome or death due to dissection plus positive skeletal features in a first-degree relative.

*In families in which a firm phenotypic diagnosis of the Marfan syndrome has been established, mutation or linkage analysis for fibrillin-1 can be used to diagnose Marfan syndrome on a molecular basis in equivocally affected relatives or prenatally (349).

abnormalities is echocardiography, this tool is an essential element of screening for Marfan syndrome. When screening is performed, it is essential to use normal values corrected for body size and age. In adult cases where a thorough multifaceted evaluation excludes diagnosis, no subsequent screening is necessary. However, in borderline cases and young children of a clearly affected parent, repeat evaluation in 12 months is appropriate because skeletal, aortic, and ocular abnormalities may evolve.

The current approach to screening for Marfan syndrome and to guiding treatment of patients diagnosed with this disorder is echocardiographic assessment of the aorta. Improved medical and surgical therapy has increased life expectancy in these patients (350).

Other heritable conditions of the heart include other connective tissue disorders and tuberous sclerosis. In patients in whom transthoracic imaging is inadequate, TEE provides an alternative approach (353). Another accepted recommendation for echocardiographic screening is in the evaluation of potential donor hearts for transplantation (352). The overall yield for conditions that eliminate the heart as a donor is approximately one of four patients.

Noninvasive screening of LV function before the initiation of chemotherapy with cardiotoxic agents is also accepted clinical practice. Both echocardiography and nuclear gated blood pool scanning have been used for this purpose. Similarly, either modality may be used to monitor ventricular function serially during treatment. In this regard it is notable that two small prospective studies have reported Doppler-defined abnormalities of diastolic function that preceded detectable changes in systolic performance in patients with doxorubicin cardiotoxicity (225,226).

Although a number of systemic diseases, such as sarcoidosis and systemic lupus erythematosus have the potential to involve the heart, there appears to be little role and generally few options for treatment of asymptomatic cardiac disease in

this setting. Thus, the role of echocardiographic screening of these subjects is debatable.

In contrast to its utility in screening selected relatively high-risk populations, echocardiographic testing cannot be justified when asymptomatic cardiovascular disease is sought in larger lower-risk groups. For example, in two large screening studies the prevalence of echocardiographically manifest hypertrophic cardiomyopathy in an adult population was reported to be 0.2% (354,355), with the majority of individuals thus identified having mild manifestations of the disease. Similarly, although there is considerable public awareness of athletes dying from unrecognized heart disease, studies (356,686) have shown that the prevalence of these and other conditions appears to be too low to justify widespread screening (Table 20).

Recommendations for Echocardiography to Screen for the Presence of Cardiovascular Disease

Class I

1. **Patients with a family history of genetically transmitted cardiovascular disease.**
2. **Potential donors for cardiac transplantation.**
3. **Patients with phenotypic features of Marfan syndrome or related connective tissue diseases.**
4. **Baseline and re-evaluations of patients undergoing chemotherapy with cardiotoxic agents.**
5. **First-degree relatives (parents, siblings, children) of patients with unexplained dilated cardiomyopathy in whom no etiology has been identified.**

Class IIb

Patients with systemic disease that may affect the heart.

Class III

1. **The general population.**
2. **Routine screening echocardiogram for participation in competitive sports in patients with normal cardiovascular history, ECG, and examination.**

XIII. ECHOCARDIOGRAPHY IN THE CRITICALLY ILL

Numerous applications of TTE and TEE to clinical conditions discussed elsewhere in these guidelines also apply to the hemodynamically unstable patient who is evaluated in either the emergency department or critical care unit. Chest pain, hypotension, or shock of unknown cause may not have the usual clinical findings that clearly define the diagnosis. Among the specific conditions detectable in the acutely ill patient are acute myocardial infarction and its complications, cardiac tamponade, aortic dissection, mechanical or infective complications of native or prosthetic valves, and source of embolism (359,360). In the critically ill patient there are significant differences in the relative value of TEE versus TTE.

The critically ill patient in the emergency department or intensive care unit is often managed by intubation and mechanical ventilation, frequently utilizing positive end-

Table 20. Echocardiographic Screening for Athletes

Author (Ref)	Year	n	Methods	Results	Study Conclusion
Maron (357)	1987	90	501 collegiate athletes were screened with history, physical examination, and ECG. Those with positive findings (n = 90) were referred for echocardiography (2D and M-mode)	14 (15%) mild MVP 3 mild septal hypertrophy (HCM vs athlete's heart?) 0 Marfan 0 definite HCM 0 any other CVD with risk for death or disease progression with athletic competition	Echocardiographic screening not justified
Lewis (356)	1989	265 (all but 3 black)	265 collegiate athletes 2D and M-mode echocardiography	30 (11%) MVP 1 small ASD 29 (11%) septal hypertrophy (HCM vs athlete's heart?)	Echocardiographic screening not justified
Weidenbener (358)	1995	2997	Limited: parasternal 2D long and short axis, estimated cost less than \$14	2% abnormal (64) 40 MVP 10 bicuspid AV 4 aortic dilatation 2 ventricular septal defect 2 dilated CS 1 AI 1 ASD 1 RV mass 1 septal hypertrophy	Can be performed inexpensively; however, "uncovered no abnormalities that precluded participation in athletic events," although "some conditions allowed only limited participation"
Murry (856)	1995	125	Parasternal LA+SA M-mode, 2-D color	90% normal, 11 (9%) MVP	Preliminary data, possible role for screening
Zeppili (857)	1998	3650 screened for anomalous cor aa (AOCA)	Apicals added if parasternal abnormal parasternal views for cor ostia	2 (1%) bicuspid AV	Anomalous CA rare in asymptomatic athletes
Kinoshita (858)	2000	1929	Parasternal screening for aortic root size	5 (0.26%) total group 4/415 (0.96%) basketball and volleyball players	Routine screening not justified? Role in tall athletes

AI indicates aortic insufficiency; AOCA, anomalous coronary artery; ASD, atrial septal defect; AV, aortic valve; CA, coronary artery; CS, coronary sinus; CVD, cardiovascular disease; ECG indicates electrocardiogram; HCM, hypertrophic cardiomyopathy; LA, long-axis view; MVP, mitral valve prolapse; RV, right ventricular; SA, short-axis view; 2D, two-dimensional.

expiratory pressure (PEEP). Up to one half of such patients cannot be adequately imaged by TTE, especially those requiring more than 10 cm PEEP (361). Furthermore, many patients in intensive care units cannot be appropriately positioned, have sustained chest injury, or are postoperative patients with dressings and tubes preventing adequate TTE. Because of these considerations, TEE is often required to make the diagnosis.

In the critically ill patient without myocardial infarction, significant left-sided valve or ventricular disease, or known pulmonary disease, the finding of RV dilation or hypokinesis on TTE indicates a high probability of pulmonary embolism. Although in some series the presence of RV hypokinesis identifies patients with 30% or more of the lung nonperfused who may receive significant benefit from thrombolysis (362), others have noted a lack of correlation between the extent of perfusion abnormalities on lung scan and the degree of RV dilatation or dysfunction (687). These authors also found that RV enlargement and systolic dysfunction are present and persist despite treatment with heparin and warfarin or vena caval interruption. The degree of RV dysfunction on TTE does seem to serve as a predictor of mortality rate (688). In patients who are hypotensive in the ICU setting, large main pulmonary thrombus may rarely be diagnosed by TEE even when unsuspected (689). One study (690) found that central pulmonary embolism could be detected in 80% using TEE and 90% using spiral CT (690,691).

The majority of studies of echocardiography in the clinically ill have been retrospective analyses. In most, both TTE and TEE results were available, allowing a comparison between the two. In some of the studies both the critically ill and injured were evaluated, and in others only postoperative patients were included. In general, there is an improved yield of critical findings by TEE in patients in whom the standard two-dimensional Doppler TTE study provided inadequate information. TEE often resulted in a change in treatment or surgery (86,88,363-370,692-694) (Table 21). Recently the first prospective but nonrandomized trial comparing the value of TTE and TEE for evaluating unexplained hypotension found that 64% of 45 TTE studies were inadequate, compared with 3% of 61 TEE studies. Transesophageal studies contributed new clinically significant diagnoses (not seen by TTE) in 17 patients (28%), leading to operation in 12 (20%) (86).

Although TEE appears to be of special advantage in the critically ill, when the overall utility of TTE echocardiography was evaluated in 500 patients, changes in treatment occurred because of the TTE finding more commonly in patients in the ICU (54%) versus patients not in the ICU (37%) (695). Recent excellent reviews comprehensively cover the use of TEE in the critically ill and traumatized patients (664,696).

New modalities using contrast injection are improving the usefulness of TTE in ICU and mechanically ventilated patients even beyond the technical improvements offered by harmonics. Two recent studies have shown that wall motion scoring and ejection fraction calculation can be improved to over 80% of such patients with contrast imaging (697,698).

TTE or TEE may help to define pathophysiological abnormalities in patients even when there is constant invasive monitoring of pulmonary artery pressures by the Swan-Ganz technique. In several series echocardiography was found to be more reliable than Swan-Ganz catheter pressure in determining the cause of hypotension (86,371-373). Although the measurement of cardiac output by TEE and Doppler using special views appears to be feasible (374-376), clinical use on a continuous basis is not yet available. It is not a realistic expectation at this point that echocardiography and Doppler measurements will replace thermodilution-determined cardiac output or pulmonary artery catheter monitoring (5), although with severe tricuspid regurgitation, thermodilution cardiac outputs can be misleading. In this situation, the cardiac output can be checked by a Doppler-derived cardiac output. Other measurements of function can be obtained using TEE and Doppler, including pulmonary venous flow determination, which may assist in separating various cardiovascular conditions responsible for hemodynamic instability (87).

TEE is valuable in the hypotensive postoperative cardiac surgery patient to detect treatable conditions (372,373). Other potential advantages of TEE in the surgical patient are addressed in practice guidelines for perioperative TEE (377), and the specific role of intraoperative TEE has been covered recently in reviews (699) and in ASE/SCA guidelines (700) and in the new section (XVI) of this guideline.

Although no fatal and few serious complications of TEE were reported in the studies cited, there are significant special technical considerations that must be taken into account in these critically ill patients (87,378).

Complication rates of TEE undertaken in the emergency department (ED) have been found to be much higher than for TEEs undertaken in ICUs (1% to 3%). In one series of 142 ED TEEs, there were 18 complications (12.6%): death (1), respiratory insufficiency/failure (7), hypotension (3), emesis (4), agitation (2), and cardiac dysrhythmia (1) (701).

A. Echocardiography in the Trauma Patient

Both TTE or TEE methods have been found to be useful in the severely injured patient in whom cardiac, pericardial, mediastinal, or major intrathoracic vascular injury has occurred. Myocardial contusion or rupture, pericardial effusion, tamponade, major vascular disruption, septal defects or fistulae, and valvular regurgitation may all result from either blunt or penetrating trauma. Assessment of the patient's volume status and detection of significant underlying heart disease, especially in the elderly patient, is possible through standard Doppler echocardiography techniques (702).

These patients represent a diagnostic challenge as they often present with serious multisystem trauma or major chest injury and are hemodynamically unstable. The ECG is helpful but often nonspecific, and serum enzymes have not been found reliable. TTE has been used since the early 1980s to evaluate cardiac trauma (379,380). In both blunt and penetrating chest trauma, 87% of patients could be imaged satisfactorily by TTE, with significant abnormalities found in 50%, the most common of which was pericardial effusion

Table 21. Role of Echocardiography in the Critically Ill and Injured

Author (Ref)	Year and Type of Study	TTE Pts or Studies	TTE	TTE Comment	% TEE Diagnosis or Number Not Available by TTE	Type of Unit	Primary Reasons for Examination
Pearson (363)	1990 retrospective	61	All	Suboptimal in most	44%	Multiple	Aortic dissection 29% Source of embolism 26% Complication of CAD 10% Miscellaneous 19% Ventricular function 8% Infective endocarditis 8%
Oh (364)	1990 retrospective	51	All	Suboptimal TTE reason for TEE	59%	Multiple	25—hemodynamic instability 49% No. of pts Infective endocarditis 10 Cardiac contusion 10 Heart donor 5 Aortic dissection, source of embolus, LV function, chest pain, other 10
Font (365)	1991 retrospective	112	All	68% fair to poor image	<i>By TEE</i> 131 new lesions compared with 95 for TTE: TTE/TEE=73% overall Equal for tamponade Detailed analysis of TTE/TEE frequency for each diagnosis	Multiple 56% postoperative cardiac or noncardiac surgery	RO vegetations 46% RO valve dysfunction 43% Assess ventricular function 19% Source of embolus 12% RO dissection 9% Other (congenital heart disease, tamponade, LVOT obstruction, constriction) 14%
Foster (366)	1992 retrospective	83	34% had TTE	TEE with Doppler not available TTE with Doppler complementary	25 “new findings” in TEE pts who had TTE 19% cardiac surgery prompted by TEE		Endocarditis 43% Embolic source 14% Mitral regurgitation 11% Hypotension 11% Other (LV function, aortic dissection, prosthetic valve, etc) 21%
Hwang (367)	1993 retrospective	80	All	50% did not provide critical information provided by TEE	Aortic dissection 27 sensitivity TEE 100% 12 sensitivity TTE 44% Hemodynamic instability TEE 20 pts TTE 11 pts Embolic source TEE 9 pts TTE 0 pts Cardiac surgery prompted in 18%	Multiple 48 pts Emergency department 32 pts	Aortic dissection 27 Hemodynamic instability 20 Embolic source 9 Evaluation of MR 7 Endocarditis 3 Other remainder

Continued on next page

Table 21. (Continued)

Author (Ref)	Year and Type of Study	TTE Pts or Studies	TTE TTE	Comment	% TEE Diagnosis or Number Not Available by TTE	Type of Unit	Primary Reasons for Examination
Khoury (368)	1994 retrospective	77	All	Technically poor or inconclusive in 77 (100%), thus reason for TEE	Same Echocardiography resulted in change in treatment of 46 of 77 pts (100%); 48% (37) were due solely to TEE findings TEE led to surgery in 22 pts (29%)	Multiple	Hemodynamic instability 41% Endocarditis 34% Embolic source 21% Aortic dissection 4%
Poelaert (369)	1995 retrospective	108	TEE only in pts with inadequate TTE	Not compared	TEE excluded abnormalities in 27% TTE not compared; primary reason for study to evaluate TEE in comparison with pulmonary artery catheter	Medical-surgical ICU	Multiple reasons No postoperative pts
Heidenreich (86)	1995 prospective	61	In 45 of 61 pts	Adequate visualization in only 36% vs 97% for TEE	17 (28%) new diagnoses by TEE not observed by TTE Multiple unexpected findings by TEE leading to a change in management in 48% of pts, resulting in improved BP in 24%	Multiple	Unexplained hypotension: multiple different causes found
Sohn (88)	1995 retrospective	122 new pts (25 in 1990 group)	All	Suboptimal TTE reason for TEE	98% In 59% a cause was found, resulting in urgent surgery in 21%	Multiple	Hemodynamic instability and inadequate TTE No. of pts 53 global or regional LV function (34%) 31 severe valvular disease (20%) 22 endocarditis (14%) 12 suspected aortic dissection (8%) 11 shunt lesions (7%) 7 mass lesions (4%) 30 had 2 reasons

BP indicates blood pressure; CAD, coronary artery disease; ICU, intensive care unit; LV, left ventricular; LVOT, left ventricular outflow tract; MR, mitral regurgitation; Pts, patients; RO, rule out; TEE, transesophageal echocardiogram; TTE transthoracic echocardiogram.

(27%) (381). In a prospective study of 336 patients over 6 years, young patients with minor blunt thoracic trauma and a normal or minimally abnormal ECG have a good prognosis, and further diagnostic studies and monitoring are seldom necessary (382). Others have proposed a similar triage scheme for blunt cardiac trauma using both TTE and TEE (383-385). Both TTE and TEE are being used with increasing frequency in EDs in patients with blunt thoracic trauma (703), prompting the publication of guidelines for echocardiography in emergency medicine (704).

Blunt cardiac injury may result in cardiac contusion significant enough to produce serious dysrhythmias (386), cardiac dysfunction, or tamponade. The majority of serious injuries result in death from rupture of the ventricle or aorta before the patient can be transported (387). In patients with serious blunt trauma who reach the hospital, even if in profound shock or cardiac arrest, survival is possible if the injury is recognized and immediate surgery undertaken, even in cardiac rupture (388). In certain cases, TTE done emergently in the ED may assist in the diagnosis and result in salvage. The sequelae of blunt cardiac trauma may not be immediately evident and require close follow-up. The diagnosis may eventually be made by various means, including cardiac echocardiography (389).

It is often difficult to image patients with severe blunt trauma with TTE. Most studies have found that TEE was valuable when TTE images were suboptimal and when aortic injury was suspected (390-392). In one study of intubated multiple injury patients not confined to blunt chest trauma, TEE evaluation detected unsuspected myocardial contusion, pericardial effusion, and aortic injury (393).

Thoracic aortic disruption usually occurs in a sudden deceleration injury or serious blunt trauma in which torsion forces are brought to bear upon the aorta, resulting in tears in the intima or transection of the aorta. The most common sites of rupture or partial rupture in those patients surviving to reach the hospital are the descending aorta just distal to the left subclavian artery (aortic isthmus) and the ascending aorta just proximal to the origin of the brachiocephalic vessels. Of the 20% who survive to reach the emergency room, 40% die within the first 24 hours. Radiological signs in these patients include widening of the mediastinum on chest radiograph, fracture of first and second ribs with an apical cap, or multiple types of thoracic trauma. Occasional patients with multi-system trauma without evidence of chest trauma sustain rupture.

While aortography has been the gold standard, computed tomography and MRI have also been used in an attempt to differentiate patients with trauma and a widened mediastinum. TEE is becoming the first approach in many centers because of the utility and speed with which it can be accomplished and because of its superiority in evaluating aortic disease such as dissection. This is especially so with the widespread use of biplane and multiplane TEE (384,391,392). Obviously, the value of TEE depends on its availability in a timely manner and the expertise of the operators to perform a comprehensive evaluation of the aorta without serious com-

plications in the traumatized patient (394,395). Several series of patients undergoing TEE have been reported in which most patients have had aortography or surgery to confirm the diagnosis (266,390,392,396,397). The use of TEE as a primary diagnostic modality in traumatic aortic rupture appears to be rapid, safe, and accurate as a bedside method. Although widespread use of TEE has not been documented in large series from many different institutions, aortography may be avoided except in those patients in whom TEE results are equivocal, when TEE is not tolerated or contraindicated, or when other vascular injuries of arch vessels or lower portions of the descending aorta are suspected. TEE, aortography, computed tomography, and MRI are reviewed in a recent publication (398).

Blunt aortic injury is the second most common cause of death in studies of blunt trauma deaths. Early studies, cited above, indicate an increasing utilization of TEE in these patients in many centers. An initial prospective study at 50 trauma centers throughout North America of 274 blunt aortic injury cases seen between 1993 and 1996 revealed that chest computed tomography (CT) and TEE were applied in 88 and 30 cases, respectively, and were 75% and 80% diagnostic, respectively (705). However, the increasing frequency of use of TEE in the assessment of blunt aortic injury is apparent from the literature, introducing a challenge to echocardiographers and systems caring for these patients. Smith *et al.* reported 101 cases of suspected traumatic rupture of the aorta in which TEE was attempted. Ninety-three patients were successfully studied with a sensitivity of 100% and a specificity of 98% (706). Vignon *et al.* signaled that TEE should be routinely performed in victims of violent deceleration collisions even when the chest X-ray appeared normal (707,708). The same authors and others (709) have more recently pointed out limitations of TEE in various types of aortic injury, concluding aortography is superior, especially for branch and proximal arch disruption. The rapidly evolving diagnostic modality of contrast-enhanced spiral thoracic computed tomography (CEST-T) has demonstrated a higher degree of accuracy. In one study from the Maryland Shock-Trauma Center, in 1104 prospectively studied blunt trauma patients, CEST-T had an overall diagnostic accuracy of 99.7% (710). Despite the rapid evolution in the use of ultrasound and spiral CT in the evaluation of aortic injuries, aortography appears at this point in time to be the most frequently used imaging modality. Which techniques are used appears to largely depend on a specific institution's individual algorithm and the expertise they can mobilize in the evaluation of these acute patients until such time as a large enough prospective comparative study of aortography, spiral CT, and/or TEE is completed in multiple trauma centers (711).

Penetrating chest trauma, whether by gunshot, stabbing, or other means, has usually required surgical exploration using a subxiphoid pericardial approach to exclude cardiac injury. The subxiphoid exploration, however, carries a negative exploration rate of 80%. TTE, when compared to subxiphoid pericardiotomy, is 96% accurate, 97% specific, and 90% sen-

sitive in detecting pericardial fluid in juxtacardiac penetrating chest wounds (399). Thus, TTE may prevent unnecessary exploratory thoracotomy or subxiphoid pericardiectomy (400). In a report in which TTE was used in the ED of a large metropolitan hospital, survival in the group who had TTE was 100%; for the nonechocardiography group, survival was 57.1% (401). In another series of patients with penetrating chest injury, TTE had an accuracy of 99.2% and positive and negative predictive values of 100% and 98% (402). Others, however, have reported that a normal echocardiogram (TTE) does not always exclude major intrapericardial injury, and that even small effusions in penetrating chest trauma may be associated with significant injury (403). When hemothorax is associated with penetrating injury, cardiac echocardiography does not have adequate sensitivity and specificity to avoid the necessity of subxiphoid exploration (404).

Late sequelae of penetrating injuries are not uncommon, and thus routine TTE is recommended in all patients with penetrating cardiac injuries (405,406). The detection and location of bullet fragments is also possible with TEE (407). While no large series of penetrating cardiac wounds studied by TEE has been reported, initial reports support its routine use in the perioperative period (408).

Iatrogenic penetrating cardiac injury in the catheterization laboratory is rare, occurring in 0.12% of procedures. Whether by guidewires, pacemaker catheters, balloon valvulotomy, PTCA, or pericardiocentesis, tamponade is the result in many of these, recognizable by fluoroscopy at the time and confirmed by cardiac echocardiography in the laboratory or at the bedside. Pericardiocentesis is the definitive treatment in most, and surgery is rarely necessary (409).

In summary, echocardiography and Doppler techniques are extremely valuable in delineating pathology and hemodynamics in the critically ill or injured patient and in certain perioperative situations. TEE appears to have a distinct advantage in certain settings and conditions.

Conditions and Settings in Which TEE Provides the Most Definitive Diagnosis in the Critically Ill and Injured

- **The hemodynamically unstable patient with suboptimal TTE images**
- **The hemodynamically unstable patient on a ventilator**
- **Major trauma or postoperative patients (unable to be positioned for adequate TTE)**
- **Suspected aortic dissection**
- **Suspected aortic injury**
- **Other conditions in which TEE is superior (see section on valvular disease)**

Because of the highly variable nature of these patients, the differing clinical circumstances in reported series, and the evolving utilization of either TTE or TEE and Doppler techniques, the relative merit and recommendations may vary among institutions.

Recommendations for Echocardiography in the Critically Ill

Class I

1. **The hemodynamically unstable patient.**
2. **Suspected aortic dissection (TEE).**

Class III

1. **The hemodynamically stable patient not expected to have cardiac disease.**
2. **Re-evaluation follow-up studies on hemodynamically stable patients.**

Recommendations for Echocardiography in the Critically Injured*

Class I

1. **Serious blunt or penetrating chest trauma (suspected pericardial effusion or tamponade).**
2. **Mechanically ventilated multiple-trauma or chest trauma patient.**
3. **Suspected pre-existing valvular or myocardial disease in the trauma patient.**
4. **The hemodynamically unstable multiple-injury patient without obvious chest trauma but with a mechanism of injury suggesting potential cardiac or aortic injury (deceleration or crush).**
5. **Widening of the mediastinum, postinjury suspected aortic injury (TEE).**
6. **Potential catheter, guidewire, pacer electrode, or pericardiocentesis needle injury with or without signs of tamponade.**

Class IIa

1. **Evaluation of hemodynamics in multiple-trauma or chest trauma patients with pulmonary artery catheter monitoring and data disparate with clinical situation.**
2. **Follow-up study on victims of serious blunt or penetrating trauma.**

Class III

Suspected myocardial contusion in the hemodynamically stable patient with a normal ECG who has no abnormal cardiac/thoracic physical findings and/or lacks a mechanism of injury suggesting cardiovascular contusion.

*The use of TTE or TEE includes Doppler techniques when indicated and available and with appropriately trained and experienced sonographer and interpreter.

TEE is indicated when TTE images are suboptimal. TEE often provides incremental information.

XIV. TWO-DIMENSIONAL ECHOCARDIOGRAPHY IN THE ADULT PATIENT WITH CONGENITAL HEART DISEASE

The adult patient with congenital heart disease is referred for echocardiography either because the problem was not dis-

covered in childhood or more often because the patient was previously diagnosed as having congenital heart disease and is stable or inoperable or has had one or more palliative or corrective surgical procedures (712,713).

As a general rule, all patients with congenital heart disease must be followed indefinitely (672), even those who have had “corrective” procedures to return them to physiologically normal status. The only potential cures are in repaired patent ductus arteriosus and in some patients a repaired atrial septal defect. Adult patients with congenital heart disease are seen by the cardiologist because they

- Have been undiagnosed in the past
- Have recognized congenital heart disease that is presently inoperable, eg, hypoplastic pulmonary arteries or systemic level pulmonary hypertension and due to severe pulmonary vascular disease, and
 - Progressive clinical deterioration, such as ventricular dysfunction or arrhythmias due to the natural history of the disease
 - Become pregnant or have other stresses such as non-cardiac surgery or infection, including infective endocarditis
- Have residual defects after a palliative or corrective operation
- Develop arrhythmias (including ventricular tachycardia, atrial flutter, or atrial fibrillation) that may result in syncope or sudden death
- Have progressive deterioration of ventricular function with congestive heart failure
- Have progressive hypoxemia because of inadequacy of palliative shunt or development of pulmonary vascular disease
- Require monitoring and prospective management to maintain ventricular or valvar function and/or to prevent arrhythmic or thrombotic complications

Table 22 lists the late complications that occur in patients with surgically treated congenital heart disease. Echocardiography has been so accurate in the diagnosis of congenital heart disease that in many centers, patients with echocardiographic diagnosis alone are sent for complete repair of major congenital heart defects (714).

Transthoracic and transesophageal echocardiography are extremely useful in monitoring patients who have had surgical palliation or “correction” by detecting and quantifying the severity of residual defects such as shunts, valvular and conduit obstruction, valvular regurgitation, and ventricular function (715-717). TEE is better at defining the detailed anatomy of the atrioventricular junction and atria than TTE (718).

Magnetic resonance imaging also is very accurate in defining intracardiac anatomy and diagnosing congenital heart disease. It is complementary to echocardiography in that it is more accurate than echocardiography in diagnosing extracar-

diac abnormalities such as anomalous pulmonary veins and vascular rings (718,719). Three-dimensional echocardiography is becoming more useful in defining complex intracardiac anatomy in patients with congenital heart disease, but techniques are still evolving, and at present, its availability is extremely limited (720,721). Finally, echocardiography is increasingly being used in intracardiac interventional procedures such as valvotomy, device closure of atrial and ventricular septal defects, and intracardiac ablation for arrhythmias (722,723).

Of special importance is the recognition that congenital heart disease is relatively infrequent in the practice of the cardiologist who sees adults. Most cardiologists and echocardiographic technicians have insufficient experience with the wide variety of congenital heart disease lesions that exist. It is likely that they will recognize that something is abnormal but not recognize the specifics of the congenital heart lesion. For this reason, it is necessary that both the cardiac sonographer and interpreting cardiologist have special competencies in congenital heart disease or refer the patient to a cardiologist (adult or pediatric) experienced in the area.

Echocardiography is useful in

- Demonstrating chamber size and atrial septum
- Evaluating LV systolic and diastolic function and RV systolic function
- Defining the presence, site, and relative magnitude of intracardiac and/or systemic-to-pulmonary artery shunts
- Defining the presence, magnitude, and site of LV and RV outflow tract and valvular obstruction
- Evaluating valvar regurgitation
- Estimating pulmonary artery pressure
- Defining the relation of veins, atria, ventricles, and arteries
- Visualizing coarctation of the aorta and estimated degree of obstruction
- Defining the presence, site, and relative magnitude of intracardiac or vascular shunts using contrast echocardiography and color Doppler.
- Demonstrating intracardiac and/or central vascular mural thrombi as well as coronary fistulas
- Assessment of atrioventricular valve anatomy and function
- Visualizing conduits and intracardiac baffles in patients who have had surgical palliation (Mustard, Rastelli, and Fontan procedures)
- Identifying the site of origin and initial course of coronary arteries

Recommendations for Echocardiography in the Adult Patient With Congenital Heart Disease

Class I

1. Patients with clinically suspected congenital heart dis-

Table 22. Late Postoperative Complications in Patients With Congenital Heart Disease

<p>1. Atrial Septal Defects Atrial arrhythmias: atrial fibrillation, atrial flutter, sick-sinus syndrome Mitral regurgitation (ostium primum defect; occasionally other types of atrial defects) Heart block (ostium primum defect) (rare) Residual left-to-right shunt (rare) Right ventricular dysfunction</p>	<p>9. Tetralogy of Fallot With Conduit RV to PA Mural calcification of homograft Degeneration of valve in conduit—stenosis or regurgitation Ventricular arrhythmias—sudden death Aneurysm of proximal attachment of right ventricular outflow graft Aortic valve regurgitation Left ventricular dysfunction (previous or present large palliative shunts or aortopulmonary collaterals or poor myocardial preservation during surgery)</p>
<p>2. Atrioventricular Septal Defect Residual interatrial or interventricular left-to-right shunt Mitral and/or tricuspid regurgitation Left ventricular inflow and outflow tract obstruction Heart block</p>	<p>10. Tricuspid Atresia, Single Ventricle (Fontan Procedure) Atrial tachyarrhythmias (atrial flutter, atrial fibrillation) Intracardiac or central vascular mural thrombi Systemic venous hypertension, manifests as ascites, pleural effusion, hepatomegaly, superior vena caval syndrome, protein losing enteropathy Pulmonary arteriovenous fistula (rare except with associated Glenn procedure) Ventricular dysfunction Subaortic obstruction</p>
<p>3. Ventricular Septal Defect Residual left-to-right shunt Heart block (rare) Ventricular arrhythmias, including ventricular tachycardia and sudden death Aortic regurgitation Left ventricular dysfunction</p>	<p>11. Transposition of the Great Vessels Senning and Mustard procedure Sinus and atrioventricular nodal dysfunction Right ventricular (systemic ventricle) failure Intra-atrial caval baffle obstruction Baffle leaks with intra-atrial shunt Obstruction of neo-left atrium and pulmonary venous hypertension Fixed subpulmonic stenosis (discrete fibrous band on septal surface of left ventricular outflow or dynamic left ventricular outflow tract obstruction) Systemic embolization Arterial switch operation Supravalvular aortic obstruction Aortic valve regurgitation Coronary arterial obstruction, myocardial ischemia Left ventricular failure Supravalvular pulmonic stenosis</p>
<p>4. Patent Ductus Arteriosus Recanalization when ligated (rare) With coil embolization: residual left-to-right shunt Embolized coils to pulmonary artery or systemically (rare): occurs early within 24 hours of placement</p>	<p>12. Congenitally Corrected Transposition (ventricular inversion with L-transposition) Heart block Left-sided atrioventricular valve regurgitation Right (systemic) ventricular failure Residual ventricular septal defect and residual subpulmonic obstruction</p>
<p>5. Aortic Stenosis Recurrent or residual aortic stenosis Aortic regurgitation Ventricular septal defect (with fibromuscular tunnel) Aortic-to-right ventricular fistula (with Kono procedure) Heart block Coronary ostial obstruction (supravalvular aortic stenosis) (rare) Prosthetic valve dysfunction Prosthetic valve leak Prosthetic valve infection</p>	<p>13. Ebstein Disease Tricuspid regurgitation, progressive supraventricular tachycardias, Wolff-Parkinson-White syndrome Residual atrial shunts</p>
<p>6. Pulmonic Valve Stenosis Residual pulmonic valve stenosis Pulmonic valve regurgitation</p>	<p>14. Coarctation of the Aorta Hypertension Bicuspid aortic valve (regurgitation, stenosis) Vascular aneurysm rupture (cerebrovascular accident, berry aneurysm) Aortic dissection Residual or recurrent coarctation Mitral valve anomalies that can result in stenosis or regurgitation (rare) Premature coronary artery disease</p>
<p>7. Palliative Shunts Infective endocarditis (all shunts) Inadequate left-to-right shunt (Blalock-Taussig) Pulmonary hypertension (Potts, Waterston): can be unilateral due to pulmonary artery kinking protecting the opposite lung Congestive heart failure (Potts, Waterston) Obstruction of right pulmonary artery (Waterston) or left pulmonary artery (Potts)</p>	
<p>8. Tetralogy of Fallot Residual right ventricular outflow tract obstruction, valvar or subvalvar Residual branch pulmonary artery stenosis Residual ventricular septal defect Pulmonic valve regurgitation—right-heart failure, associated tricuspid regurgitation Aortic regurgitation Ventricular arrhythmias, including ventricular tachycardia and sudden death Heart block (rare) Right ventricular outflow tract aneurysm Calcification of homograft patches Aortic dilation Left ventricular dysfunction (previous or present large palliative shunts or aortopulmonary collaterals or poor myocardial preservation during surgery)</p>	

These are late postoperative complications in the more common congenital heart lesions in patients who survive to adulthood. There may be occasional patients with other lesions not included in this list.

RV indicates right ventricle; PA, pulmonary artery.

ease, as evidenced by signs and symptoms such as a murmur, cyanosis, or unexplained arterial desaturation, and an abnormal ECG or radiograph suggesting congenital heart disease.

2. Patients with known congenital heart disease on follow-up when there is a change in clinical findings.
3. Patients with known congenital heart disease for whom there is uncertainty as to the original diagnosis or when the precise nature of the structural abnormalities or hemodynamics is unclear.
4. Periodic echocardiograms in patients with known congenital heart lesions and for whom ventricular function and atrioventricular valve regurgitation must be followed (eg, patients with a functional single ventricle after Fontan procedure, transposition of the great vessels after Mustard procedure, L-transposition and ventricular inversion, and palliative shunts).
5. Patients with known congenital heart disease for whom following pulmonary artery pressure is important (eg, patients with hemodynamically important, moderate, or large ventricular septal defects, atrial septal defects, single ventricle, or any of the above with an additional risk factor for pulmonary hypertension).
6. Periodic echocardiography in patients with repaired (or palliated) congenital heart disease with the following: change in clinical condition or clinical suspicion of residual defects, obstruction of conduits and baffles, LV or RV function that must be followed, or when there is a possibility of hemodynamic progression or a history of pulmonary hypertension.
7. To direct interventional catheter valvotomy, radiofrequency ablation, and interventions in the presence of complex cardiac anatomy.
8. Identification of site of origin and initial course of coronary arteries (TEE may be indicated in some patients).*

Class IIb

A follow-up echocardiographic study, annually or once every 2 years, in patients with known hemodynamically significant congenital heart disease without evident change in clinical condition.

Class III

1. Multiple repeat echocardiography in patients with repaired patent ductus arteriosus, atrial septal defect, ventricular septal defect, coarctation of the aorta, or bicuspid aortic valve without change in clinical condition.
2. Repeat echocardiography in patients with known hemodynamically insignificant congenital heart lesions (eg, small atrial septal defect, small ventricular septal defect) without a change in clinical condition.

* TEE may be necessary to image both coronary origins in adults.

XV. ECHOCARDIOGRAPHY IN THE PEDIATRIC PATIENT

Congenital structural heart disease is the most common type of cardiovascular disease in the pediatric population. However, acquired heart disease also contributes to the cardiovascular morbidity of this population. Historically identified with rheumatic fever and endocarditis, acquired pediatric heart disease now includes Kawasaki disease and other coronary arterial diseases, human immunodeficiency virus (HIV) and other viral-related cardiac disease, dilated cardiomyopathy with or without acute-onset congestive heart failure, hypertrophic cardiomyopathy, and an increasing pediatric and young adult population with clinical cardiovascular issues related to surgical palliation/correction of structural heart disease and cardiac transplantation.

Two-dimensional Doppler echocardiography has become the definitive diagnostic method for the recognition and assessment of congenital and acquired heart disease in the pediatric population. Its use has eliminated the need for invasive or other noninvasive studies in some and decreased the frequency and improved the timing and performance of invasive studies in other patients (714,724,725). Echocardiographic re-evaluation in some candidates improves medical or surgical management. For the child with insignificant cardiac disease, an echocardiographic evaluation should reduce the frequency of pediatric cardiology surveillance and provides reassurance to the family (726). For those patients with a significant cardiac abnormality, early and accurate echocardiographic evaluation improves clinical outcome. Most echocardiographers who deal with adult patients have little experience with congenital heart disease, especially as it is seen in the pediatric and neonatal patient. Echocardiographers should have appropriate training and experience before attempting to perform or interpret echocardiograms in patients with congenital heart disease.

Re-evaluation echocardiographic examinations are frequently used to monitor cardiovascular adaptation to surgical repair or palliation and identify recurrence of abnormalities. Such longitudinal follow-up allows facilitates proactive surgical and/or medical intervention (416-422,424-426,430,431,727-729). For these reasons, echocardiography provides improved outcome and lowers healthcare costs by streamlining the use of medical resources, guiding management decisions, and providing early education and support for the family.

A. Resource Utilization and Age

Guidelines for pediatric echocardiography utilization must be stratified by age to accommodate the unique cardiovascular physiology of the neonate. Such guidelines must recognize the newborn's transitional circulation and the frequent coexistence of confounding pulmonary disease. The transitional circulation in the perinatal age group may obscure hemodynamically important, even critical, cardiovascular abnormalities. Due to the rapid changes in pulmonary vascu-

lar resistance and the patency of the ductus arteriosus, re-evaluation echocardiographic examinations of the critically ill neonate are often required. Newborns with pulmonary hypertension (persistent pulmonary hypertension of the neonate) will require repeated echocardiographic evaluation of the cardiovascular response to medical interventions modulating pulmonary artery pressure. Those undergoing extracorporeal cardiopulmonary therapy require echocardiographic monitoring of ventricular function (432) and surveillance for intracardiac thrombus formation. Newborn infants with noncardiac anomalies requiring urgent surgical intervention undergo preoperative echocardiographic screening, even in the absence of clinically manifest cardiovascular disease, to exclude associated cardiovascular anomalies (730). This knowledge facilitates perioperative treatment of these patients and focuses both on noncardiac and cardiac therapy. For neonates with multiple congenital abnormalities and severe anatomic and/or functional neurological impairment, echocardiographic identification of cardiac anomalies will better define survivability and help guide difficult management decisions regarding life support and palliation (433).

B. Cardiovascular Disease in the Neonate

1. Structural Congenital Cardiovascular Disease

Two-dimensional echocardiography provides essential structural information in all forms of cardiac and great vessel disease in pediatric patients. Doppler echocardiography provides important physiological information that, when combined with anatomic data, guides therapeutic management in some diagnostic categories. Re-evaluation examinations allow tracking of hemodynamic changes such as those occurring during the transition phase from fetal to newborn and infancy periods (434). Echocardiography provides clinical information to guide medical or surgical intervention and provide prognostic information. It is also valuable to track evolutionary changes in the cardiovascular system and to determine management subsequent to medical or surgical intervention.

Perinatal physiological changes often mask or obscure the presence of hemodynamically important cardiovascular lesions on physical examination of the neonatal or young infant (731). Echocardiography allows early recognition of cardiac lesions in the neonate with presumed sepsis or pulmonary disease in which either the pulmonary or the systemic circulation depends on the patency of the ductus arteriosus (435-437). Definitive diagnosis in these lesions before ductal closure may prevent severe morbidity or death. Infants with a loud murmur, signs of congestive heart failure, cyanosis, or failure to thrive have a high probability of significant heart disease and along with a general examination by a qualified pediatric cardiologist should undergo immediate echocardiographic evaluation under his/her supervision.

The common categories of structural congenital cardiovascular disease encountered in the neonate and information provided by echocardiography are summarized as follows:

1. Intracardiac shunts: location, morphology and size of defect, direction of flow and gradient across defect, pulmonary/systemic flow profile, ventricular compensation, associated lesions (438,732)
2. Obstructive lesions: location, morphology, pressure gradient, ventricular compensation, associated lesions (437,440,733-735)
3. Regurgitant lesions: valve morphology, assessment of severity, atrial/ventricular dilation, ventricular compensation, associated lesions (420,444,446,736)
4. Anomalous venous connections: location and connections of proximal systemic and pulmonary veins, assessment of left-to-right and right-to-left shunts, presence of venous obstruction, and associated lesions (424,447-449)
5. Conotruncal abnormalities: position of great arteries, ventriculoarterial connections, spatial and hemodynamic relation of great arteries to coexisting ventricular septal defect, nature of subarterial obstruction, great artery anatomy, associated lesions, ventricular compensation (450-454)
6. Coronary anomalies: origin, size and flow in coronary arteries, presence of coronary artery fistulae, ventricular compensation (455,737,738)
7. Complex lesions: cardiac segmental analysis of situs and connections, size and location of all cardiac chambers, atrioventricular valve morphology and function, subarterial and arterial obstruction, interatrial and interventricular communications, venous and great artery anatomy, ventricular compensation

C. Cardiopulmonary Disease

The hemodynamic transition from the fetal to the extrauterine environment influences clinical expression of cardiovascular and pulmonary disease in the neonate. Premature infants may have respiratory failure based on a combination of processes: lung immaturity, hyaline membrane disease, persistence of the ductus arteriosus, inflammatory disease, alveolar capillary dysplasia, or congenital heart disease. Echocardiography indicates the direction and degree of shunting across the interatrial septum or patent ductus arteriosus and estimation of pulmonary artery pressure. In premature babies, diagnosis and monitoring of a patent ductus arteriosus is achieved by echocardiography. Echocardiography identifies occult ductal-dependent cardiovascular lesions, thereby avoiding undesirable pharmacological or surgical closure of a patent ductus arteriosus.

Neonates with pulmonary hypertension (persistent pulmonary hypertension of the neonate) may present with or without perinatally acquired pulmonary parenchymal disease. Differentiation of this entity from cyanotic heart disease can be accomplished by echocardiography. Inhaled nitric oxide increases systemic oxygen saturation by causing

a reduction in pulmonary vascular resistance and right-to-left shunting across the interatrial septum and ductus arteriosus in such neonates (739,740). In addition to excluding structural abnormalities, Doppler echocardiography provides additional information about atrial and ductal shunting, pulmonary artery pressure, and ventricular function in response to therapeutic interventions. Re-evaluation studies are useful for monitoring the efficacy of therapeutic interventions and the response to withdrawal of therapy. Adverse rebound pulmonary hypertension can accompany withdrawal of nitric oxide therapy (741-743). In patients with severe disease progressing to extracorporeal membrane oxygenation (744,745), this information is useful in assessing the contribution of extracorporeal circulation to ventricular output, alteration in myocardial function (746), and changes in ductus arteriosus flow (432).

D. Arrhythmias/Conduction Disturbances

Electrophysiological anomalies may be present in the newborn period. Arrhythmias may occur as an isolated clinical problem; however, some neonatal rhythm abnormalities are associated with structural cardiac or systemic disease. Intracardiac tumors, particularly the rhabdomyomas of tuberous sclerosis (458), can present with supraventricular or ventricular tachyarrhythmias. Perinatal arrhythmia may present as nonimmune fetal hydrops or acute-onset congestive heart failure. Echocardiography is integrated into the treatment of these patients to identify the hemodynamic sequelae of the dysrhythmia and coexisting systemic disease.

E. Acquired Cardiovascular Disease in the Neonate

Myocardial abnormalities in the neonate are most commonly related to transplacentally acquired pathogens, metabolic abnormalities, structural congenital heart disease, maternal systemic disease, or peripartum injury (459,460). Echocardiography is used to identify reversible structural anomalies contributing to myocardial dysfunction, monitor the response of the myocardium to medical intervention, and document recovery from peripartum injury. Premature infants receiving steroids for pulmonary disease should undergo echocardiography at intervals to screen for the appearance of hypertrophic cardiomyopathy (747).

Recommendations for Neonatal Echocardiography

Class I

- 1. Cyanosis, respiratory distress, congestive heart failure, or abnormal arterial pulses.**
- 2. Chromosomal abnormality or major extracardiac abnormality associated with a high incidence of coexisting cardiac abnormality.**
- 3. Lack of expected improvement in cardiopulmonary status in a premature infant with a clinical diagnosis of pulmonary disease.**
- 4. Systemic maternal disease associated with neonatal comorbidity.**

- 5. Loud or abnormal murmur or other abnormal cardiac finding in an infant.**
- 6. Presence of a syndrome associated with cardiovascular disease and dominant inheritance or multiple affected family members.**
- 7. Presence of a syndrome associated with heart disease, with or without abnormal cardiac findings, for which an urgent management decision is needed.**
- 8. Cardiomegaly on chest radiograph.**
- 9. Dextrocardia, abnormal pulmonary or visceral situs by clinical, electrocardiographic, or radiographic examination.**
- 10. Arrhythmias or other abnormalities on standard ECG suggesting structural heart disease or peripartum myocardial injury.**
- 11. Clinical suspicion of residual or recurrent abnormality, poor ventricular function, pulmonary artery hypertension, thrombus, sepsis, or pericardial effusion after cardiovascular surgical therapy for congenital heart disease.**
- 12. Re-evaluation after initiation or termination of medical therapy for pulmonary artery hypertension.**
- 13. Re-evaluation during initiation or withdrawal of extracorporeal cardiopulmonary support.**
- 14. Nonimmunologic fetal hydrops.**
- 15. Follow-up assessment of a neonate with patent ductus arteriosus who has undergone medical or surgical intervention.**

Class IIa

- 1. Short, soft murmur at the lower left sternal border in the neonate.**
- 2. Failure to thrive in the absence of definite abnormal clinical findings.**
- 3. Presence of a syndrome associated with a high incidence of congenital heart disease for which there are no abnormal cardiac findings and no urgency of management decisions.**

Class III

- 1. History of nonsustained fetal ectopy in the absence of postpartum arrhythmias.**
- 2. Acrocyanosis with normal upper-and-lower extremity pulsed oximetry oxygen saturations.**

F. Congenital Cardiovascular Disease in the Infant, Child, and Adolescent

Cardiovascular disease in the infant, child, and adolescent includes anomalies of cardiac anatomy, function, morphogenesis, and rhythm. While these problems often present as an asymptomatic heart murmur, the cardiac murmurs of this age group are more commonly functional than pathological. History and physical examination by a skilled observer are usually sufficient to distinguish functional from pathological murmurs and are more cost-effective than referral for an echocardiogram (461). Echocardiography provides useful guidance for the primary practitioner (520,748-751) con-

fronted with ambiguous historical and clinical findings. Echocardiography can demonstrate the presence or absence of abnormalities such as an interatrial septal defect, bicuspid aortic valve, mildly obstructive subaortic stenosis, MVP, or functionally occult cardiomyopathy. Such determination clarifies the need for further evaluation or endocarditis prophylaxis, or both. For patients with clinical findings of hemodynamically important heart disease, anatomic and physiological data provided by re-evaluation and two-dimensional Doppler echocardiography may establish a definitive diagnosis and allow the most efficient selection of adjuvant diagnostic procedures or medical/invasive intervention. Referral, acquisition, and appropriate interpretation of the echocardiogram must consider the compliance of the patient, include relevant medical history, and identify the clinical objective of the examination. The need for conscious sedation of infants and toddlers should be identified and requisite protocols implemented in a setting that permits mandated surveillance.

1. Structural Cardiovascular Disease

The categories of structural cardiovascular disease in the infant, child, or adolescent are identical to those encountered in the neonate (see previous section). Physical findings may become more obvious in the older children and adolescents. In this population, echocardiography may play a less important role in screening for heart disease than it does in the neonatal period. The more important role for echocardiography in this age group is in fully characterizing a cardiac lesion once an abnormality is suspected. Echocardiography provides essential information, particularly for the child and adolescent, regarding the natural history of the abnormality (528) and responses to medical and surgical management (752). Contributing to the successful management of these children is the early recognition and prevention of secondary functional changes in the cardiovascular system, and echocardiography is often the most direct and cost-effective way to acquire this information.

Echocardiography enhances patient selection, endovascular device implantation, and surveillance of patients undergoing therapeutic cardiac catheterization. Interventions including transcatheter closure of interatrial septal defects or the ductus arteriosus and endovascular stent implantation are guided by anatomic and Doppler-based echocardiographic imaging of intracardiac and central vascular structures before, during, and after deployment of devices (753-761).

Echocardiography also provides important information in patients with systemic connective tissue disorders, eg, Marfan syndrome or Ehlers-Danlos syndrome. Re-evaluation and examination of patients with these disorders identifies acute and chronic changes in great artery size, semilunar and atrioventricular valve function, and ventricular compensation (462-464). Functional murmurs are commonly encountered in this pediatric population. The contribution of echocardiography to an experienced clinician's evaluation of an asymptomatic patient with this finding on routine examination is limited. Such murmurs can usually be diagnosed by an expe-

rienced clinician without the need for echocardiography. Referral of infants, children, and adolescents with functional murmurs for echocardiographic examination should be guided by evidence of coexisting congenital or acquired cardiovascular disease.

Recommendations for Echocardiography in the Infant, Child, and Adolescent

Class I

1. **Atypical or pathological murmur or other abnormal cardiac finding in an infant or older child.**
2. **Cardiomegaly on chest radiograph.**
3. **Dextrocardia, abnormal pulmonary or visceral situs on clinical, electrocardiographic, or radiographic examination.**
4. **Patients with a known cardiac defect, to assess timing of medical or surgical therapy.**
5. **Selection, placement, patency, and monitoring of endovascular devices, as well as identification of intracardiac or intravascular shunting before, during, and after interventional cardiac catheterization.**
6. **Immediate assessment after percutaneous interventional cardiac catheterization procedure.**
7. **Immediate preoperative evaluation for cardiac surgery of a patient with a known cardiac defect to guide cardiac surgical management and inform the patient and family of risks of surgery.**
8. **Patient with known cardiac lesion and change in physical finding.**
9. **Postoperative congenital or acquired heart disease with clinical suspicion of residual or recurrent abnormality, poor ventricular function, pulmonary artery hypertension, thrombus, sepsis, or pericardial effusion.**
10. **Presence of a syndrome associated with cardiovascular disease and dominant inheritance or multiple affected family members (eg, Marfan syndrome or Ehlers-Danlos syndrome).**
11. **Patients with a family history of genetically transmitted myocardial disease, with or without abnormal cardiac finding.**
12. **Baseline and follow-up examinations of patients with neuromuscular disorders having known myocardial involvement.**
13. **Exercise-induced precordial chest pain or syncope.**

Class IIb

Failure to thrive in the absence of definite abnormal clinical findings.

Class III

1. **In a child or adolescent, an asymptomatic heart murmur identified by an experienced observer as functional or an insignificant cardiovascular abnormality.**
2. **In an otherwise asymptomatic child or adolescent, chest pain identified by an experienced observer as musculoskeletal in origin.**

G. Arrhythmias/Conduction Disturbances

Frequent, sustained, or complex rhythm abnormalities in the pediatric population may be associated with Ebstein's anomaly of the tricuspid valve, cardiac tumor, dilated or hypertrophic cardiomyopathy, arrhythmogenic RV cardiomyopathy, MVP, glycogen storage disease, or stimulation from migrated central venous catheters. Thus, exclusion of these lesions by echocardiography is an important component in evaluation. Mild rhythm disturbances, such as sinus arrhythmias and isolated supraventricular ectopic beats or brief and infrequent runs of supraventricular tachycardia, are rarely associated with cardiac pathology. Echocardiography is generally indicated only when abnormal findings are also present. Occasionally, echocardiography aids in the characterization of an arrhythmia when surface ECG findings are ambiguous. Echocardiography after radiofrequency catheter ablation is discretionary (762,763). Re-evaluation echocardiogram after initiation of medical therapy or radiofrequency ablation of patients with ectopic atrial tachycardia and secondary dilated cardiomyopathy identifies recovery of ventricular function (764). Persistent ventricular dilatation after successful ablation or effective medical control of the heart rate may indicate an arrhythmogenic primary cardiomyopathy.

Recommendations for Echocardiography in Pediatric Patients With Arrhythmias/Conduction Disturbances

Class I

1. Arrhythmia in the presence of an abnormal cardiac finding.
2. Arrhythmia in a patient with a family history of a genetically transmitted cardiac lesion associated with arrhythmia, such as tuberous sclerosis or hypertrophic cardiomyopathy.
3. Complete atrioventricular block or advanced second-degree atrioventricular block.
4. Complete or high-degree secondary atrioventricular block.
5. Arrhythmia requiring treatment.

Class IIa

1. Ventricular arrhythmia in a patient referred for evaluation for competitive sports.
2. Evidence of pre-excitation on ECG with symptoms.

Class IIb

1. Pre-excitation on ECG in the absence of abnormal cardiac findings.
2. Recurring arrhythmia not requiring treatment in the presence of normal findings on examination.
3. Examination immediately after radiofrequency ablation.

Class III

Sinus arrhythmia or isolated extrasystoles in a child with otherwise normal cardiac findings and no family history of a genetically transmitted abnormality associated with arrhythmia.

H. Acquired Cardiovascular Disease

Acquired cardiovascular disease occurs with systemic disease processes associated with inflammation, renal disease and related systemic hypertension, cardiotoxic drug therapy, pulmonary parenchymal disease, and after heart transplantation. Patients receiving anthracycline or other cardiotoxic agents should have baseline and re-evaluation follow-up studies. Echocardiographic assessment of patients with renal disease provides guidance in management of hemodialysis and hypertensive medications.

Echocardiography provides information for the common categories of acquired pediatric heart disease regarding acute and chronic changes in ventricular size, ventricular wall thickness, ventricular wall motion, ventricular systolic and diastolic function, ventricular wall stress, atrioventricular and semilunar valve anatomy and function, pericardial anatomy, and the presence of intracardiac masses.

The common categories of pediatric acquired heart disease are summarized as follows:

- Kawasaki disease can result in abnormalities of the coronary circulation, myocarditis, pericarditis, and myocardial infarction. Baseline and re-evaluations by echocardiography are recommended in all patients with clinical stigmata of this disease to guide management decisions (465-469). Since long-term abnormalities of the coronary arteries have been noted after resolution of initial aneurysms, these patients may require lifelong follow-up studies (470). Stress echocardiography may be a means to follow these patients serially and chronically (765-767).
- Endocarditis is encountered in the pediatric population with and without structural congenital heart disease. The increased use of central venous catheters for hemodynamic monitoring, parenteral alimentation, and chemotherapy expands the population at risk for endocarditis. Echocardiography identifies intracardiac masses and valve regurgitation associated with infectious valvulitis. Echocardiography offers supportive evidence for bacterial or rickettsial endocarditis but does not necessarily confirm or exclude the diagnosis. Children with suspected bacterial and rickettsial diseases associated with myocardial depression should have echocardiographic assessment of ventricular size and function, particularly because the acutely ill presentation of these disorders may mask the contribution of myocardial dysfunction to low cardiac output.
- Rheumatic fever is a persistent cause of acquired pediatric cardiac disease in the United States. Newer diagnostic criteria include echocardiographic assessment of mitral valve function, ventricular function, and pericarditis. Echocardiography is an important component of the diagnostic and sequential evaluation of children with fever, new cardiac murmur, migratory polyarthritis, and chorea (471,768).

- In children, HIV infection acquired during the fetal or newborn period is aggressive, with early and prominent myocardial involvement. Therefore, a baseline study and re-evaluation follow-up studies should be done as indicated by the appearance of tachycardia, congestive heart failure, and respiratory distress (470,769).
- Dilated cardiomyopathy with or without acute-onset congestive heart failure occurs in association with metabolic disorders after viral myopericarditis (473,474) or cardiotoxic chemotherapy. Frequently no etiology is identified to account for an occult dilated cardiomyopathy (475,476). Echocardiography identifies pericardial disease and myocardial dysfunction and permits surveillance of ventricular function during acute and convalescent phases of myocarditis. Identification of occult ventricular and atrial mural thrombi allows prompt anticoagulation therapy, possibly reducing further systemic morbidity. Echocardiographic follow-up of patients receiving cardiotoxic chemotherapy identifies at-risk subjects and helps guide subsequent therapy (477,478). Stress echocardiography may be useful in detecting subclinical LV dysfunction (770).
- Echocardiography is useful in detecting hypertrophic cardiomyopathy and determining the presence and nature of subaortic and subpulmonary obstruction, mitral insufficiency, and diastolic compliance abnormalities. Echocardiography is useful in screening family members for all types of cardiomyopathy associated with a dominant or recessive pattern of inheritance, eg, isolated noncompaction of the myocardium (771), and in screening patients with multisystem disorders associated with cardiomyopathy, eg, muscular dystrophy and Friedreich's ataxia (480,481). Re-evaluation studies measuring septal and ventricular wall thickness as well as systolic and diastolic function are required to monitor the sequelae of hypertrophic or dilated cardiomyopathies in the pediatric population. Hypertrophic cardiomyopathy also occurs in response to systemic hypertension (482) secondary to chronic renal disease, the Noonan syndrome, and obliterative arteriopathies and after cardiac transplantation (772-774). Echocardiographic surveillance of LV wall thickness, systolic function, and diastolic function permits appropriate adjustments in medical therapy (483,484). The leading cause of death after the first posttransplant year is transplant-related CAD. There is evidence that stress echocardiography identifies subclinical ischemia (773).

Recommendations for Echocardiography in Pediatric Acquired Cardiovascular Disease

Class I

- 1. Baseline studies and re-evaluation as clinically indicated on all pediatric patients with suspected or documented Kawasaki disease, myopericarditis, HIV, or rheumatic fever.**

- 2. After cardiac or cardiopulmonary transplant to monitor for signs of acute or chronic rejection, thrombus, and cardiac growth.**
- 3. Baseline and re-evaluation examinations of patients receiving cardiotoxic chemotherapeutic agents.**
- 4. Patients with clinical evidence of myocardial disease.**
- 5. Patients with severe renal disease and/or systemic hypertension.**
- 6. Donors undergoing evaluation for cardiac transplantation.**

Class IIa

An acutely ill child with suspected bacterial sepsis or rickettsial disease.

Class IIb

- 1. Follow-up examinations after acute rheumatic fever in patients with normal cardiac findings.**
- 2. A single late follow-up study after acute pericarditis with no evidence of recurrence or chronic pericardial disease.**

Class III

- 1. Routine screening echocardiogram for participation in competitive sports in patients with normal cardiovascular examination.**
- 2. Long-term follow-up studies in patients with Kawasaki disease who have no coronary abnormalities during the acute phase of the disease process.**

I. Pediatric Acquired Cardiopulmonary Cardiovascular Disease

Disease states in older infants and children with diseases that cause secondary pulmonary hypertension require documentation of pulmonary hypertension when there are suggestive clinical, electrocardiographic, or radiographic findings. These include bronchopulmonary dysplasia, adult-onset respiratory distress syndrome, cystic fibrosis, and chronic upperairway obstruction (775). Clinical expression of primary pulmonary artery hypertension in the pediatric population may initially include atypical fatigue, seizures and/or syncope without antecedent history of structural cardiopulmonary disease. Echocardiography provides documentation of pulmonary artery hypertension and estimation of severity by the presence of RV dilation or hypertrophy, the presence of tricuspid or pulmonic valvular regurgitation, and Doppler estimation of RV systolic pressure (776). Continuous intravenous epoprostenol therapy for patients with severe primary pulmonary hypertension has produced symptomatic and hemodynamic improvement as well as improved survival (777). Follow-up studies reflect response to medical and/or surgical therapy and are useful in guiding management.

Acquired cardiopulmonary disease in the pediatric population includes acute respiratory failure, idiopathic dilated cardiomyopathy, and septic shock, as well as low cardiac output syndrome after congenital heart surgery. Extracorporeal life support improves systemic oxygenation and perfusion during rapidly progressive, potentially self-limiting cardiopul-

monary failure (778,779). Echocardiography provides noninvasive indices of LV and atrioventricular valve function and RV systolic pressure to guide patient selection and separation from extracorporeal life support.

Recommendations for Echocardiography in Pediatric Acquired Cardiopulmonary Disease

Class I

1. Any patient with clinical findings of pulmonary artery hypertension.
2. Re-evaluation after surgical intervention or initiation of oral and/or parenteral vasodilator therapy for pulmonary artery hypertension.
3. Re-evaluation during withdrawal of extracorporeal cardiopulmonary support.

Class IIa

Baseline study of patients with cystic fibrosis and no findings of cor pulmonale.

J. Thrombus/Tumor

Stroke and other manifestations of thromboembolism that occur in childhood may result from intracardiac thrombus, tumor, or vegetation. In some groups of patients, long-term indwelling catheters in the central veins or atria may predispose to thrombus formation or infection. Because children have a lower incidence of peripheral vascular disease as a cause of stroke or loss of pulse, the yield of echocardiography in finding an intracardiac cause may be somewhat higher than for adults. Situations in which there is a high suspicion of intracardiac thrombus include late-onset arrhythmias after Fontan palliation of congenital heart disease (485), severe dilated cardiomyopathy or other causes of severely reduced ventricular function, noncompaction of the myocardium, and patients on ventricular assist or extracorporeal cardiopulmonary membrane oxygenation devices. In addition, the presence of aortic thrombus should be sought in neonates with transumbilical aortic catheters and the appearance of hypertension, low cardiac output, or renal failure (629).

Patients with longstanding indwelling catheters and evidence for sepsis, cyanosis, or right-heart failure should be screened for the presence of thrombus or vegetation on the catheter. The patient with intracardiac right-to-left shunting and indwelling catheter should be evaluated by echocardiography when there are suggestive symptoms or findings of systemic embolization.

Echocardiographic screening for cardiac tumor is indicated in the fetus, newborn, or child with clinical evidence or familial history of tuberous sclerosis (780,781). Screening in the second and third trimester of gestation as well as during infancy and again in childhood is warranted because this lesion may appear at any of these times. Older children and adolescents with evidence of peripheral embolization should be screened for the presence of myxoma.

Recommendations for Echocardiography in Pediatric Thromboembolic Disease States

Class I

1. Thromboembolic event in an infant, child, or adolescent.
2. Finding or family history of tuberous sclerosis.
3. Appearance of sepsis, cyanosis, or right-heart failure in a patient with a long-standing indwelling catheter.
4. Systemic embolization or acute-onset hypertension in a patient with right-to-left-shunting and an indwelling catheter.
5. Superior vena caval syndrome in the presence of central venous catheter.

Class IIb

Patient with indwelling catheter and fever but without evidence of pulmonary or systemic embolization.

Class III

Routine surveillance of asymptomatic patients with indwelling catheter.

K. Transesophageal Echocardiography

Transthoracic echocardiography, using high-frequency imaging probes and multiple parasternal, apical, suprasternal, and subcostal projections offers excellent resolution of intracardiac and paracardiac structures in the infant and young child. Transesophageal echocardiography, however, adds important clinical information regarding these structures in the older pediatric patient and in subjects of all ages during or after thoracic instrumentation. Because the potential for airway compromise and coexistence of complex gastroesophageal anomalies is increased in smaller patients, the procedure should only be performed by persons skilled in TEE and trained in the care of infants and children.

Transesophageal echocardiography has become particularly useful in the intraoperative management of neonates and children undergoing cardiovascular surgery. This is true for patients undergoing repair of shunts, valvular insufficiency, obstruction, and univentricular repairs (782-784). The development of smaller transesophageal echocardiographic probes has extended its use to the smaller neonates (785,786).

Transesophageal echocardiography may be used in concert with cardiac catheterization to limit the quantity of radiographic contrast material. This is indicated in the presence of significant pulmonary artery hypertension or in complex cases when an unsafe amount of radiographic contrast material would be required for adequate documentation of the lesion.

The placement of intracardiac and intravascular devices can be aided by echocardiographic guidance (781,782). Transesophageal echocardiography has become particularly helpful in guiding placement of catheter-deployed devices used in closing atrial septal defects. It is essential in ensuring proper positioning of the device in the defect and in determining whether there are residual shunts or abnormal device occlusion of venous inflow into the atria or encroachment on

the atrioventricular valves. Likewise, placement of catheters for radiofrequency ablation of arrhythmogenic pathways can be facilitated by TEE when there are intracardiac abnormalities (787).

Direct atrial-pulmonary and intracardiac and extracardiac caval-pulmonary Fontan palliations are associated with maladaptations to functional single ventricle physiology. Right atrial dilatation with pulmonary venous compression and right atrial venous stasis are associated with disturbances in pulmonary blood flow and atrial rhythm (788). Spontaneous closure of Fontan baffle fenestration results in elevated systemic venous pressure, reduced systemic cardiac output, and persistent effusions (789). Anomalies in coagulation (790) predispose patients with modified Fontan palliations to thrombotic events (791,792). Transthoracic echocardiographic imaging of caval-pulmonary channels is limited by their posterior location and anterior prosthetic material. Transesophageal echocardiography provides a retrocardiac acoustical window for assessing caval, atrial, and central pulmonary artery anatomy/flow and baffle fenestration patency as well as occult mural thrombi.

Recommendations for TEE in Pediatric Patients

Class I

1. **Any patient with congenital or acquired heart disease needing echocardiography when significant diagnostic information cannot be obtained by TTE.**
2. **Monitoring and guidance during cardiothoracic surgical procedures.**
3. **Guidance of catheter/device placement during interventional catheterization/radiofrequency ablation in patients with congenital heart disease.**
4. **Study of patients with intra-atrial baffle in whom the potential for thrombus is of concern because of elevated central venous pressures, atrial chamber dilation, increasing cyanosis, or the appearance of arrhythmia.**
5. **Patients with long-term placement of intravascular devices in whom thrombus or vegetation is suspected.**
6. **Patients with a prosthetic valve in whom thrombus or vegetation is suspected.**
7. **Any patient with suspected endocarditis and inadequate transthoracic acoustical window.**
8. **Patients with right atrial to pulmonary artery Fontan connection for identification of atrial thrombus.**

Class IIa

Patients with lateral tunnel Fontan palliation.

Class III

1. **Performing TEE in a patient who has not previously had careful study by TTE.**
2. **Patients with structural esophageal abnormality.**

L. Fetal Echocardiography

Widespread use of general fetal ultrasound examinations among women receiving prenatal care has resulted in increased referrals for specific cardiac analysis. Definition of

fetal cardiac structures is currently possible at 10 to 12 weeks of gestation with the use of vaginal probes with high-resolution transducers (793). By 16 to 18 weeks, accurate segmental analysis of cardiac structure is possible with a conventional transabdominal approach at the current state of technology (494,495). Doppler examination provides important information about blood flow across the cardiac valves, great arteries, ductus arteriosus, and umbilical arteries (496). A general fetal ultrasound examination usually includes a four-chamber or inflow view of the fetal heart (497). This view is sensitive to abnormalities of the inflow portions of the heart but is insensitive to some septal defects, outflow lesions, and conotruncal abnormalities (498,794). Patients are referred for specific fetal echocardiographic examination because of an abnormality of structure or rhythm noted on ultrasound examination or because the patient is in a high-risk group for fetal heart disease (499-502). Early recognition of fetal heart disease allows the opportunity for transplacental therapy, as in the case of arrhythmias (503-505). When a potentially life-threatening cardiac anomaly is found (506-508), the delivery can be planned at a tertiary care center where supportive measures can be instituted before severe hypoxia, shock, or acidosis ensues (509). The effect of antenatal diagnosis of life-threatening congenital heart disease on surgical outcome is multifactorial. Conflicting observations regarding the impact of prenatal diagnosis on surgical outcome for hypoplastic left heart syndrome and D transposition of the great arteries have been reported. The experience in larger series suggests that prenatal diagnosis of life-threatening congenital heart disease improves preoperative condition and surgical outcome (795,796). Prenatal diagnosis of life-threatening cardiovascular anomalies permits early education of the parents so that complex therapeutic choices can be reviewed and informed consent obtained (510-512).

Antenatal diagnosis of congenital heart disease can be influenced by the palliative effect of fetal circulation and morphometric changes in the heart and great vessels occurring throughout gestation. The severity of pulmonary stenosis cannot be assessed by quantitation of valve gradient because of the variability in RV output and the patency of the ductus arteriosus. The outcome of fetal heart disease is often suggested only after re-evaluation studies to determine growth of cardiac chambers and vascular structures and changes in blood flow patterns (797). The spectrum of antenatal cardiac lesions is broader than that seen in neonates and infants because of the presence of nonviable subcategories of congenital heart disease. A knowledge of prenatal maternal history (513,798) is as necessary as good imaging in providing proper antenatal and postnatal care to the mother, fetus, and neonate.

In skilled hands the diagnostic accuracy of fetal echocardiography may reach the high sensitivity and specificity of echocardiography in the neonate; however, not all pediatric cardiology centers have specially trained fetal echocardiographers (514). Such experts may be pediatric cardiologists, obstetricians, or radiologists with special training or experience in fetal ultrasound imaging and a comprehensive

knowledge of congenital heart disease, fetal cardiac anatomy and physiology, and arrhythmias. When specific expertise in fetal echocardiography does not exist, close collaboration between a pediatric cardiologist/echocardiographer and a fetal ultrasonographer may produce similar results once a learning curve has been completed. The collaboration of a multidisciplinary perinatal team provides support for diagnostic and therapeutic decisions.

Recommendations for Fetal Echocardiography

Class I

1. **Abnormal-appearing heart on general fetal ultrasound examination.**
2. **Fetal tachycardia, bradycardia, or persistent irregular rhythm on clinical or screening ultrasound examination.**
3. **Maternal/family risk factors for cardiovascular disease, such as a parent, sibling, or first-degree relative with congenital heart disease.**
4. **Maternal diabetes.**
5. **Maternal systemic lupus erythematosus.**
6. **Teratogen exposure during a vulnerable period.**
7. **Other fetal system abnormalities (including chromosomal).**
8. **Performance of transplacental therapy or presence of a history of significant but intermittent arrhythmia. Re-evaluation examinations are required in these conditions.**

Class IIa

Fetal distress or dysfunction of unclear etiology.

Class IIb

1. **Previous history of multiple fetal losses.**
2. **Multiple gestation.**

Class III

1. **Low-risk pregnancies with normal anatomic findings on ultrasound examination.**
2. **Occasional premature contractions without sustained tachycardia or signs of dysfunction or distress.**
3. **Presence of a noncardiovascular system abnormality when evaluation of the cardiovascular system will not alter either management decisions or fetal outcome.**

XVI. INTRAOPERATIVE ECHOCARDIOGRAPHY

Over the past 15 years, the application of intraoperative echocardiography (IOE) has grown enormously, and it is now used routinely in most cardiac surgical centers in North America. Although its usefulness often seems obvious to its users, the demonstration of its impact on patient outcomes remains a significant challenge. In 1996, a task force of the American Society of Anesthesiologists/Society of Cardiovascular Anesthesiologists (ASA/SCA) published practice guidelines for perioperative TEE (799). The guidelines were

evidence-based and focused on the effectiveness of perioperative TEE in improving clinical outcomes. A literature search conducted at that time retrieved 1844 articles, of which 588 were considered relevant to the perioperative setting. A more recent literature search identified an additional 118 articles related to the intraoperative use of echocardiography. The current text makes reference only to the latter. However, the recommendations for IOE that are provided in these guidelines are based on the initial ASA/SCA guidelines as well as the newer information.

A. General Usefulness in Cardiac Surgery

1. Adult Surgery

Several recent studies have reported on the usefulness of IOE in adult cardiac surgery (800-804). The studies have usually examined whether IOE yielded new information and how frequently the new information had an impact on anesthetic or surgical management (Table 23). The incidence of new information ranged from 12.8% to 38.6%, whereas the impact on treatment ranged from 9.7% to 14.6%.

Intraoperative TEE is, however, not without risks. Hogue *et al.* studied independent predictors of swallowing dysfunction after cardiac surgery (805). In addition to age and length of intubation after surgery, intraoperative use of TEE was a highly significant (P less than 0.003) predictor of swallowing dysfunction. In another study of 838 consecutive cardiac surgical patients, significant factors causing postoperative dysphagia were studied by multiple logistic regression (806). After controlling for other significant factors such as stroke, left ventricular ejection fraction, intubation time, and duration of operation, the patients with intraoperative TEE had 7.8 times greater odds of dysphagia than those without. In a more recent case series of intraoperative TEE performed in 7200 cardiac surgical patients, no mortality and a morbidity of only 0.2% were observed (807).

2. Pediatric Surgery

As for adult cardiac surgery, the use of IOE has become routine in many pediatric cardiac surgery centers. Whereas epicardial echocardiography was used most commonly in the early years, the use of TEE has increased with the development of smaller TEE probes. Several recent studies have documented the utility of intraoperative TEE, particularly for the detection of residual defects after cardiopulmonary bypass (CPB) (808-811). The detection of significant residual defects after CPB ranged from 4.4% to 12.8% (Table 24).

Greene *et al.* evaluated the safety of TEE in pediatric cardiac surgery by performing an endoscopic examination of the esophagus after TEE (812). In 50 patients undergoing repair of congenital cardiac defects, the endoscopic examination was performed after removal of the TEE probe. In 32 patients, mild mucosal injury was observed, but none resulted in long-term feeding or swallowing difficulties.

Table 23. Usefulness of Intraoperative Echocardiography in Adult Cardiac Surgery

Author (Ref)	Year	N	New Information	Change in Management
Click (800)	2000	3245	15%	14%
Couture (802)	2000	851	—	14.6%
Michel-Cherqui (803)	2000	203	12.8%	10.8%
Mishra (801)	1998	5016	22.9%	—
Sutton (804)	1998	238	38.6%	9.7%

B. Usefulness in Specific Lesions or Procedures

1. Adult Cardiac Surgery

a. Mitral Valve Repair

Intraoperative echocardiography is used extensively in mitral valve repair. It allows the detailed evaluation of mitral valvular pathology at the time of surgery, the intraoperative recognition of systolic anterior motion after repair, and the assessment of residual regurgitation. Two recent studies from Japan have confirmed the usefulness of intraoperative TEE for the assessment of residual regurgitation after mitral valve repair (813,814). In one study, it was observed that 5 of 34 patients had 1+ regurgitation on postoperative ventriculography. Four of these patients demonstrated a maximal mosaic area greater than 2 cm² on color flow Doppler by TEE immediately after cardiopulmonary bypass. They all developed rapidly progressing mitral regurgitation in the postoperative period (813). In another study, 40 of 42 patients with no or trivial mitral regurgitation (mosaic area less than or equal to 2 cm²) also had no or trivial MR early and late postoperatively (814). The two other patients in whom no or trivial mitral regurgitation was detected intraoperatively by TEE evolved to moderate regurgitation 3 months later.

b. Valve Replacement

Morehead et al. studied the significance of paravalvular jets detected by IOE after valve replacement (815). In 27 patients, multiple jets were detected after valve replacement. They were more common and larger in the mitral position and after insertion of mechanical valves. Reversal of anticoagulation with protamine reduced the incidence and size of the jets in all patients.

c. Ischemic Heart Disease

Bergquist et al. studied how TEE guides clinical decision making in myocardial revascularization (816). Among the

Table 24. Usefulness of Intraoperative Echocardiography in Pediatric Cardiac Surgery

Author (Ref)	Year	N	Residual Defects
Rosenfeld (808)	1998	86	12.8%
Sheil (809)	1999	200	10.5%
Stevenson (810)	1995	667	6.6%
Ungerleider (811)	1995	1000	4.4%

584 intraoperative interventions that were recorded, TEE was the single most important guiding factor in 98 instances (17%). TEE was the single most important monitor influencing fluid administration, anti-ischemic therapy, vasoactive medications, inotropes, and antiarrhythmic therapy. In two patients, critical surgical interventions were made solely on the basis of TEE. In high-risk coronary artery bypass grafting (CABG), Savage et al. observed that in 33% of patients, at least one major surgical management alteration was initiated on the basis of TEE whereas in 51% of patients, at least one major anesthetic/hemodynamic change was initiated by a TEE finding (817).

Arruda et al. evaluated the role of power Doppler imaging to assess the patency of CABG anastomosis (818). In 11 of 12 patients, the flow in the left anterior descending coronary artery could be visualized before and after the anastomosis. In one patient, the graft was revised because of worsened flow after CPB.

d. Minimally Invasive Cardiac Surgery

With the growing interest in minimally invasive cardiac surgery, the role of IOE in these procedures has been evaluated. Applebaum et al. reported that TEE facilitated the placement of intravascular catheters during port-access surgery, thereby avoiding the use of fluoroscopy (819). Fluoroscopy was only helpful as an aide to TEE for placement of the coronary sinus catheter. Falk et al. observed that TEE was particularly useful for monitoring the placement and positioning of the endoaortic clamp that is used in these procedures (820). In patients undergoing coronary bypass without CPB, Moises et al. detected 31 new regional wall motion abnormalities during 48 coronary artery clampings (821). At the time of chest closure, 16 segments had partial recovery, and 5 of these had not recovered. Seven days later, the regional wall motion abnormalities persisted in the five without recovery and in two with partial recovery. These patients had more clinical problems postoperatively. In minimally invasive valve surgery, Secknus et al. noted intracardiac air in all patients (822). New LV dysfunction was more common in patients with extensive air by IOE. Second CPB runs were required in 6% of patients.

e. Air Embolization

In a study of 20 patients undergoing CABG, Yao et al. observed intraluminal aortic air emboli in all patients (823). Although embolization was unevenly distributed throughout the procedure, 42% of emboli were detected within 4 min-

utes of aortic cross-clamp release and 24% after partial occlusion clamp release. Tingleff *et al.* studied two groups of 15 patients: group I consisted of patients undergoing true open heart procedures, whereas patients in group II underwent CABG (824). Air embolism was detected in all patients in group I, with episodes occurring up to 28 minutes after termination of CPB. In most cases, TEE clearly demonstrated that the air originated in the lung veins and was not retained in the heart. For patients in group II, air embolism was noted in only half and was seen only in the period between cross-clamp removal and termination of CPB.

f. Aortic Atheromatous Disease

The relationship between the severity of aortic atheromatous disease and postoperative dysfunction has been established previously. Choudhary *et al.* documented severe atheromatous disease in 12 of 126 patients undergoing CABG (825). Protruding atheromas were significantly more common in patients over 60 years of age. Of 4 patients with grade V atheromas, 2 developed right hemiplegia postoperatively. To determine the optimal method to detect ascending aortic atheromas intraoperatively, manual palpation, TEE, and epiaortic scanning were compared in 100 patients (826). Age older than 70 years and hypertension were significant risk factors for severe ascending atheromas. Epiaortic scanning was found to be superior to both manual palpation and TEE.

2. Pediatric Cardiac Surgery

a. Mitral Regurgitation

Lee *et al.* studied the validity of intraoperative TEE for predicting the degree of MR at follow-up in 47 patients with atrioventricular defects (827). Intraoperative TEE was useful in detecting severe MR that required further repair at the same time. In 21 of the patients, however, there was a discrepancy between the intraoperative and follow-up grades of MR. The authors noted that blood pressures were significantly lower and heart rates significantly higher intraoperatively.

b. Aortic Regurgitation

Fourteen patients who underwent repair of ventricular septal defect with aortic regurgitation were studied by intraoperative TEE (828). The severity of prolapse of each aortic cusp and its adjacent sinus was assessed. The valvar regurgitation was quantified by Doppler-derived regurgitant indices. TEE detected prolapse of the aortic valve and its sinus in all patients. On the basis of the TEE findings, an aortic valve exploration was executed in 12 patients. No residual aortic regurgitation was observed after CPB, but a residual ventricular septal defect was detected in 5 patients.

c. Transposition of the Great Vessels

Less than perfect coronary artery translocation accounts for the majority of perioperative deaths after the arterial switch

procedure for transposition of the great vessels. Shankar *et al.* used epicardial echocardiography to study four neonates with a failing left ventricle or difficulty of weaning from CPB (829). In 2 patients, coronary arterial problems in the form of kinking of the proximal left coronary artery and extrinsic compression of the artery by the neopulmonary trunk were identified and corrected. In 2 patients, supravalvar aortic stenosis was recognized, leading to prompt revision.

d. Patent Ductus Arteriosus Interruption

The efficacy of intraoperative TEE in reducing the incidence of residual ductal flow after video-assisted thoracoscopic patent ductus arteriosus interruption was studied by Lavoie *et al.* (830). In 2 of 30 consecutive patients (mean age 2.4 years; mean weight 11.2 kg), intraoperative TEE detected residual flow after placement of the vascular clip, requiring placement of a second clip. At one-month follow-up, three patients had residual duct flow.

Recommendations for Intraoperative Echocardiography

Class I

- 1. Evaluation of acute, persistent, and life-threatening hemodynamic disturbances in which ventricular function and its determinants are uncertain and have not responded to treatment.**
- 2. Surgical repair of valvular lesions, hypertrophic obstructive cardiomyopathy, and aortic dissection with possible aortic valve involvement.**
- 3. Evaluation of complex valve replacements requiring homografts or coronary reimplantation, such as the Ross procedure.**
- 4. Surgical repair of most congenital heart lesions that require CPB.**
- 5. Surgical intervention for endocarditis when preoperative testing was inadequate or extension to perivalvular tissue is suspected.**
- 6. Placement of intracardiac devices and monitoring of their position during port-access and other cardiac surgical interventions.**
- 7. Evaluation of pericardial window procedures in patients with posterior or loculated pericardial effusions.**

Class IIa

- 1. Surgical procedures in patients at increased risk of myocardial ischemia, myocardial infarction, or hemodynamic disturbances.**
- 2. Evaluation of valve replacement, aortic atheromatous disease, the Maze procedure, cardiac aneurysm repair, removal of cardiac tumors, intracardiac thrombectomy, and pulmonary embolectomy.**
- 3. Detection of air emboli during cardiectomy, heart transplant operations, and upright neurosurgical procedures.**

Class IIb

1. Evaluation of suspected cardiac trauma, repair of acute thoracic aortic dissection without valvular involvement, and anastomotic sites during heart and/or lung transplantation.
2. Evaluation of regional myocardial function during and after off-pump CABG procedures.
3. Evaluation of pericardiectomy, pericardial effusions, and pericardial surgery.
4. Evaluation of myocardial perfusion, coronary anatomy, or graft patency.
5. Dobutamine stress testing to detect inducible demand ischemia or to predict functional changes after myocardial revascularization.
6. Assessment of residual duct flow after interruption of patent ductus arteriosus (831).

Class III

Surgical repair of uncomplicated secundum atrial septal defect.

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Appendix. ACC/AHA/ASE Writing Committee to Update the 1997 Guidelines on the Clinical Application of Echocardiography—Relationships with Industry

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This table represents the actual or potential relationships with industry that were reported orally at the initial writing committee conference call in April 2000 and updated in conjunction with all meetings and conference calls of the writing committee. It does not reflect any actual or potential relationships with industry at the time of publication.

ORIGINAL REFERENCES

- Fryback DG, Thornbury JR. The efficacy of diagnostic imaging. *Med Decis Making*. 1991;11:88-94.
- Guyatt GH, Tugwell PX, Feeny DH, Haynes RB, Drummond M. A framework for clinical evaluation of diagnostic technologies. *Can Med Assoc J*. 1986;134:587-94.
- Douglas PS. Justifying echocardiography: the role of outcomes research in evaluating a diagnostic test. *J Am Soc Echocardiogr*. 1996;9:577-81.
- Deleted during update.
- Cunio RE, Natanson C. Echocardiography, pulmonary artery catheterization, and radionuclide cineangiography in septic shock. *Intensive Care Med*. 1994;20:535-7.
- Peller OG, Wallerson DC, Devereux RB. Role of Doppler and imaging echocardiography in selection of patients for cardiac valvular surgery. *Am Heart J*. 1987;114:1445-61.
- Smith MD. Evaluation of valvular regurgitation by Doppler echocardiography. *Cardiol Clin*. 1991;9:193-228.
- Richards KL. Doppler echocardiography in the diagnosis and quantification of valvular heart disease. *Mod Concepts Cardiovasc Dis*. 1987;56:43-8.
- Aurigemma G, Reichek N, Schiebler M, Axel L. Evaluation of aortic regurgitation by cardiac cine magnetic resonance imaging: planar analysis and comparison to Doppler echocardiography. *Cardiology*. 1991;78:340-7.
- Kilner PJ, Manzara CC, Mohiaddin RH, et al. Magnetic resonance jet velocity mapping in mitral and aortic valve stenosis. *Circulation*. 1993;87:1239-48.
- Miyake T, Yokoyama T. Evaluation of transient heart murmur resembling pulmonary artery stenosis in term infants by Doppler and M-mode echocardiography. *Jpn Circ J*. 1993;57:77-83.
- Auerback ML. High tech in a low tech country. *West J Med*. 1993;159:93-4.
- Mishra M, Chambers JB, Jackson G. Murmurs in pregnancy: an audit of echocardiography. *BMJ*. 1992;304:1413-4.
- Northcote RJ, Knight PV, Ballantyne D. Systolic murmurs in pregnancy: value of echocardiographic assessment. *Clin Cardiol*. 1985;8:327-8.
- Fink JC, Schmid CH, Selker HP. A decision aid for referring patients with systolic murmurs for echocardiography. *J Gen Intern Med*. 1994;9:479-84.
- Deleted during update.
- Xu M, McHaffie DJ. Nonspecific systolic murmurs: an audit of the clinical value of echocardiography. *N Z Med J*. 1993;106:54-6.
- McKillop GM, Stewart DA, Burns JM, Ballantyne D. Doppler echocardiography in elderly patients with ejection systolic murmurs. *Postgrad Med J*. 1991;67:1059-61.
- Smythe JF, Teixeira OH, Vlad P, Demers PP, Feldman W. Initial evaluation of heart murmurs: are laboratory tests necessary? *Pediatrics*. 1990;86:497-500.
- Martin RP, Rakowski H, Kleiman JH, Beaver W, London E, Popp RL. Reliability and reproducibility of two dimensional echocardiograph measurement of the stenotic mitral valve orifice area. *Am J Cardiol*. 1979;43:560-8.
- Stamm RB, Martin RP. Quantification of pressure gradients across stenotic valves by Doppler ultrasound. *J Am Coll Cardiol*. 1983;2:707-18.
- Hatle L, Angelsen B, Tromsdal A. Noninvasive assessment of atri-ventricular pressure half-time by Doppler ultrasound. *Circulation*. 1979;60:1096-104.
- Thomas JD, Wilkins GT, Choong CY, et al. Inaccuracy of mitral pressure half-time immediately after percutaneous mitral valvotomy. Dependence on transmitral gradient and left atrial and ventricular compliance. *Circulation*. 1988;78:980-93.
- Cherix EC, Pieters FA, Janssen JH, de Swart H, Palmans-Meulemans A. Value of exercise Doppler-echocardiography in patients with mitral stenosis. *Int J Cardiol*. 1994;45:219-26.
- Gordon SP, Douglas PS, Come PC, Manning WJ. Two-dimensional and Doppler echocardiographic determinants of the natural history of mitral valve narrowing in patients with rheumatic mitral stenosis: implications for follow-up. *J Am Coll Cardiol*. 1992;19:968-73.
- Wilkins GT, Weyman AE, Abascal VM, Block PC, Palacios IF. Percutaneous balloon dilatation of the mitral valve: an analysis of echocardiographic variables related to outcome and the mechanism of dilatation. *Br Heart J*. 1988;60:299-308.
- Goldstein SA, Campbell A, Mintz GS, Pichard A, Leon M, Lindsay J Jr. Feasibility of on-line transesophageal echocardiography during balloon mitral valvulotomy: experience with 93 patients. *J Heart Valve Dis*. 1994;3:136-48.
- Perez JE, Ludbrook PA, Ahumada GG. Usefulness of Doppler echocardiography in detecting tricuspid valve stenosis. *Am J Cardiol*. 1985;55:601-3.
- Otto CM, Pearlman AS. Doppler echocardiography in adults with symptomatic aortic stenosis. Diagnostic utility and cost-effectiveness. *Arch Intern Med*. 1988;148:2553-60.
- Otto CM, Davis KB, Holmes DR, et al. Methodologic issues in clinical evaluation of stenosis severity in adults undergoing aortic or mitral balloon valvuloplasty. The NHLBI Balloon Valvuloplasty Registry. *Am J Cardiol*. 1992;69:1607-16.
- Burwash IG, Pearlman AS, Kraft CD, Miyake-Hull C, Healy NL, Otto CM. Flow dependence of measures of aortic stenosis severity during exercise. *J Am Coll Cardiol*. 1994;24:1342-50.
- deFilippi CR, Willet 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;75:191-4.
- Nakatani S, Imanishi T, Terasawa A, Beppu S, Nagata S, Miyatake K. Clinical application of transpulmonary contrast-enhanced Doppler technique in the assessment of severity of aortic stenosis. *J Am Coll Cardiol*. 1992;20:973-8.
- Sahn DJ, Maciel BC. Physiological valvular regurgitation. Doppler echocardiography and the potential for iatrogenic heart disease. *Circulation*. 1988;78:1075-7.
- Yoshida K, Yoshikawa J, Shakudo M, et al. Color Doppler evaluation of valvular regurgitation in normal subjects. *Circulation*. 1988;78:840-7.
- Rahko PS. Prevalence of regurgitant murmurs in patients with valvular regurgitation detected by Doppler echocardiography. *Ann Intern Med*. 1989;111:466-72.
- Smith MD, Grayburn PA, Spain MG, DeMaria AN. Observer variability in the quantitation of Doppler color flow jet areas for mitral and aortic regurgitation. *J Am Coll Cardiol*. 1988;11:579-84.
- Yoshikawa J, Yoshida K, Akasaka T, Shakudo M, Kato H. Value and limitations of color Doppler flow mapping in the detection and semiquantification of valvular regurgitation. *Int J Card Imaging*. 1987;2:85-91.
- Wilkenshoff UM, Kruck I, Gast D, Schroder R. Validity of continuous wave Doppler and colour Doppler in the assessment of aortic regurgitation. *Eur Heart J*. 1994;15:1227-34.
- Grayburn PA, Fehske W, Omran H, Brickner ME, Luderitz B.

- Multiphase transesophageal echocardiographic assessment of mitral regurgitation by Doppler color flow mapping of the vena contracta. *Am J Cardiol.* 1994;74:912-7.
41. Rivera JM, Vandervoort PM, Mele D, et al. Quantification of tricuspid regurgitation by means of the proximal flow convergence method: a clinical study. *Am Heart J.* 1994;127:1354-62.
 42. Xie GY, Berk MR, Smith MD, DeMaria AN. A simplified method for determining regurgitant fraction by Doppler echocardiography in patients with aortic regurgitation. *J Am Coll Cardiol.* 1994;24:1041-5.
 43. Enriquez-Sarano M, Seward JB, Bailey KR, Tajik AJ. Effective regurgitant orifice area: a noninvasive Doppler development of an old hemodynamic concept. *J Am Coll Cardiol.* 1994;23:443-51.
 44. Chen C, Koschyk D, Brockhoff C, et al. Noninvasive estimation of regurgitant flow rate and volume in patients with mitral regurgitation by Doppler color mapping of accelerating flow field. *J Am Coll Cardiol.* 1993;21:374-83.
 45. Cohen GI, Davison MB, Klein AL, Salcedo EE, Stewart WJ. A comparison of flow convergence with other transthoracic echocardiographic indexes of prosthetic mitral regurgitation. *J Am Soc Echocardiogr.* 1992;5:620-7.
 46. Teague SM, Heinsimer JA, Anderson JL, et al. Quantification of aortic regurgitation utilizing continuous wave Doppler ultrasound. *J Am Coll Cardiol.* 1986;8:592-9.
 47. Labovitz AJ, Ferrara RP, Kern MJ, Bryg RJ, Mrosek DG, Williams GA. Quantitative evaluation of aortic insufficiency by continuous wave Doppler echocardiography. *J Am Coll Cardiol.* 1986;8:1341-7.
 48. Klein AL, Obarski TP, Stewart WJ, et al. Transesophageal Doppler echocardiography of pulmonary venous flow: a new marker of mitral regurgitation severity. *J Am Coll Cardiol.* 1991;18:518-26.
 49. Rosen SE, Borer JS, Hochreiter C, et al. Natural history of the asymptomatic/minimally symptomatic patient with severe mitral regurgitation secondary to mitral valve prolapse and normal right and left ventricular performance. *Am J Cardiol.* 1994;74:374-80.
 50. Bonow RO, Lakatos E, Maron BJ, Epstein SE. Serial long-term assessment of the natural history of asymptomatic patients with chronic aortic regurgitation and normal left ventricular systolic function. *Circulation.* 1991;84:1625-35.
 51. Enriquez-Sarano M, Tajik AJ, Schaff HV, Orszulak TA, Bailey KR, Frye RL. Echocardiographic prediction of survival after surgical correction of organic mitral regurgitation. *Circulation.* 1994;90:830-7.
 52. Stewart WJ, Currie PJ, Salcedo EE, et al. Evaluation of mitral leaflet motion by echocardiography and jet direction by Doppler color flow mapping to determine the mechanisms of mitral regurgitation. *J Am Coll Cardiol.* 1992;20:1353-61.
 53. Levine RA, Stathogiannis E, Newell JB, Harrigan P, Weyman AE. Reconsideration of echocardiographic standards for mitral valve prolapse: lack of association between leaflet displacement isolated to the apical four chamber view and independent echocardiographic evidence of abnormality. *J Am Coll Cardiol.* 1988;11:1010-9.
 54. Nishimura RA, McGoon MD, Shub C, Miller FA, Ilstrup DM, Tajik AJ. Echocardiographically documented mitral-valve prolapse. Long-term follow-up of 237 patients. *N Engl J Med.* 1985;313:1305-9.
 55. Zuppiroli A, Mori F, Favilli S, et al. Arrhythmias in mitral valve prolapse: relation to anterior mitral leaflet thickening, clinical variables, and color Doppler echocardiographic parameters. *Am Heart J.* 1994;128:919-27.
 56. Babuty D, Cosnay P, Breuillac JC, et al. Ventricular arrhythmia factors in mitral valve prolapse. *PACE Pacing Clin Electrophysiol.* 1994;17:1090-9.
 57. Takamoto T, Nitta M, Tsujibayashi T, Taniguchi K, Marumo F. The prevalence and clinical features of pathologically abnormal mitral valve leaflets (myxomatous mitral valve) in the mitral valve prolapse syndrome: an echocardiographic and pathological comparative study. *J Cardiol Suppl.* 1991;25:75-86.
 58. Marks AR, Choong CY, Sanfilippo AJ, Ferre M, Weyman AE. Identification of high-risk and low-risk subgroups of patients with mitral-valve prolapse. *N Engl J Med.* 1989;320:1031-6.
 59. Chandraratna PAN, Nimalasuriya A, Kawanishi D, Duncan P, Rosin B, Rahimtoola SH. Identification of the increased frequency of cardiovascular abnormalities associated with mitral valve prolapse by two-dimensional echocardiography. *Am J Cardiol.* 1984;54:1283-5.
 60. Daniel WG, Mugge A, Martin RP, et al. Improvement in the diagnosis of abscesses associated with endocarditis by transesophageal echocardiography. *N Engl J Med.* 1991;324:795-800.
 61. Shapiro SM, Young E, De Guzman S, et al. Transesophageal echocardiography in diagnosis of infective endocarditis. *Chest.* 1994;105:377-82.
 62. Mugge A, Daniel WG, Frank G, Lichtlen PR. Echocardiography in infective endocarditis: reassessment of prognostic implications of vegetation size determined by the transthoracic and the transesophageal approach. *J Am Coll Cardiol.* 1989;14:631-8.
 63. Rubenson DS, Tucker CR, Stinson EB, et al. The use of echocardiography in diagnosing culture-negative endocarditis. *Circulation.* 1981;64:641-6.
 64. Heinle S, Wilderman N, Harrison JK, et al. Value of transthoracic echocardiography in predicting embolic events in active infective endocarditis. Duke Endocarditis Service. *Am J Cardiol.* 1994;74:799-801.
 65. Sanfilippo AJ, Picard MH, Newell JB, et al. Echocardiographic assessment of patients with infectious endocarditis: prediction of risk for complications. *J Am Coll Cardiol.* 1991;18:1191-9.
 66. Hecht SR, Berger M. Right-sided endocarditis in intravenous drug users. Prognostic features in 102 episodes. *Ann Intern Med.* 1992;117:560-6.
 67. Rohmann S, Erbel R, Gorge G, et al. Clinical relevance of vegetation localization by transoesophageal echocardiography in infective endocarditis. *Eur Heart J.* 1992;13:446-52.
 68. Vuille C, Nidorf M, Weyman AE, Picard MH. Natural history of vegetations during successful medical treatment of endocarditis. *Am Heart J.* 1994;128:1200-9.
 69. Lindner JR, Case RA, Dent JM, Abbott RD, Scheld WM, Kaul S. Diagnostic value of echocardiography in suspected endocarditis. An evaluation based on the pretest probability of disease. *Circulation.* 1996;93:730-6.
 70. Werner GS, Schulz R, Fuchs JB, et al. Infective endocarditis in the elderly in the era of transesophageal echocardiography: clinical features and prognosis compared with younger patients. *Am J Med.* 1996;100:90-7.
 71. Job FP, Franke S, Lethen H, Flachskampf FA, Hanrath P. Incremental value of biplane and multiphase transesophageal echocardiography for the assessment of active infective endocarditis. *Am J Cardiol.* 1995;75:1033-7.
 72. Lowry RW, Zoghbi WA, Baker WB, Wray RA, Quinones MA. Clinical impact of transesophageal echocardiography in the diagnosis and management of infective endocarditis. *Am J Cardiol.* 1994;73:1089-91.
 73. Birmingham GD, Rahko PS, Ballantyne F III. Improved detection

- of infective endocarditis with transesophageal echocardiography. *Am Heart J.* 1992;123:774-81.
74. Watanakunakorn C, Burkert T. Infective endocarditis at a large community teaching hospital, 1980-1990. A review of 210 episodes. *Medicine (Baltimore).* 1993;72:90-102.
 75. Shively BK, Gurule FT, Roldan CA, Leggett JH, Schiller NB. Diagnostic value of transesophageal compared with transthoracic echocardiography in infective endocarditis. *J Am Coll Cardiol.* 1991;18:391-7.
 76. Burger AJ, Peart B, Jabi H, Touchon RC. The role of two-dimensional echocardiography in the diagnosis of infective endocarditis [corrected] [published erratum appears in *Angiology.* 1991;42:765]. *Angiology.* 1991;42:552-60.
 77. Mohr-Kahaly S, Kupferwasser I, Erbel R, et al. Value and limitations of transesophageal echocardiography in the evaluation of aortic prostheses. *J Am Soc Echocardiogr.* 1993;6:12-20.
 78. Reisner SA, Meltzer RS. Normal values of prosthetic valve Doppler echocardiographic parameters: a review. *J Am Soc Echocardiogr.* 1988;1:201-10.
 79. Come PC. Pitfalls in the diagnosis of periprosthetic valvular regurgitation by pulsed Doppler echocardiography. *J Am Coll Cardiol.* 1987;9:1176-9.
 80. Gaasch WH. Diagnosis and treatment of heart failure based on left ventricular systolic or diastolic dysfunction. *JAMA.* 1994;271:1276-80.
 81. Byrd BF III, O'Kelly BF, Schiller NB. Contrast echocardiography enhances tricuspid but not mitral regurgitation. *Clin Cardiol.* 1991;14:V10-4.
 82. Terasawa A, Miyatake K, Nakatani S, Yamagishi M, Matsuda H, Beppu S. Enhancement of Doppler flow signals in the left heart chambers by intravenous injection of sonicated albumin. *J Am Coll Cardiol.* 1993;21:737-42.
 83. Horowitz RS, Morganroth J, Parrotto C, Chen CC, Soffer J, Pauletto FJ. Immediate diagnosis of acute myocardial infarction by two-dimensional echocardiography. *Circulation.* 1982;65:323-9.
 84. Sabia P, Afrookteh A, Touchstone DA, Keller MW, Esquivel L, Kaul S. Value of regional wall motion abnormality in the emergency room diagnosis of acute myocardial infarction. A prospective study using two-dimensional echocardiography. *Circulation.* 1991;84(suppl I):I-85-92.
 85. Peels CH, Visser CA, Kupper AJ, Visser FC, Roos JP. Usefulness of two-dimensional echocardiography for immediate detection of myocardial ischemia in the emergency room. *Am J Cardiol.* 1990;65:687-91.
 86. Heidenreich PA, Stainback RF, Redberg RF, Schiller NB, Cohen NH, Foster E. Transesophageal echocardiography predicts mortality in critically ill patients with unexplained hypotension. *J Am Coll Cardiol.* 1995;26:152-8.
 87. Foster E, Schiller NB. Transesophageal echocardiography in the critical care patient. *Cardiol Clin.* 1993;11:489-503.
 88. Sohn DW, Shin GJ, Oh JK, et al. Role of transesophageal echocardiography in hemodynamically unstable patients. *Mayo Clin Proc.* 1995;70:925-31.
 89. Cox D, Taylor J, Nanda NC. Refractory hypoxemia in right ventricular infarction from right-to-left shunting via a patent foramen ovale: efficacy of contrast transesophageal echocardiography. *Am J Med.* 1991;91:653-5.
 90. Heger JJ, Weyman AE, Wann LS, Rogers EW, Dillon JC, Feigenbaum H. Cross-sectional echocardiographic analysis of the extent of left ventricular asynergy in acute myocardial infarction. *Circulation.* 1980;61:1113-8.
 91. Gibson RS, Bishop HL, Stamm RB, Crampton RS, Beller GA, Martin RP. Value of early two dimensional echocardiography in patients with acute myocardial infarction. *Am J Cardiol.* 1982;49:1110-9.
 92. Kerber RE, Abboud FM. Echocardiographic detection of regional myocardial infarction: an experimental study. *Circulation.* 1973;47:997-1005.
 93. Weiss JL, Bulkley BH, Hutchins GM, Mason SJ. Two-dimensional echocardiographic recognition of myocardial injury in man: comparison with postmortem studies. *Circulation.* 1981;63:401-8.
 94. Nixon JV, Narahara KA, Smitherman TC. Estimation of myocardial involvement in patients with acute myocardial infarction by two-dimensional echocardiography. *Circulation.* 1980;62:1248-55.
 95. Distante A, Picano E, Moscarelli E, Palombo C, Benassi A, L'Abbate A. Echocardiographic versus hemodynamic monitoring during attacks of variant angina pectoris. *Am J Cardiol.* 1985;55:1319-22.
 96. Shibata J, Takahashi H, Itaya M, et al. Cross-sectional echocardiographic visualization of the infarcted site in myocardial infarction: correlation with electrocardiographic and coronary angiographic findings. *J Cardiol.* 1982;12:885-94.
 97. Tennant R, Wiggers CJ. The effect of coronary artery occlusion on myocardial contraction. *Am J Physiol.* 1935;112:351-61.
 98. Jaarsma W, Visser CA, Eenige van MJ, Verheugt FW, Kupper AJ, Roos JP. Predictive value of two-dimensional echocardiographic and hemodynamic measurements on admission with acute myocardial infarction. *J Am Soc Echocardiogr.* 1988;1:187-93.
 99. Nishimura RA, Tajik AJ, Shub C, Miller FA, Ilstrup DM, Harrison CE. Role of two-dimensional echocardiography in the prediction of in-hospital complications after acute myocardial infarction. *J Am Coll Cardiol.* 1984;4:1080-7.
 100. Visser CA, Lie KI, Kan G, Meltzer R, Durrer D. Detection and quantification of acute, isolated myocardial infarction by two dimensional echocardiography. *Am J Cardiol.* 1981;47: 1020-5.
 101. Saeian K, Rhyne TL, Sagar KB. Ultrasonic tissue characterization for diagnosis of acute myocardial infarction in the coronary care unit. *Am J Cardiol* 1994;74:1211-5.
 102. Gibler WB, Runyon JP, Levy RC, et al. A rapid diagnostic and treatment center for patients with chest pain in the emergency department. *Ann Emerg Med.* 1995;25:1-8.
 103. Weaver WD, Cerqueira M, Hallstrom AP, et al. Prehospital-initiated vs hospital-initiated thrombolytic therapy. The Myocardial Infarction Triage and Intervention Trial. *JAMA.* 1993;270:1211-6.
 104. Kereiakes DJ, Weaver WD, Anderson JL, et al. Time delays in the diagnosis and treatment of acute myocardial infarction: a tale of eight cities. Report from the Pre-hospital Study Group and the Cincinnati Heart Project. *Am Heart J.* 1990;120:773-80.
 105. Long-term effects of intravenous thrombolysis in acute myocardial infarction: final report of the GISSI study. Gruppo Italiano per lo Studio della Streptochi-nasi nell'Infarto Miocardico (GISSI). *Lancet.* 1987;2:871-4.
 106. Emergency department: rapid identification and treatment of patients with acute myocardial infarction. National Heart Attack Alert Program Coordinating Committee, 60 Minutes to Treatment Working Group. *Ann Emerg Med.* 1994;23:311-29.
 107. Parisi AF, Moynihan PF, Folland ED, Strauss WE, Sharma GV, Sasahara AA. Echocardiography in acute and remote myocardial infarction. *Am J Cardiol.* 1980;46:1205-14.
 108. Shen W, Khandheria BK, Edwards WD, et al. Value and limitations of two-dimensional echocardiography in predicting myocar-

- dial infarct size. *Am J Cardiol.* 1991;68:1143-9.
109. Oh JK, Gibbons RJ, Christian TF, et al. Correlation of regional wall motion abnormalities detected by two-dimensional echocardiography with perfusion defect determined by technetium 99m sestamibi imaging in patients treated with reperfusion therapy during acute myocardial infarction. *Am Heart J.* 1996;131:32-7.
110. 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;103:814-22.
111. Bhatnagar SK, Moussa MA, Al-Yusuf AR. The role of prehospital discharge two-dimensional echocardiography in determining the prognosis of survivors of first myocardial infarction. *Am Heart J.* 1985;109:472-7.
112. Nelson GR, Cohn PF, Gorlin R. Prognosis in medically-treated coronary artery disease: influence of ejection fraction compared to other parameters. *Circulation.* 1975;52:408-12.
113. Sabia P, Abbott RD, Afrookteh A, Keller MW, Touchstone DA, Kaul S. Importance of two-dimensional echocardiographic assessment of left ventricular systolic function in patients presenting to the emergency room with cardiac-related symptoms. *Circulation.* 1991;84:1615-24.
114. Fleischmann KE, Goldman L, Robiolio PA, et al. Echocardiographic correlates of survival in patients with chest pain. *J Am Coll Cardiol.* 1994;23:1390-6.
115. Nishimura RA, Schaff HV, Shub C, Gersh BJ, Edwards WD, Tajik AJ. Papillary muscle rupture complicating acute myocardial infarction: analysis of 17 patients. *Am J Cardiol.* 1983;51:373-7.
116. Kono T, Sabbah HN, Rosman H, et al. Mechanism of functional mitral regurgitation during acute myocardial ischemia. *J Am Coll Cardiol.* 1992;19:1101-5.
117. Godley RW, Wann LS, Rogers EW, Feigenbaum H, Weyman AE. Incomplete mitral leaflet closure in patients with papillary muscle dysfunction. *Circulation.* 1981;63:565-71.
118. Kaul S, Spotnitz WD, Glasheen WP, Touchstone DA. Mechanism of ischemic mitral regurgitation. An experimental evaluation. *Circulation.* 1991;84:2167-80.
119. Visser CA, Kan G, Meltzer RS, Koolen JJ, Dunning AJ. Incidence, timing and prognostic value of left ventricular aneurysm formation after myocardial infarction: a prospective, serial echocardiographic study of 158 patients. *Am J Cardiol.* 1986;57:729-32.
120. Erlebacher JA, Weiss JL, Weisfeldt ML, Bulkley BH. Early dilation of the infarcted segment in acute transmural myocardial infarction: role of infarct expansion in acute left ventricular enlargement. *J Am Coll Cardiol.* 1984;4:201-8.
121. Miyatake K, Okamoto M, Kinoshita N, et al. Doppler echocardiographic features of ventricular septal rupture in myocardial infarction. *J Am Coll Cardiol.* 1985;5:182-7.
122. Rogers EW, Glassman RD, Feigenbaum H, Weyman AE, Godley RW. Aneurysms of the posterior interventricular septum with postinfarction ventricular septal defect. Echocardiographic identification. *Chest.* 1980;78:741-6.
123. Chandraratna PAN, Balachandran PK, Shah PM, Hodges M. Echocardiographic observations on ventricular septal rupture complicating acute myocardial infarction. *Circulation.* 1975;51:506-10.
124. Raitt MH, Kraft CD, Gardner CJ, Pearlman AS, Otto CM. Subacute ventricular free wall rupture complicating myocardial infarction. *Am Heart J.* 1993;126:946-55.
125. Gatewood RP, Nanda NC. Differentiation of left ventricular pseudoaneurysm from true aneurysm with two dimensional echocardiography. *Am J Cardiol.* 1980;46:869-78.
126. Roelandt JR, Sutherland GR, Yoshida K, Yoshikawa J. Improved diagnosis and characterization of left ventricular pseudoaneurysm by Doppler color flow imaging. *J Am Coll Cardiol.* 1988;12:807-11.
127. Keren A, Goldberg S, Gottlieb S, et al. Natural history of left ventricular thrombi: their appearance and resolution in the posthospitalization period of acute myocardial infarction. *J Am Coll Cardiol.* 1990;15:790-800.
128. Visser CA, Kan G, Meltzer RS, Dunning AJ, Roelandt J. Embolic potential of left ventricular thrombus after myocardial infarction: a two-dimensional echocardiographic study of 119 patients. *J Am Coll Cardiol.* 1985;5:1276-80.
129. DeMaria AN, Bommer W, Neumann A, Grehl T, Weinart L, DeNardo S, Amsterdam EA, Mason DT. Left ventricular thrombi identified by cross-sectional echocardiography. *Ann Intern Med.* 1979;90:14-8.
130. Spirito P, Bellotti P, Chiarella F, Domenicucci S, Sementa A, Vecchio C. Prognostic significance and natural history of left ventricular thrombi in patients with acute anterior myocardial infarction: a two-dimensional echocardiographic study. *Circulation.* 1985;72:774-80.
131. Gueret P, Dubourg O, Ferrier A, Farcot JC, Rigaud M, Bourdarias JP. Effects of full-dose heparin anticoagulation on the development of left ventricular thrombosis in acute transmural myocardial infarction. *J Am Coll Cardiol.* 1986;8:419-26.
132. Keating EC, Gross SA, Schlamowitz RA, et al. Mural thrombi in myocardial infarctions. Prospective evaluation by two-dimensional echocardiography. *Am J Med.* 1983;74:989-95.
133. Stratton JR, Lighty GW, Pearlman AS, Ritchie JL. Detection of left ventricular thrombus by two-dimensional echocardiography: sensitivity, specificity, and causes of uncertainty. *Circulation.* 1982;66:156-66.
134. Isner JM, Roberts WC. Right ventricular infarction complicating left ventricular infarction secondary to coronary heart disease. Frequency, location, associated findings and significance from analysis of 236 necropsy patients with acute or healed myocardial infarction. *Am J Cardiol.* 1978;42:885-94.
135. D'Arcy B, Nanda NC. Two-dimensional echocardiographic features of right ventricular infarction. *Circulation.* 1982;65:167-73.
136. Smart SC, Sawada S, Ryan T, et al. Low-dose dobutamine echocardiography detects reversible dysfunction after thrombolytic therapy of acute myocardial infarction. *Circulation.* 1993;88:405-15.
137. Pierard LA, De Landsheere CM, Berthe C, Rigo P, Kulbertus HE. Identification of viable myocardium by echocardiography during dobutamine infusion in patients with myocardial infarction after thrombolytic therapy: comparison with positron emission tomography. *J Am Coll Cardiol.* 1990;15:1021-31.
138. Barilla F, Gheorghide M, Alam M, Khaja F, Goldstein S. Low-dose dobutamine in patients with acute myocardial infarction identifies viable but not contractile myocardium and predicts the magnitude of improvement in wall motion abnormalities in response to coronary revascularization. *Am Heart J.* 1991;122:1522-31.
139. Ellis SG, Wynne J, Braunwald E, Henschke CI, Sandor T, Kloner RA. Response of reperfusion-salvaged, stunned myocardium to inotropic stimulation. *Am Heart J.* 1984;107:13-9.
140. Bolli R, Zhu WX, Myers ML, Hartley CJ, Roberts R. Beta-adrenergic stimulation reverses postischemic myocardial dysfunction without producing subsequent functional deterioration. *Am J Cardiol.* 1985;56:964-8.

141. Picano E, Pingitore A, Sicari R, et al. Stress echocardiographic results predict risk of reinfarction early after uncomplicated acute myocardial infarction: large-scale multicenter study. Echo Persantine International Cooperative (EPIC) Study Group. *J Am Coll Cardiol.* 1995;26:908-13.
142. Ryan T, Armstrong WF, O'Donnell JA, Feigenbaum H. Risk stratification after acute myocardial infarction by means of exercise two-dimensional echocardiography. *Am Heart J.* 1987;114:1305-16.
143. Jaarsma W, Visser CA, Kupper AJ, Res JC, van Eenige MJ, Roos JP. Usefulness of two-dimensional exercise echocardiography shortly after myocardial infarction. *Am J Cardiol.* 1986;57:86-90.
144. Applegate RJ, Dell'Italia LJ, Crawford MH. Usefulness of two-dimensional echocardiography during low-level exercise testing early after uncomplicated acute myocardial infarction. *Am J Cardiol.* 1987;60:10-4.
145. Bolognese L, Rossi L, Sarasso G, et al. Silent versus symptomatic dipyridamole-induced ischemia after myocardial infarction: clinical and prognostic significance. *J Am Coll Cardiol.* 1992;19:953-9.
146. Quintana M, Lindvall K, Ryden L, Brolund F. Prognostic value of predischARGE exercise stress echocardiography after acute myocardial infarction. *Am J Cardiol.* 1995;76:1115-21.
147. Weyman AE, Peskoe SM, Williams ES, Dillon JC, Feigenbaum H. Detection of left ventricular aneurysms by cross-sectional echocardiography. *Circulation.* 1976;54:936-44.
148. Visser CA, Kan G, David GK, Lie KI, Durrer D. Echocardiographic-cineangiographic correlation in detecting left ventricular aneurysm: a prospective study of 422 patients. *Am J Cardiol.* 1982;50:337-41.
149. Barrett MJ, Charuzi Y, Corday E. Ventricular aneurysm: cross-sectional echocardiographic approach. *Am J Cardiol.* 1980;46:1133-7.
150. Marwick T, Willemart B, D'Hondt AM, et al. Selection of the optimal nonexercise stress for the evaluation of ischemic regional myocardial dysfunction and malperfusion. Comparison of dobutamine and adenosine using echocardiography and 99mTc-MIBI single photon emission computed tomography. *Circulation.* 1993;87:345-54.
151. McNeill AJ, Fioretti PM, el-Said SM, Salustri A, de Feyter PJ, Roelandt JR. Dobutamine stress echocardiography before and after coronary angioplasty. *Am J Cardiol.* 1992;69:740-5.
152. Sawada SG, Ryan T, Fineberg NS, et al. Exercise echocardiographic detection of coronary artery disease in women. *J Am Coll Cardiol.* 1989;14:1440-7.
153. Marangelli V, Iliceto S, Piccinni G, De Martino G, Sorgente L, Rizzon P. Detection of coronary artery disease by digital stress echocardiography: comparison of exercise, transesophageal atrial pacing and dipyridamole echocardiography. *J Am Coll Cardiol.* 1994;24:117-24.
154. Armstrong WF, O'Donnell J, Ryan T, Feigenbaum H. Effect of prior myocardial infarction and extent and location of coronary disease on accuracy of exercise echocardiography. *J Am Coll Cardiol.* 1987;10:531-8.
155. Ryan T, Vasey CG, Presti CF, O'Donnell JA, Feigenbaum H, Armstrong WF. Exercise echocardiography: detection of coronary artery disease in patients with normal left ventricular wall motion at rest. *J Am Coll Cardiol.* 1988;11:993-9.
156. Labovitz AJ, Lewen M, Kern MJ, et al. The effects of successful PTCA on left ventricular function: assessment by exercise echocardiography. *Am Heart J.* 1989;117:1003-8.
157. Crouse LJ, Harbrecht JJ, Vacek JL, Rosamond TL, Kramer PH. Exercise echocardiography as a screening test for coronary artery disease and correlation with coronary arteriography. *Am J Cardiol.* 1991;67:1213-8.
158. Pozzoli MM, Fioretti PM, Salustri A, Reijs AE, Roelandt JR. Exercise echocardiography and technetium-99m MIBI single-photon emission computed tomography in the detection of coronary artery disease. *Am J Cardiol.* 1991;67:350-5.
159. Galanti G, Sciagra R, Comeglio M, et al. Diagnostic accuracy of peak exercise echocardiography in coronary artery disease: comparison with thallium-201 myocardial scintigraphy. *Am Heart J.* 1991;122:1609-16.
160. Marwick TH, Nemecek JJ, Pashkow FJ, Stewart WJ, Salcedo EE. Accuracy and limitations of exercise echocardiography in a routine clinical setting. *J Am Coll Cardiol.* 1992;19:74-81.
161. Quinones MA, Verani MS, Haichin RM, Mahmorian JJ, Suarez J, Zoghbi WA. Exercise echocardiography versus ²⁰¹Tl single-photon emission computed tomography in evaluation of coronary artery disease. Analysis of 292 patients. *Circulation.* 1992;85: 1026-31.
162. Salustri A, Pozzoli MM, Hermans W, et al. Relationship between exercise echocardiography and perfusion single-photon emission computed tomography in patients with single-vessel coronary artery disease. *Am Heart J.* 1992;124:75-83.
163. Amanullah AM, Lindvall K, Bevegard S. Exercise echocardiography after stabilization of unstable angina: correlation with exercise thallium-201 single photon emission computed tomography. *Clin Cardiol.* 1992;15:585-9.
164. Ryan T, Segar DS, Sawada SG, et al. Detection of coronary artery disease with upright bicycle exercise echocardiography. *J Am Soc Echocardiogr.* 1993;6:186-97.
165. Mertes H, Erbel R, Nixdorff U, Mohr-Kahaly S, Kruger S, Meyer J. Exercise echocardiography for the evaluation of patients after nonsurgical coronary artery revascularization. *J Am Coll Cardiol.* 1993;21:1087-93.
166. Hoffmann R, Lethen H, Kleinhans E, Weiss M, Flachskampf FA, Hanrath P. Comparative evaluation of bicycle and dobutamine stress echocardiography with perfusion scintigraphy and bicycle electrocardiogram for identification of coronary artery disease. *Am J Cardiol.* 1993;72:555-9.
167. Cohen JL, Ottenweller JE, George AK, Duvvuri S. Comparison of dobutamine and exercise echocardiography for detecting coronary artery disease. *Am J Cardiol.* 1993;72:1226-31.
168. Hecht HS, DeBord L, Shaw R, et al. Digital supine bicycle stress echocardiography: a new technique for evaluating coronary artery disease. *J Am Coll Cardiol.* 1993;21:950-6.
169. Roger VL, Pellikka PA, Oh JK, Bailey KR, Tajik AJ. Identification of multivessel coronary artery disease by exercise echocardiography. *J Am Coll Cardiol.* 1994;24:109-14.
170. Roger VL, Pellikka PA, Oh JK, Miller FA, Seward JB, Tajik AJ. Stress echocardiography. Part I. Exercise echocardiography: techniques, implementation, clinical applications, and correlations. *Mayo Clin Proc.* 1995;70:5-15.
171. Berthe C, Pierard LA, Hiernaux M, et al. Predicting the extent and location of coronary artery disease in acute myocardial infarction by echocardiography during dobutamine infusion. *Am J Cardiol.* 1986;58:1167-72.
172. Salustri A, Fioretti PM, McNeill AJ, Pozzoli MM, Roelandt JR. Pharmacological stress echocardiography in the diagnosis of coronary artery disease and myocardial ischaemia: a comparison between dobutamine and dipyridamole. *Eur Heart J.* 1992;13: 1356-62.
173. Previtali M, Lanzarini L, Ferrario M, Tortorici M, Mussini A, Montemartini C. Dobutamine versus dipyridamole echocardiography

- raphy in coronary artery disease. *Circulation*. 1991;83(suppl III):III-27-31.
174. Sawada SG, Segar DS, Ryan T, et al. Echocardiographic detection of coronary artery disease during dobutamine infusion. *Circulation*. 1991;83:1605-14.
175. Cohen JL, Greene TO, Ottenweller J, Binenbaum SZ, Wilchfort SD, Kim CS. Dobutamine digital echocardiography for detecting coronary artery disease. *Am J Cardiol*. 1991;67:1311-8.
176. Mazeika PK, Nadazdin A, Oakley CM. Dobutamine stress echocardiography for detection and assessment of coronary artery disease. *J Am Coll Cardiol*. 1992;19:1203-11.
177. Marcovitz PA, Armstrong WF. Accuracy of dobutamine stress echocardiography in detecting coronary artery disease. *Am J Cardiol*. 1992;69:1269-73.
178. Segar DS, Brown SE, Sawada SG, Ryan T, Feigenbaum H. Dobutamine stress echocardiography: correlation with coronary lesion severity as determined by quantitative angiography. *J Am Coll Cardiol*. 1992;19:1197-202.
179. Marwick T, D'Hondt AM, Baudhuin T, et al. Optimal use of dobutamine stress for the detection and evaluation of coronary artery disease: combination with echocardiography or scintigraphy, or both? *J Am Coll Cardiol*. 1993;22:159-67.
180. Forster T, McNeill AJ, Salustri A, et al. Simultaneous dobutamine stress echocardiography and technetium-99m isonitrite single-photon emission computed tomography in patients with suspected coronary artery disease. *J Am Coll Cardiol*. 1993;21:1591-6.
181. Günalp B, Dokumaci B, Uyan C, et al. Value of dobutamine technetium-99m-sestamibi SPECT and echocardiography in the detection of coronary artery disease compared with coronary angiography. *J Nucl Med*. 1993;34:889-94.
182. Pellikka PA, Roger VL, Oh JK, Miller FA, Seward JB, Tajik AJ. Stress echocardiography. Part II. Dobutamine stress echocardiography: techniques, implementation, clinical applications, and correlations. *Mayo Clin Proc*. 1995;70:16-27.
183. Beleslin BD, Ostojic M, Stepanovic J, et al. Stress echocardiography in the detection of myocardial ischemia. Head-to-head comparison of exercise, dobutamine, and dipyridamole tests. *Circulation*. 1994;90:1168-76.
184. Dagianti A, Penco M, Agati L, Sciomer S, Rosanio S, Dagianti A, Fedele F. Stress echocardiography: comparison of exercise, dipyridamole and dobutamine in detecting and predicting the extent of coronary artery disease. *J Am Coll Cardiol*. 1995;26:18-25.
185. Sawada SG, Ryan T, Conley MJ, Corya BC, Feigenbaum H, Armstrong WF. Prognostic value of a normal exercise echocardiogram. *Am Heart J*. 1990;120:49-55.
186. Krivokapich J, Child JS, Gerber RS, Lem V, Moser D. Prognostic usefulness of positive or negative exercise stress echocardiography for predicting coronary events in ensuing twelve months. *Am J Cardiol*. 1993;71:646-51.
187. Mazeika PK, Nadazdin A, Oakley CM. Prognostic value of dobutamine echocardiography in patients with high pretest likelihood of coronary artery disease. *Am J Cardiol*. 1993;71:33-9.
188. Severi S, Picano E, Michelassi C, et al. Diagnostic and prognostic value of dipyridamole echocardiography in patients with suspected coronary artery disease. Comparison with exercise electrocardiography. *Circulation*. 1994;89:1160-73.
189. Coletta C, Galati A, Greco G, et al. Prognostic value of high dose dipyridamole echocardiography in patients with chronic coronary artery disease and preserved left ventricular function. *J Am Coll Cardiol*. 1995;26:887-94.
190. Williams MJ, Odabashian J, Lauer MS, Thomas JD, Marwick TH. Prognostic value of dobutamine echocardiography in patients with left ventricular dysfunction. *J Am Coll Cardiol*. 1996;27:132-9.
191. Afridi I, Quinones MA, Zoghbi WA, Cheirif J. Dobutamine stress echocardiography: sensitivity, specificity, and predictive value for future cardiac events. *Am Heart J*. 1994;127:1510-15.
192. Kamaran M, Teague SM, Finkelhor RS, Dawson N, Bahler RC. Prognostic value of dobutamine stress echocardiography in patients referred because of suspected coronary artery disease. *Am J Cardiol*. 1995;76:887-91.
193. Marcovitz PA, Shayna V, Horn RA, Hepner A, Armstrong WF. Value of dobutamine stress echocardiography in determining the prognosis of patients with known or suspected coronary disease. *Am J Cardiol*. 1996;78:404-8.
194. Dilsizian V, Bonow RO. Current diagnostic techniques of assessing myocardial viability in patients with hibernating and stunned myocardium [published erratum appears in *Circulation*. 1993;87:2070]. *Circulation*. 1993;87:1-20.
195. Cigarroa CG, deFilippi CR, Brickner ME, Alvarez LG, Wait MA, Grayburn PA. Dobutamine stress echocardiography identifies hibernating myocardium and predicts recovery of left ventricular function after coronary revascularization. *Circulation*. 1993;88:430-6.
196. Afridi I, Kleiman NS, Raizner AE, Zoghbi WA. Dobutamine echocardiography in myocardial hibernation. Optimal dose and accuracy in predicting recovery of ventricular function after coronary angioplasty. *Circulation*. 1995;91:663-70.
197. La Canna G, Alfieri O, Giubbini R, Gargano M, Ferrari R, Visioli O. Echocardiography during infusion of dobutamine for identification of reversible dysfunction in patients with chronic coronary artery disease. *J Am Coll Cardiol*. 1994;23:617-26.
198. Arnese M, Cornel JH, Salustri A, et al. Prediction of improvement of regional left ventricular function after surgical revascularization. A comparison of low-dose dobutamine echocardiography with 201 Tl single-photon emission computed tomography. *Circulation*. 1995;91:2748-52.
199. Perrone-Filardi P, Pace L, Prastaro M, et al. Dobutamine echocardiography predicts improvement of hypoperfused dysfunctional myocardium after revascularization in patients with coronary artery disease. *Circulation*. 1995;91:2556-65.
200. deFilippi CR, Willett DL, Irani WN, Eichhorn EJ, Velasco CE, Grayburn PA. Comparison of myocardial contrast echocardiography and low-dose dobutamine stress echocardiography in predicting recovery of left ventricular function after coronary revascularization in chronic ischemic heart disease. *Circulation*. 1995;92:2863-8.
201. Mock MB, Ringqvist I, Fisher LD, et al. Survival of medically treated patients in the Coronary Artery Surgery Study (CASS) registry. *Circulation*. 1982;66:562-8.
202. McConahay DR, Valdes M, McCallister BD, et al. Accuracy of treadmill testing in assessment of direct myocardial revascularization. *Circulation*. 1977;56:548-52.
203. Krumholz HM, Douglas PS, Goldman L, Waksmonski C. Clinical utility of transthoracic two-dimensional and Doppler echocardiography. *J Am Coll Cardiol*. 1994;24:125-31.
204. 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;2:358-67.
205. Sahn DJ, DeMaria A, Kisslo J, Weyman A. Recommendations regarding quantitation in M-mode echocardiography: results of a

- survey of echocardiographic measurements. *Circulation*. 1978;58:1072-83.
206. Vuille C, Weyman AE. Left ventricle I: general considerations, assessment of chamber size and function. In: *Principles and Practice of Echocardiography*. 2nd ed. Philadelphia: Lea & Febiger; 1994.
 207. Stamm RB, Carabello BA, Mayers DL, Martin RP. Two-dimensional echocardiographic measurement of left ventricular ejection fraction: prospective analysis of what constitutes an adequate determination. *Am Heart J*. 1982;104:136-44.
 208. Amico AF, Lichtenberg GS, Reisner SA, Stone CK, Schwartz RG, Meltzer RS. Superiority of visual versus computerized echocardiographic estimation of radionuclide left ventricular ejection fraction. *Am Heart J*. 1989;118:1259-65.
 209. Quinones MA, Waggoner AD, Reduto LA, et al. A new, simplified and accurate method for determining ejection fraction with two-dimensional echocardiography. *Circulation*. 1981;64:744-53.
 210. Aurigemma GP, Gaasch WH, Villegas B, Meyer TE. Noninvasive assessment of left ventricular mass, chamber volume, and contractile function. *Curr Probl Cardiol*. 1995;20:361-440.
 211. Martin RP. Real time ultrasound quantification of ventricular function: has the eyeball been replaced or will the subjective become objective? *J Am Coll Cardiol*. 1992;19:321-3.
 212. Borow KM, Neumann A, Wynne J. Sensitivity of end-systolic pressure-dimension and pressure-volume relations to the inotropic state in humans. *Circulation*. 1982;65:988-97.
 213. Huntsman LL, Stewart DK, Barnes SR, Franklin SB, Colocousis JS, Hessel EA. Noninvasive Doppler determination of cardiac output in man. Clinical validation. *Circulation*. 1983;67:593-602.
 214. Hoit BD, Rashwan M, Watt C, Sahn DJ, Bhargava V. Calculating cardiac output from transmitral volume flow using Doppler and M-mode echocardiography. *Am J Cardiol*. 1988;62:131-5.
 215. Brandenburg RO, Chazov E, Cherian G, et al. Report of the WHO/ISFC Task Force on Definition and Classification of Cardiomyopathies. *Circulation*. 1981;64:437A-8A.
 216. Echeverria HH, Bilsker MS, Myerburg RJ, Kessler KM. Congestive heart failure: echocardiographic insights. *Am J Med*. 1983;75:750-5.
 217. Aguirre FV, Pearson AC, Lewen MK, McCluskey M, Labovitz AJ. Usefulness of Doppler echocardiography in the diagnosis of congestive heart failure. *Am J Cardiol*. 1989;63:1098-102.
 218. Shah PM. Echocardiography in congestive or dilated cardiomyopathy. *J Am Soc Echocardiogr*. 1988;1:20-30.
 219. Rihal CS, Nishimura RA, Hatle LK, Bailey KR, Tajik AJ. Systolic and diastolic dysfunction in patients with clinical diagnosis of dilated cardiomyopathy. Relation to symptoms and prognosis. *Circulation*. 1994;90:2772-9.
 220. Pinamonti B, Di Lenarda A, Sinagra G, Camerini F. Restrictive left ventricular filling pattern in dilated cardiomyopathy assessed by Doppler echocardiography: clinical, echocardiographic and hemodynamic correlations and prognostic implications. Heart Muscle Disease Study Group. *J Am Coll Cardiol*. 1993;22:808-15.
 221. Pfeffer MA, Braunwald E, Moye LA, et al. Effect of captopril on mortality and morbidity in patients with left ventricular dysfunction after myocardial infarction. Results of the Survival and Ventricular Enlargement Trial. The SAVE Investigators. *N Engl J Med*. 1992;327:669-77.
 222. Effect of enalapril on mortality and the development of heart failure in asymptomatic patients with reduced left ventricular ejection fractions. The SOLVD Investigators. *N Engl J Med*. 1992;327:685-91.
 223. Lipshultz SE, Colan SD, Gelber RD, Perez-Atayde AR, Sallan SE, Sanders SP. Late cardiac effects of doxorubicin therapy for acute lymphoblastic leukemia in childhood. *N Engl J Med*. 1991;324:808-15.
 224. Rosenthal DS, Braunwald E. Hematological-oncological disorders and heart disease. In: Braunwald E, ed. *Heart Disease: A Textbook of Cardiovascular Medicine*. Philadelphia, Pa: WB Saunders; 1992:1756-7.
 225. Stoddard MF, Seeger J, Liddell NE, Hadley TJ, Sullivan DM, Kupersmith J. Prolongation of isovolumetric relaxation time as assessed by Doppler echocardiography predicts doxorubicin-induced systolic dysfunction in humans. *J Am Coll Cardiol*. 1992;20:62-9.
 226. Marchandise B, Schroeder E, Bosly A, et al. Early detection of doxorubicin cardiotoxicity: interest of Doppler echocardiographic analysis of left ventricular filling dynamics. *Am Heart J*. 1989;118:92-8.
 227. Lee BH, Goodenday LS, Muswick GJ, Yasnoff WA, Leighton RF, Skeel RT. Alterations in left ventricular diastolic function with doxorubicin therapy. *J Am Coll Cardiol*. 1987;9:184-8.
 228. Maron BJ, Gottdiener JS, Epstein SE. Patterns and significance of distribution of left ventricular hypertrophy in hypertrophic cardiomyopathy. A wide angle, two dimensional echocardiographic study of 125 patients. *Am J Cardiol*. 1981;48:418-28.
 229. Rakowski H, Sasson Z, Wigle ED. Echocardiographic and Doppler assessment of hypertrophic cardiomyopathy. *J Am Soc Echocardiogr*. 1988;1:31-47.
 230. Fananapazir L, Cannon RO, Tripodi D, Panza JA. Impact of dual-chamber permanent pacing in patients with obstructive hypertrophic cardiomyopathy with symptoms refractory to verapamil and beta-adrenergic blocker therapy. *Circulation*. 1992;85:2149-61.
 231. Appleton CP, Hatle LK, Popp RL. Demonstration of restrictive ventricular physiology by Doppler echocardiography. *J Am Coll Cardiol*. 1988;11:757-68.
 232. Hatle LK, Appleton CP, Popp RL. Differentiation of constrictive pericarditis and restrictive cardiomyopathy by Doppler echocardiography. *Circulation*. 1989;79:357-70.
 233. Klein AL, Hatle LK, Burstow DJ, et al. Doppler characterization of left ventricular diastolic function in cardiac amyloidosis. *J Am Coll Cardiol*. 1989;13:1017-26.
 234. Nishimura R, Abel MD, Hatle LK, Tajik AJ. Assessment of diastolic function of the heart: background and current applications of Doppler echocardiography. Part II. *Mayo Clin Proc*. 1989;64:181-204.
 235. Soufer R, Wohlgeleit D, Vita NA, et al. Intact systolic left ventricular function in clinical congestive heart failure. *Am J Cardiol*. 1985;55:1032-6.
 236. Kitzman DW, Higginbotham MB, Cobb FR, Sheikh KH, Sullivan MJ. Exercise intolerance in patients with heart failure and preserved left ventricular systolic function: failure of the Frank-Starling mechanism. *J Am Coll Cardiol*. 1991;17:1065-72.
 237. Choong CY, Herrmann HC, Weyman AE, Fifer MA. Preload dependence of Doppler-derived indexes of left ventricular diastolic function in humans. *J Am Coll Cardiol*. 1987;10:800-8.
 238. Harrison MR, Clifton GD, Pennell AT, DeMaria AN. Effect of heart rate on left ventricular diastolic transmitral flow velocity patterns assessed by Doppler echocardiography in normal subjects. *Am J Cardiol*. 1991;67:622-7.
 239. Levine RA, Gibson TC, Aretz T, et al. Echocardiographic measurement of right ventricular volume. *Circulation*. 1984;69:497-505.

240. Feigenbaum H, Waldhausen JA, Hyde LP. Ultrasound diagnosis of pericardial effusion. *JAMA*. 1965;191:107-10.
241. Clark JG, Berberich SN, Zager JR. Echocardiographic findings of pericardial effusion mimicked by fibrocalcific pericardial disease. *Echocardiography*. 1985;2:475-80.
242. Armstrong WF, Schilt BF, Helper DJ, Dillon JC, Feigenbaum H. Diastolic collapse of the right ventricle with cardiac tamponade: an echocardiographic study. *Circulation*. 1982;65:1491-6.
243. 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.
244. Singh S, Wann LS, Schuchard GH, et al. Right ventricular and right atrial collapse in patients with cardiac tamponade—a combined echocardiographic and hemodynamic study. *Circulation*. 1984;70:966-71.
245. Shina S, Yaginuma T, Kando K, Kawai N, Hosada S. Echocardiographic evaluation of impending cardiac tamponade. *J Cardiol*. 1979;9:555-69.
246. 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;12:1470-7.
247. D'Cruz IA, Cohen HC, Prabhu R, Glick G. Diagnosis of cardiac tamponade by echocardiography: changes in mitral valve motion and ventricular dimensions, with special reference to paradoxical pulse. *Circulation*. 1975;52:460-5.
248. Appleton CP, Hatle LK, Popp RL. Cardiac tamponade and pericardial effusion: respiratory variation in transvalvular flow velocities studied by Doppler echocardiography. *J Am Coll Cardiol*. 1988;11:1020-30.
249. Burstow DJ, Oh JK, Bailey KR, Seward JB, Tajik AJ. Cardiac tamponade: characteristic Doppler observations. *Mayo Clin Proc*. 1989;64:312-24.
250. Pandian NG, Skorton DJ, Kieso RA, Kerber RE. Diagnosis of constrictive pericarditis by two-dimensional echocardiography: studies in a new experimental model and in patients. *J Am Coll Cardiol*. 1984;4:1164-73.
251. Chandraratna PA. Echocardiography and Doppler ultrasound in the evaluation of pericardial disease. *Circulation*. 1991;84(suppl D):I-303-10.
252. Thurber DL, Edwards JE, Achor RWP. Secondary malignant tumors of the pericardium. *Circulation*. 1962;26:228-41.
253. Chandraratna PA, Aronow WS. Detection of pericardial metastases by cross-section echocardiography. *Circulation*. 1981;63:197-9.
254. Hynes JK, Tajik AJ, Osborn MJ, Orszulak TA, Seward JB. Two-dimensional echocardiographic diagnosis of pericardial cyst. *Mayo Clin Proc*. 1983;58:60-3.
255. Gibson TC, Grossman W, McLaurin LP, Moos S, Craige E. An echocardiographic study of the interventricular septum in constrictive pericarditis. *Br Heart J*. 1976;38:738-43.
256. Schnittger I, Bowden RE, Abrams J, Popp RL. Echocardiography: pericardial thickening and constrictive pericarditis. *Am J Cardiol*. 1978;42:388-95.
257. Tei C, Child JS, Tanaka H, Shah PM. Atrial systolic notch on the interventricular septal echogram: an echocardiographic sign of constrictive pericarditis. *J Am Coll Cardiol*. 1983;3:907-12.
258. Pool PE, Seagren SC, Abbasi AS, Charuzi Y, Kraus R. Echocardiographic manifestations of constrictive pericarditis. *Chest*. 1975;68:684-8.
259. Oh JK, Hatle LK, Seward JB, et al. Diagnostic role of Doppler echocardiography in constrictive pericarditis. *J Am Coll Cardiol*. 1994;23:154-62.
260. Kansal S, Roitman D, Sheffield LT. Two-dimensional echocardiography of congenital absence of pericardium. *Am Heart J*. 1985;109:912-5.
261. Ruys F, Paulus W, Stevens C, Brutsaert D. Expansion of the left atrial appendage is a distinctive cross-sectional echocardiographic feature of congenital defect of the pericardium. *Eur Heart J*. 1983;4:738-41.
262. Banning AP, Masani ND, Ikram S, Fraser AG, Hall RJ. Transoesophageal echocardiography as the sole diagnostic investigation in patients with suspected thoracic aortic dissection. *Br Heart J*. 1994;72:461-5.
263. Erbel R, Engberding R, Daniel W, Roelandt J, Visser C, Rennollet H. Echocardiography in diagnosis of aortic dissection. *Lancet*. 1989;1:457-61.
264. Mohr-Kahaly S, Erbel R, Rennollet H, et al. Ambulatory follow-up of aortic dissection by transesophageal two-dimensional and color-coded Doppler echocardiography. *Circulation*. 1989;80:24-33.
265. Wiet SP, Pearce WH, McCarthy WJ, Joob AW, Yao JS, McPherson DD. Utility of transesophageal echocardiography in the diagnosis of disease of the thoracic aorta. *J Vasc Surg*. 1994;20:613-20.
266. Smith MD, Cassidy JM, Souther S, et al. Transesophageal echocardiography in the diagnosis of traumatic rupture of the aorta. *N Engl J Med*. 1995;332:356-62.
267. Yock PG, Popp RL. Noninvasive estimation of right ventricular systolic pressure by Doppler ultrasound in patients with tricuspid regurgitation. *Circulation*. 1984;70:657-62.
268. Skjaerpe T, Hatle L. Noninvasive estimation of pulmonary artery pressure by Doppler ultrasound in tricuspid regurgitation. In: Spencer MP, ed. *Cardiac Doppler Diagnosis*. Boston, Mass: Martinus Nijhoff; 1983:274-354.
269. Deleted during update.
270. Kannel WB, Gordon T, Offutt D. Left ventricular hypertrophy by electrocardiogram. Prevalence, incidence, and mortality in the Framingham study. *Ann Intern Med*. 1969;71:89-105.
271. Devereux RB, Koren MJ, de Simone G, Okin PM, Kligfield P. Methods for detection of left ventricular hypertrophy: application to hypertensive heart disease. *Eur Heart J*. 1993;14(suppl D):8-15.
272. Gottdiener JS, Livengood SV, Meyer PS, Chase GA. Should echocardiography be performed to assess effects of antihypertensive therapy? Test-retest reliability of echocardiography for measurement of left ventricular mass and function. *J Am Coll Cardiol*. 1995;25:424-30.
273. Grandits GA, Liebson PR, Dianzumba S, Prineas RJ. Echocardiography in multicenter clinical trials: experience from the Treatment of Mild Hypertension Study. *Control Clin Trials*. 1994;15:395-410.
274. Devereux RB, Casale PN, Wallerson DC, et al. Cost-effectiveness of echocardiography and electrocardiography for detection of left ventricular hypertrophy in patients with systemic hypertension. *Hypertension*. 1987;9(pt 2):II-69-76.
275. Devereux RB, Reichek N. Echocardiographic determination of left ventricular mass in man. Anatomic validation of the method. *Circulation*. 1977;55:613-8.
276. Devereux RB, Alonso DR, Lutas EM, et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. *Am J Cardiol*. 1986;57:450-8.
277. Liebson PR, Devereux RB, Horan MJ. Hypertension research. Echocardiography in the measurement of left ventricular wall

- mass. *Hypertension*. 1987;9(pt 2):II-2-5.
278. Germain P, Roul G, Kastler B, Mossard JM, Bareiss P, Sacrez A. Inter-study variability in left ventricular mass measurement. Comparison between M-mode echography and MRI. *Eur Heart J*. 1992;13:1011-9.
 279. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Left ventricular mass and incidence of coronary heart disease in an elderly cohort. The Framingham Heart Study. *Ann Intern Med*. 1989;110:101-7.
 280. Ghali JK, Liao Y, Simmons B, Castaner A, Cao G, Cooper RS. The prognostic role of left ventricular hypertrophy in patients with or without coronary artery disease. *Ann Intern Med*. 1992;117:831-6.
 281. Devereux RB, Koren MJ, de Simone G, Roman MJ, Laragh JH. Left ventricular mass as a measure of preclinical hypertensive disease. *Am J Hypertens*. 1992;5(suppl):175S-81S.
 282. Dahlof B, Pennert K, Hansson L. Reversal of left ventricular hypertrophy in hypertensive patients. A metaanalysis of 109 treatment studies. *Am J Hypertens*. 1992;5:95-110.
 283. Liebson PR. Clinical studies of drug reversal of hypertensive left ventricular hypertrophy. *Am J Hypertens*. 1990;3:512-7.
 284. Liebson PR, Grandits GA, Dianzumba S, et al. Comparison of five antihypertensive monotherapies and placebo for change in left ventricular mass in patients receiving nutritional-hygienic therapy in the Treatment of Mild Hypertension Study (TOMHS). *Circulation*. 1995;91:698-706.
 285. Liebson PR, Savage DD. Echocardiography in hypertension: a review, II: echocardiographic studies of the effects of antihypertensive agents on left ventricular wall mass and function. *Echocardiography*. 1987;4:215-49.
 286. Shub C, Tajik A, Sheps S. Clinical impact of echocardiographic left ventricular mass determination in hypertensive patients. *Am J Hypertens*. 1993;6:25A. Abstract.
 287. Cardiogenic brain embolism. The second report of the Cerebral Embolism Task Force. *Arch Neurol*. 1989;46:727-43.
 288. Lee RJ, Bartzokis T, Yeoh TK, Grogan HR, Choi D, Schnittger I. Enhanced detection of intracardiac sources of cerebral emboli by transesophageal echocardiography. *Stroke*. 1991;22:734-9.
 289. Pop G, Sutherland GR, Koudstaal PJ, Sit TW, de Jong G, Roelandt JR. Transesophageal echocardiography in the detection of intracardiac embolic sources in patients with transient ischemic attacks. *Stroke*. 1990;21:560-5.
 290. Hofmann T, Kasper W, Meinertz T, Geibel A, Just H. Echocardiographic evaluation of patients with clinically suspected arterial emboli. *Lancet*. 1990;336:1421-4.
 291. Zeiler K, Siostrzonek P, Lang W, et al. Different risk factor profiles in young and elderly stroke patients with special reference to cardiac disorders. *J Clin Epidemiol*. 1992;45:1383-9.
 292. Cujec B, Polasek P, Voll C, Shuaib A. Transesophageal echocardiography in the detection of potential cardiac source of embolism in stroke patients. *Stroke*. 1991;22:727-33.
 293. Vandenbogaerde J, De Bleecker J, Decoo D, et al. Transesophageal echo-Doppler in patients suspected of a cardiac source of peripheral emboli. *Eur Heart J*. 1992;13:88-94.
 294. Labovitz AJ, Camp A, Castello R, et al. Usefulness of transesophageal echocardiography in unexplained cerebral ischemia. *Am J Cardiol*. 1993;72:1448-52.
 295. Comess KA, DeRook FA, Beach KW, Lytle NJ, Golby AJ, Albers GW. Transesophageal echocardiography and carotid ultrasound in patients with cerebral ischemia: prevalence of findings and recurrent stroke risk. *J Am Coll Cardiol*. 1994;23:1598-603.
 296. Albers GW, Comess KA, DeRook FA, et al. Transesophageal echocardiographic findings in stroke subtypes. *Stroke*. 1994;25:23-8.
 297. Lindgren A, Roijer A, Norrving B, Wallin L, Eskilsson J, Johansson BB. Carotid artery and heart disease in subtypes of cerebral infarction. *Stroke*. 1994;25:2356-62.
 298. Mitusch R, Lange V, Stierle U, Maurer B, Sheikhzadeh A. Transesophageal echocardiographic determinants of embolism in nonrheumatic atrial fibrillation. *Int J Card Imaging*. 1995;11:27-34.
 299. Pearson AC, Labovitz AJ, Tatineni S, Gomez CR. Superiority of transesophageal echocardiography in detecting cardiac source of embolism in patients with cerebral ischemia of uncertain etiology. *J Am Coll Cardiol*. 1991;17:66-72.
 300. Sansoy V, Abbott RD, Jayaweera AR, Kaul S. Low yield of transthoracic echocardiography for cardiac source of embolism. *Am J Cardiol*. 1995;75:166-9.
 301. Jones EF, Donnan GA, Calafiore P, Tonkin AM. Transoesophageal echocardiography in the investigation of stroke: experience in 135 patients with cerebral ischaemic events. *Aust N Z J Med*. 1993;23:477-83.
 302. Chimowitz MI, DeGeorgia MA, Poole RM, Hepner A, Armstrong WM. Left atrial spontaneous echo contrast is highly associated with previous stroke in patients with atrial fibrillation or mitral stenosis. *Stroke*. 1993;24:1015-9.
 303. Cabanes L, Mas JL, Cohen A, et al. Atrial septal aneurysm and patent foramen ovale as risk factors for cryptogenic stroke in patients less than 55 years of age. A study using transesophageal echocardiography. *Stroke*. 1993;24:1865-73.
 304. Hwang JJ, Kuan P, Chen JJ, et al. Significance of left atrial spontaneous echo contrast in rheumatic mitral valve disease as a predictor of systemic arterial embolization: a transesophageal echocardiographic study. *Am Heart J*. 1994;127:880-5.
 305. Leung DY, Black IW, Cranney GB, Hopkins AP, Walsh WF. Prognostic implications of left atrial spontaneous echo contrast in nonvalvular atrial fibrillation. *J Am Coll Cardiol*. 1994;24:755-62.
 306. Zabalgoitia M, Norris LP, Garcia M. Atrial septal aneurysm as a potential source of neurological ischemic events. *Am J Card Imaging*. 1994;8:39-44.
 307. Pearson AC, Nagelhout D, Castello R, Gomez CR, Labovitz AJ. Atrial septal aneurysm and stroke: a transesophageal echocardiographic study. *J Am Coll Cardiol*. 1991;18:1223-9.
 308. Demopoulos LA, Tunick PA, Bernstein NE, Perez JL, Kronzon I. Protruding atheromas of the aortic arch in symptomatic patients with carotid artery disease. *Am Heart J*. 1995;129:40-4.
 309. Tunick PA, Perez JL, Kronzon I. Protruding atheromas in the thoracic aorta and systemic embolization. *Ann Intern Med*. 1991;115:423-7.
 310. Katz ES, Tunick PA, Rusinek H, Ribakove G, Spencer FC, Kronzon I. Protruding aortic atheromas predict stroke in elderly patients undergoing cardiopulmonary bypass: experience with intraoperative transesophageal echocardiography. *J Am Coll Cardiol*. 1992;20:70-7.
 311. Amarenco P, Cohen A, Tzourio C, et al. Atherosclerotic disease of the aortic arch and the risk of ischemic stroke. *N Engl J Med*. 1994;331:1474-9.
 312. Jones EF, Kalman JM, Calafiore P, Tonkin AM, Donnan GA. Proximal aortic atheroma. An independent risk factor for cerebral ischemia. *Stroke*. 1995;26:218-24.
 313. Karalis DG, Chandrasekaran K, Victor MF, Ross JJ, Mintz GS. Recognition and embolic potential of intraaortic atherosclerotic debris. *J Am Coll Cardiol*. 1991;17:73-8.
 314. Klotzsch C, Janssen G, Berlit P. Transesophageal echocardiogra-

- phy and contrast-TCD in the detection of a patent foramen ovale: experiences with 111 patients. *Neurology*. 1994;44:1603-6.
315. Hanna JP, Sun JP, Furlan AJ, Stewart WJ, Sila CA, Tan M. Patent foramen ovale and brain infarct. Echocardiographic predictors, recurrence, and prevention. *Stroke*. 1994;25:782-6.
316. Homma S, Di Tullio MR, Sacco RL, Mihalatos D, Li Mandri G, Mohr JP. Characteristics of patent foramen ovale associated with cryptogenic stroke. A biplane transesophageal echocardiographic study. *Stroke*. 1994;25:582-6.
317. Lechat P, Mas JL, Lascault G, et al. Prevalence of patent foramen ovale in patients with stroke. *N Engl J Med*. 1988;318:1148-52.
318. Hausmann D, Mugge A, Becht I, Daniel WG. Diagnosis of patent foramen ovale by transesophageal echocardiography and association with cerebral and peripheral embolic events. *Am J Cardiol*. 1992;70:668-72.
319. Godtfredsen J, Egeblad H, Berning J. Echocardiography in lone atrial fibrillation. *Acta Med Scand*. 1983;213:111-3.
320. Kupari M, Leinonen H, Koskinen P. Value of routine echocardiography in new-onset atrial fibrillation. *Int J Cardiol*. 1987;16:106-8.
321. Patterson MW, De Souza E. Two-dimensional echocardiographic diagnosis of arrhythmogenic right ventricular dysplasia presenting as frequent ventricular extrasystoles in a child. *Pediatr Cardiol*. 1988;9:41-3.
322. Mehta D, Odawara H, Ward DE, McKenna WJ, Davies MJ, Camm AJ. Echocardiographic and histologic evaluation of the right ventricle in ventricular tachycardias of left bundle branch block morphology without overt cardiac abnormality. *Am J Cardiol*. 1989;63:939-44.
323. Morgera T, Salvi A, Alberti E, Silvestri F, Camerini F. Morphological findings in apparently idiopathic ventricular tachycardia. An echocardiographic haemodynamic and histologic study. *Eur Heart J*. 1985;6:323-34.
324. Sanfilippo AJ, Abascal VM, Sheehan M, et al. Atrial enlargement as a consequence of atrial fibrillation. A prospective echocardiographic study. *Circulation*. 1990;82:792-7.
325. Iwase M, Sotobata I, Yokota M, et al. Evaluation by pulsed Doppler echocardiography of the atrial contribution to left ventricular filling in patients with DDD pacemakers. *Am J Cardiol*. 1986;58:104-9.
326. Egeblad H, Rasmussen V. Analysis of arrhythmias based on atrial wall motion. Usefulness and feasibility of recording left and right atrial systole by echocardiography. *Acta Med Scand*. 1986;219:283-9.
327. Drinkovic N. Subcostal M-mode echocardiography of the right atrial wall for differentiation of supraventricular tachyarrhythmias with aberration from ventricular tachycardia. *Am Heart J*. 1984;107:326-31.
328. Goldbaum TS, Goldstein SA, Lindsay J Jr. Subcostal M-mode echocardiography of atrial septum for diagnosis of atrial flutter. *Am J Cardiol*. 1984;54:1143-5.
329. Windle JR, Armstrong WF, Feigenbaum H, Miles WM, Prystowsky EN. Determination of the earliest site of ventricular activation in Wolff-Parkinson-White syndrome: application of digital continuous loop two-dimensional echocardiography. *J Am Coll Cardiol*. 1986;7:1286-94.
330. Dittrich HC, Erickson JS, Schneiderman T, Blacky AR, Savides T, Nicod PH. Echocardiographic and clinical predictors for outcome of elective cardioversion of atrial fibrillation. *Am J Cardiol*. 1989;63:193-7.
331. Dethy M, Chassat C, Roy D, Mercier LA. Doppler echocardiographic predictors of recurrence of atrial fibrillation after cardioversion. *Am J Cardiol*. 1988;62:723-6.
332. Hoglund C, Rosenhamer G. Echocardiographic left atrial dimension as a predictor of maintaining sinus rhythm after conversion of atrial fibrillation. *Acta Med Scand*. 1985;217:411-5.
333. Henry WL, Morganroth J, Pearlman AS, et al. Relation between echocardiographically determined left atrial size and atrial fibrillation. *Circulation*. 1976;53:273-9.
334. Ewy GA, Ulfers L, Hager WD, Rosenfeld AR, Roeske WR, Goldman S. Response of atrial fibrillation to therapy: role of etiology and left atrial diameter. *J Electrocardiol*. 1980;13:119-24.
335. Halpern SW, Ellrod G, Singh BN, Mandel WJ. Efficacy of intravenous procainamide infusion in converting atrial fibrillation to sinus rhythm: relation to left atrial size. *Br Heart J*. 1980;44:589-95.
336. Flugelman MY, Hasin Y, Katznelson N, Kriwisky M, Shefer A, Gotsman MS. Restoration and maintenance of sinus rhythm after mitral valve surgery for mitral stenosis. *Am J Cardiol*. 1984;54:617-9.
337. Manning WJ, Silverman DI, Gordon SP, Krumholz HM, Douglas PS. Cardioversion from atrial fibrillation without prolonged anticoagulation with use of transesophageal echocardiography to exclude the presence of atrial thrombi. *N Engl J Med*. 1993;328:750-5.
338. Black IW, Fatkin D, Sagar KB, et al. Exclusion of atrial thrombus by transesophageal echocardiography does not preclude embolism after cardioversion of atrial fibrillation. A multicenter study. *Circulation*. 1994;89:2509-13.
339. Fatkin D, Kuchar DL, Thorburn CW, Feneley MP. Transesophageal echocardiography before and during direct current cardioversion of atrial fibrillation: evidence for atrial stunning as a mechanism of thromboembolic complications. *J Am Coll Cardiol*. 1994;23:307-16.
340. Laupacis A, Albers G, Dalen J, Dunn M, Feinberg W, Jacobson A. Antithrombotic therapy in atrial fibrillation. *Chest*. 1995;108(suppl):352S-9S.
341. Stoddard MF, Dawkins PR, Prince CR, Ammash NM. Left atrial appendage thrombus is not uncommon in patients with acute atrial fibrillation and a recent embolic event: a transesophageal echocardiographic study. *J Am Coll Cardiol*. 1995;25:452-9.
342. Santiago D, Warshofsky M, Li Mandri G, et al. Left atrial appendage function and thrombus formation in atrial fibrillation-flutter: a transesophageal echocardiographic study. *J Am Coll Cardiol*. 1994;24:159-64.
343. Deleted during update.
344. Deleted during update.
345. Deleted during update.
346. Maron BJ, Nichols PF, Pickle LW, Wesley YE, Mulvihill JJ. Patterns of inheritance in hypertrophic cardiomyopathy: assessment by M-mode and two-dimensional echocardiography. *Am J Cardiol*. 1984;53:1087-94.
347. Maron BJ, Spirito P, Wesley Y, Arce J. Development and progression of left ventricular hypertrophy in children with hypertrophic cardiomyopathy. *N Engl J Med*. 1986;315:610-4.
348. Spirito P, Maron BJ. Absence of progression of left ventricular hypertrophy in adult patients with hypertrophic cardiomyopathy. *J Am Coll Cardiol*. 1987;9:1013-7.
349. De Paep A, Devereux RB, Dietz HC, Hennekam RC, Pyeritz RE. Revised diagnostic criteria for the Marfan syndrome. *Am J Med Genet*. 1996;62:417-26.
350. Silverman DI, Burton KJ, Gray J, et al. Life expectancy in the Marfan syndrome. *Am J Cardiol*. 1995;75:157-60.
351. Michels VV, Moll PP, Miller FA, et al. The frequency of familial

- dilated cardiomyopathy in a series of patients with idiopathic dilated cardiomyopathy. *N Engl J Med.* 1992;326:77-82.
352. Gilbert EM, Krueger SK, Murray JL, et al. Echocardiographic evaluation of potential cardiac transplant donors. *J Thorac Cardiovasc Surg.* 1988;95:1003-7.
 353. Stoddard MF, Longaker RA. The role of transesophageal echocardiography in cardiac donor screening. *Am Heart J.* 1993;125:1676-81.
 354. Hada Y, Sakamoto T, Amano K, et al. Prevalence of hypertrophic cardiomyopathy in a population of adult Japanese workers as detected by echocardiographic screening. *Am J Cardiol.* 1987;59:183-4.
 355. Maron BJ, Gardin JM, Flack JM, Gidding SS, Kurosaki TT, Bild DE. Prevalence of hypertrophic cardiomyopathy 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;92:785-9.
 356. Lewis JF, Maron BJ, Diggs JA, Spencer JE, Mehrotra PP, Curry CL. Preparticipation echocardiographic screening for cardiovascular disease in a large, predominantly black population of collegiate athletes. *Am J Cardiol.* 1989;64:1029-33.
 357. Maron BJ, Bodison SA, Wesley YE, Tucker E, Green KJ. Results of screening a large group of intercollegiate competitive athletes for cardiovascular disease. *J Am Coll Cardiol.* 1987;10:1214-21.
 358. Weidenbener EJ, Krauss MD, Waller BF, Taliercio CP. Incorporation of screening echocardiography in the preparticipation exam. *Clin J Sport Med.* 1995;5:86-9.
 359. Balogun MO, Omotoso AB, Bell E, et al. An audit of emergency echocardiography in a district general hospital. *Int J Cardiol.* 1993;41:65-8.
 360. Sanfilippo A, Weyman A. The role of echocardiography in managing critically ill patients. *J Crit Illness.* 1988;3:27-44.
 361. Parker MM, Cunnion RE, Parrillo JE. Echocardiography and nuclear cardiac imaging in the critical care unit. *JAMA.* 1985;254:2935-9.
 362. Wolfe MW, Lee RT, Feldstein ML, Parker JA, Come PC, Goldhaber SZ. Prognostic significance of right ventricular hypokinesis and perfusion lung scan defects in pulmonary embolism. *Am Heart J.* 1994;127:1371-5.
 363. Pearson AC, Castello R, Labovitz AJ. Safety and utility of transesophageal echocardiography in the critically ill patient. *Am Heart J.* 1990;119:1083-9.
 364. Oh JK, Seward JB, Khandheria BK, et al. Transesophageal echocardiography in critically ill patients. *Am J Cardiol.* 1990;66:1492-5.
 365. Font VE, Obarski TP, Klein AL, et al. Transesophageal echocardiography in the critical care unit. *Cleve Clin J Med.* 1991;58:315-22.
 366. Foster E, Schiller NB. The role of transesophageal echocardiography in critical care: UCSF experience. *J Am Soc Echocardiogr.* 1992;5:368-74.
 367. Hwang JJ, Shyu KG, Chen JJ, Tseng YZ, Kuan P, Lien WP. Usefulness of transesophageal echocardiography in the treatment of critically ill patients. *Chest.* 1993;104:861-6.
 368. Khoury AF, Afridi I, Quinones MA, Zoghbi WA. Transesophageal echocardiography in critically ill patients: feasibility, safety, and impact on management. *Am Heart J.* 1994;127:1363-71.
 369. 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;107:774-9.
 370. Pearson AC. Non-invasive evaluation of the hemodynamically unstable patient: the advantages of seeing clearly. *Mayo Clin Proc.* 1995;70:1012-14.
 371. Jardin F, Valtier B, Beauchet A, Dubourg O, Bourdarias JP. Invasive monitoring combined with two-dimensional echocardiographic study in septic shock. *Intensive Care Med.* 1994;20:550-4.
 372. Reichert CL, Visser CA, Koolen JJ, et al. Transesophageal echocardiography in hypotensive patients after cardiac operations. Comparison with hemodynamic parameters. *J Thorac Cardiovasc Surg.* 1992;104:321-6.
 373. Chan KL. Transesophageal echocardiography for assessing cause of hypotension after cardiac surgery. *Am J Cardiol.* 1988;62:1142-3.
 374. Stoddard MF, Prince CR, Ammash N, Goad JL, Vogel RL. Pulsed Doppler transesophageal echocardiographic determination of cardiac output in human beings: comparison with thermodilution technique. *Am Heart J.* 1993;126:956-62.
 375. Feinberg MS, Hopkins WE, Davila-Roman VG, Barzilai B. Multiplane transesophageal echocardiographic doppler imaging accurately determines cardiac output measurements in critically ill patients. *Chest.* 1995;107:769-73.
 376. Dabaghi SF, Rokey R, Rivera JM, Saliba WI, Majid PA. Comparison of echocardiographic assessment of hemodynamics in the intensive care unit with right-sided cardiac catheterization. *Am J Cardiol.* 1995;76:392-5.
 377. Practice guidelines for perioperative transesophageal echocardiography: a report by the American Society of Anesthesiologists and the Society of Cardiovascular Anesthesiologists Task Force on Transesophageal Echocardiography. *Anesthesiology.* 1996;84:986-1006.
 378. Oh JK, Freeman WK. Transesophageal echocardiography in critically ill patients. *Transesophageal Echocardiography.* Boston, Mass: Little, Brown & Co; 1994;17:549-75.
 379. Miller FA, Seward JB, Gersh BJ, Tajik AJ, Mucha P Jr. Two-dimensional echocardiographic findings in cardiac trauma. *Am J Cardiol.* 1982;50:1022-7.
 380. Beggs CW, Helling TS, Evans LL, Hays LV, Kennedy FR, Crouse LJ. Early evaluation of cardiac injury by two-dimensional echocardiography in patients suffering blunt chest trauma. *Ann Emerg Med.* 1987;16:542-5.
 381. Reid CL, Kawanishi DT, Rahimtoola SH, Chandraratna PA. Chest trauma: evaluation by two-dimensional echocardiography. *Am Heart J.* 1987;113:971-6.
 382. Cachecho R, Grindlinger GA, Lee VW. The clinical significance of myocardial contusion. *J Trauma.* 1992;33:68-73.
 383. Hiatt JR, Yeatman LA, Child JS. The value of echocardiography in blunt chest trauma. *J Trauma.* 1988;28:914-22.
 384. Johnson SB, Kearney PA, Smith MD. Echocardiography in the evaluation of thoracic trauma. *Surg Clin North Am.* 1995;75:193-205.
 385. Paone RF, Peacock JB, Smith DL. Diagnosis of myocardial contusion. *South Med J.* 1993;86:867-70.
 386. Maron BJ, Poliac LC, Kaplan JA, Mueller FO. Blunt impact to the chest leading to sudden death from cardiac arrest during sports activities. *N Engl J Med.* 1995;333:337-42.
 387. Malangoni MA, McHenry CR, Jacobs DG. Outcome of serious blunt cardiac injury. *Surgery.* 1994;116:628-33.
 388. Schiavone WA, Ghumrauri BK, Catalano DR, et al. The use of echocardiography in the emergency management of nonpenetrating traumatic myocardial rupture. *Ann Emerg Med.* 1991;20:1248-50.
 389. End A, Rodler S, Oturanlar D, et al. Elective surgery for blunt car-

- diac trauma. *J Trauma*. 1994;37:798-802.
390. Brooks SW, Young JC, Cmolik B, et al. The use of transesophageal echocardiography in the evaluation of chest trauma. *J Trauma*. 1992;32:761-5.
391. Karalis DG, Victor MF, Davis GA, et al. The role of echocardiography in blunt chest trauma: a transthoracic and transesophageal echocardiographic study. *J Trauma*. 1994;36:53-8.
392. Shapiro MJ, Yanofsky SD, Trapp J, et al. Cardiovascular evaluation in blunt thoracic trauma using transesophageal echocardiography (TEE). *J Trauma*. 1991;31:835-9.
393. Catoire P, Orliaguet G, Liu N, et al. Systematic transesophageal echocardiography for detection of mediastinal lesions in patients with multiple injuries. *J Trauma*. 1995;38:96-102.
394. Vlahakes GJ, Warren RL. Traumatic rupture of the aorta. *N Engl J Med*. 1995;332:389-90.
395. Goldstein SA, Mintz GS, Lindsay J Jr. Aorta: comprehensive evaluation by echocardiography and transesophageal echocardiography. *J Am Soc Echocardiogr*. 1993;6:634-59.
396. Buckmaster MJ, Kearney PA, Johnson SB, Smith MD, Sapin PM. Further experience with transesophageal echocardiography in the evaluation of thoracic aortic injury. *J Trauma*. 1994;37:989-95.
397. Young RJ, Joynt GM, Gomersall CD. Notice of redundant publication: transesophageal echocardiography in the diagnosis of traumatic rupture of the aorta. *N Engl J Med*. 1995;333:457.
398. Fernandez LG, Lain KY, Messersmith RN, et al. Transesophageal echocardiography for diagnosing aortic injury: a case report and summary of current imaging techniques. *J Trauma*. 1994;36:877-80.
399. Jimenez E, Martin M, Krukenkamp I, Barrett J. Subxiphoid pericardiotomy versus echocardiography: a prospective evaluation of the diagnosis of occult penetrating cardiac injury. *Surgery*. 1990;108:676-9.
400. Freshman SP, Wisner DH, Weber CJ. 2-D echocardiography: emergent use in the evaluation of penetrating precordial trauma. *J Trauma*. 1991;31:902-5.
401. Plummer D, Brunette D, Asinger R, Ruiz E. Emergency department echocardiography improves outcome in penetrating cardiac injury. *Ann Emerg Med*. 1992;21:709-12.
402. Nagy KK, Lohmann C, Kim DO, Barrett J. Role of echocardiography in the diagnosis of occult penetrating cardiac injury. *J Trauma*. 1995;38:859-62.
403. Bolton JW, Bynoe RP, Lazar HL, Almond CH. Two-dimensional echocardiography in the evaluation of penetrating intrapericardial injuries. *Ann Thorac Surg*. 1993;56:506-9.
404. Meyer DM, Jessen ME, Grayburn PA. Use of echocardiography to detect occult cardiac injury after penetrating thoracic trauma: a prospective study. *J Trauma*. 1995;39:902-7.
405. Cha EK, Mittal V, Allaben RD. Delayed sequelae of penetrating cardiac injury. *Arch Surg*. 1993;128:836-9.
406. Demetriades D, Charalambides C, Sareli P, Pantanowitz D. Late sequelae of penetrating cardiac injuries. *Br J Surg*. 1990;77:813-4.
407. Xie SW, Picard MH. Two-dimensional and color Doppler echocardiographic diagnosis of penetrating missile wounds of the heart: chronic complications from intracardiac course of a bullet. *J Am Soc Echocardiogr*. 1992;5:81-4.
408. Porembka DT, Johnson DJ II, Hoit BD, Reising J III, Davis K Jr, Koutlas T. Penetrating cardiac trauma: a perioperative role for transesophageal echocardiography. *Anesth Analg*. 1993;77:1275-7.
409. Friedrich SP, Berman AD, Baim DS, Diver DJ. Myocardial perforation in the cardiac catheterization laboratory: incidence, presentation, diagnosis, and management. *Cathet Cardiovasc Diagn*. 1994;32:99-107.
410. Huhta JC, Glasow P, Murphy DJ, et al. Surgery without catheterization for congenital heart defects: management of 100 patients. *J Am Coll Cardiol*. 1987;9:823-9.
411. Krabill KA, Ring WS, Foker JE, et al. Echocardiographic versus cardiac catheterization diagnosis of infants with congenital heart disease requiring cardiac surgery. *Am J Cardiol*. 1987;60:351-4.
412. George B, DiSessa TG, Williams R, Friedman WF, Laks H. Coarctation repair without cardiac catheterization in infants. *Am Heart J*. 1987;114:1421-5.
413. Alboliras ET, Seward JB, Hagler DJ, Danielson GK, Puga FJ, Tajik AJ. Impact of two-dimensional and Doppler echocardiography on care of children aged two years and younger. *Am J Cardiol*. 1988;61:166-9.
414. Leung MP, Mok CK, Lau KC, Lo R, Yeung CY. The role of cross sectional echocardiography and pulsed Doppler ultrasound in the management of neonates in whom congenital heart disease is suspected. A prospective study. *Br Heart J*. 1986;56:73-82.
415. Lipshultz SE, Sanders SP, Mayer JE, Colan SD, Lock JE. Are routine preoperative cardiac catheterization and angiography necessary before repair of ostium primum atrial septal defect? *J Am Coll Cardiol*. 1988;11:373-8.
416. Gewitz MH, Werner JC, Kleinman CS, Hellenbrand WE, Talner NS. Role of echocardiography in aortic stenosis: pre- and postoperative studies. *Am J Cardiol*. 1979;43:67-73.
417. Smallhorn JF, Pauperio H, Benson L, Freedom RM, Rowe RD. Pulsed Doppler assessment of pulmonary vein obstruction. *Am Heart J*. 1985;110:483-6.
418. Vick GW, Murphy DJ, Ludomirsky A, et al. Pulmonary venous and systemic ventricular inflow obstruction in patients with congenital heart disease: detection by combined two-dimensional and Doppler echocardiography. *J Am Coll Cardiol*. 1987;9:580-7.
419. Valdes-Cruz LM, Pieroni DR, Roland JM, Shematek JP. Recognition of residual postoperative shunts by contrast echocardiographic techniques. *Circulation*. 1977;55:148-52.
420. Meijboom EJ, Ebels T, Anderson RH, et al. Left atrioventricular valve after surgical repair in atrioventricular septal defect with separate valve orifices (ostium primum atrial septal defect): an echo-Doppler study. *Am J Cardiol*. 1986;57:433-6.
421. Meijboom EJ, Wyse RK, Ebels T, et al. Doppler mapping of postoperative left atrioventricular valve regurgitation. *Circulation*. 1988;77:311-5.
422. Chin AJ, Sanders SP, Williams RG, Lang P, Norwood WI, Castaneda AR. Two-dimensional echocardiographic assessment of caval and pulmonary venous pathways after the Senning operation. *Am J Cardiol*. 1983;52:118-26.
423. Satomi G, Nakamura K, Takao A, Imai Y. Two-dimensional echocardiographic detection of pulmonary venous channel stenosis after Senning's operation. *Circulation*. 1983;68:545-9.
424. Smallhorn JF, Burrows P, Wilson G, Coles J, Gilday DL, Freedom RM. Two-dimensional and pulsed Doppler echocardiography in the postoperative evaluation of total anomalous pulmonary venous connection. *Circulation*. 1987;76:298-305.
425. Fyfe DA, Currie PJ, Seward JB, et al. Continuous-wave Doppler determination of the pressure gradient across pulmonary artery bands: hemodynamic correlation in 20 patients. *Mayo Clin Proc*. 1984;59:744-50.
426. Stevenson JG, Kawabori I, Bailey WW. Noninvasive evaluation of Blalock-Taussig shunts: determination of patency and differentiation from patent ductus arteriosus by Doppler echocardiography. *Am Heart J*. 1983;106:1121-32.

427. Marx GR, Allen HD, Goldberg SJ. Doppler echocardiographic estimation of systolic pulmonary artery pressure in patients with aortic-pulmonary shunts. *J Am Coll Cardiol.* 1986;7:880-5.
428. Hagler DJ, Seward JB, Tajik AJ, Ritter DG. Functional assessment of the Fontan operation: combined M-mode, two-dimensional and Doppler echocardiographic studies. *J Am Coll Cardiol.* 1984;4:756-64.
429. DiSessa TG, Child JS, Perloff JK, et al. Systemic venous and pulmonary arterial flow patterns after Fontan's procedure for tricuspid atresia or single ventricle. *Circulation.* 1984;70:898-902.
430. Nakazawa M, Nojima K, Okuda H, et al. Flow dynamics in the main pulmonary artery after the Fontan procedure in patients with tricuspid atresia or single ventricle [published erratum appears in *Circulation.* 1987;76:609]. *Circulation.* 1987;75:1117-23.
431. Borow KM, Colan SD, Neumann A. Altered left ventricular mechanics in patients with valvular aortic stenosis and coarctation of the aorta: effects on systolic performance and late outcome. *Circulation.* 1985;72:515-22.
432. Martin GR, Short BL. Doppler echocardiographic evaluation of cardiac performance in infants on prolonged extracorporeal membrane oxygenation. *Am J Cardiol.* 1988;62:929-34.
433. Musewe NN, Alexander DJ, Teshima I, Smallhorn JF, Freedom RM. Echocardiographic evaluation of the spectrum of cardiac anomalies associated with trisomy 13 and trisomy 18. *J Am Coll Cardiol.* 1990;15:673-7.
434. Musewe NN, Smallhorn JF, Benson LN, Burrows PE, Freedom RM. Validation of Doppler-derived pulmonary arterial pressure in patients with ductus arteriosus under different hemodynamic states. *Circulation.* 1987;76:1081-91.
435. Leung MP, Mok CK, Hui PW. Echocardiographic assessment of neonates with pulmonary atresia and intact ventricular septum. *J Am Coll Cardiol.* 1988;12:719-25.
436. Bash SE, Huhta JC, Vick GW, Gutgesell HP, Ott DA. Hypoplastic left heart syndrome: is echocardiography accurate enough to guide surgical palliation? *J Am Coll Cardiol.* 1986;7:610-6.
437. Huhta JC, Gutgesell HP, Latson LA, Huffines FD. Two-dimensional echocardiographic assessment of the aorta in infants and children with congenital heart disease. *Circulation.* 1984;70:417-24.
438. Bierman FZ, Williams RG. Subxiphoid two-dimensional imaging of the interatrial septum in infants and neonates with congenital heart disease. *Circulation.* 1979;60:80-90.
439. Bierman FZ, Fellows K, Williams RG. Prospective identification of ventricular septal defects in infancy using subxiphoid two-dimensional echocardiography. *Circulation.* 1980;62:807-17.
440. Lima CO, Sahn DJ, Valdes-Cruz LM, et al. Noninvasive prediction of transvalvular pressure gradient in patients with pulmonary stenosis by quantitative two-dimensional echocardiographic Doppler studies. *Circulation.* 1983;67:866-71.
441. Simpson IA, Sahn DJ, Valdes-Cruz LM, Chung KJ, Sherman FS, Swensson RE. Color Doppler flow mapping in patients with coarctation of the aorta: new observations and improved evaluation with color flow diameter and proximal acceleration as predictors of severity. *Circulation.* 1988;77:736-44.
442. Murphy DJ, Ludomirsky A, Huhta JC. Continuous-wave Doppler in children with ventricular septal defect: noninvasive estimation of interventricular pressure gradient. *Am J Cardiol.* 1986;57:428-32.
443. Huhta JC, Latson LA, Gutgesell HP, Cooley DA, Kearney DL. Echocardiography in the diagnosis and management of symptomatic aortic valve stenosis in infants. *Circulation.* 1984;70:438-44.
444. Roberson DA, Silverman NH. Ebstein's anomaly: echocardiographic and clinical features in the fetus and neonate. *J Am Coll Cardiol.* 1989;14:1300-7.
445. Silverman NH, Hudson S. Evaluation of right ventricular volume and ejection fraction in children by two-dimensional echocardiography. *Pediatr Cardiol.* 1983;4:197-203.
446. Colan SD, Borow KM, Neumann A. Left ventricular end-systolic wall stress-velocity of fiber shortening relation: a load-independent index of myocardial contractility. *J Am Coll Cardiol.* 1984;4:715-24.
447. van der Velde ME, Parness IA, Colan SD, et al. Two-dimensional echocardiography in the pre- and postoperative management of totally anomalous pulmonary venous connection. *J Am Coll Cardiol.* 1991;18:1746-51.
448. Van Hare GF, Schmidt KG, Cassidy SC, Gooding CA, Silverman NH. Color Doppler flow mapping in the ultrasound diagnosis of total anomalous pulmonary venous connection. *J Am Soc Echocardiogr.* 1988;1:341-7.
449. Huhta JC, Gutgesell HP, Nihill MR. Cross sectional echocardiographic diagnosis of total anomalous pulmonary venous connection. *Br Heart J.* 1985;53:525-34.
450. Pasquini L, Sanders SP, Parness IA, et al. Conal anatomy in 119 patients with d-loop transposition of the great arteries and ventricular septal defect: an echocardiographic and pathologic study. *J Am Coll Cardiol.* 1993;21:1712-21.
451. Sanders SP, Bierman FZ, Williams RG. Conotruncal malformations: diagnosis in infancy using subxiphoid 2-dimensional echocardiography. *Am J Cardiol.* 1982;50:1361-7.
452. Trowitzsch E, Colan SD, Sanders SP. Global and regional right ventricular function in normal infants and infants with transposition of the great arteries after Senning operation. *Circulation.* 1985;72:1008-14.
453. Borow KM, Keane JF, Castaneda AR, Freed MD. Systemic ventricular function in patients with tetralogy of fallot, ventricular septal defect and transposition of the great arteries repaired during infancy. *Circulation.* 1981;64:878-85.
454. van Doesburg NH, Bierman FZ, Williams RG. Left ventricular geometry in infants with d-transposition of the great arteries and intact interventricular septum. *Circulation.* 1983;68:733-9.
455. Pasquini L, Sanders SP, Parness IA, et al. Coronary echocardiography in 406 patients with d-loop transposition of the great arteries. *J Am Coll Cardiol.* 1994;24:763-8.
456. Day RW, Laks H, Drinkwater DC. The influence of coronary anatomy on the arterial switch operation in neonates. *J Thorac Cardiovasc Surg.* 1992;104:706-12.
457. Koike K, Musewe NN, Smallhorn JF, Freedom RM. Distinguishing between anomalous origin of the left coronary artery from the pulmonary trunk and dilated cardiomyopathy: role of echocardiographic measurement of the right coronary artery diameter. *Br Heart J.* 1989;61:192-7.
458. Smythe JF, Dyck JD, Smallhorn JF, Freedom RM. Natural history of cardiac rhabdomyoma in infancy and childhood. *Am J Cardiol.* 1990;66:1247-9.
459. Reller MD, Tsang RC, Meyer RA, Braun CP. Relationship of prospective diabetes control in pregnancy to neonatal cardiorespiratory function. *J Pediatr.* 1985;106:86-90.
460. Ino T, Sherwood WG, Benson LN, Wilson GJ, Freedom RM, Rowe RD. Cardiac manifestations in disorders of fat and carnitine metabolism in infancy. *J Am Coll Cardiol.* 1988;11:1301-8.
461. Newburger JW, Rosenthal A, Williams RG, Fellows K, Miettinen OS. Noninvasive tests in the initial evaluation of heart murmurs in children. *N Engl J Med.* 1983;308:61-4.

462. Geva T, Hegesh J, Frand M. The clinical course and echocardiographic features of Marfan's syndrome in childhood. *Am J Dis Child.* 1987;141:1179-82.
463. Geva T, Sanders SP, Diogenes MS, Rockenmacher S, Van Praagh R. Two-dimensional and Doppler echocardiographic and pathologic characteristics of the infantile Marfan syndrome. *Am J Cardiol.* 1990;65:1230-7.
464. Pan CW, Chen CC, Wang SP, Hsu TL, Chiang BN. Echocardiographic study of cardiac abnormalities in families of patients with Marfan's syndrome. *J Am Coll Cardiol.* 1985;6:1016-20.
465. Capannari TE, Daniels SR, Meyer RA, Schwartz DC, Kaplan S. Sensitivity, specificity and predictive value of two-dimensional echocardiography in detecting coronary artery aneurysms in patients with Kawasaki disease. *J Am Coll Cardiol.* 1986;7:355-60.
466. Rowley AH, Gonzalez-Crussi F, Gidding SS, Duffy CE, Shulman ST. Incomplete Kawasaki disease with coronary artery involvement. *J Pediatr.* 1987;110:409-13.
467. Vogel M, Smallhorn JF, Freedom RM. Serial analysis of regional left ventricular wall motion by two-dimensional echocardiography in patients with coronary artery enlargement after Kawasaki disease. *J Am Coll Cardiol.* 1992;20:915-9.
468. Satomi G, Nakamura K, Narai S, Takao A. Systematic visualization of coronary arteries by two-dimensional echocardiography in children and infants: evaluation in Kawasaki's disease and coronary arteriovenous fistulas. *Am Heart J.* 1984;107:497-505.
469. Anderson TM, Meyer RA, Kaplan S. Long-term echocardiographic evaluation of cardiac size and function in patients with Kawasaki disease. *Am Heart J.* 1985;110:107-15.
470. Dajani AS, Taubert KA, Takahashi M, et al. Guidelines for long-term management of patients with Kawasaki disease. Report from the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, American Heart Association. *Circulation.* 1994;89:916-22.
471. Guidelines for the diagnosis of rheumatic fever. Jones Criteria, 1992 update. Special Writing Group of the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease of the Council on Cardiovascular Disease in the Young of the American Heart Association. *JAMA.* 1993;269:73.
472. Lipshultz SE, Chanock S, Sanders SP, Colan SD, Perez-Atayde A, McIntosh K. Cardiovascular manifestations of human immunodeficiency virus infection in infants and children. *Am J Cardiol.* 1989;63:1489-97.
473. Gagliardi MG, Bevilacqua M, Squitieri C, Boldrini R, Di Julio DP, Marcelletti C. Dilated cardiomyopathy caused by acute myocarditis in pediatric patients: evolution of myocardial damage in a group of potential heart transplant candidates. *J Heart Lung Transplant.* 1993;12(suppl):S224-9.
474. Wiles HB, Gillette PC, Harley RA, Upshur JK. Cardiomyopathy and myocarditis in children with ventricular ectopic rhythm. *J Am Coll Cardiol.* 1992;20:359-62.
475. Lewis AB. Prognostic value of echocardiography in children with idiopathic dilated cardiomyopathy. *Am Heart J.* 1994;128:133-6.
476. Griffin ML, Hernandez A, Martin TC, et al. Dilated cardiomyopathy in infants and children. *J Am Coll Cardiol.* 1988;11:139-44.
477. Steinherz LJ, Graham T, Hurwitz R, et al. Guidelines for cardiac monitoring of children during and after anthracycline therapy: report of the Cardiology Committee of the Children's Cancer Study Group. *Pediatrics.* 1992;89:942-9.
478. Lipshultz SE, Sanders SP, Goorin AM, Krischer JP, Sallan SE, Colan SD. Monitoring for anthracycline cardiotoxicity. *Pediatrics.* 1994;93:433-7.
479. Chin TK, Perloff JK, Williams RG, et al. Isolated noncompaction of left ventricular myocardium. *Circulation.* 1990;82:507-13.
480. Alboliras ET, Shub C, Gomez MR, et al. Spectrum of cardiac involvement in Friedreich's ataxia: clinical, electrocardiographic and echocardiographic observations. *Am J Cardiol.* 1986;58:518-24.
481. Melacini P, Fanin M, Danieli GA, et al. Cardiac involvement in Becker muscular dystrophy. *J Am Coll Cardiol.* 1993;22:1927-34.
482. Kimball TR, Daniels SR, Loggie JM, Khoury P, Meyer RA. Relation of left ventricular mass, preload, afterload and contractility in pediatric patients with essential hypertension. *J Am Coll Cardiol.* 1993;21:997-1001.
483. Boucek MM, Mathis CM, Boucek RJ, et al. Prospective evaluation of echocardiography for primary rejection surveillance after infant heart transplantation: comparison with endomyocardial biopsy. *J Heart Lung Transplant.* 1994;13:66-73.
484. Bernstein D, Kolla S, Miner M, et al. Cardiac growth after pediatric heart transplantation. *Circulation.* 1992;85:1433-9.
485. Dobell AR, Trusler GA, Smallhorn JF, Williams WG. Atrial thrombi after the Fontan operation. *Ann Thorac Surg.* 1986;42:664-7.
486. Deleted during update.
487. Fyfe DA, Kline CH, Sade RM, Gillette PC. Transesophageal echocardiography detects thrombus formation not identified by transthoracic echocardiography after the Fontan operation. *J Am Coll Cardiol.* 1991;18:1733-7.
488. Fyfe DA, Ritter SB, Snider AR, et al. Guidelines for transesophageal echocardiography in children. *J Am Soc Echocardiogr.* 1992;5:640-4.
489. Wolfe LT, Rossi A, Ritter SB. Transesophageal echocardiography in infants and children: use and importance in the cardiac intensive care unit. *J Am Soc Echocardiogr.* 1993;6:286-9.
490. Takamoto S, Kyo S, Adachi H, Matsumura M, Yokote Y, Omoto R. Intraoperative color flow mapping by real-time two-dimensional Doppler echocardiography for evaluation of valvular and congenital heart disease and vascular disease. *J Thorac Cardiovasc Surg.* 1985;90:802-12.
491. Gussenhoven EJ, van Herwerden LA, Roelandt J, Ligtvoet KM, Bos E, Witsenburg M. Intraoperative two-dimensional echocardiography in congenital heart disease. *J Am Coll Cardiol.* 1987;9:565-72.
492. Czer LS, Maurer G, Bolger AF, et al. Intraoperative evaluation of mitral regurgitation by Doppler color flow mapping. *Circulation.* 1987;76(suppl III):III-108-16.
493. Deleted during update.
494. Sahn DJ, Lange LW, Allen HD, et al. Quantitative real-time cross-sectional echocardiography in the developing normal human fetus and newborn. *Circulation.* 1980;62:588-97.
495. Schmidt KG, de Araujo LMD, Silverman NH. Evaluation of structural and functional abnormalities of the fetal heart by echocardiography. *J Cardiac Imag.* 1988;2:57-76.
496. Kenny JF, Plappert T, Doubilet P, et al. Changes in intracardiac blood flow velocities and right and left ventricular stroke volumes with gestational age in the normal human fetus: a prospective Doppler echocardiographic study. *Circulation.* 1986;74:1208-16.
497. Tegnander E, Eik-Nes SH, Johansen OJ, Linker DT. Prenatal detection of heart defects at the routine fetal examination at 18 weeks in a non-selected population. *Ultrasound Obstet Gynecol.* 1995;5:372-80.
498. Ott WJ. The accuracy of antenatal fetal echocardiography screen-

- ing in high- and low-risk patients. *Am J Obstet Gynecol.* 1995;172:1741-7.
499. Bromley B, Estroff JA, Sanders SP, et al. Fetal echocardiography: accuracy and limitations in a population at high and low risk for heart defects. *Am J Obstet Gynecol.* 1992;166:1473-81.
500. Parness IA, Yeager SB, Sanders SP, Benacerraf B, Colan SD, VanPraagh R. Echocardiographic diagnosis of fetal heart defects in mid trimester. *Arch Dis Child.* 1988;63:1137-45.
501. Holley DG, Martin GR, Brenner JJ, et al. Diagnosis and management of fetal cardiac tumors: a multicenter experience and review of published reports. *J Am Coll Cardiol.* 1995;26:516-20.
502. Allan LD, Crawford DC, Chita SK, Anderson RH, Tynan MJ. Familial recurrence of congenital heart disease in a prospective series of mothers referred for fetal echocardiography. *Am J Cardiol.* 1986;58:334-7.
503. Strasburger JF, Huhta JC, Carpenter RJ, Garson AJ, McNamara DG. Doppler echocardiography in the diagnosis and management of persistent fetal arrhythmias. *J Am Coll Cardiol.* 1986;7:1386-91.
504. Kleinman CS, Donnerstein RL, Jaffe CC, et al. Fetal echocardiography. A tool for evaluation of in utero cardiac arrhythmias and monitoring of in utero therapy: analysis of 71 patients. *Am J Cardiol.* 1983;51:237-43.
505. Machado MV, Tynan MJ, Curry PV, Allan LD. Fetal complete heart block. *Br Heart J.* 1988;60:512-5.
506. Martin GR, Ruckman RN. Fetal echocardiography: a large clinical experience and follow-up. *J Am Soc Echocardiogr.* 1990;3:4-8.
507. Silverman NH, Golbus MS. Echocardiographic techniques for assessing normal and abnormal fetal cardiac anatomy. *J Am Coll Cardiol.* 1985;5(suppl):20S-9S.
508. Schmidt KG, Birk E, Silverman NH, Scagnelli SA. Echocardiographic evaluation of dilated cardiomyopathy in the human fetus. *Am J Cardiol.* 1989;63:599-605.
509. Chang AC, Huhta JC, Yoon GY, et al. Diagnosis, transport, and outcome in fetuses with left ventricular outflow tract obstruction. *J Thorac Cardiovasc Surg.* 1991;102:841-8.
510. Smythe JF, Copel JA, Kleinman CS. Outcome of prenatally detected cardiac malformations. *Am J Cardiol.* 1992;69:1471-4.
511. Blake DM, Copel JA, Kleinman CS. Hypoplastic left heart syndrome: prenatal diagnosis, clinical profile, and management. *Am J Obstet Gynecol.* 1991;165:529-34.
512. Crawford DC, Wright VM, Drake DP, Allan LD. Fetal diaphragmatic hernia: the value of fetal echocardiography in the prediction of postnatal outcome. *Br J Obstet Gynaecol.* 1989;96:705-10.
513. Jacobson SJ, Jones K, Johnson K, et al. Prospective multicentre study of pregnancy outcome after lithium exposure during first trimester. *Lancet.* 1992;339:530-3.
514. Meyer RA, Hagler D, Huhta J, et al. Guidelines for physician training in fetal echocardiography: recommendations of the Society of Pediatric Echocardiography Committee on Physician Training. *J Am Soc Echocardiogr.* 1990;3:1-3.
515. Smith SC Jr, Dove JT, Jacobs AK, et al. ACC/AHA guidelines for percutaneous coronary interventions (revision of the 1993 PTCA guidelines)—executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1993 Guidelines for Percutaneous Transluminal Coronary Angioplasty). *J Am Coll Cardiol.* 2001;37:2215-39.
516. Mintz GS, Nissen SE, Anderson WD, et al. American College of Cardiology Clinical Expert Consensus Document on Standards for Acquisition, Measurement and Reporting of Intravascular Ultrasound Studies (IVUS). A report of the American College of Cardiology Task Force on Clinical Expert Consensus Documents. *J Am Coll Cardiol.* 2001;37:1478-92.
517. Eagle KA, Berger PB, Calkins H, et al. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1996 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). 2002; Available at: www.acc.org. Accessed: 6/26/02.
518. Sutherland GR, Stewart MJ, Groundstroem KW, et al. Color Doppler myocardial imaging: a new technique for the assessment of myocardial function. *J Am Soc Echocardiogr.* 1994;7:441-58.
519. Isaza K. Pulsed Doppler tissue imaging [letter; comment]. *Am J Cardiol.* 1998;81:663.
520. Attenhofer JCH, Turina J, Mayer K, et al. Echocardiography in the evaluation of systolic murmurs of unknown cause. *Am J Med.* 2000;108:614-20.
521. Monin JL, Monchi M, Gest V, Duval-Moulin AM, Dubois-Randé JL, Gueret P. Aortic stenosis with severe left ventricular dysfunction and low transvalvular pressure gradients: risk stratification by low-dose dobutamine echocardiography. *J Am Coll Cardiol.* 2001;37:2101-7.
522. Corti R, Binggeli C, Turina M, Jenni R, Luscher TF, Turina J. Predictors of long-term survival after valve replacement for chronic aortic regurgitation: is M-mode echocardiography sufficient? *Eur Heart J.* 2001;22:866-73.
523. Flemming MA, Oral H, Rothman ED, Briesmiester K, Petrusha JA, Starling MR. Echocardiographic markers for mitral valve surgery to preserve left ventricular performance in mitral regurgitation. *Am Heart J.* 2000;140:476-82.
524. 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;75:191-4.
525. Lin SS, Roger VL, Pascoe R, Seward JB, Pellikka PA. Dobutamine stress Doppler hemodynamics in patients with aortic stenosis: feasibility, safety, and surgical correlations. *Am Heart J.* 1998;136:1010-6.
526. Gardin JM, Schumacher D, Constantine G, Davis KD, Leung C, Reid CL. Valvular abnormalities and cardiovascular status following exposure to dexfenfluramine or phentermine/fenfluramine. *JAMA.* 2000;283:1703-9.
527. Jick H. Heart valve disorders and appetite-suppressant drugs [editorial; comment]. *JAMA.* 2000;283:1738-40.
528. Bonow RO, Carabello BA, Cheitlin MD. American College of Cardiology/American Heart Association Practice Guidelines for the Management of Patients with Valvular Heart Disease. *J Am Coll Cardiol.* 1998;32:1486-588.
529. Shah PM. Echocardiographic diagnosis of mitral valve prolapse. *J Am Soc Echocardiogr.* 1994;7:286-93.
530. Freed LA, Levy D, Levine RA, et al. Prevalence and clinical outcome of mitral-valve prolapse. *N Engl J Med.* 1999;341:1-7.
531. Devereux RB. Recent developments in the diagnosis and management of mitral valve prolapse. *Curr Opin Cardiol.* 1995;10:107-16.
532. Durack DT, Lukes AS, Bright DK. New criteria for diagnosis of

NEW REFERENCES

- infective endocarditis: utilization of specific echocardiographic findings. Duke Endocarditis Service. *Am J Med.* 1994;96:200-9.
533. Rosen AB, Fowler VG Jr., Corey GR, et al. Cost-effectiveness of transesophageal echocardiography to determine the duration of therapy for intravascular catheter-associated *Staphylococcus aureus* bacteremia. *Ann Intern Med.* 1999;130:810-20.
534. Sasaki H, Charuzi Y, Beeder C, Sugiki Y, Lew AS. Utility of echocardiography for the early assessment of patients with nondiagnostic chest pain. *Am Heart J.* 1986;112:494-7.
535. Parisi AF, Moynihan PF, Folland ED, Feldman CL. Quantitative detection of regional left ventricular contraction abnormalities by two-dimensional echocardiography. II. Accuracy in coronary artery disease. *Circulation.* 1981;63:761-7.
536. Stamm RB, Gibson RS, Bishop HL, Carabello BA, Beller GA, Martin RP. Echocardiographic detection of infarct-localized asynergy and remote asynergy during acute myocardial infarction: correlation with the extent of angiographic coronary disease. *Circulation.* 1983;67:233-44.
537. Lundgren C, Bourdillon PD, Dillon JC, Feigenbaum H. Comparison of contrast angiography and two-dimensional echocardiography for the evaluation of left ventricular regional wall motion abnormalities after acute myocardial infarction. *Am J Cardiol.* 1990;65:1071-77.
538. Prevaliti M, Poli A, Lanzarini L, Fetiveau R, Mussini A, Ferrario M. Dobutamine stress echocardiography for assessment of myocardial viability and ischemia in acute myocardial infarction treated with thrombolysis. *Am J Cardiol.* 1993;72:124G-130G.
539. Watada H, Ito H, Oh H, et al. Dobutamine stress echocardiography predicts reversible dysfunction and quantitates the extent of irreversibly damaged myocardium after reperfusion of anterior myocardial infarction. *J Am Coll Cardiol.* 1994;24:624-30.
540. Salustri A, Elhendy A, Garyfallydis P, et al. Prediction of improvement of ventricular function after first acute myocardial infarction using low-dose dobutamine stress echocardiography. *Am J Cardiol.* 1994;74:853-6.
541. Poli A, Prevaliti M, Lanzarini L, et al. Comparison of dobutamine stress echocardiography with dipyridamole stress echocardiography for detection of viable myocardium after myocardial infarction treated with thrombolysis. *Heart.* 1996;75:240-6.
542. Bolognese L, Antoniucci D, Rovai D, et al. Myocardial contrast echocardiography versus dobutamine echocardiography for predicting functional recovery after acute myocardial infarction treated with primary coronary angioplasty. *J Am Coll Cardiol.* 1996;28:1677-83.
543. Minardi G, Di Segni M, Manzara CC, et al. Diagnostic and prognostic value of dipyridamole and dobutamine stress echocardiography in patients with Q-wave acute myocardial infarction. *Am J Cardiol.* 1997;80:847-51.
544. Smart S, Wynsen J, Sagar K. Dobutamine-atropine stress echocardiography for reversible dysfunction during the first week after acute myocardial infarction: limitations and determinants of accuracy. *J Am Coll Cardiol.* 1997;30:1669-78.
545. Orlandini AD, Tuero EI, Diaz R, Vilamajo OA, Paolasso EA. Acute cardiac rupture during dobutamine-atropine echocardiography stress test. *J Am Soc Echocardiogr.* 2000;13:152-3.
546. Carlos ME, Smart SC, Wynsen JC, Sagar KB. Dobutamine stress echocardiography for risk stratification after myocardial infarction. *Circulation.* 1997;95:1402-10.
547. Greco CA, Salustri A, Seccareccia F, et al. Prognostic value of dobutamine echocardiography early after uncomplicated acute myocardial infarction: a comparison with exercise electrocardiography. *J Am Coll Cardiol.* 1997;29:261-7.
548. Sclavo MG, Noussan P, Pallisco O, Presbitero P. Usefulness of dipyridamole-echocardiographic test to identify jeopardized myocardium after thrombolysis. Limited clinical predictivity of dipyridamole-echocardiographic test in convalescing acute myocardial infarction: correlation with coronary angiography. *Eur Heart J.* 1992;13:1348-55.
549. Picano E, Landi P, Bolognese L, et al. Prognostic value of dipyridamole echocardiography early after uncomplicated myocardial infarction: a large-scale, multicenter trial. The EPIC Study Group. *Am J Med.* 1993;95:608-18.
550. van Daele ME, McNeill AJ, Fioretti PM, et al. Prognostic value of dipyridamole sestamibi single-photon emission computed tomography and dipyridamole stress echocardiography for new cardiac events after an uncomplicated myocardial infarction. *J Am Soc Echocardiogr.* 1994;7:370-80.
551. Sicari R, Picano E, Landi P, et al. Prognostic value of dobutamine-atropine stress echocardiography early after acute myocardial infarction. Echo Dobutamine International Cooperative (EDIC) Study. *J Am Coll Cardiol.* 1997;29:254-60.
552. Prevaliti M, Fetiveau R, Lanzarini L, Cavalotti C, Klersy C. Prognostic value of myocardial viability and ischemia detected by dobutamine stress echocardiography early after acute myocardial infarction treated with thrombolysis. *J Am Coll Cardiol.* 1998;32:380-6.
553. Volpi A, De Vita C, Franzosi MG, et al. Determinants of 6-month mortality of survivors of myocardial infarction after thrombolysis. Results of the GISSI-2 data base. The Ad Hoc Working Group of the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI)-2 Data Base. *Circulation.* 1993;88:416-29.
554. Goldberg RJ, Gore JM, Alpert JS, Dalen JE. Recent changes in attack and survival rates of acute myocardial infarction (1975 through 1981). The Worcester Heart Attack Study. *JAMA.* 1986;255:2774-9.
555. Limacher MC, Quinones MA, Poliner LR, Nelson JG, Winters WL Jr., Waggoner AD. Detection of coronary artery disease with exercise two-dimensional echocardiography. Description of a clinically applicable method and comparison with radionuclide ventriculography. *Circulation.* 1983;67:1211-18.
556. Armstrong WF, O'Donnell J, Dillon JC, McHenry PL, Morris SN, Feigenbaum H. Complementary value of two-dimensional exercise echocardiography to routine treadmill exercise testing. *Ann Intern Med.* 1986;105:829-35.
557. Sheikh KH, Bengtson JR, Helmy S, et al. Relation of quantitative coronary lesion measurements to the development of exercise-induced ischemia assessed by exercise echocardiography. *J Am Coll Cardiol.* 1990;15:1043-51.
558. Marwick TH, D'Hondt AM, Mairesse GH, et al. Comparative ability of dobutamine and exercise stress in inducing myocardial ischaemia in active patients [published erratum appears in *Br Heart J* 1994; 72:590]. *Br Heart J.* 1994;72:31-8.
559. Williams MJ, Marwick TH, O'Gorman D, Foale RA. Comparison of exercise echocardiography with an exercise score to diagnose coronary artery disease in women. *Am J Cardiol.* 1994;74:435-8.
560. Marwick TH, Anderson T, Williams MJ, et al. Exercise echocardiography is an accurate and cost-efficient technique for detection of coronary artery disease in women. *J Am Coll Cardiol.* 1995;26:335-41.
561. Bjornstad K, Aakhus S, Hatle L. Comparison of digital dipyridamole stress echocardiography and upright bicycle stress echocardiography for identification of coronary artery stenosis. *Cardiology.* 1995;86:514-20.
562. Marwick TH, Torelli J, Harjai K, et al. Influence of left ventricu-

- lar hypertrophy on detection of coronary artery disease using exercise echocardiography. *J Am Coll Cardiol.* 1995;26:1180-6.
563. Tawa CB, Baker WB, Kleiman NS, Trakhtenbroit A, Desir R, Zoghbi WA. Comparison of adenosine echocardiography, with and without isometric handgrip, to exercise echocardiography in the detection of ischemia in patients with coronary artery disease. *J Am Soc Echocardiogr.* 1996;9:33-43.
564. Luotolahti M, Saraste M, Hartiala J. Exercise echocardiography in the diagnosis of coronary artery disease. *Ann Med.* 1996;28:73-7.
565. Tian J, Zhang G, Wang X, Cui J, Xiao J. Exercise echocardiography: feasibility and value for detection of coronary artery disease. *Chin Med J (Engl).* 1996;109:381-4.
566. Martin TW, Seaworth JF, Johns JP, Pupa LE, Condos WR. Comparison of adenosine, dipyridamole, and dobutamine in stress echocardiography. *Ann Intern Med.* 1992;116:190-6.
567. McNeill AJ, Fioretti PM, el Said EM, Salustri A, Forster T, Roelandt JRTC. Enhanced sensitivity for detection of coronary artery disease by addition of atropine to dobutamine stress echocardiography. *Am J Cardiol.* 1992;70:41-6.
568. Previtali M, Lanzarini L, Fèveau R, et al. Comparison of dobutamine stress echocardiography, dipyridamole stress echocardiography and exercise stress testing for diagnosis of coronary artery disease. *Am J Cardiol.* 1993;72:865-70.
569. Takeuchi M, Araki M, Nakashima Y, Kuroiwa A. Comparison of dobutamine stress echocardiography and stress thallium-201 single-photon emission computed tomography for detecting coronary artery disease. *J Am Soc Echocardiogr.* 1993;6:593-602.
570. Ostojic M, Picano E, Beleslin B, et al. Dipyridamole-dobutamine echocardiography: a novel test for the detection of milder forms of coronary artery disease. *J Am Coll Cardiol.* 1994;23:1115-22.
571. Ho FM, Huang PJ, Liau CS, et al. Dobutamine stress echocardiography compared with dipyridamole thallium-201 single-photon emission computed tomography in detecting coronary artery disease. *Eur Heart J.* 1995;16:570-5.
572. Daoud EG, Pitt A, Armstrong WF. Electrocardiographic response during dobutamine stress echocardiography. *Am Heart J.* 1995;129:672-77.
573. Pingitore A, Picano E, Colosso MQ, et al. The atropine factor in pharmacologic stress echocardiography. Echo Persantine (EPIC) and Echo Dobutamine International Cooperative (EDIC) Study Groups. *J Am Coll Cardiol.* 1996;27:1164-70.
574. Schroder K, Voller H, Dingerkus H, et al. Comparison of the diagnostic potential of four echocardiographic stress tests shortly after acute myocardial infarction: submaximal exercise, transesophageal atrial pacing, dipyridamole, and dobutamine-atropine. *Am J Cardiol.* 1996;77:909-14.
575. Anthopoulos LP, Bonou MS, Kardaras FG, et al. Stress echocardiography in elderly patients with coronary artery disease: applicability, safety and prognostic value of dobutamine and adenosine echocardiography in elderly patients. *J Am Coll Cardiol.* 1996;28:52-9.
576. Ling LH, Pellikka PA, Mahoney DW, et al. Atropine augmentation in dobutamine stress echocardiography: role and incremental value in a clinical practice setting. *J Am Coll Cardiol.* 1996;28:551-7.
577. Dionisopoulos PN, Collins JD, Smart SC, Knickelbine TA, Sagar KB. The value of dobutamine stress echocardiography for the detection of coronary artery disease in women. *J Am Soc Echocardiogr.* 1997;10:811-7.
578. Ho YL, Wu CC, Huang PJ, et al. Assessment of coronary artery disease in women by dobutamine stress echocardiography: comparison with stress thallium-201 single-photon emission computed tomography and exercise electrocardiography. *Am Heart J.* 1998;135:655-62.
579. Takeuchi M, Sonoda S, Miura Y, Kuroiwa A. Comparative diagnostic value of dobutamine stress echocardiography and stress thallium-201 single-photon-emission computed tomography for detecting coronary artery disease in women. *Coron Artery Dis.* 1996;7:831-5.
580. Roger VL, Pellikka PA, Bell MR, Chow CW, Bailey KR, Seward JB. Sex and test verification bias. Impact on the diagnostic value of exercise echocardiography. *Circulation.* 1997;95:405-10.
581. Elhendy A, Geleijnse ML, van Domburg RT, et al. Gender differences in the accuracy of dobutamine stress echocardiography for the diagnosis of coronary artery disease. *Am J Cardiol.* 1997;80:1414-8.
582. Sharp SM, Sawada SG, Segar DS, et al. Dobutamine stress echocardiography: detection of coronary artery disease in patients with dilated cardiomyopathy. *J Am Coll Cardiol.* 1994;24:934-9.
583. Garber AM, Solomon NA. Cost-effectiveness of alternative test strategies for the diagnosis of coronary artery disease. *Ann Intern Med.* 1999;130:719-28.
584. Kuntz KM, Fleischmann KE, Hunink MG, Douglas PS. Cost-effectiveness of diagnostic strategies for patients with chest pain. *Ann Intern Med.* 1999;130:709-18.
585. Kim C, Kwok YS, Saha S, Redberg RF. Diagnosis of suspected coronary artery disease in women: a cost-effectiveness analysis. *Am Heart J.* 1999;137:1019-27.
586. Melin JA, Wijns W, Vanbutsele RJ, et al. Alternative diagnostic strategies for coronary artery disease in women: demonstration of the usefulness and efficiency of probability analysis. *Circulation.* 1985;71:535-42.
587. Mulvagh SL, DeMaria AN, Feinstein SB, et al. Contrast echocardiography: current and future applications. *J Am Soc Echocardiogr.* 2000;13:331-42.
588. Prince CR, Stoddard MF, Morris GT, et al. Dobutamine two-dimensional transesophageal echocardiographic stress testing for detection of coronary artery disease. *Am Heart J.* 1994;128:36-41.
589. Panza JA, Laurienzo JM, Curiel RV, Quyyumi AA, Cannon RO III. Transesophageal dobutamine stress echocardiography for evaluation of patients with coronary artery disease. *J Am Coll Cardiol.* 1994;24:1260-67.
590. Laurienzo JM, Cannon RO III, Quyyumi AA, Dilsizian V, Panza JA. Improved specificity of transesophageal dobutamine stress echocardiography compared to standard tests for evaluation of coronary artery disease in women presenting with chest pain. *Am J Cardiol.* 1997;80:1402-7.
591. Frohwein S, Klein JL, Lane A, Taylor WR. Transesophageal dobutamine stress echocardiography in the evaluation of coronary artery disease. *J Am Coll Cardiol.* 1995;25:823-9.
592. Cortigiani L, Dodi C, Paolini EA, Bernardi D, Bruno G, Nannini E. Prognostic value of pharmacological stress echocardiography in women with chest pain and unknown coronary artery disease. *J Am Coll Cardiol.* 1998;32:1975-81.
593. Poldermans D, Fioretti PM, Boersma E, et al. Dobutamine-atropine stress echocardiography and clinical data for predicting late cardiac events in patients with suspected coronary artery disease. *Am J Med.* 1994;97:119-25.
594. Heupler S, Mehta R, Lobo A, Leung D, Marwick TH. Prognostic implications of exercise echocardiography in women with known or suspected coronary artery disease. *J Am Coll Cardiol.* 1997;30:414-20.
595. McCully RB, Roger VL, Mahoney DW, et al. Outcome after normal exercise echocardiography and predictors of subsequent cardiac events: follow-up of 1,325 patients. *J Am Coll Cardiol.* 1998;31:144-9.
596. Chuah SC, Pellikka PA, Roger VL, McCully RB, Seward JB.

- Role of dobutamine stress echocardiography in predicting outcome in 860 patients with known or suspected coronary artery disease. *Circulation*. 1998;97:1474-80.
597. Davar JI, Brull DJ, Bulugahipitiya S, Coghlan JG, Lipkin DP, Evans TR. Prognostic value of negative dobutamine stress echo in women with intermediate probability of coronary artery disease. *Am J Cardiol*. 1999;83:100-2, A8.
598. Picano E, Severi S, Michelassi C, et al. Prognostic importance of dipyridamole-echocardiography test in coronary artery disease. *Circulation*. 1989;80:450-57.
599. Gibbons RJ, Abrams JA, Chatterjee K, et al. ACC/AHA 2002 guideline update for the management of patients with chronic stable angina: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1999 Guidelines for the Management of Patients With Chronic Stable Angina). 2003. Available at: www.acc.org. Accessed: February 24, 2003.
600. Kwok Y, Kim C, Grady D, Segal M, Redberg R. Meta-analysis of exercise testing to detect coronary artery disease in women. *Am J Cardiol*. 1999;83:660-6.
601. Tong AT, Douglas PS. Stress echocardiography in women. *Cardiol Clin*. 1999;17:573-82.
602. Fang JC, Rocco T, Jarcho J, Ganz P, Mudge GH. Noninvasive assessment of transplant-associated arteriosclerosis. *Am Heart J*. 1998;135:980-7.
603. Collings CA, Pinto FJ, Valantine HA, Popylisen S, Puryear JV, Schnittger I. Exercise echocardiography in heart transplant recipients: a comparison with angiography and intracoronary ultrasonography. *J Heart Lung Transplant*. 1994;13:604-13.
604. Cohn JM, Wilensky RL, O'Donnell JA, Bourdillon PD, Dillon JC, Feigenbaum H. Exercise echocardiography, angiography, and intracoronary ultrasound after cardiac transplantation. *Am J Cardiol*. 1996;77:1216-9.
605. Spes CH, Mudra H, Schnaack SD, et al. Dobutamine stress echocardiography for noninvasive diagnosis of cardiac allograft vasculopathy: a comparison with angiography and intravascular ultrasound. *Am J Cardiol*. 1996;78:168-74.
606. Ciliberto GR, Massa D, Mangiacavalli M, et al. High-dose dipyridamole echocardiography test in coronary artery disease after heart transplantation. *Eur Heart J*. 1993;14:48-52.
607. Lewis JF, Selman SB, Murphy JD, Mills RM Jr, Geiser EA, Conti CR. Dobutamine echocardiography for prediction of ischemic events in heart transplant recipients. *J Heart Lung Transplant*. 1997;16:390-3.
608. Rahimtoola SH. Concept and evaluation of hibernating myocardium. *Annu Rev Med*. 1999;50:75-86.
609. Shan K, Nagueh SF, Zoghbi WA. Assessment of myocardial viability with stress echocardiography. *Cardiol Clin*. 1999;17:539-53, ix.
610. Yoshida K, Gould KL. Quantitative relation of myocardial infarct size and myocardial viability by positron emission tomography to left ventricular ejection fraction and 3-year mortality with and without revascularization. *J Am Coll Cardiol*. 1993;22:984-97.
611. Lee KS, Marwick TH, Cook SA, et al. Prognosis of patients with left ventricular dysfunction, with and without viable myocardium after myocardial infarction. Relative efficacy of medical therapy and revascularization. *Circulation*. 1994;90:2687-94.
612. Di Carli MF, Asgarzadie F, Schelbert HR, et al. Quantitative relation between myocardial viability and improvement in heart failure symptoms after revascularization in patients with ischemic cardiomyopathy. *Circulation*. 1995;92:3436-44.
613. Nagueh SF. Dobutamine echocardiography versus nuclear cardiac imaging for evaluation of myocardial viability. *Curr Opin Cardiol*. 1997;12:547-52.
614. Marzullo P, Parodi O, Reichenhofer B, et al. Value of rest thallium-201/technetium-99m sestamibi scans and dobutamine echocardiography for detecting myocardial viability. *Am J Cardiol*. 1993;71:166-72.
615. Alfieri O, La Canna G, Giubbini R, Pardini A, Zogno M, Fucci C. Recovery of myocardial function. The ultimate target of coronary revascularization. *Eur J Cardiothorac Surg*. 1993;7:325-30.
616. Charney R, Schwinger ME, Chun J, et al. Dobutamine echocardiography and resting-redistribution thallium-201 scintigraphy predicts recovery of hibernating myocardium after coronary revascularization. *Am Heart J*. 1994;128:864-69.
617. Senior R, Glenville B, Basu S, et al. Dobutamine echocardiography and thallium-201 imaging predict functional improvement after revascularisation in severe ischaemic left ventricular dysfunction. *Br Heart J*. 1995;74:358-64.
618. Haque T, Furukawa T, Takahashi M, Kinoshita M. Identification of hibernating myocardium by dobutamine stress echocardiography: comparison with thallium-201 reinjection imaging. *Am Heart J*. 1995;130:553-63.
619. Iliceto S, Galiuto L, Marchese A, et al. Analysis of microvascular integrity, contractile reserve, and myocardial viability after acute myocardial infarction by dobutamine echocardiography and myocardial contrast echocardiography. *Am J Cardiol*. 1996;77:441-5.
620. Varga A, Ostojic M, Djordjevic-Dikic A, et al. Infra-low dose dipyridamole test. A novel dose regimen for selective assessment of myocardial viability by vasodilator stress echocardiography. *Eur Heart J*. 1996;17:629-34.
621. Baer FM, Voth E, Deutsch HJ, et al. Predictive value of low dose dobutamine transesophageal echocardiography and fluorine-18 fluorodeoxyglucose positron emission tomography for recovery of regional left ventricular function after successful revascularization. *J Am Coll Cardiol*. 1996;28:60-9.
622. Vanoverschelde JL, D'Hondt AM, Marwick T, et al. Head-to-head comparison of exercise-redistribution-reinjection thallium single-photon emission computed tomography and low dose dobutamine echocardiography for prediction of reversibility of chronic left ventricular ischemic dysfunction. *J Am Coll Cardiol*. 1996;28:432-42.
623. Gerber BL, Vanoverschelde JL, Bol A, et al. Myocardial blood flow, glucose uptake, and recruitment of inotropic reserve in chronic left ventricular ischemic dysfunction. Implications for the pathophysiology of chronic myocardial hibernation. *Circulation*. 1996;94:651-9.
624. Bax JJ, Cornel JH, Visser FC, et al. Prediction of recovery of myocardial dysfunction after revascularization. Comparison of fluorine-18 fluorodeoxyglucose/thallium-201 SPECT, thallium-201 stress-reinjection SPECT and dobutamine echocardiography. *J Am Coll Cardiol*. 1996;28:558-64.
625. Perrone-Filardi P, Pace L, Prastaro M, et al. Assessment of myocardial viability in patients with chronic coronary artery disease. Rest-4-hour-24-hour 201Tl tomography versus dobutamine echocardiography. *Circulation*. 1996;94:2712-9.
626. Qureshi U, Nagueh SF, Afridi I, et al. Dobutamine echocardiography and quantitative rest-redistribution 201Tl tomography in myocardial hibernation. Relation of contractile reserve to 201Tl uptake and comparative prediction of recovery of function. *Circulation*. 1997;95:626-35.
627. Furukawa T, Haque T, Takahashi M, Kinoshita M. An assessment of dobutamine echocardiography and end-diastolic wall thickness

- for predicting post-revascularization functional recovery in patients with chronic coronary artery disease. *Eur Heart J*. 1997;18:798-806.
628. Cornel JH, Bax JJ, Fioretti PM, et al. Prediction of improvement of ventricular function after revascularization. 18F-fluorodeoxyglucose single-photon emission computed tomography vs low-dose dobutamine echocardiography. *Eur Heart J*. 1997;18:941-48.
629. Grayburn P, Weiss JL, Hack TC, et al. Phase III multicenter trial comparing the efficacy of 2% dodecafluoropentane emulsion (EchoGen) and sonicated 5% human albumin (Albunex) as ultrasound contrast agents in patients with suboptimal echocardiograms. *J Am Coll Cardiol*. 1998;32:230-6.
630. Vasan RS, Larson MG, Benjamin EJ, Evans JC, Reiss CK, Levy D. Congestive heart failure in subjects with normal versus reduced left ventricular ejection fraction: prevalence and mortality in a population-based cohort. *J Am Coll Cardiol*. 1999;33:1948-55.
631. Aurigemma GP, Gottdiener JS, Shemanski L, Gardin J, Kitzman D. Predictive value of systolic and diastolic function for incident congestive heart failure in the elderly: the Cardiovascular Health Study. *J Am Coll Cardiol*. 2001;37:1042-8.
632. Rossvoll O, Hatle LK. Pulmonary venous flow velocities recorded by transthoracic Doppler ultrasound: relation to left ventricular diastolic pressures. *J Am Coll Cardiol*. 1993;21:1687-96.
633. Nagueh SF, Middleton KJ, Kopelen HA, Zoghbi WA, Quinones MA. Doppler tissue imaging: a noninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressures. *J Am Coll Cardiol*. 1997;30:1527-33.
634. 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;102:1788-94.
635. Sivaram CA, Jugdutt BI, Amy RW, Basualdo CA, Haraphongse M, Shnitka TK. Cardiac amyloidosis: combined use of two-dimensional echocardiography and electrocardiography in noninvasive screening before biopsy. *Clin Cardiol*. 1985;8:511-8.
636. Bhandari AK, Nanda NC. Myocardial texture characterization by two-dimensional echocardiography. *Am J Cardiol*. 1983;51:817-25.
637. Maggiolini S, Bozzano A, Russo P, et al. Echocardiography-guided pericardiocentesis with probe-mounted needle: report of 53 cases. *J Am Soc Echocardiogr*. 2001;14:821-4.
638. Tsang TS, Freeman WK, Sinak LJ, Seward JB. Echocardiographically guided pericardiocentesis: evolution and state-of-the-art technique. *Mayo Clin Proc*. 1998;73:647-52.
639. Ha JW, Oh JK, Ling LH, Nishimura RA, Seward JB, Tajik AJ. Annulus paradoxus: transmitral flow velocity to mitral annular velocity ratio is inversely proportional to pulmonary capillary wedge pressure in patients with constrictive pericarditis. *Circulation*. 2001;104:976-8.
640. Erbel R, Alfonso F, Boileau C, et al. Diagnosis and management of aortic dissection. *Eur Heart J*. 2001;22:1642-81.
641. David TE. Aortic valve-sparing operations for aortic root aneurysm. *Semin Thorac Cardiovasc Surg*. 2001;13:291-6.
642. Maraj R, Rerkpattanapipat P, Jacobs LE, Makornwattana P, Kotler MN. Meta-analysis of 143 reported cases of aortic intramural hematoma. *Am J Cardiol*. 2000;86:664-8.
643. Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA*. 2000;283:897-903.
644. Wittlich N, Erbel R, Eichler A, et al. Detection of central pulmonary artery thromboemboli by transesophageal echocardiography in patients with severe pulmonary embolism. *J Am Soc Echocardiogr*. 1992;5:515-24.
645. Verdecchia P, Schillaci G, Borgioni C, et al. Prognostic significance of serial changes in left ventricular mass in essential hypertension. *Circulation*. 1998;97:48-54.
646. Palmieri V, Dahlof B, DeQuattro V, et al. Reliability of echocardiographic assessment of left ventricular structure and function: the PRESERVE study. Prospective Randomized Study Evaluating Regression of Ventricular Enlargement. *J Am Coll Cardiol*. 1999;34:1625-32.
647. McNamara RL, Lima JA, Whelton PK, Powe NR. Echocardiographic identification of cardiovascular sources of emboli to guide clinical management of stroke: a cost-effectiveness analysis. *Ann Intern Med*. 1997;127:775-87.
648. Agmon Y, Khandheria BK, Meissner I, et al. Frequency of atrial septal aneurysms in patients with cerebral ischemic events. *Circulation*. 1999;99:1942-44.
649. Steiner MM, Di Tullio MR, Rundek T, et al. Patent foramen ovale size and embolic brain imaging findings among patients with ischemic stroke. *Stroke*. 1998;29:944-8.
650. Homma S, Di Tullio MR, Sacco RL, Sciacca RR, Smith C, Mohr JP. Surgical closure of patent foramen ovale in cryptogenic stroke patients. *Stroke*. 1997;28:2376-81.
651. Transesophageal echocardiographic correlates of thromboembolism in high-risk patients with nonvalvular atrial fibrillation. The Stroke Prevention in Atrial Fibrillation Investigators Committee on Echocardiography. *Ann Intern Med*. 1998;128:639-47.
652. Zabalgoitia M, Halperin JL, Pearce LA, Blackshear JL, Asinger RW, Hart RG. Transesophageal echocardiographic correlates of clinical risk of thromboembolism in nonvalvular atrial fibrillation. Stroke Prevention in Atrial Fibrillation III Investigators. *J Am Coll Cardiol*. 1998;31:1622-6.
653. Panagiotopoulos K, Tomanidis S, Saridakis N, Vemmos K, Mouloupoulos S. Left atrial and left atrial appendage functional abnormalities in patients with cardioembolic stroke in sinus rhythm and idiopathic atrial fibrillation. *J Am Soc Echocardiogr*. 1998;11:711-9.
654. Homma S, Sacco RL, Di Tullio MR, Sciacca RR, Mohr JP. Effect of medical treatment in stroke patients with patent foramen ovale: patent foramen ovale in Cryptogenic Stroke Study. *Circulation*. 2002;105:2625-31.
655. Mas JL, Arquizan C, Lamy C, et al. Recurrent cerebrovascular events associated with patent foramen ovale, atrial septal aneurysm, or both. *N Engl J Med*. 2001;345:1740-6.
656. Saxon LA, Stevenson WG, Fonarow GC, et al. Transesophageal echocardiography during radiofrequency catheter ablation of ventricular tachycardia. *Am J Cardiol*. 1993;72:658-661.
657. Tucker KJ, Curtis AB, Murphy J, et al. Transesophageal echocardiographic guidance of transseptal left heart catheterization during radiofrequency ablation of left-sided accessory pathways in humans. *Pacing Clin Electrophysiol*. 1996;19:272-81.
658. Chu E, Kalman JM, Kwasman MA, et al. Intracardiac echocardiography during radiofrequency catheter ablation of cardiac arrhythmias in humans. *J Am Coll Cardiol*. 1994;24:1351-7.
659. Fisher WG, Pelini MA, Bacon ME. Adjunctive intracardiac echocardiography to guide slow pathway ablation in human atrioventricular nodal reentrant tachycardia: anatomic insights. *Circulation*. 1997;96:3021-9.
660. Pires LA, Huang SK, Wagshal AB, Mazzola F, Young PG, Moser S. Clinical utility of routine transthoracic echocardiographic stud-

- ies after uncomplicated radiofrequency catheter ablation: a prospective multicenter study. The Atakr Investigators Group. *Pacing Clin Electrophysiol.* 1996;19:1502-7.
661. Cox JL, Schuessler RB, Lappas DG, Boineau JP. An 8 1/2-year clinical experience with surgery for atrial fibrillation. *Ann Surg.* 1996;224:267-73.
662. Albirini A, Scalia GM, Murray RD, et al. Left and right atrial transport function after the Maze procedure for atrial fibrillation: an echocardiographic Doppler follow-up study. *J Am Soc Echocardiogr.* 1997;10:937-45.
663. Danias PG, Caulfield TA, Weigner MJ, Silverman DI, Manning WJ. Likelihood of spontaneous conversion of atrial fibrillation to sinus rhythm. *J Am Coll Cardiol.* 1998;31:588-92.
664. Poelaert J, Schmidt C, Colardyn F. Transoesophageal echocardiography in the critically ill. *Anaesthesia.* 1998;53:55-68.
665. Verhorst PM, Kamp O, Welling RC, Van Eenige MJ, Visser CA. Transesophageal echocardiographic predictors for maintenance of sinus rhythm after electrical cardioversion of atrial fibrillation. *Am J Cardiol.* 1997;79:1355-9.
666. Perez Y, Duval AM, Carville C, et al. Is left atrial appendage flow a predictor for outcome of cardioversion of nonvalvular atrial fibrillation? A transthoracic and transesophageal echocardiographic study. *Am Heart J.* 1997;134:745-51.
667. Omran H, Jung W, Schimpf R, et al. Echocardiographic parameters for predicting maintenance of sinus rhythm after internal atrial defibrillation. *Am J Cardiol.* 1998;81:1446-9.
668. Manning WJ, Silverman DI, Keighley CS, Oettgen P, Douglas PS. Transesophageal echocardiographically facilitated early cardioversion from atrial fibrillation using short-term anticoagulation: final results of a prospective 4.5-year study. *J Am Coll Cardiol.* 1995;25:1354-1361.
669. Stoddard MF, Dawkins PR, Prince CR, Longaker RA. Transesophageal echocardiographic guidance of cardioversion in patients with atrial fibrillation. *Am Heart J.* 1995;129:1204-15.
670. Klein AL, Grimm RA, Black IW, et al. Cardioversion guided by transesophageal echocardiography: the ACUTE Pilot Study. A randomized, controlled trial. Assessment of Cardioversion Using Transesophageal Echocardiography. *Ann Intern Med.* 1997;126:200-9.
671. Weigner MJ, Thomas LR, Patel U, et al. Early cardioversion of atrial fibrillation facilitated by transesophageal echocardiography: short-term safety and impact on maintenance of sinus rhythm at 1 year. *Am J Med.* 2001;110:694-702.
672. Pfammatter JP, Berdat P, Hammerli M, Carrel T. Pediatric cardiac surgery after exclusively echocardiography-based diagnostic work-up. *Int J Cardiol.* 2000;74:185-90.
673. Klein AL, Grimm RA, Murray RD, et al. Use of transesophageal echocardiography to guide cardioversion in patients with atrial fibrillation. *N Engl J Med.* 2001;344:1411-20.
674. Laupacis A, Albers G, Dalen J, Dunn MI, Jacobson AK, Singer DE. Antithrombotic therapy in atrial fibrillation. *Chest.* 1998;114:579S-589S.
675. Fuster V, Ryden LE, Asinger RW, et al. ACC/AHA/ESC guidelines for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines and Policy Conferences (Committee to Develop Guidelines for the Management of Patients With Atrial Fibrillation). *J Am Coll Cardiol.* 2003;38:1266i-1xx.
676. Recchia D, Barzilai B. Echocardiography in the evaluation of patients with syncope. *J Gen Intern Med.* 1995;10:649-55.
677. Sarasin FP, Junod AF, Carballo D, Slama S, Unger PF, Louis-Simonet M. Role of echocardiography in the evaluation of syncope: a prospective study. *Heart.* 2002;88:363-7.
678. Charron P, Dubourg O, Desnos M, et al. Diagnostic value of electrocardiography and echocardiography for familial hypertrophic cardiomyopathy in a genotyped adult population. *Circulation.* 1997;96:214-9.
679. Grunig E, Tasman JA, Kucherer H, Franz W, Kubler W, Katus HA. Frequency and phenotypes of familial dilated cardiomyopathy. *J Am Coll Cardiol.* 1998;31:186-94.
680. Mestroni L, Rocco C, Gregori D, et al. Familial dilated cardiomyopathy: evidence for genetic and phenotypic heterogeneity. Heart Muscle Disease Study Group. *J Am Coll Cardiol.* 1999;34:181-90.
681. Baig MK, Goldman JH, Caforio AL, Coonar AS, Keeling PJ, McKenna WJ. Familial dilated cardiomyopathy: cardiac abnormalities are common in asymptomatic relatives and may represent early disease. *J Am Coll Cardiol.* 1998;31:195-201.
682. Crispell KA, Wray A, Ni H, Nauman DJ, Hershberger RE. Clinical profiles of four large pedigrees with familial dilated cardiomyopathy: preliminary recommendations for clinical practice. *J Am Coll Cardiol.* 1999;34:837-47.
683. Corrado D, Fontaine G, Marcus FI, et al. Arrhythmogenic right ventricular dysplasia/cardiomyopathy: need for an international registry. Study Group on Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy of the Working Groups on Myocardial and Pericardial Disease and Arrhythmias of the European Society of Cardiology and of the Scientific Council on Cardiomyopathies of the World Heart Federation. *Circulation.* 2000;101:E101-6.
684. Coonar AS, Protonotarios N, Tsatsopoulou A, et al. Gene for arrhythmogenic right ventricular cardiomyopathy with diffuse nonepidermolytic palmoplantar keratoderma and woolly hair (Naxos disease) maps to 17q21. *Circulation.* 1998;97:2049-58.
685. Maron BJ, Moller JH, Seidman CE, et al. Impact of laboratory molecular diagnosis on contemporary diagnostic criteria for genetically transmitted cardiovascular diseases: hypertrophic cardiomyopathy, long-QT syndrome, and Marfan syndrome. A statement for healthcare professionals from the Councils on Clinical Cardiology, Cardiovascular Disease in the Young, and Basic Science, American Heart Association]. *Circulation.* 1998;98:1460-71.
686. Fuller CM. Cost effectiveness analysis of screening of high school athletes for risk of sudden cardiac death. *Med Sci Sports Exerc.* 2000;32:887-90.
687. Miller RL, Das S, Anandarangam T, et al. Association between right ventricular function and perfusion abnormalities in hemodynamically stable patients with acute pulmonary embolism. *Chest.* 1998;113:665-70.
688. Ribeiro A, Lindmarker 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;134:479-87.
689. Ritchie ME, Srivastava BK. Use of transesophageal echocardiography to detect unsuspected massive pulmonary emboli. *J Am Soc Echocardiogr.* 1998;11:751-4.
690. Pruszczyk P, Torbicki A, Pacho R, et al. Noninvasive diagnosis of suspected severe pulmonary embolism: transesophageal echocardiography vs spiral CT. *Chest.* 1997;112:722-8.
691. Perrier A, Howarth N, Didier D, et al. Performance of helical computed tomography in unselected outpatients with suspected pulmonary embolism. *Ann Intern Med.* 2001;135:88-97.

692. Cicek S, Demirlic U, Kuralay E, Tatar H, Ozturk O. Transesophageal echocardiography in cardiac surgical emergencies. *J Card Surg.* 1995;10:236-44.
693. Slama MA, Novara A, Van de PP, et al. Diagnostic and therapeutic implications of transesophageal echocardiography in medical ICU patients with unexplained shock, hypoxemia, or suspected endocarditis. *Intensive Care Med.* 1996;22:916-22.
694. Alam M. Transesophageal echocardiography in critical care units: Henry Ford Hospital experience and review of the literature. *Prog Cardiovasc Dis.* 1996;38:315-28.
695. Tam JW, Nichol J, MacDiarmid AL, Lazarow N, Wolfe K. What is the real clinical utility of echocardiography? A prospective observational study. *J Am Soc Echocardiogr.* 1999;12:689-97.
696. Tousignant C. Transesophageal echocardiographic assessment in trauma and critical care. *Can J Surg.* 1999;42:171-5.
697. Reilly JP, Tunick PA, Timmermans RJ, Stein B, Rosenzweig BP, Kronzon I. Contrast echocardiography clarifies uninterpretable wall motion in intensive care unit patients. *J Am Coll Cardiol.* 2000;35:485-90.
698. Kornbluth M, Liang DH, Brown P, Gessford E, Schnittger I. Contrast echocardiography is superior to tissue harmonics for assessment of left ventricular function in mechanically ventilated patients. *Am Heart J.* 2000;140:291-6.
699. Brandt RR, Oh JK, Abel MD, Click RL, Orszulak TA, Seward JB. Role of emergency intraoperative transesophageal echocardiography. *J Am Soc Echocardiogr.* 1998;11:972-7.
700. 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.
701. Gendreau MA, Triner WR, Bartfield J. Complications of transesophageal echocardiography in the ED. *Am J Emerg Med.* 1999;17:248-51.
702. Pretre R, Chilcott M. Blunt trauma to the heart and great vessels. *N Engl J Med.* 1997;336:626-32.
703. Chan D. Echocardiography in thoracic trauma. *Emerg Med Clin North Am.* 1998;16:191-207.
704. Stewart WJ, Douglas PS, Sagar K, et al. Echocardiography in emergency medicine: a policy statement by the American Society of Echocardiography and the American College of Cardiology. The Task Force on Echocardiography in Emergency Medicine of the American Society of Echocardiography and the Echocardiography TPEC Committees of the American College of Cardiology. *J Am Soc Echocardiogr.* 1999;12:82-4.
705. Fabian TC, Richardson JD, Croce MA, et al. Prospective study of blunt aortic injury: Multicenter Trial of the American Association for the Surgery of Trauma. *J Trauma.* 1997;42:374-80.
706. Smith MD, Cassidy JM, Souther S, et al. Transesophageal echocardiography in the diagnosis of traumatic rupture of the aorta. *N Engl J Med.* 1995;332:356-62.
707. Vignon P, Gueret P, Vedrinne JM, et al. Role of transesophageal echocardiography in the diagnosis and management of traumatic aortic disruption. *Circulation.* 1995;92:2959-68.
708. Vignon P, Lagrange P, Boncoeur MP, Francois B, Gastinne H, Lang RM. Routine transesophageal echocardiography for the diagnosis of aortic disruption in trauma patients without enlarged mediastinum. *J Trauma.* 1996;40:422-27.
709. Ben Menachem Y. Assessment of blunt aortic-brachiocephalic trauma: should angiography be supplanted by transesophageal echocardiography?. *J Trauma.* 1997;42:969-72.
710. Mirvis SE, Shanmuganathan K, Buell J, Rodriguez A. Use of spiral computed tomography for the assessment of blunt trauma patients with potential aortic injury. *J Trauma.* 1998;45:922-30.
711. Patel NH, Stephens KE, Jr., Mirvis SE, Shanmuganathan K, Mann FA. Imaging of acute thoracic aortic injury due to blunt trauma: a review. *Radiology.* 1998;209:335-48.
712. Brickner ME, Hillis LD, Lange RA. Congenital heart disease in adults. First of two parts. *N Engl J Med.* 2000;342:256-63.
713. Brickner ME, Hillis LD, Lange RA. Congenital heart disease in adults. Second of two parts [published erratum appears in N Engl J Med 2000;342:988]. *N Engl J Med.* 2000;342:334-42.
714. Tworetzky W, McElhinney DB, Brook MM, Reddy VM, Hanley FL, Silverman NH. Echocardiographic diagnosis alone for the complete repair of major congenital heart defects. *J Am Coll Cardiol.* 1999;33:228-33.
715. Marelli AJ, Child JS, Perloff JK. Transesophageal echocardiography in congenital heart disease in the adult. *Cardiol Clin.* 1993;11:505-20.
716. Simpson IA, Sahn DJ. Adult congenital heart disease: use of transthoracic echocardiography versus magnetic resonance imaging scanning. *Am J Card Imaging.* 1995;9:29-37.
717. Sreeram N, Sutherland GR, Geuskens R, et al. The role of transoesophageal echocardiography in adolescents and adults with congenital heart defects. *Eur Heart J.* 1991;12:231-40.
718. Hoppe UC, Dederichs B, Deutsch HJ, Theissen P, Schicha H, Sehtem U. Congenital heart disease in adults and adolescents: comparative value of transthoracic and transesophageal echocardiography and MR imaging. *Radiology.* 1996;199:669-77.
719. Hartnell GG, Cohen MC, Meier RA, Finn JP. Magnetic resonance angiography demonstration of congenital heart disease in adults. *Clin Radiol.* 1996;51:851-7.
720. Bartel T, Muller S, Erbel R. Dynamic three-dimensional echocardiography using parallel slicing: a promising diagnostic procedure in adults with congenital heart disease. *Cardiology.* 1998;89:140-7.
721. Li J, Sanders SP. Three-dimensional echocardiography in congenital heart disease. *Curr Opin Cardiol.* 1999;14:53-9.
722. Harrison DA, McLaughlin PR. Interventional cardiology for the adult patient with congenital heart disease: the Toronto Hospital experience. *Can J Cardiol.* 1996;12:965-71.
723. Triedman JK, Bergau DM, Saul JP, Epstein MR, Walsh EP. Efficacy of radiofrequency ablation for control of intraatrial reentrant tachycardia in patients with congenital heart disease. *J Am Coll Cardiol.* 1997;30:1032-38.
724. Sreeram N, Colli AM, Monro JL, et al. Changing role of non-invasive investigation in the preoperative assessment of congenital heart disease: a nine year experience. *Br Heart J.* 1990;63:345-9.
725. Pfammatter JP, Berdat PA, Carrel TP, Stocker FP. Pediatric open heart operations without diagnostic cardiac catheterization. *Ann Thorac Surg.* 1999;68:532-6.
726. McCrindle BW, Shaffer KM, Kan JS, Zahka KG, Rowe SA, Kidd L. An evaluation of parental concerns and misperceptions about heart murmurs. *Clin Pediatr (Phila).* 1995;34:25-31.
727. Schulze-Neick I, Bultmann M, Werner H, et al. Right ventricular function in patients treated with inhaled nitric oxide after cardiac surgery for congenital heart disease in newborns and children. *Am J Cardiol.* 1997;80:360-3.
728. Rychik J, Jacobs ML, Norwood WI. Early changes in ventricular geometry and ventricular septal defect size following Rastelli

- operation or intraventricular baffle repair for conotruncal anomaly. A cause for development of subaortic stenosis. *Circulation*. 1994;90(suppl II):II13-19.
729. Salzer-Muhar U, Marx M, Ties M, Proll E, Wimmer M. Doppler flow profiles in the right and left pulmonary artery in children with congenital heart disease and a bidirectional cavopulmonary shunt. *Pediatr Cardiol*. 1994;15:302-7.
730. Suda K, Bigras JL, Bohn D, Hornberger LK, McCrindle BW. Echocardiographic predictors of outcome in newborns with congenital diaphragmatic hernia. *Pediatrics*. 2000;105:1106-9.
731. Wren C, Richmond S, Donaldson L. Presentation of congenital heart disease in infancy: implications for routine examination. *Arch Dis Child Fetal Neonatal Ed*. 1999;80:F49-53.
732. Magee AG, Boutin C, McCrindle BW, Smallhorn JF. Echocardiography and cardiac catheterization in the preoperative assessment of ventricular septal defect in infancy. *Am Heart J*. 1998;135:907-13.
733. Krauser DG, Rutkowski M, Phoon CK. Left ventricular volume after correction of isolated aortic coarctation in neonates. *Am J Cardiol*. 2000;85:904-7, A10.
734. Tani LY, Minich LL, Hawkins JA, Pagotto LT, Shaddy RE. Influence of left ventricular cavity size on interventricular shunt timing and outcome in neonates with coarctation of the aorta and ventricular septal defect. *Am J Cardiol*. 1999;84:750-2, A9.
735. Kovalchin JP, Brook MM, Rosenthal GL, Suda K, Hoffman JJ, Silverman NH. Echocardiographic hemodynamic and morphometric predictors of survival after two-ventricle repair in infants with critical aortic stenosis [published erratum appears in *J Am Coll Cardiol* 1999;33:591]. *J Am Coll Cardiol*. 1998;32:237-44.
736. Tamura M, Menahem S, Brizard C. Clinical features and management of isolated cleft mitral valve in childhood. *J Am Coll Cardiol*. 2000;35:764-70.
737. Jureidini SB, Marino CJ, Singh GK, Fiore A, Balfour IC. Main coronary artery and coronary ostial stenosis in children: detection by transthoracic color flow and pulsed Doppler echocardiography. *J Am Soc Echocardiogr*. 2000;13:255-63.
738. Garcia JA, Zellers TM, Weinstein EM, Mahony L. Usefulness of Doppler echocardiography in diagnosing right ventricular coronary arterial communications in patients with pulmonary atresia and intact ventricular septum and comparison with angiography. *Am J Cardiol*. 1998;81:103-4.
739. Roberts JD Jr, Fineman JR, Morin FC III, et al. Inhaled nitric oxide and persistent pulmonary hypertension of the newborn. The Inhaled Nitric Oxide Study Group. *N Engl J Med*. 1997;336:605-10.
740. Davidson D, Barefield ES, Kattwinkel J, et al. Inhaled nitric oxide for the early treatment of persistent pulmonary hypertension of the term newborn: a randomized, double-masked, placebo-controlled, dose-response, multicenter study. The I-NO/PPHN Study Group. *Pediatrics*. 1998;101:325-34.
741. Davidson D, Barefield ES, Kattwinkel J, et al. Safety of withdrawing inhaled nitric oxide therapy in persistent pulmonary hypertension of the newborn. *Pediatrics*. 1999;104:231-36.
742. Lavoie A, Hall JB, Olson DM, Wylam ME. Life-threatening effects of discontinuing inhaled nitric oxide in severe respiratory failure. *Am J Respir Crit Care Med*. 1996;153:1985-87.
743. Francoise M, Gouyon JB, Mercier JC. Hemodynamics and oxygenation changes induced by the discontinuation of low-dose inhalational nitric oxide in newborn infants. *Intensive Care Med*. 1996;22:477-81.
744. UK collaborative randomised trial of neonatal extracorporeal membrane oxygenation. UK Collaborative ECMO Trail Group. *Lancet*. 1996;348:75-82.
745. Green TP, Timmons OD, Fackler JC, Moler FW, Thompson AE, Sweeney MF. The impact of extracorporeal membrane oxygenation on survival in pediatric patients with acute respiratory failure. Pediatric Critical Care Study Group. *Crit Care Med*. 1996;24:323-9.
746. Martin GR, Short BL, Abbott C, O'Brien AM. Cardiac stun in infants undergoing extracorporeal membrane oxygenation. *J Thorac Cardiovasc Surg*. 1991;101:607-11.
747. Fritz KI, Bhat AM. Effect of beta-blockade on symptomatic dexamethasone-induced hypertrophic obstructive cardiomyopathy in premature infants: three case reports and literature review. *J Perinatol*. 1998;18:38-44.
748. Van Oort A, Blanc-Botden M, De Boo T, Van Der WT, Rohmer J, Daniels O. The vibratory innocent heart murmur in schoolchildren: difference in auscultatory findings between school medical officers and a pediatric cardiologist. *Pediatr Cardiol*. 1994;15:282-7.
749. Gaskin PR, Owens SE, Talner NS, Sanders SP, Li JS. Clinical auscultation skills in pediatric residents. *Pediatrics*. 2000;105:1184-7.
750. Steinberger J, Moller JH, Berry JM, Sinaiko AR. Echocardiographic diagnosis of heart disease in apparently healthy adolescents. *Pediatrics*. 2000;105:815-818.
751. Danford DA, Martin AB, Fletcher SE, et al. Children with heart murmurs: can ventricular septal defect be diagnosed reliably without an echocardiogram? *J Am Coll Cardiol*. 1997;30:243-6.
752. Tsang TS, Barnes ME, Hayes SN, et al. Clinical and echocardiographic characteristics of significant pericardial effusions following cardiothoracic surgery and outcomes of echo-guided pericardiocentesis for management: Mayo Clinic experience, 1979-1998. *Chest*. 1999;116:322-31.
753. McMahon CJ, Feltes TF, Fraley JK, et al. Natural history of growth of secundum atrial septal defects and implications for transcatheter closure. *Heart*. 2002;87:256-9.
754. Rao PS, Langhough R. Relationship of echocardiographic, shunt flow, and angiographic size to the stretched diameter of the atrial septal defect. *Am Heart J*. 1991;122:505-8.
755. Jan SL, Hwang B, Lee PC, Fu YC, Chiu PS, Chi CS. Intracardiac ultrasound assessment of atrial septal defect: comparison with transthoracic echocardiographic, angiographic, and balloon-sizing measurements. *Cardiovasc Intervent Radiol*. 2001;24:84-9.
756. Mazic U, Gavora P, Masura J. The role of transesophageal echocardiography in transcatheter closure of secundum atrial septal defects by the Amplatzer septal occluder. *Am Heart J*. 2001;142:482-8.
757. Taeed R, Shim D, Kimball TR, et al. One-year follow-up of the amplatzer device to close atrial septal defects. *Am J Cardiol*. 2001;87:116-8, A9.
758. Stromberg D, Pignatelli R, Rosenthal GL, Ing FF. Does ductal occlusion with the gianturco coil cause left pulmonary artery and/or descending aorta obstruction? *Am J Cardiol*. 1999;83:1229-35.
759. Schroeder VA, Shim D, Spicer RL, Pearl JM, Manning PJ, Beekman RH III. Surgical emergencies during pediatric interventional catheterization. *J Pediatr*. 2002;140:570-5.
760. Formigari R, Toscano A, Herraiz I, et al. Late follow-up of occlusion of the patent ductus arteriosus with the Rashkind device with emphasis on long-term efficacy and risk for infections. *Am J Cardiol*. 2001;88:586-8.
761. Powell AJ, Lock JE, Keane JF, Perry SB. Prolongation of RV-PA

- conduit life span by percutaneous stent implantation. Intermediate-term results. *Circulation*. 1995;92:3282-8.
762. Tanel RE, Walsh EP, Triedman JK, Epstein MR, Bergau DM, Saul JP. Five-year experience with radiofrequency catheter ablation: implications for management of arrhythmias in pediatric and young adult patients. *J Pediatr*. 1997;131:878-87.
763. Calkins H, Yong P, Miller JM, et al. Catheter ablation of accessory pathways, atrioventricular nodal reentrant tachycardia, and the atrioventricular junction: final results of a prospective, multicenter clinical trial. The Atakr Multicenter Investigators Group. *Circulation*. 1999;99:262-70.
764. De Giovanni JV, Dindar A, Griffith MJ, et al. Recovery pattern of left ventricular dysfunction following radiofrequency ablation of incessant supraventricular tachycardia in infants and children. *Heart*. 1998;79:588-92.
765. Kimball TR, Witt SA, Daniels SR. Dobutamine stress echocardiography in the assessment of suspected myocardial ischemia in children and young adults. *Am J Cardiol*. 1997;79:380-4.
766. Noto N, Ayusawa M, Karasawa K, et al. Dobutamine stress echocardiography for detection of coronary artery stenosis in children with Kawasaki disease. *J Am Coll Cardiol*. 1996;27:1251-6.
767. Pahl E, Sehgal R, Chrystof D, et al. Feasibility of exercise stress echocardiography for the follow-up of children with coronary involvement secondary to Kawasaki disease. *Circulation*. 1995;91:122-8.
768. Minich LL, Tani LY, Pagotto LT, Shaddy RE, Veasy LG. Doppler echocardiography distinguishes between physiologic and pathologic "silent" mitral regurgitation in patients with rheumatic fever. *Clin Cardiol*. 1997;20:924-6.
769. Lipshultz SE, Easley KA, Orav EJ, et al. Left ventricular structure and function in children infected with human immunodeficiency virus: the prospective P2C2 HIV Multicenter Study. Pediatric Pulmonary and Cardiac Complications of Vertically Transmitted HIV Infection (P2C2 HIV) Study Group. *Circulation*. 1998;97:1246-56.
770. De Wolf D, Suys B, Maurus R, et al. Dobutamine stress echocardiography in the evaluation of late anthracycline cardiotoxicity in childhood cancer survivors. *Pediatr Res*. 1996;39:504-12.
771. Ichida F, Hamamichi Y, Miyawaki T, et al. Clinical features of isolated noncompaction of the ventricular myocardium: long-term clinical course, hemodynamic properties, and genetic background. *J Am Coll Cardiol*. 1999;34:233-40.
772. Kimball TR, Witt SA, Daniels SR, Khoury PR, Meyer RA. Frequency and significance of left ventricular thickening in transplanted hearts in children. *Am J Cardiol*. 1996;77:77-80.
773. Larsen RL, Applegate PM, Dyar DA, et al. Dobutamine stress echocardiography for assessing coronary artery disease after transplantation in children. *J Am Coll Cardiol*. 1998;32:515-20.
774. Pahl E, Crawford SE, Swenson JM, et al. Dobutamine stress echocardiography: experience in pediatric heart transplant recipients. *J Heart Lung Transplant*. 1999;18:725-32.
775. Jacobs IN, Teague WG, Bland JWJ. Pulmonary vascular complications of chronic airway obstruction in children. *Arch Otolaryngol Head Neck Surg*. 1997;123:700-4.
776. Subhedar NV, Shaw NJ. Changes in oxygenation and pulmonary haemodynamics in preterm infants treated with inhaled nitric oxide. *Arch Dis Child Fetal Neonatal Ed*. 1997;77:F191-7.
777. Barst RJ, Rubin LJ, Long WA, et al. A comparison of continuous intravenous epoprostenol (prostacyclin) with conventional therapy for primary pulmonary hypertension. The Primary Pulmonary Hypertension Study Group. *N Engl J Med*. 1996;334:296-302.
778. Carcillo JA, Fields AI. Clinical practice parameters for hemodynamic support of pediatric and neonatal patients in septic shock. *Crit Care Med*. 2002;30:1365-78.
779. Wessel DL. Managing low cardiac output syndrome after congenital heart surgery. *Crit Care Med*. 2001;29:S220-30.
780. Podnar T, Martanovic P, Gavora P, Masura J. Morphological variations of secundum-type atrial septal defects: feasibility for percutaneous closure using Amplatzer septal occluders. *Catheter Cardiovasc Interv*. 2001;53:386-91.
781. Vogel M, Berger F, Dahnert I, Ewert P, Lange PE. Treatment of atrial septal defects in symptomatic children aged less than 2 years of age using the Amplatzer septal occluder. *Cardiol Young*. 2000;10:534-7.
782. Siwik ES, Spector ML, Patel CR, Zahka KG. Costs and cost-effectiveness of routine transesophageal echocardiography in congenital heart surgery. *Am Heart J*. 1999;138:771-6.
783. Stevenson JG, Sorensen GK, Gartman DM, Hall DG, Rittenhouse EA. Transesophageal echocardiography during repair of congenital cardiac defects: identification of residual problems necessitating reoperation. *J Am Soc Echocardiogr*. 1993;6:356-65.
784. Chaliki HP, Click RL, Abel MD. Comparison of intraoperative transesophageal echocardiographic examinations with the operative findings: prospective review of 1918 cases. *J Am Soc Echocardiogr*. 1999;12:237-40.
785. Shiota T, Lewandowski R, Piel JE, et al. Micromultiplane transesophageal echocardiographic probe for intraoperative study of congenital heart disease repair in neonates, infants, children, and adults. *Am J Cardiol*. 1999;83:292-5, A7.
786. Fyfe DA, Eklund CH, Sade RM, et al. Transesophageal echocardiography detects thrombus formation not identified by transthoracic echocardiography after the Fontan operation. *J Am Coll Cardiol*. 1991;18:1733-7.
787. Drant SE, Klitzner TS, Shannon KM, Wetzel GT, Williams RG. Guidance of radiofrequency catheter ablation by transesophageal echocardiography in children with palliated single ventricle. *Am J Cardiol*. 1995;76:1311-2.
788. Kreutzer J, Keane JF, Lock JE, et al. Conversion of modified Fontan procedure to lateral atrial tunnel cavopulmonary anastomosis. *J Thorac Cardiovasc Surg*. 1996;111:1169-76.
789. Kreutzer J, Lock JE, Jonas RA, Keane JF. Transcatheter fenestration dilation and/or creation in postoperative Fontan patients. *Am J Cardiol*. 1997;79:228-32.
790. van Nieuwenhuizen RC, Peters M, Lubbers LJ, Trip MD, Tijssen JG, Mulder BJ. Abnormalities in liver function and coagulation profile following the Fontan procedure. *Heart*. 1999;82:40-6.
791. Kaulitz R, Luhmer I, Bergmann F, Rodeck B, Hausdorf G. Sequelae after modified Fontan operation: postoperative haemodynamic data and organ function. *Heart*. 1997;78:154-9.
792. Veldtman GR, Nishimoto A, Siu S, et al. The Fontan procedure in adults. *Heart*. 2001;86:330-5.
793. Yagel S, Weissman A, Rotstein Z, et al. Congenital heart defects: natural course and in utero development. *Circulation*. 1997;96:550-5.
794. Buskens E, Grobbee DE, Frohn-Mulder IM, et al. Efficacy of routine fetal ultrasound screening for congenital heart disease in normal pregnancy. *Circulation*. 1996;94:67-72.
795. Bonnet D, Coltri A, Butera G, et al. Detection of transposition of the great arteries in fetuses reduces neonatal morbidity and mortality. *Circulation*. 1999;99:916-8.
796. Kumar RK, Newburger JW, Gauvreau K, Kamenir SA, Hornberger LK. Comparison of outcome when hypoplastic left heart syndrome and transposition of the great arteries are diagnosed prenatally versus when diagnosis of these two conditions is

- made only postnatally. *Am J Cardiol.* 1999;83:1649-53.
797. Hornberger LK, Sanders SP, Rein AJ, Spevak PJ, Parness IA, Colan SD. Left heart obstructive lesions and left ventricular growth in the midtrimester fetus. A longitudinal study. *Circulation.* 1995;92:1531-8.
798. Zuber M, Gautschi N, Oechslin E, Widmer V, Kiowski W, Jenni R. Outcome of pregnancy in women with congenital shunt lesions. *Heart.* 1999;81:271-5.
799. Practice guidelines for perioperative transesophageal echocardiography. A report by the American Society of Anesthesiologists and the Society of Cardiovascular Anesthesiologists Task Force on Transesophageal Echocardiography. *Anesthesiology.* 1996;84:986-1006.
800. Click RL, Abel MD, Schaff HV. Intraoperative transesophageal echocardiography: 5-year prospective review of impact on surgical management. *Mayo Clin Proc.* 2000;75:241-7.
801. Mishra M, Chauhan R, Sharma KK, et al. Real-time intraoperative transesophageal echocardiography—how useful? Experience of 5,016 cases. *J Cardiothorac Vasc Anesth.* 1998;12:625-32.
802. Couture P, Denault AY, McKenty S, et al. Impact of routine use of intraoperative transesophageal echocardiography during cardiac surgery. *Can J Anaesth.* 2000;47:20-6.
803. Michel-Cherqui M, Ceddaha A, Liu N, et al. Assessment of systematic use of intraoperative transesophageal echocardiography during cardiac surgery in adults: a prospective study of 203 patients. *J Cardiothorac Vasc Anesth.* 2000;14:45-50.
804. Sutton DC, Kluger R. Intraoperative transoesophageal echocardiography: impact on adult cardiac surgery. *Anaesth Intensive Care.* 1998;26:287-93.
805. Hogue CW Jr, Lappas GD, Creswell LL, et al. Swallowing dysfunction after cardiac operations. Associated adverse outcomes and risk factors including intraoperative transesophageal echocardiography. *J Thorac Cardiovasc Surg.* 1995;110:517-22.
806. Rousou JA, Tighe DA, Garb JL, et al. Risk of dysphagia after transesophageal echocardiography during cardiac operations. *Ann Thorac Surg.* 2000;69:486-9.
807. Kallmeyer IJ, Collard CD, Fox JA, Body SC, Shernan SK. The safety of intraoperative transesophageal echocardiography: a case series of 7200 cardiac surgical patients. *Anesth Analg.* 2001;92:1126-30.
808. Rosenfeld HM, Gentles TL, Wernovsky G, et al. Utility of intraoperative transesophageal echocardiography in the assessment of residual cardiac defects. *Pediatr Cardiol.* 1998;19:346-51.
809. Sheil ML, Baines DB. Intraoperative transoesophageal echocardiography for pediatric cardiac surgery—an audit of 200 cases. *Anaesth Intensive Care.* 1999;27:591-5.
810. Stevenson JG. Role of intraoperative transesophageal echocardiography during repair of congenital cardiac defects. *Acta Paediatr Suppl.* 1995;410:23-33.
811. Ungerleider RM, Kisslo JA, Greeley WJ, et al. Intraoperative echocardiography during congenital heart operations: experience from 1,000 cases. *Ann Thorac Surg.* 1995;60:S539-42.
812. Greene MA, Alexander JA, Knauf DG, et al. Endoscopic evaluation of the esophagus in infants and children immediately following intraoperative use of transesophageal echocardiography. *Chest.* 1999;116:1247-50.
813. Kawano H, Mizoguchi T, Aoyagi S. Intraoperative transesophageal echocardiography for evaluation of mitral valve repair. *J Heart Valve Dis.* 1999;8:287-93.
814. Saiki Y, Kasegawa H, Kawase M, Osada H, Ootaki E. Intraoperative TEE during mitral valve repair: does it predict early and late postoperative mitral valve dysfunction? *Ann Thorac Surg.* 1998;66:1277-81.
815. Morehead AJ, Firstenberg MS, Shiota T, et al. Intraoperative echocardiographic detection of regurgitant jets after valve replacement. *Ann Thorac Surg.* 2000;69:135-9.
816. Bergquist BD, Bellows WH, Leung JM. Transesophageal echocardiography in myocardial revascularization: II. Influence on intraoperative decision making. *Anesth Analg.* 1996;82:1139-45.
817. Savage RM, Lytle BW, Aronson S, et al. Intraoperative echocardiography is indicated in high-risk coronary artery bypass grafting. *Ann Thorac Surg.* 1997;64:368-73.
818. Arruda AM, Dearani JA, Click RL, Ishikura F, Seward JB. Intraoperative application of power Doppler imaging: visualization of myocardial perfusion after anastomosis of left internal thoracic artery to left anterior descending coronary artery. *J Am Soc Echocardiogr.* 1999;12:650-4.
819. Applebaum RM, Kasliwal RR, Kanojia A, et al. Utility of three-dimensional echocardiography during balloon mitral valvuloplasty. *J Am Coll Cardiol.* 1998;32:1405-9.
820. Falk V, Walther T, Diegeler A, et al. Echocardiographic monitoring of minimally invasive mitral valve surgery using an endoaortic clamp. *J Heart Valve Dis.* 1996;5:630-7.
821. Moises VA, Mesquita CB, Campos O, et al. Importance of intraoperative transesophageal echocardiography during coronary artery surgery without cardiopulmonary bypass. *J Am Soc Echocardiogr.* 1998;11:1139-44.
822. Secknus MA, Asher CR, Scalia GM, Cosgrove DM III, Stewart WJ. Intraoperative transesophageal echocardiography in minimally invasive cardiac valve surgery. *J Am Soc Echocardiogr.* 1999;12:231-6.
823. Yao FS, Barbut D, Hager DN, Trifiletti RR, Gold JP. Detection of aortic emboli by transesophageal echocardiography during coronary artery bypass surgery. *J Cardiothorac Vasc Anesth.* 1996;10:314-7.
824. Tingleff J, Joyce FS, Pettersson G. Intraoperative echocardiographic study of air embolism during cardiac operations. *Ann Thorac Surg.* 1995;60:673-7.
825. Choudhary SK, Bhan A, Sharma R, et al. Aortic atherosclerosis and perioperative stroke in patients undergoing coronary artery bypass: role of intra-operative transesophageal echocardiography. *Int J Cardiol.* 1997;61:31-8.
826. Sylvris S, Calafiore P, Matalanis G, et al. The intraoperative assessment of ascending aortic atheroma: epiaortic imaging is superior to both transesophageal echocardiography and direct palpation. *J Cardiothorac Vasc Anesth.* 1997;11:704-7.
827. Lee HR, Montenegro LM, Nicolson SC, Gaynor JW, Spray TL, Rychik J. Usefulness of intraoperative transesophageal echocardiography in predicting the degree of mitral regurgitation secondary to atrioventricular defect in children. *Am J Cardiol.* 1999;83:750-3.
828. Leung MP, Chau KT, Chiu C, Yung TC, Mok CK. Intraoperative TEE assessment of ventricular septal defect with aortic regurgitation. *Ann Thorac Surg.* 1996;61:854-60.
829. Shankar S, Sreeram N, Brawn WJ, Sethia B. Intraoperative ultrasonographic troubleshooting after the arterial switch operation. *Ann Thorac Surg.* 1997;63:445-8.
830. Lavoie J, Javorski JJ, Donahue K, Sanders SP, Burke RP, Burrows FA. Detection of residual flow by transesophageal echocardiography during video-assisted thoracoscopic patent ductus arteriosus interruption. *Anesth Analg.* 1995;80:1071-5.
831. Le Bret E, Papadatos S, Folliguet T, et al. Interruption of patent ductus arteriosus in children: robotically assisted versus videotho-

- racoscopic surgery. *J Thorac Cardiovasc Surg.* 2002;123:973-6.
832. 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;105:539-42.
833. Zoghbi WA, Enriquez-Sarano M, Foster E, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and doppler echocardiography (a report from the American Society of Echocardiography's Nomenclature and Standards Committee and the Task Force on Valvular Regurgitation). *JASE* 2003;16:777-802.
834. Moss AJ, Zareba W, Hall WJ, et al. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *N Engl J Med.* 2002; 346:877-83.
835. Gregoratos G, Abrams J, Epstein AE, et al. ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices – summary article: a report of the American College of Cardiology/American Heart Association/Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines). *J Am Coll Cardiol.* 2002;40:1703-19.
836. Nagueh SF, Bachinski LL, Meyer D, Hill R, Zoghbi WA, Tam JW, Quinones MA, Roberts R, Marian AJ. Tissue Doppler imaging consistently detects myocardial abnormalities in patients with hypertrophic cardiomyopathy and provides a novel means for an early diagnosis before and independently of hypertrophy. *Circulation.* 2001;104:128-30.
837. Ho CY, Sweitzer NK, McDonough B, et al. Assessment of diastolic function with Doppler tissue imaging to predict genotype in preclinical hypertrophic cardiomyopathy. *Circulation.* 2002;105:2992-7.
838. Lakkis NM, Nagueh SF, Kleiman NS, et al. Echocardiography-guided ethanol septal reduction for hypertrophic obstructive cardiomyopathy. *Circulation.* 1998; 98:1750-5.
839. Faber L, Ziemssen P, Seggewiss H. Targeting percutaneous transluminal septal ablation for hypertrophic obstructive cardiomyopathy by intraprocedural echocardiographic monitoring. *J Am Soc Echocardiography.* 2000;13:1074-9.
840. Tei C, Dujardin KS, Hodge DO, Bailey KR, McGoon MD, Tajik AJ, Deward SB. Doppler echocardiographic index for assessment of global right ventricular function. *J Am Soc Echocardiogr.* 1996; 9:838-47.
841. Burgess MI, Mogulkoc N, Bright-Thomas RJ, et al. Comparison of echocardiographic markers of right ventricular function in determining prognosis in chronic pulmonary disease. *J Am Soc Echocardiogr.* 2002;15:633-9.
842. Roman MJ, Devereux RB, Kramer-Fox R, O'Loughlin J. Two-dimensional echocardiographic aortic root dimensions in normal children and adults. *Am J Cardiol.* 1989;64:507-12.
843. Zuppiroli A, Rinaldi M, Kramer-Fox R, Favilli S, Roman MJ, Devereux RB. Natural history of mitral valve prolapse. *Am J Cardiol.* 1995;75:1028-32.
844. Masini M, Picano E, Lattanzi F, Distante A, L'Abbate A. High dose dipyridamole-echocardiography test in women: correlation with exercise-electrocardiography test and coronary arteriography. *J Am Coll Cardiol.* 1988;12:682-85.
845. Lewis JF, Lin L, McGorray S, et al. Dobutamine stress echocardiography in women with chest pain: pilot phase data from the National Heart, Lung and Blood Institute Women's Ischemia Syndrome Evaluation (WISE). *J Am Coll Cardiol.* 1999;33:1462-8.
846. Akosah KO, Mohanty PK, Funai JT, et al. Noninvasive detection of transplant coronary artery disease by dobutamine stress echocardiography. *J Heart Lung Transplant.* 1994;13:1024-38.
847. Herregods MC, Anastassiou I, Van Cleemput J, et al. Dobutamine stress echocardiography after heart transplantation. *J Heart Lung Transplant.* 1994;13:1039-44.
848. Derumeaux G, Redonnet M, Mouton-Schleifer D, et al. Dobutamine stress echocardiography in orthotopic heart transplant recipients. VACOMED Research Group. *J Am Coll Cardiol.* 1995;25:1665-72.
849. Akosah KO, Olsovsky M, Kirchberg D, Salter D, Mohanty PK. Dobutamine stress echocardiography predicts cardiac events in heart transplant patients. *Circulation.* 1996;94:II283-8.
850. Derumeaux G, Redonnet M, Soyer R, Cribier A, Letac B. Assessment of the progression of cardiac allograft vasculopathy by dobutamine stress echocardiography. *J Heart Lung Transplant.* 1998;17:259-67.
851. Akosah KO, McDaniel S, Hanrahan JS, Mohanty PK. Dobutamine stress echocardiography early after heart transplantation predicts development of allograft coronary artery disease and outcome. *J Am Coll Cardiol.* 1998;31:1607-14.
852. Spes CH, Klauss V, Mudra H, et al. Diagnostic and prognostic value of serial dobutamine stress echocardiography for noninvasive assessment of cardiac allograft vasculopathy: a comparison with coronary angiography and intravascular ultrasound. *Circulation.* 1999;100:509-15.
853. Nagueh SF, Vaduganathan P, Ali N, et al. Identification of hibernating myocardium: comparative accuracy of myocardial contrast echocardiography, rest-redistribution thallium-201 tomography and dobutamine echocardiography. *J Am Coll Cardiol.* 1997;29:985-93.
854. Meluzin J, Cerny J, Frelich M, et al. on behalf of the investigators of the multicenter study. Prognostic value of the amount of dysfunctional but viable myocardium in revascularized patients with coronary artery disease and left ventricular dysfunction. *J Am Coll Cardiol.* 1998;32:912-20.
855. Afridi I, Grayburn PA, Panza JA, Oh JK, Zoghbi WA, Marwick TH. Myocardial viability during dobutamine echocardiography predicts survival in patients with coronary artery disease and severe left ventricular systolic dysfunction. *J Am Coll Cardiol.* 1998;32:921-6.
856. Murry PM, Cantwell JD, Heath DL, Shoop J. The role of limited echocardiography in screening athletes. *Am J Cardiol* 1995;76:849-50.
857. Zeppilli P, dello RA, Santini C, et al. In vivo detection of coronary artery anomalies in asymptomatic athletes by echocardiography screening. *Chest* 1998;114:89-93.
858. Kinoshita N, Mimura J, Obayashi C, Katsukawa F, Onishi S, Yamazaki H. Aortic root dilatation among young competitive athletes: echocardiography screening of 1929 athletes between 15 and 34 years of age. *Am Heart J* 2000;139:723-8.