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# ACC/AHA Guidelines for the Clinical Application of Echocardiography

**A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Clinical Application of Echocardiography)**

**Developed in Collaboration With the American Society of Echocardiography**

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# Preamble

It is clearly important that the medical profession plays a significant role in critically 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 Committee on Clinical Application of Echocardiography was chaired by Melvin D. Cheitlin, MD, FACC, and included the following members: Joseph S. Alpert, MD, FACC, William F. Armstrong, MD, FACC, Gerard P. Aurigemma, MD, FACC, George A. Beller, MD, FACC, Fredrick Z. Bierman, MD, FACC, Thomas W. Davidson, MD, FAAFP, Jack L. Davis, MD, FACC, Pamela S. Douglas, MD, FACC, Linda D. Gillam, MD, FACC, Richard P. Lewis, MD, FACC, Alan S. Pearlman, MD, FACC, John T. Philbrick, MD, FACP, Pravin M. Shah, MD, FACC, and Roberta G. Williams, MD, FACC.

The committee is composed of both university-affiliated and practicing physicians and those with specific echocardiographic expertise and senior clinicians who use the technique. Two general physicians (one general internal medicine and one family practitioner) also served on the committee.

The document was reviewed by three outside reviewers nominated by the ACC and three outside reviewers nominated by the AHA as well as other individuals from the American Society of Echocardiography, Society of Pediatric Echocardiography, American College of Physicians, and American Academy of Family Physicians.

The executive summary and recommendations are published in the March 15, 1997, issue of *Journal of the American College of Cardiology.* The full text is published in *Circulation* . Reprints of both the full text and the executive summary and recommendations are available from both organizations. The document will be reviewed 2 years after publication and yearly thereafter and considered current unless the task force revises or withdraws it from distribution. The document was endorsed by the American Society of Echocardiography.

*James L. Ritchie, MD, FACC*
*Chair, ACC/AHA Task Force on Practice Guidelines*

# I. Introduction, General Considerations, and Scope

The previous guidelines for the use of echocardiography were published in December 1990. Since that time there have been significant advances in the technology of Doppler echocardiography and growth in its clinical use and in the scientific evidence leading to recommendations for its proper use.

The recommendations 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 search yielded over 3000 references, which the committee reviewed. This document includes recommendations for the use of Doppler echocardiography in both adult and pediatric patients. The pediatric guidelines also include recommendations for fetal Doppler echocardiography, an increasingly important field. The guidelines include recommendations for the use of Doppler 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 Doppler echocardiographic techniques in the evaluation of such common clinical problems.

The recommendations concerning the use of Doppler echocardiography follow the indication classification system (eg, Class 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.

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

**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.

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]

## 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 on either 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 here but will be reviewed in the revised guidelines for coronary angiography (in preparation). 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.[4] The techniques of three-dimensional echocardiography are in the developmental stages and also are not considered here. Intraoperative TEE is not considered in this document because it is the subject of practice guidelines for perioperative TEE.[5]

New techniques that are still rapidly evolving are also not addressed in this document. Echocardiography-contrast substances that can pass through the pulmonary circulation and opacify the left heart are in development. Echocardiography-contrast injections into the coronary artery to quantitate myocardium at risk and perfusion territories and the second harmonic echocardiogram to enhance echocardiographic contrast 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 quantitate 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 Doppler 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, two-dimensional Doppler 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 Doppler echocardiographic techniques reveal details of structure and function such as filamentous strands on valves, valvular prolapse, and jet velocities representing minimal and at times transient valvular insufficiency that could generate unnecessary further testing or inappropriate and potentially detrimental therapy.

These guidelines contain recommendations concerning not only indications for the use of these techniques but also specific circumstances when Doppler 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 a Doppler 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 then 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 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 addressing this issue are available.

The use of two-dimensional Doppler echocardiography in establishing cardiac diagnoses and making therapeutic decisions 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 Doppler 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 Doppler echocardiographic studies in following 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.

This document assumes that Doppler 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 American Society of Echocardiography, the American College of Cardiology, 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 reevaluation 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.

## 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 $S_2$, 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.[12,16]

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 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 determining the etiology and judging the severity of lesions, particularly in pediatric and elderly patients.[15,17-19]

| Indications for Echocardiography in the Evaluation of Heart Murmurs | Class |
|---|---|
| **1. A murmur in a patient with cardiorespiratory symptoms.** | **I** |
| **2. A murmur in an asymptomatic patient if the clinical features indicate at least a moderate probability that the murmur is reflective of structural heart disease.** | **I** |

| | |
|---|---|
| **3. 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.** | **IIa** |
| **4. In an adult, an asymptomatic heart murmur that has been identified by an experienced observer as functional or innocent.** | **III** |

## 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] Prognostic information is obtained from assessment of the hemodynamic response to exercise[24] and/or delineation of morphological characteristics,[25] which in turn help guide the selection of therapeutic interventions.[26]

| Indications for Echocardiography in Valvular Stenosis | Class |
|---|---|
| **1. Diagnosis; assessment of hemodynamic severity.** | **I** |
| **2. Assessment of left ventricular (LV) and right ventricular (RV) size, function, and/or hemodynamics.** | **I** |
| **3. Reevaluation of patients with known valvular stenosis with changing symptoms or signs.** | **I** |
| **4. Assessment of changes in hemodynamic severity and ventricular compensation in patients with known valvular stenosis during pregnancy.** | **I** |
| **5. Reevaluation of asymptomatic patients with severe stenosis.** | **I** |
| **6. Assessment of the hemodynamic significance of mild to moderate valvular stenosis by stress Doppler echocardiography.** | **IIa** |
| **7. Reevaluation of patients with mild to moderate aortic stenosis with LV dysfunction or hypertrophy even without clinical symptoms.** | **IIa** |
| **8. Reevaluation of patients with mild to moderate aortic valvular stenosis with stable signs and symptoms.** | **IIb** |

| | |
|---|---|
| **9. Routine reevaluation of asymptomatic adult patients with mild aortic stenosis having stable physical signs and normal LV size and function.** | **III** |
| **10. Routine reevaluation of asymptomatic patients with mild to moderate mitral stenosis and stable physical signs.** | **III** |

**(See also "Indications 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] 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] 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]

## 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 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 reevaluation of regurgitant lesions and in determination of the timing of operative intervention.[49-51] 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]

| **Indications for Echocardiography in Native Valvular Regurgitation** | **Class** |
|---|---|
| **1. Diagnosis; assessment of hemodynamic severity.** | **I** |
| **2. Initial assessment and reevaluation (when indicated) of LV and RV size, function, and/or hemodynamics.** | **I** |
| **3. Reevaluation of patients with mild to moderate valvular regurgitation with changing symptoms.** | **I** |
| **4. Reevaluation of asymptomatic patients with severe regurgitation.** | **I** |
| **5. Assessment of changes in hemodynamic severity and ventricular compensation in patients with known valvular regurgitation during pregnancy.** | **I** |
| **6. Reevaluation of patients with mild to moderate regurgitation with ventricular dilation without clinical symptoms.** | **I** |
| **7. Assessment of the effects of medical therapy on the severity of regurgitation and ventricular compensation and function.** | **I** |
| **8. Reevaluation of patients with mild to moderate mitral regurgitation without chamber dilation and without clinical symptoms.** | **IIb** |
| **9. Reevaluation of patients with moderate aortic regurgitation without chamber dilation and without clinical symptoms.** | **IIb** |
| **10. Routine reevaluation in asymptomatic patients with mild valvular regurgitation having stable physical signs and normal LV size and function.** | **III** |

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

## 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 reevaluation at a shorter interval. (See "Indications for Echocardiography in Valvular Stenosis," "Indications for Echocardiography in Native Valvular Regurgitation," and "Indications for Echocardiography in Interventions for Valvular Heart Disease and Prosthetic Valves.")

## 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. 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 no 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.

| Indications for Echocardiography in Mitral Valve Prolapse | Class |
|---|---|
| **1. Diagnosis; assessment of hemodynamic severity, leaflet morphology, and/or ventricular compensation in patients with physical signs of MVP.** | **I** |
| **2. To exclude MVP in patients who have been diagnosed but without clinical evidence to support the diagnosis.** | **IIa** |
| **3. To exclude MVP in patients with first-degree relatives with known myxomatous valve disease.** | **IIa** |
| **4. Risk stratification in patients with physical signs of MVP or known MVP.** | **IIa** |
| **5. 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.** | **III** |

| | |
|---|---|
| **6. Routine repetition of echocardiography in patients with MVP with no or mild regurgitation and no changes in clinical signs or symptoms.** | **III** |

## Infective Endocarditis: Native Valves

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 excrecenses, 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 myocardial involvement are important for risk stratification and prognosis (Table 3).[66-68] These features, along with clinical characteristics such as persistent fever, infecting organism, etc, 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 indication to perform TEE. Indications 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 due to 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]

| **Indications for Echocardiography in Infective Endocarditis: Native Valves** | **Class** |
|---|---|
| **1. Detection and characterization of valvular lesions, their hemodynamic severity, and/or ventricular compensation.*** | **I** |
| **2. Detection of vegetations and characterizations of lesions in patients with congenital heart disease suspected of having infective endocarditis.** | **I** |

| | |
|---|---|
| **3. Detection of associated abnormalities (eg, abscesses, shunts, etc).*** | **I** |
| **4. Reevaluation studies in complex endocarditis (eg, virulent organism, severe hemodynamic lesion, aortic valve involvement, persistent fever or bacteremia, clinical change, or symptomatic deterioration).** | **I** |
| **5. Evaluation of patients with high clinical suspicion of culture-negative endocarditis.*** | **I** |
| **6. Evaluation of bacteremia without a known source.*** | **IIa** |
| **7. Risk stratification in established endocarditis.*** | **IIa** |
| **8. Routine reevaluation in uncomplicated endocarditis during antibiotic therapy.** | **IIb** |
| **9. Evaluation of fever and nonpathological murmur without evidence of bacteremia.** | **III** |

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

## 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. 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, obtaining baseline postoperative studies can be useful 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, reevaluation is indicated even in the absence of a changing clinical situation, as in some cases reoperation may be dictated by echocardiographic findings alone.

| **Indications for Echocardiography in Interventions for Valvular Heart Disease and Prosthetic Valves** | **Class** |
|---|---|
| **1. Assessment of the timing of valvular intervention based on ventricular compensation, function, and/or severity of primary and secondary lesions.** | **I** |

| | |
|---|---|
| **2. Selection of alternative therapies for mitral valve disease (such as balloon valvuloplasty, operative valve repair, valve replacement).*** | **I** |
| **3. Use of echocardiography (especially TEE) in performing interventional techniques (eg, balloon valvotomy) for valvular disease.** | **I** |
| **4. Postintervention baseline studies for valve function (early) and ventricular remodeling (late).** | **I** |
| **5. Reevaluation of patients with valve replacement with changing clinical signs and symptoms; suspected prosthetic dysfunction (stenosis, regurgitation) or thrombosis.*** | **I** |
| **6. Routine reevaluation study after baseline studies of patients with valve replacements with mild to moderate ventricular dysfunction without changing clinical signs or symptoms.** | **IIa** |
| **7. Routine reevaluation at the time of increased failure rate of a bioprosthesis without clinical evidence of prosthetic dysfunction.** | **IIb** |
| **8. Routine reevaluation of patients with valve replacements without suspicion of valvular dysfunction and unchanged clinical signs and symptoms.** | **III** |
| **9. Patients whose clinical status precludes therapeutic interventions.** | **III** |

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

## 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 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 seen in some prosthetic valves due to pressure recovery and may not

accurately represent the hemodynamic gradient. 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] Reevaluation 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 vegetations. 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.

| **Indications for Echocardiography in Infective Endocarditis: Prosthetic Valves** | **Class** |
|---|---|
| **1. Detection and characterization of valvular lesions, their hemodynamic severity, and/or ventricular compensation.*** | **I** |
| **2. Detection of associated abnormalities (eg, abscesses, shunts, etc).*** | **I** |
| **3. Reevaluation in complex endocarditis (eg, virulent organism, severe hemodynamic lesion, aortic valve involvement, persistent fever or bacteremia, clinical change, or symptomatic deterioration).*** | **I** |
| **4. Evaluation of suspected endocarditis and negative cultures.*** | **I** |
| **5. Evaluation of bacteremia without known source.*** | **I** |
| **6. Evaluation of persistent fever without evidence of bacteremia or new murmur.*** | **IIa** |

| | |
|---|---|
| **7. Routine reevaluation in uncomplicated endocarditis during antibiotic therapy.*** | **IIb** |
| **8. Evaluation of transient fever without evidence of bacteremia or new murmur.** | **III** |

***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 (see section IV, "Ischemic Heart Disease"). Other cardiovascular abnormalities that frequently cause chest pain, 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 coronary artery disease, the role of echocardiography has grown over the last 5 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 coronary artery disease 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 an acute infarction[83-85] with a pooled negative predictive accuracy of about 95%. In a patient with previous myocardial infarction (either clinically evident or silent), the resting echocardiogram can confirm that event and evaluate its functional significance.

| **Indications for Echocardiography in Patients With Chest Pain** | **Class** |
|---|---|
| **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).** | **I** |
| **2. Evaluation of chest pain in patients with suspected acute myocardial ischemia, when baseline ECG is nondiagnostic and when study can be obtained during pain or soon after its abatement (see section IV).** | **I** |
| **3. Evaluation of chest pain in patients with suspected aortic dissection (see section VIII).** | **I** |

| | |
|---|---|
| **4. Chest pain in patients with severe hemodynamic instability (see section XIII).** | **I** |
| **5. Evaluation of chest pain for which a noncardiac etiology is apparent.** | **III** |
| **6. Diagnosis of chest pain in a patient with electrocardiographic changes diagnostic of myocardial ischemia/infarction.** | **III** |

# Sections IV and V

## IV. Ischemic Heart Disease

Echocardiography has become an established and powerful tool for diagnosing the presence of coronary artery disease 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 compromise 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 risk stratification and prognostication.

### 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.

#### *Diagnosis*

The use of echocardiography for diagnosis of acute myocardial infarction is most helpful 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 coronary artery disease and pathological evidence of infarction.[83,90-97] 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 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 coronary artery disease 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] However, the absence of segmental abnormalities (ie, the presence of either normal wall motion or diffuse abnormalities) has a high negative predictive value. 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 coronary artery disease is usually present in patients with unstable angina. These patients generally are identified 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 reversible segmental wall motion abnormalities confirms the diagnosis of unstable angina.

### *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] 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[110] and late[111] complications and mortality.[90,112] 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 echocardiograms are excellent for evaluating these changes 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 but do merit careful observation. Relatively mild and localized wall motion abnormalities indicate a low risk of complications.

### *Assessment of Complications*

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

1. *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.

2. *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 LV remodeling characterized by progressive chamber dilation and further deterioration in global systolic function.

3. *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 preexisting or the result of RV infarction.

4. *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. 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.

5. *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.

6. *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]

7. *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."

### *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.

### *Predischarge Evaluation Using Stress Echocardiography*

Graded stress echocardiography using intravenous dobutamine can help in assessing myocardial viability early after myocardial infarction.[136-138] Available data suggest 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 2 to 10 days after acute myocardial infarction. Myocardial stunning may occur when acute ischemia is followed by restoration of adequate blood flow and may last for days to months. Reperfusion-salvaged, stunned (but not functioning at rest) myocardium 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] (suggesting that these segments are "stunned"). However, when segments with hypokinesis or akinesis at rest show no improvement during dobutamine infusion, functional recovery is less common (suggesting that most of these segments are infarcted). Segments with initial improvement during low-dose dobutamine infusion but deterioration of function with higher doses 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] There are few data on long-term event rates in patients studied by predischarge stress echocardiography after an acute myocardial infarction in both those who have and those who have not undergone thrombolytic or other reperfusion therapy. 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 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 the longer the time between resolution of chest pain and acquisition of echocardiographic images. 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 indications for echocardiography in acute myocardial ischemic syndromes are summarized below.

| **Indications for Echocardiography in the Diagnosis of Acute Myocardial Ischemic Syndromes** | **Class** |
|---|---|

| | |
|---|---|
| **1. Diagnosis of suspected acute ischemia or infarction not evident by standard means.** | **I** |
| **2. Measurement of baseline LV function.** | **I** |
| **3. Patients with inferior myocardial infarction and bedside evidence suggesting possible RV infarction.** | **I** |
| **4. Assessment of mechanical complications and mural thrombus.*** | **I** |
| **5. Identification of location/severity of disease in patients with ongoing ischemia.** | **IIa** |
| **6. Diagnosis of acute myocardial infarction already evident by standard means.** | **III** |

***TEE is indicated when TTE studies are not diagnostic**.

| **Indications for Echocardiography in Risk Assessment, Prognosis, and Assessment of Therapy in Acute Myocardial Ischemic Syndromes** | **Class** |
|---|---|
| **1. Assessment of infarct size and/or extent of jeopardized myocardium.** | **I** |
| **2. In-hospital assessment of ventricular function when the results are used to guide therapy.** | **I** |
| **3. In-hospital or early postdischarge assessment of the presence/extent of inducible ischemia whenever baseline abnormalities are expected to compromise electrocardiographic interpretation.*** | **I** |
| **4. In-hospital or early postdischarge assessment of the presence/extent of inducible ischemia in the absence of baseline abnormalities expected to compromise ECG interpretation.*** | **IIa** |
| **5. Assessment of myocardial viability when required to define potential efficacy of revascularization.°** | **IIa** |
| **6. Reevaluation of ventricular function during recovery when results are used to guide therapy.** | **IIa** |
| **7. Assessment of ventricular function after revascularization.** | **IIa** |

| | |
|---|---|
| **8. Assessment of long-term prognosis (≥ 2 years after acute myocardial infarction).** | **IIb** |
| **9. Routine reevaluation in the absence of any change in clinical status.** | **III** |

***Exercise or pharmacological stress echocardiogram.**

**ºDobutamine stress echocardiogram.**

## Chronic Ischemic Heart Disease

In patients with chronic ischemic heart disease, echocardiography is useful for a range of indications, including diagnosis, risk stratification, and clinical management decisions. Quantitative indexes 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 and Assessment of Left Ventricular Function").

### *Diagnostic Accuracy of Echocardiographic Techniques in Chronic Coronary Artery Disease*

1. *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 normal, also can be evaluated (see section V, "Cardiomyopathy and Assessment of Left Ventricular Function").

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]

2. *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 coronary artery disease. 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. 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 those with >70% stenosis

compared with those with more moderate lesions.[150-184] Compared with standard treadmill exercise testing, stress echocardiography is of significant additive clinical value for detecting and localizing myocardial ischemia.

In patients with a significant clinical suspicion of coronary artery disease, stress echocardiography is appropriate when standard exercise testing is likely to be nondiagnostic. 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.[150,183,184] 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. 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]

### *Special Issues With Regard to Stress Echocardiography for the Diagnosis of Coronary Artery Disease*

1. *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 coronary artery disease is intermediate. 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 coronary artery disease. In patients with a very low pretest likelihood for coronary artery disease (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 coronary artery disease (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.[185-193] Table 9 summarizes the prognostic value of stress echocardiography. Additional prognostic studies are desirable.

2. *Pharmacological stress echocardiography.* Pharmacological agents can be used to increase cardiac workload in lieu of treadmill or bicycle exercise or cause coronary arterial 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 heterogenous 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 adenosine compared with dobutamine.[150,183,184] However, in several studies pharmacological stress echocardiography using vasodilator agents does appear to be valuable in detecting inducible myocardial ischemia and in determining prognosis.[141,145,153,188,189]

3. *Detection of coronary artery disease in asymptomatic patients.* Stress echocardiography is not recommended for screening in asymptomatic patients without known coronary artery disease 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 coronary artery disease.

4. *Stress echocardiography for preoperative evaluation.* This topic is discussed in another ACC/AHA guideline.[4]

### *Diagnosis of Myocardial Viability in Chronic Coronary Artery Disease*

In patients with chronic stable coronary artery disease, myocardial contractile function can be impaired because of irreversible myocardial necrosis or as a result of hibernating myocardium.[194] 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. Since this condition is potentially reversible, identifying it accurately has important clinical value; revascularization of hibernating myocardium can lead to functional recovery and presumably improved long-range outlook. In patients with multivessel coronary artery disease and depressed LV function, improvement in regional LV function during dobutamine stress echocardiography is considered to indicate contractile reserve and to be predictive of improved ventricular function after revascularization.[195-200] The lack of contractile reserve during dobutamine infusion denotes a low likelihood of improvement after bypass surgery. Table 6 summarizes the role of dobutamine stress echocardiography for evaluating hibernating myocardium. Larger studies in patients with chronic ischemic LV dysfunction are needed to confirm the accuracy of this approach.

### *Assessment of Disease Severity/Risk Stratification/ Prognosis in Chronic Coronary Artery Disease*

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 coronary artery disease and new or changing symptoms.

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 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 knowing the state of LV systolic and diastolic function, valvular function, and right-heart hemodynamics. These indications are discussed in section II, "Murmurs and Valvular Heart Disease," and section V, "Cardiomyopathy and Assessment of Left Ventricular Function."

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 denotes a low cardiovascular event rate during follow-up.[185-193] 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] 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 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).

### *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. In a symptomatic patient or when there are other clinical indications, 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 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] can help in choosing revascularization rather than heart transplantation.

After successful bypass surgery, routine follow-up testing generally is not necessary in the asymptomatic individual. Improvement in patient outcomes by identifying asymptomatic 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 indications for echocardiography in chronic ischemic heart disease are summarized below.

| **Indications for Echocardiography in Diagnosis and Prognosis of Chronic Ischemic Heart Disease** | **Class** |
|---|---|
| **1. Diagnosis of myocardial ischemia in symptomatic individuals.*** | **I** |
| **2. Assessment of global ventricular function at rest.** | **I** |
| **3. Assessment of myocardial viability (hibernating myocardium) for planning revascularization.°** | **I** |
| **4. Assessment of functional significance of coronary lesions (if not already known) in planning percutaneous transluminal coronary angioplasty.*** | **I** |
| **5. Diagnosis of myocardial ischemia in selected patients with an intermediate or high pretest likelihood of coronary artery disease.*** | **IIb** |
| **6. Assessment of an asymptomatic patient with positive results from a screening treadmill test.** | **IIb** |
| **7. Assessment of global ventricular function with exercise.*** | **IIb** |
| **8. Screening of asymptomatic persons with a low likelihood of coronary artery disease.** | **III** |
| **9. Routine periodic reassessment of stable patients for whom no change in therapy is contemplated.** | **III** |

| | |
|---|---|
| **10. Routine substitution for treadmill exercise testing in patients for whom ECG analysis is expected to suffice.** | **III** |

***Exercise or pharmacological stress echocardiogram.**

**ºDobutamine stress echocardiogram.**

**Other: Risk stratification before noncardiac surgery. (See Eagle KA, Brundage BH, Chaitman BR, et al. ACC/AHA guidelines for perioperative cardiovascular evaluation for noncardiac surgery. *J Am Coll Cardiol*. 1996;27:910-948.)**

| Indications for Echocardiography in Assessment of Interventions in Chronic Ischemic Heart Disease | Class |
|---|---|
| **1. Assessment of LV function when needed to guide institution and modification of drug therapy in patients with known or suspected LV dysfunction.** | **I** |
| **2. Assessment for restenosis after revascularization in patients with atypical recurrent symptoms.*** | **I** |
| **3. Assessment for restenosis after revascularization in patients with typical recurrent symptoms.*** | **IIa** |
| **4. Routine assessment of asymptomatic patients after revascularization.** | **III** |

***Exercise or pharmacological stress echocardiography.**

# V. Cardiomyopathy and Assessment of Left Ventricular Function: Echocardiographic Parameters

Of all the indications for echocardiography, the evaluation of ventricular systolic function is perhaps the most common. In the recent prospective study by Krumholz et al,[203] evaluation of LV systolic function was the primary indication for TTE in 26% of inpatients studied, a frequency at least twice that of the next most common indication. Current echocardiographic 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 size[204] as well as LV volumes and ejection fraction. An advantage of two-dimensional (compared with M-mode) echocardiography is that the myocardial mass, chamber volumes, and ejection fraction of an abnormally shaped ventricle can be measured. 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]

### Assessment of Ejection Fraction

M-mode echocardiographic methods can be used to define many indexes of global LV function; the most widely used parameters are ejection phase indexes, 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; mildly, moderately, or severely reduced; or it may be quantitated. When performed by skilled observers, visual estimation has been reported to yield ejection fractions that correspond closely to those 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 clinical 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 indication for testing, duplicate estimates of ejection fraction with multiple modalities should be discouraged.

All ejection phase indexes of myocardial contractile performance are limited by their load dependence. Load-independent indexes, including end-systolic pressure-volume relations,[210] preload recruitable stroke work, and end-systolic stress-length relations,[212] can be derived echocardiographically. Although these indexes may be used in the clinical setting, practically speaking, their use is limited by the need for simultaneous LV pressure and difficult 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 clinically. The determination of cardiac output is a potentially more useful Doppler application,[213,214] but clinically, thermodilution methods are usually used for this purpose.

### 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 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 clinically determined jugular venous pressure is definitely not 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 usually denotes 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 elucidate the origin of dyspnea by documenting or ruling out the common cardiac causes of pulmonary congestion: left-sided valvular disease, depressed systolic function, diastolic function, and cardiomyopathy. In this regard, echocardiography may be the preferred initial diagnostic test when the history, physical examination, and routine laboratory tests suggest or cannot eliminate cardiac disease.

### Regional Left Ventricular 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]

### Ventricular Dysfunction

Clinically most instances of systolic dysfunction are due to ischemic heart disease, hypertensive disease, or valvular heart disease. However, primary disorders of the heart muscle are 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. 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[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,[217] who prospectively studied 151 consecutive patients undergoing Doppler echocardiography who had a clinical diagnosis of congestive heart failure; a normal ejection fraction (>55%) was observed in 34% of patients.

### Dilated Cardiomyopathy

Echocardiography demonstrates dilation of ventricular chambers, usually without increased wall thickness. 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 reevaluation 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 (<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 <450 to 500 mg/m$^2$.[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/m$^2$ show either increased wall stress or evidence of reduced contractility by stress-shortening analysis.[223] For this reason, reevaluation 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/m$^2$ of doxorubicin.[226,227]

### 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 distribution of hypertrophy.[228] Doppler techniques may be used to localize and quantitate intraventricular gradients at rest and with provocative maneuvers, evaluate diastolic filling, and quantitate associated mitral regurgitation.[229] 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 dual-chamber pacing.[230]

### 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 the etiology of one type of restrictive cardiomyopathy.

### Heart Failure With Normal Systolic Function (Diastolic Dysfunction)

Diastolic dysfunction is thought to be present when a patient with heart failure has a systolic ejection fraction >40%.[80,217,218,235] There are other, subtler manifestations of diastolic dysfunction, including failure to augment cardiac output with exercise.[236] Given that the prognosis and optimal management for the patient with diastolic dysfunction is likely to be quite different from the heart failure patient with systolic dysfunction, it is critical that the proper diagnosis be made. A large number of indexes of diastolic function based on information from M-mode and two-dimensional echocardiography (and more recently Doppler mitral and pulmonary flow profiles) have been investigated. However, experience and theoretic considerations have tempered early enthusiasm that simple Doppler indexes could adequately describe as complex a phenomenon as diastolic function. This is due to several factors, including

- Load and heart rate dependence of the commonly described echocardiographic variables [237,238]
- The broad overlap in diastolic filling parameters for normal and abnormal subjects
- The inability to apply most methods in atrial fibrillation and other rhythm and conduction disturbances

For these reasons, echocardiographic indexes of diastolic function are most useful in the reevaluation examination of a given patient or in characterizing large groups of subjects. Nonetheless, when these indexes are interpreted in the context of clinical variables with the recognition of all the potentially confounding influences, they may provide valuable information in individual subjects.

### 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 as well as difficulty in obtaining standardized imaging planes. 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. Echocardiographic methods of evaluating RV diastolic function have not been reported.

| Indications for Echocardiography in Patients With Dyspnea, Edema, or Cardiomyopathy | Class |
|---|---|
| 1. Assessment of LV size and function in patients with suspected cardiomyopathy or clinical diagnosis of heart failure.* | I |
| 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.* | I |
| 3. Dyspnea with clinical signs of heart disease. | I |
| 4. Patients with unexplained hypotension, especially in the intensive care unit.* | I |
| 5. Patients exposed to cardiotoxic agents, to determine the advisability of additional or increased dosages. | I |
| 6. Reevaluation of LV function in patients with established cardiomyopathy when there has been a documented change in clinical status or to guide medical therapy. | I |
| 7. Reevaluation of patients with established cardiomyopathy when there is no change in clinical status. | IIb |
| 8. Reevaluation of patients with edema when a potential cardiac cause has already been demonstrated. | IIb |
| 9. Evaluation of LV ejection fraction in patients with recent (contrast or radionuclide) angiographic determination of ejection fraction. | III |
| 10. Routine reevaluation in clinically stable patients in whom no change in management is contemplated. | III |
| 11. In patients with edema, normal venous pressure, and no evidence of heart disease. | III |

*TEE is indicated when TTE studies are not diagnostic.

# Sections VI. - XII.

## VI. Pericardial Disease

One of the earliest clinical applications of echocardiography was in the detection of pericardial effusion,[240] 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. 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.

### 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, 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.

### 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.

### 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]

### 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]

### 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] 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.

### 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.

| Indications for Echocardiography in Pericardial Disease | Class |
|---|---|
| **1. Patients with suspected pericardial disease, including effusion, constriction, or effusive-constrictive process.** | **I** |
| **2. Patients with suspected bleeding in the pericardial space, eg, trauma, perforation, etc.** | **I** |
| **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.** | **I** |
| **4. Pericardial friction rub developing in acute myocardial infarction accompanied by symptoms such as persistent pain, hypotension, and nausea.** | **I** |
| **5. 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.** | **IIa** |
| **6. Echocardiographic guidance and monitoring of pericardiocentesis.** | **IIa** |
| **7. Postsurgical pericardial disease, including postpericardiotomy syndrome, with potential for hemodynamic impairment.** | **IIb** |
| **8. In the presence of a strong clinical suspicion and nondiagnostic TTE, TEE assessment of pericardial thickness to support a diagnosis of constrictive pericarditis.** | **IIb** |
| **9. Routine follow-up of small pericardial effusion in clinically stable patients.** | **III** |
| **10. Follow-up studies in patients with cancer or other terminal illness for whom management would not be influenced by echocardiographic findings.** | **III** |
| **11. Assessment of pericardial thickness in patients without clinical evidence of constrictive pericarditis.** | **III** |
| **12. Pericardial friction rub in early uncomplicated myocardial infarction or early postoperative period after cardiac surgery.** | **III** |

# 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 diagnosis.

Intracardiac masses should be suspected in the context of the clinical presentation. Examples of this include suspicion of vegetative lesions in patients with underlying connective tissue diseases and new or varying murmurs and obviously in patients with signs and symptoms suggesting infective endocarditis. Intracardiac thrombi should be suspected in several clinical situations. These include a suspicion of LV thrombi in patients after extensive anterior myocardial infarction and left atrial thrombi in patients with atrial fibrillation, especially 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. 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 actual detection of the mass. Patients for whom echocardiography may be appropriate include those in whom there is a high clinical suspicion that a cardiac mass is present. 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 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.

| Indications for Echocardiography in Patients With Cardiac Masses and Tumors | Class |
|---|---|
| **1. Evaluation of patients with clinical syndromes and events suggesting an underlying cardiac mass.** | **I** |

| | |
|---|---|
| **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.** | **I** |
| **3. Follow-up or surveillance studies after surgical removal of masses known to have a high likelihood of recurrence (ie, myxoma).** | **I** |
| **4. Patients with known primary malignancies when echocardiographic surveillance for cardiac involvement is part of the disease staging process.** | **I** |
| **5. Screening persons with disease states likely to result in mass formation but for whom no clinical evidence for the mass exists.** | **IIb** |
| **6. Patients for whom the results of echocardiography will have no impact on diagnosis or clinical decision making.** | III |

# 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 upper ascending portion adjacent to the tracheobronchial tree. Using transthoracic imaging, good visualization of the main pulmonary artery segment and the proximal right and left pulmonary 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 supra- clavicular 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.

### Aortic Dissection

Acute aortic dissection is a life-threatening emergency, and an early and prompt diagnosis is mandatory for appropriate patient care. 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. Biplane or 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 LV size and function. TEE is also suited for postoperative evaluation of patients with aortic dissection.[264]

### 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 reevaluation of patients with ascending aortic aneurysms (especially in patients with Marfan syndrome) for determining 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]

### 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.

| Indications for Echocardiography in Suspected Thoracic Aortic Disease | Class | |
|---|---|---|
| | TTE | TEE |
| 1. Aortic dissection. | IIa | I |
| 2. Aortic aneurysm. | I* | I |
| 3. Aortic rupture. | IIb | I |
| 4. Aortic root dilatation in Marfan or other connective tissue syndromes. | I | IIb |
| 5. Degenerative or traumatic aortic disease with clinical atheroembolism. | IIb | I |
| 6. Follow-up of aortic dissection, especially after surgical repair without suspicion of complication or progression. | I | IIa |

| | | |
|---|---|---|
| **7. Follow-up of aortic dissection especially after surgical repair when complication or progression is suspected.** | **IIa** | **I** |
| **8. First-degree relative of a patient with Marfan syndrome or other connective tissue disorder.** | **I** | **IIb** |

***Especially for aortic root aneurysm.**

### 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 permits clear visualization of the pulmonary vein connections.

## IX. Pulmonary 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 can 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 distorted, 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.

### Pulmonary Thromboembolism

Echocardiography has aided a diagnosis of central pulmonary artery thromboembolic disorders, especially in patients with severe or massive pulmonary embolism. However, echocardiography has low sensitivity and specificity for detecting pulmonary emboli.[269]

| Indications for Echocardiography in Pulmonary Disease | Class |
|---|---|
| **1. Suspected pulmonary hypertension.** | **I** |
| **2. Pulmonary emboli and suspected clots in the right atrium or ventricle or main pulmonary artery branches.*** | **I** |
| **3. For distinguishing cardiac versus noncardiac etiology of dyspnea in patients in whom all clinical and laboratory clues are ambiguous.*** | **I** |
| **4. Follow-up of pulmonary artery pressures in patients with pulmonary hypertension to evaluate response to treatment.** | **I** |
| **5. Lung disease with clinical suspicion of cardiac involvement (suspected cor pulmonale).** | **I** |
| **6. Measurement of exercise pulmonary artery pressure.** | **IIa** |
| **7. Patients being considered for lung transplantation or other surgical procedure for advanced lung disease.*** | **IIa** |
| **8. Lung disease without any clinical suspicion of cardiac involvement.** | **III** |

| | |
|---|---|
| **9. Reevaluation studies of RV function in patients with chronic obstructive lung disease without a change in clinical status.** | **III** |

***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 coronary artery disease and degenerative valve disease, especially in the elderly. Stress echocardiography is indicated in the diagnosis and assessment of the functional severity of concomitant coronary artery disease. 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] the impact on patient morbidity and mortality is unknown, as is the potential effectiveness of echocardiography in guiding therapy aimed at reducing LV mass.[272,286] Despite this, a possible indication for reevaluation quantitation of LV mass in assessing drug therapy for hypertension is evaluation of regression of LV hypertrophy

after adequate blood pressure control.[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, following LV hypertrophy by echocardiography cannot be supported.

| Indications for Echocardiography in Hypertension | Class |
|---|---|
| **1. When assessment of resting LV function, hypertrophy, or concentric remodeling is important in clinical decision making (see LV function).** | **I** |
| **2. Detection and assessment of functional significance of concomitant coronary artery disease (see coronary disease).*** | **I** |
| **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.** | **I** |
| **4. Identification of LV diastolic filling abnormalities with or without systolic abnormalities.** | **IIa** |
| **5. 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.** | **IIa** |
| **6. Risk stratification for prognosis by determination of LV performance.** | **IIb** |
| **7. Reevaluation to guide antihypertensive therapy based on LV mass regression.** | **III** |
| **8. Reevaluation in asymptomatic patients to assess LV function.** | **III** |

***Stress echocardiography.**

# 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. Cardioembolic disease refers to the heart as the origin of an occlusive embolus with subsequent migration to a target organ. 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 in those with vascular disease, also have a potential cardiac source of embolus (Table 10). Proving 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 deriving 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 derived. The available data are all concordant, however, in suggesting a high prevalence of potential cardiac sources of embolus and other neurological embolic events.

Many studies have evaluated the frequency of a cardioembolic source of an acute neurological syndrome. 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 Cerebal Embolism Task Force has previously 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] 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, 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 cryptogenic. 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 >50% for younger persons.[303] 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 responsible pathology.

Additionally, a decision to use echocardiographic techniques 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, nonrheumatic atrial fibrillation,

mitral annular calcification, and anterior myocardial infarction with aneurysm formation. 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 of embolic potential) suggests that echocardiographic screening may be applicable to a broader range of patients than only those persons with clinically suspected disease.

Two-dimensional echocardiography is the only technique that is easily applied and widely available for evaluation of potential cardioembolic source. Rapid intravenous injection of agitated saline floods the right heart with microbubbles and can detect right-to-left shunting across a patent foramen ovale. Examinations can be performed 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 compared with TTE.[299,300] Table 13 outlines the relation between TEE and TTE in a wide range 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 is probably not warranted. Conversely, TEE is uniquely suited for detection of left atrial spontaneous contrast,[302,304,305] left atrial thrombi, atrial septal aneurysm,[306,307] right-to-left shunting through a patent foramen ovale, and atheroma of the aortic arch[308-313] as well as several other more recently described anomalies.

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 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. 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. Recent 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; however, it should be understood that this does not represent a distinct break. 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.

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. 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 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.

| Indications for Echocardiography in Patients With Neurological Events or Other Vascular Occlusive Events | Class |
|---|---|
| **1. Patients of any age with abrupt occlusion of a major peripheral or visceral artery.** | **I** |
| **2. Younger patients (typically <45 years) with cerebrovascular events.** | **I** |
| **3. Older patients (typically >45 years) with neurological events without evidence of cerebrovascular disease or other obvious cause.** | **I** |
| **4. Patients for whom a clinical therapeutic decision (anticoagulation, etc) will depend on the results of echocardiography.** | **I** |
| **5. Patients with suspicion of embolic disease and with cerebrovascular disease of questionable significance.** | **IIa** |
| **6. Patients with a neurological event and intrinsic cerebrovascular disease of a nature sufficient to cause the clinical event.** | **IIb** |
| **7. 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.** | **III** |

# XII. Arrhythmias and Palpitation

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, reentrant 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.

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 indications 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 indication 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 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 been suggested. Thus, TEE has been reported to be helpful in guiding transseptal catheterization during radiofrequency ablative procedures.[329] Early studies also proposed routine postprocedural evaluation of patients undergoing ablation. However, the yield was low enough in laboratories with established ablative programs that the test is no longer recommended in uncomplicated cases.

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."

| Indications for Echocardiography in Patients With Arrhythmias and Palpitations | Class |
|---|---|
| **1. Arrhythmias with clinical suspicion of structural heart disease.** | **I** |
| **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.** | **I** |
| **3. Evaluation of patients as a component of the workup before electrophysiological ablative procedures.** | **I** |
| **4. Arrhythmia requiring treatment.** | **IIa** |
| **5. TEE guidance of transseptal catheterization and catheter placement during ablative procedures.** | **IIa** |
| **6. Arrhythmias commonly associated with, but without clinical evidence of, heart disease.** | **IIb** |
| **7. 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.)** | **IIb** |
| **8. Palpitation without corresponding arrhythmia or other cardiac signs or symptoms.** | **III** |
| **9. Isolated premature ventricular contractions for which there is no clinical suspicion of heart disease.** | **III** |

## Cardioversion of Patients With Atrial Fibrillation

Recent 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[330-335] (Table 18).

The issue of performing TEE before elective cardioversion from atrial fibrillation has recently been addressed in patients with atrial fibrillation of >48 hours' duration.[337,338] 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. Recently 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] However, the authors emphasize the importance of anticoagulation from the time of study until the time of cardioversion and subsequently until atrial mechanical function has returned after conversion. Others, however, have reported that 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] Large multicenter trials are under way to address this issue. Thus, at present, although there is controversy concerning the routine use of TEE to identify patients to undergo cardioversion without prolonged anticoagulation, there is agreement that periconversion and postconversion anticoagulation is indicated. 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. It has been the recommendation of the American College of Chest Physicians Consensus Conference on Antithrombotic Therapy[340] that anticoagulation is not necessary before cardioversion of patients with atrial fibrillation of <48 hours' duration. 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 argue that guidelines for anticoagulation and TEE in patients undergoing cardioversion of atrial fibrillation should not differentiate between those with recent versus chronic fibrillation.

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.

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

| | |
|---|---|
| **5. Evaluation of patient for whom a decision concerning cardioversion will be impacted by knowledge of prognostic factors (such as LV function, coexistent mitral valve disease, etc).** | **I** |
| **6. Patients with atrial fibrillation of <48 hours' duration and other heart disease.*** | **IIa** |
| **7. Patients with atrial fibrillation of <48 hours' duration and no other heart disease.*** | **IIb** |
| **8. Patients with mitral valve disease or hypertrophic cardiomyopathy who have been on long-term anticoagulation at therapeutic levels before cardioversion.*** | **IIb** |
| **9. Patients undergoing cardioversion from atrial flutter.** | **IIb** |
| **10. Patients requiring emergent cardioversion.** | **III** |
| **11. Patients who have been on long-term anticoagulation at therapeutic levels and who do not have mitral valve disease or hypertrophic cardiomyopathy before cardioversion.** | **III** |
| **12. Precardioversion evaluation of patients who have undergone previous TEE and with no clinical suspicion of a significant interval change.** | **III** |

***TEE only.**

## 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. Only retrospective studies published in abstract form are available on the subject,[343-345] and the conclusions differ. While two studies reported no diagnostic yield from echocardiography in patients in whom history, physical examination, and ECG failed to indicate a cause,[343,344] a third study reported that echocardiography identified what was ultimately determined to be the sole cause in 8% of subjects with syncope referred for echocardiographic evaluation.[345] However, it is impossible to determine the true yield of echocardiography in this setting, including its ability to rule out as well as rule in clinically suspected abnormalities in a retrospective study. Thus, there is a strong need for a large prospective study in this area.

| Indications for Echocardiography in the Patient With Syncope | Class |
|---|---|
| **1. Syncope in a patient with clinically suspected heart disease.** | **I** |
| **2. Periexertional syncope.** | **I** |
| **3. Syncope in a patient in a high-risk occupation (eg, pilot).** | **IIa** |
| **4. Syncope of occult etiology with no findings of heart disease on history or physical exam.** | **IIb** |
| **5. Recurrent syncope in a patient in whom previous echocardiographic or other testing demonstrated a cause of syncope.** | **III** |
| **6. Syncope in a patient for whom there is no clinical suspicion of heart disease.** | **III** |
| **7. Classic neurogenic syncope.** | **III** |

## 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 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 members of an affected individual. The most common diseases that fall into this category are hypertrophic cardiomyopathy and Marfan syndrome.

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 raise the possibility that patterns of disease progression may be similarly variable. Thus, repeat screening of adults may also be justifiable, particularly in those whose kindred have malignant forms of the disease.

Marfan syndrome is transmitted as an autosomal dominant with spontaneous mutation occurring in up to 30% of subjects. The diagnosis is 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 take into consideration the family history (Table 19). Because the primary method of diagnosing cardiovascular 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 as 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. A single report has suggested that one of five patients with dilated cardiomyopathy has a familial form of the disease, thus proposing a role for echocardiographic screening in this setting as well.[351] However, no other study has confirmed this observation.

Another accepted indication for echocardiographic screening is in the evaluation of potential donor hearts for transplantation.[352] In patients in whom transthoracic imaging is inadequate, TEE provides an alternative approach.[353] 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 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[356-358] have shown that the prevalence of these and other conditions appears to be too low to justify widespread screening (Table 20).

| **Indications for Echocardiography to Screen for the Presence of Cardiovascular Disease** | **Class** |
|---|---|
| **1. Patients with a family history of genetically transmitted cardiovascular disease.** | **I** |
| **2. Potential donors for cardiac transplantation.** | **I** |
| **3. Patients with phenotypic features of Marfan syndrome or related connective tissue diseases.** | **I** |
| **4. Baseline and reevaluations of patients undergoing chemotherapy with cardiotoxic agents.** | **I** |
| **5. Patients with systemic disease that may affect the heart.** | **IIb** |
| **6. The general population.** | **III** |
| **7. Competitive athletes without clinical evidence of heart disease.** | **III** |

# Sections XIII. - XV.

## 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-expiratory pressure (PEEP). Up to one half of such patients cannot be adequately imaged by TTE, especially those requiring >10 cm PEEP.[361] Furthermore, many patients in intensive care units cannot be appropriately positioned, have sustained chest injury, or are postoperative 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. The presence of RV hypokinesis identifies patients with 30% or more of the lung nonperfused who may receive significant benefit from thrombolysis.[362]

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. These studies total 735 patients. 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] (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]

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] 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]

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]

### *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.

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 (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]

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 emergency department 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 multisystem 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 complications 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]

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% sensitive in detecting pericardial fluid in juxtacardiac penetrating chest wounds.[399] Thus, TTE may prevent unnecessary exploratory thoracotomy or subxiphoid pericardiotomy.[400] In a report in which TTE was used in the emergency department 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 and Doppler.[407] While no large series of penetrating cardiac wounds studied by TEE and Doppler 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 lab 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 Transesophageal Echocardiography 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.

| Indications for Echocardiography in the Critically Ill | Class |
|---|---|

| | |
|---|---|
| **1. The hemodynamically unstable patient.** | **I** |
| **2. Suspected aortic dissection (TEE).** | **I** |
| **3. The hemodynamically stable patient not expected to have cardiac disease.** | **III** |
| **4. Reevaluation follow-up studies on hemodynamically stable patients.** | **III** |

| **Indications for Echocardiography in the Critically Injured*** | **Class** |
|---|---|
| **1. Serious blunt or penetrating chest trauma (suspected pericardial effusion or tamponade).** | **I** |
| **2. Mechanically ventilated multiple-trauma or chest trauma patient.** | **I** |
| **3. Suspected preexisting valvular or myocardial disease in the trauma patient.** | **I** |
| **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).** | **I** |
| **5. Widening of the mediastinum, postinjury suspected aortic injury (TEE).** | **I** |
| **6. Potential catheter, guidewire, pacer electrode, or pericardiocentesis needle injury with or without signs of tamponade.** | **I** |
| **7. Evaluation of hemodynamics in multiple-trauma or chest trauma patients with pulmonary artery catheter monitoring and data disparate with clinical situation.** | **IIa** |
| **8. Follow-up study on victims of serious blunt or penetrating trauma.** | **IIa** |
| **9. Suspected myocardial contusion in the hemodynamically stable patient with a normal ECG.** | **III** |

***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 Doppler Echocardiography in the Adult Patient With Congenital Heart Disease

The adult patient with congenital heart disease is seen either because the problem was not discovered in childhood or more often because the patient was previously diagnosed as having inoperable congenital heart disease or has had one or more palliative or corrective surgical procedures.

As a general rule, all patients with congenital heart disease must be followed indefinitely, 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 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 noncardiac 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.

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.

Two-dimensional Doppler echocardiography is useful in

- Demonstrating chamber size.
- Evaluating RV and LV 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.
- Using contrast echocardiography and color Doppler to define the presence, site, and relative magnitude of intracardiac or vascular shunts.
- Demonstrating intracardiac and/or central vascular mural thrombi as well as coronary fistulas.
- Assessment of atrioventricular valve anatomy and function.

| **Indications for Echocardiography in the Adult Patient With Congenital Heart Disease** | **Class** |
|---|---|
| **1. Patients with clinically suspected congenital heart disease, 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.** | **I** |
| **2. Patients with known congenital heart disease on follow-up when there is a change in clinical findings.** | **I** |
| **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.** | **I** |
| **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 functionally single ventricle after Fontan procedure, transposition of the great vessels after Mustard procedure, L-transposition and ventricular inversion, and palliative shunts).** | **I** |
| **5. Patients with known congenital heart disease for whom following pulmonary artery pressure is important (eg, patients with moderate or ventricular septal defects, atrial septal defects, single ventricle, or any of the above with an additional risk factor for pulmonary hypertension).** | **I** |

| | |
|---|---|
| **6. Periodic echocardiography in patients with surgically repaired (or palliated) congenital heart disease with the following: change in clinical condition or clinical suspicion of residual defects, LV or RV function that must be followed, or when there is a possibility of hemodynamic progression or a history of pulmonary hypertension.** | **I** |
| **7. To direct interventional catheter valvotomy, radiofrequency ablation valvotomy interventions in the presence of complex cardiac anatomy.** | **I** |
| **8. A follow-up Doppler echocardiographic study, annually or once every 2 years, in patients with known hemodynamically significant congenital heart disease without evident change in clinical condition.** | **IIb** |
| **9. Multiple repeat Doppler 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.** | **III** |
| **10. Repeat Doppler 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.** | **III** |

# 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, 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.

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.[410-415] Echocardiographic reevaluation in some candidates improves medical or surgical management. For the child with insignificant cardiac disease, an echocardiographic evaluation provides reassurance to the family. For those patients with significant cardiac abnormality, early and accurate echocardiographic

evaluation improves clinical outcome.

Reevaluation echocardiographic examinations are frequently used to monitor cardiovascular adaptation to surgical repair or palliation and identify recurrence of abnormalities. Such longitudinal follow-up facilitates proactive surgical and/or medical intervention. [416-431] 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.

## 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 vascular resistance and the patency of the ductus arteriosus, reevaluation echocardiographic examinations of the critically ill neonate are often required. Newborns with primary pulmonary hypertension (persistent fetal circulation) 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. 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]

## Congenital Cardiovascular Disease in the Neonate

### *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, helps guide therapeutic management in some diagnostic categories. Reevaluation 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. Echocardiography allows early recognition of 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. These evaluations should not be performed in the field.

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[438,439]
2. Obstructive lesions: location, morphology, pressure gradient, ventricular compensation, associated lesions[437,440-443]
3. Regurgitant lesions: valve morphology, assessment of severity, atrial/ventricular dilation, ventricular compensation, associated lesions[420,444-446]
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 abnormal coronary artery/ventricular fistulae, ventricular compensation[455-457]
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

## Cardiopulmonary Disease

The transition from fetal to extrauterine hemodynamics 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, or congenital heart disease. Echocardiography indicates the patency of the ductus arteriosus, direction and degree of shunting at the ductal level, and estimation of pulmonary artery pressure and its consequences. Echocardiography identifies coexisting ductal-dependent cardiovascular lesions before pharmacological or surgical closure of a patent ductus arteriosus is planned.

Neonates with primary pulmonary hypertension (persistent fetal circulation) may present with or without perinatally acquired pulmonary parenchymal disease. Differentiation of this entity from cyanotic heart disease can be accomplished by echocardiography. In addition to excluding structural abnormalities, Doppler echocardiography provides additional information about atrial and ductal shunting, pulmonary artery pressure, and ventricular function. Reevaluation studies are useful for monitoring the efficacy of therapeutic interventions. In patients with severe disease progressing to extracorporeal membrane oxygenation, this information is useful in assessing the contribution of extracorporeal circulation to ventricular output, alteration in myocardial function, and changes in ductus arteriosus flow.[432]

## Arrhythmias

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. Neonatal arrhythmia may present as acute-onset congestive heart failure or findings of nonimmune fetal hydrops. Echocardiography is integrated into the treatment of these patients to identify the hemodynamic sequelae of the dysrhythmia and coexisting systemic disease.

### Myocardial 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.

| Indications for Neonatal Echocardiography | Class |
|---|---|
| **1. Cyanosis, respiratory distress, congestive heart failure, or abnormal arterial pulses.** | **I** |
| **2. Chromosomal abnormality or major extracardiac abnormality associated with a high incidence of coexisting cardiac abnormality.** | **I** |
| **3. Lack of expected improvement in cardiopulmonary status in a premature infant with a clinical diagnosis of pulmonary disease.** | **I** |
| **4. Systemic maternal disease associated with neonatal comorbidity.** | **I** |
| **5. Loud or abnormal murmur or other abnormal cardiac finding in an infant.** | **I** |
| **6. Presence of a syndrome associated with cardiovascular disease and dominant inheritance or multiple affected family members.** | **I** |
| **7. Presence of a syndrome associated with heart disease, with or without abnormal cardiac findings, for which an urgent management decision is needed.** | **I** |
| **8. Cardiomegaly on chest radiograph.** | **I** |

| | |
|---|---|
| **9. Dextrocardia, abnormal pulmonary or visceral situs by clinical, electrocardiographic, or radiographic examination.** | **I** |
| **10. Arrhythmias or other abnormalities on standard ECG suggesting structural heart disease or peripartum myocardial injury.** | **I** |
| **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.** | **I** |
| **12. Nonimmunologic fetal hydrops.** | **I** |
| **13. Follow-up assessment of a neonate with patent ductus arteriosus who has undergone medical or surgical intervention.** | **I** |
| **14. Short, soft murmur at the lower left sternal border in the neonate.** | **IIa** |
| **15. Failure to thrive in the absence of definite abnormal clinical findings.** | **IIa** |
| **16. 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.** | **IIb** |
| **17. History of nonsustained fetal ectopy in the absence of postpartum arrhythmias.** | **III** |

## 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] However, in the presence of ambiguous 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 significant heart disease, anatomic and physiological data provided by reevaluation and two-dimensional Doppler echocardiography may provide a definitive diagnosis and allow the most efficient selection of adjuvant diagnostic procedures or medical/invasive intervention. Referral for cardiac ultrasound must provide the pediatric cardiologist and the sonographer with

relevant diagnostic data and the clinical objective for the examination.

### *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). The presence of heart disease is more likely to be recognized in the older population because of an abnormal clinical finding. Therefore, echocardiography plays a less important role in screening for heart disease than it does in the neonatal period. The more important role for echocardiography in the infant, child, and adolescent is in fully characterizing a cardiac lesion once an abnormality is suspected. It also provides essential information concerning the natural history of the abnormality and responses to medical and surgical management. 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 also provides important information in patients with systemic connective tissue disorders, eg, Marfan syndrome or Ehlers-Danlos syndrome. Reevaluation 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 the evaluation of an asymptomatic patient with this finding on routine examination by an experienced clinician is limited. Such murmurs can usually be diagnosed by an experienced 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.

| **Indications for Echocardiography in the Infant, Child, and Adolescent** | **Class** |
|---|---|
| **1. Atypical or pathological murmur or other abnormal cardiac finding in an infant or older child.** | **I** |
| **2. Cardiomegaly on chest radiograph.** | **I** |
| **3. Dextrocardia, abnormal pulmonary or visceral situs on clinical, electrocardiographic, or radiographic examination.** | **I** |
| **4. Patients with a known cardiac defect to assess timing of medical or surgical therapy.** | **I** |
| **5. 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.** | **I** |
| **6. Patient with known cardiac lesion and change in physical finding.** | **I** |

| | |
|---|---|
| **7. 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.** | **I** |
| **8. Presence of a syndrome associated with cardiovascular disease and dominant inheritance or multiple affected family members.** | **I** |
| **9. Patients with a family history of genetically transmitted myocardial disease, with or without abnormal cardiac finding.** | **I** |
| **10. Phenotypic findings of Marfan syndrome or Ehlers-Danlos syndrome.** | **I** |
| **11. Baseline and follow-up examinations of patients with neuromuscular disorders having known myocardial involvement.** | **I** |
| **12. Presence of a syndrome associated with a high incidence of congenital heart disease when there are no abnormal cardiac findings.** | **I** |
| **13. Exercise-induced precordial chest pain or syncope.** | **I** |
| **14. "Atypical," "nonvasodepressor" syncope without other cause.** | **I** |
| **15. Failure to thrive in the absence of definite abnormal clinical findings.** | **IIb** |
| **16. In a child or adolescent, an asymptomatic heart murmur identified by an experienced observer as functional or an insignificant cardiovascular abnormality.** | **III** |
| **17. In an otherwise asymptomatic child or adolescent, chest pain identified by an experienced observer as musculoskeletal in origin.** | **III** |

## 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, 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 low-grade supraventricular ectopic beats or brief and infrequent runs of supraventricular tachycardia, are rarely associated with cardiac pathology. Therefore, 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.

| Indications for Echocardiography in Pediatric Patients With Arrhythmias/Conduction Disturbances | Class |
|---|---|
| **1. Arrhythmia in the presence of an abnormal cardiac finding.** | **I** |
| **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.** | **I** |
| **3. Complete atrioventricular block or advanced second-degree atrioventricular block.** | **I** |
| **4. Complete or high-degree secondary atrioventricular block.** | **I** |
| **5. Arrhythmia requiring treatment.** | **I** |
| **6. Ventricular arrhythmia in a patient referred for evaluation for competitive sports.** | **IIa** |
| **7. Evidence of preexcitation on ECG.** | **IIa** |
| **8. Preexitation on ECG in the absence of abnormal cardiac findings.** | **IIb** |
| **9. Recurring arrhythmia not requiring treatment in the presence of normal findings on examination.** | **IIb** |
| **10. 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.** | **III** |

## 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 reevaluation 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 in the proximal or distal coronary circulation, myocarditis, pericarditis, and myocardial infarction. Baseline and reevaluations 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]
- 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]
- In children, HIV infection acquired during the fetal or newborn period is aggressive with early and prominent myocardial involvement. Therefore, a baseline study and reevaluation follow-up studies should be done as indicated by the appearance of tachycardia, congestive heart failure, and respiratory distress.[470,472]
- 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]
- 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,[479] and in screening patients with multisystem disorders associated with cardiomyopathy, eg, muscular dystrophy and Friedreich's ataxia.[480,481] Reevaluation 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.[483,484] Echocardiographic surveillance of LV wall thickness, systolic function, and diastolic function permits appropriate adjustments in medical therapy. Reevaluation studies identify changes in cardiac allograft ventricular function and wall motion in a population at risk for small-vessel coronary artery disease.

| Indications for Echocardiography in Pediatric Acquired Cardiovascular Disease | Class |
|---|---|
| **1. Baseline studies and reevaluation as clinically indicated on all pediatric patients with suspected or documented Kawasaki disease, myopericarditis, HIV, or rheumatic fever.** | **I** |
| **2. Postcardiac or cardiopulmonary transplant to monitor for signs of acute or chronic rejection, thrombus, and cardiac growth.** | **I** |
| **3. Baseline and reevaluation examinations of patients receiving cardiotoxic therapeutic agents.** | **I** |
| **4. Patients with clinical evidence of myocardial disease.** | **I** |
| **5. Patients with severe renal disease and an abnormal cardiac finding.** | **I** |
| **6. Donors undergoing evaluation for cardiac transplantation.** | **I** |
| **7. An acutely ill child with suspected bacterial sepsis or rickettsial disease.** | **IIa** |
| **8. Follow-up examinations after acute rheumatic fever in patients with normal cardiac findings.** | **IIb** |
| **9. A single late follow-up study after acute pericarditis with no evidence of recurrence or chronic pericardial disease.** | **IIb** |
| **10. Long-term follow-up studies in patients with Kawasaki disease who have no coronary abnormalities during the acute phase of the disease process.** | **III** |

## Pulmonary Diseases in Pediatric Acquired Cardiovascular Disease

Children with upper airway or chronic lung disease may have clinical or ECG evidence of pulmonary artery hypertension. Echocardiography provides indirect documentation of

pulmonary artery hypertension and estimation of severity by the presence of RV dilation and/or hypertrophy, the presence of tricuspid or pulmonic valvular regurgitation, and by Doppler estimation of RV systolic pressure. Follow-up studies reflect response to therapy and are useful in guiding management.

Disease states in older infants and children with diseases that cause secondary pulmonary hypertension require documentation of pulmonary hypertension when there are suggestive clinical, ECG, or radiographic findings. These include severe scoliosis, central hypoventilation, diaphragmatic hernia, pulmonary hypoplasia, upper airway obstruction, and familial pulmonary artery hypertension.

| **Indications for Echocardiography in Pediatric Cardiopulmonary Disease** | **Class** |
|---|---|
| **1. Any patient with clinical findings of pulmonary artery hypertension.** | **I** |
| **2. Baseline study of patients with cystic fibrosis and no findings of cor pulmonale.** | **IIa** |

### 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 distal 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 cardiac-assist or extracorporeal cardiopulmonary membrane oxygenation devices or with reduced systolic function of any cause. In addition, the presence of aortic thrombus should be sought in neonates with indwelling aortic catheters and the appearance of hypertension, low cardiac output, or renal failure.

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. 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.

| **Indications for Echocardiography in Pediatric Thromboembolic Disease States** | **Class** |
|---|---|

| | |
|---|---|
| **1. Thromboembolic event in an infant, child, or adolescent.** | **I** |
| **2. Finding or family history of tuberous sclerosis.** | **I** |
| **3. Appearance of sepsis, cyanosis, or right-heart failure in a patient with a long-standing indwelling catheter.** | **I** |
| **4. Systemic embolization or acute-onset hypertension in a patient with right-to-left-shunting and an indwelling catheter.** | **I** |
| **5. Superior vena caval syndrome in the presence of central venous catheter.** | **I** |
| **6. Patient with indwelling catheter and fever but without evidence of pulmonary or systemic embolization.** | **IIb** |
| **7. Routine surveillance of asymptomatic patients with indwelling catheter.** | **III** |

## Transesophageal Echocardiography

TTE, 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. TEE, 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.

TEE 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. For neonates requiring urgent balloon atrial septostomy, performance of this procedure under TTE or TEE guidance in the neonatal intensive care unit may be lifesaving.

The placement of intracardiac and intravascular devices can be aided by echocardiographic guidance. Likewise, placement of catheters for radiofrequency ablation of arrhythmogenic pathways can be facilitated by TEE when there are intracardiac abnormalities.

Intraoperative echocardiography has been used to provide timely information about the success of septal defect closure and valve palliation.[486-493] In some lesions the ability to scan the heart by TEE or direct transducer placement on the heart surface allows the patient to undergo surgical repair without previous cardiac catheterization.

| **Indications for Transesophageal Echocardiography in Pediatric Patients** | **Class** |
|---|---|

| | |
|---|---|
| **1. Any patient with congenital or acquired heart disease needing echocardiography when significant diagnostic information cannot be obtained by TTE.** | **I** |
| **2. Monitoring and guidance during cardiothoracic procedures when there is a risk for residual shunting, valvular insufficiency, obstruction, or myocardial dysfunction.** | **I** |
| **3. Guidance of catheter/device placement during interventional catheterization/radiofrequency ablation in patients with congenital heart disease.** | **I** |
| **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.** | **I** |
| **5. Patients with long-term placement of intravascular devices in whom thrombus or vegetation is suspected.** | **I** |
| **6. Patients with a prosthetic valve in whom thrombus or vegetation is suspected.** | **I** |
| **7. Any patient with suspected endocarditis and inadequate transthoracic acoustical window.** | **I** |
| **8. Performing TEE in a patient who has not previously had careful study by TTE.** | **III** |
| **9. Patients with structural esophageal abnormality.** | **III** |

## 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. 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] In the coming years such studies are likely to be performed even earlier than 16 to 18 weeks. 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] Patients are referred for specific fetal echo-cardiographic 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] Education of the parents can be initiated early so that complex therapeutic choices can be reviewed and informed consent obtained.[510-512] If the fetal heart appears normal, the family may be reassured.

Diagnostic difficulties may arise because of modulation of the anatomic and physiological presentation of certain lesions by the fetal circulation and dramatic changes in the heart and great vessels that may occur throughout gestation. As an example, 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 reevaluation studies to determine growth of cardiac chambers and vascular structures and changes in blood flow patterns. The spectrum of congenital cardiac lesions is broader than that seen in neonates and infants because of the presence of nonviable subcategories of disease. A knowledge of prenatal maternal history[513] is as necessary as good imaging in providing proper care for these patients. An additional degree of difficulty is imposed by the inability to see the fetus for orientation reference and the inability to examine the fetus for clinical findings that might guide the performance and interpretation of the echocardiogram.

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 .

| Indications for Fetal Echocardiography | Class |
|---|---|
| **1. Abnormal-appearing heart on general fetal ultrasound examination.** | **I** |
| **2. Fetal tachycardia, bradycardia, or persistent irregular rhythm on clinical or screening ultrasound examination.** | **I** |
| **3. Maternal/family risk factors for cardiovascular disease, such as a parent, sibling, or first-degree relative with congenital heart disease.** | **I** |
| **4. Maternal diabetes.** | **I** |
| **5. Maternal systemic lupus erythematosus.** | **I** |
| **6. Teratogen exposure during a vulnerable period.** | **I** |

| | |
|---|---|
| **7. Other fetal system abnormalities (including chromosomal).** | **I** |
| **8. Performance of transplacental therapy or presence of a history of significant but intermittent arrhythmia. Reevaluation examinations are required in these conditions.** | **I** |
| **9. Fetal distress or dysfunction of unclear etiology.** | **IIa** |
| **10. Previous history of multiple fetal losses.** | **IIb** |
| **11. Multiple gestation.** | **IIb** |
| **12. Low-risk pregnancies with normal anatomic findings on ultrasound examination.** | **III** |
| **13. Occasional premature contractions without sustained tachycardia or signs of dysfunction or distress.** | **III** |
| **14. Presence of a noncardiovascular system abnormality when evaluation of the cardiovascular system will not alter either management decisions or fetal outcome.** | **III** |

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# Tables 1 - 11

## Table 1. Doppler Echocardiography Capabilities in the Adult Patient

| | Echocardiography | | | | |
|---|---|---|---|---|---|
| | **M mode** | **2D** | **Spectral Doppler** | **Color Doppler** | **TEE** |
| *Anatomy-Pathology* | | | | | |
| 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 | ++ | ++++ | - | - | ++ |
| *Function* | | | | | |
| 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; TEE, transesophageal echocardiography; MV, mitral valve; LV, left ventricular; EF, ejection fraction; RV, right ventricular; PA, pulmonary artery.

*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.

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

| Study | n | Features Examined | Outcome | *P* value |
|---|---|---|---|---|
| Nishimura[54] | 237 | MV leaflet $\geq$ 5 mm | [up arrow] sum of sudden death, endocarditis, and cerebral embolus | *P* <.02 |
| | | LVID $\geq$ 60 mm | [up arrow] MVR (26% vs 3.1%) | *P* <.001 |
| Zuppiroli[55] | 119 | MV leaflet >5 mm | [up arrow] complex ventricular arrhythmia | *P* <.001 |
| Babuty[56] | 58 | undefined MV thickening | no relation to complex ventricular arrhythmias | NS |
| Takamoto[57] | 142 | MV leaflet $\geq$ 3 mm, redundant, low echocardiographic density | [up arrow] ruptured chordae (48% vs 5%) | |
| Marks[58] | 456 | MV leaflet $\geq$ 5 mm | [up arrow] endocarditis (3.5% vs 0%) | *P* <.02 |
| | | | [up arrow] moderate-severe MR (11.9% vs 0%) | *P* <.001 |
| | | | [up arrow] MVR (6.6% vs 0.7%) | *P* <.02 |
| | | | [up arrow] stroke (7.5% vs 5.8%) | NS |
| Chandraratna[59] | 86 | MV leaflets >5.1 mm | [up arrow] cardiovascular abnormalities (60% vs 6%) | |
| | | | (Marfan syndrome, TVP, MR, dilated ascending aorta) | *P* <.001 |

MV indicates mitral valve; LVID, left ventricular internal diameter; MVR, mitral valve replacement; NS, not significant; MR, mitral regurgitation; TVP, tricuspid valve prolapse.

**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 et al[71] | 41 patients, | 4 (10%) false-negative | No false-negative | Additional veg | Area underestimated in 60% of all veg |
| | 83 veg with IE | | | ● 23% vs monoplane | |
| | 6 abscesses | 3 missed abscesses | 1 missed abscess | ● 9% vs biplane | |
| **Negative Predictive Value of TEE** | | | **Negative Predictive Value** | | |
| Lowry et al[72] | 93 patients undergoing TEE for suspected IE | | NV IE, 100% | | With suspected PV IE, negative TEE does not rule out |
| | | | PV IE, 90% | | |
| **Sensitivity of TTE vs TEE** | | | | | |
| Shapiro et al[61] | 64 patients with suspected IE, prospective study | 34 with veg on either TTE or TEE | *P* =.004 | | |
| | | TTE 24 (70.6%) | | | |
| | | TEE 33 (97.1%) | *P* =.02 | | |
| | | 12 with veg <1 cm | | | |
| | | TTE 5 (41.7%) | | | |
| | | TEE 12 (100%) | | | |
| | | 9 with periannular complications | *P* =.001 | | |
| | | TTE 2 (22.2%) | | | |
| | | TEE 9 (100%) | | | |
| Birmingham et al[73] | 61 patients with suspected IE | Sensitivity for veg | For aortic veg | For mitral veg | |
| | | TTE 30% *P* <.01 | TTE 25% *P* <.01 | TTE 50% *P* <.01 | |
| | 31 (51%) had IE | TEE 88% | TEE 88% | TEE 100% | |
| **Sensitivity of TTE** | | | | | |

| | | | | |
|---|---|---|---|---|
| Watanakunakorn, Burkert[74] | 204 patients | | | |
| | 219 episodes of IE | 2D TTE in 164 episodes | | |
| | 148 host valves | 67 (40.9%) positive for veg | | |
| | 33 IVDU | | | |
| | 2 early PV IE | | | |
| | 27 late PV IE | | | |

## Sensitivity and Specificity TTE vs TEE

| | | | | |
|---|---|---|---|---|
| Shively et al[75] | 66 episodes of suspected IE in 62 patients | TTE 7 of 16 | sensitivity | TTE 44% *P* <.01 TEE 94% |
| | Diagnosis of IE made by clinical picture and lab studies in 16 of 66 episodes | TEE 15 of 16 | specificity | TTE 98% TEE 100% |

## Sensitivity, Specificity, and Predictive Accuracy of TTE

## TTE for Detecting IE

| | | | | |
|---|---|---|---|---|
| Burger et al[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 et al[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% | | AV IE 76% sensitivity |
| | | PV 61% | | 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 |

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

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

| Author | Patient Population | Total Pts | Criteria | Sens (%) | Spec (%) | PPV (%) | NPV (%) | Comments |
|---|---|---|---|---|---|---|---|---|
| Heger[90] 1980 | Consecutive AMI | 44 | SWMA | 100 | - | - | - | Less severe WMA predicts uncomplicated course |
| Visser[100] 1981 | Consecutive AMI | 90 | SWMA | 98 | - | - | - | Area of asynergy vs peak CK-MB, r=.87 |
| Nishimura[99] 1984 | Consecutive AMI | 61 | SWMA | 93 | - | - | - | All false-negative results had non-Q MI |
| Horowitz[83] 1982 | CP, no prior MI | 80 | SWMA | 94 | 84 | 86 | 93 | 19% technically inadequate; ECG ST[up arrow] in 45% of AMI |
| Peels[85] 1990 | CP (in ER), no prior MI | 43 | SWMA | 92 | 53 | 46 | 94 | More extensive WMA imply multivessel CAD |
| Sabia[84] 1991 | Consecutive CP (in ER) | 180 | SWMA | 93 | 60 | 31 | 98 | ECG ST[up arrow] in only 30% with AMI |
| Saeian[101] 1994 | Consecutive CP | 60 | SWMA | 88 | 94 | 91 | 92 | |
| Gibler[102] 1995 | CP (in ER), known CAD or UA excluded | 901 | any WMA | 47 | 99 | 50 | 99 | Both global and regional WMA considered abnormal |

Pts indicates patients; Sens, sensitivity; Spec, specificity; PPV, positive predictive value; NPV, negative predictive value; AMI, acute myocardial infarction, confirmed by myocardial-specific enzyme measurements; SWMA, segmental wall motion abnormalities; CK-MB, creatine kinase-MB; CP, chest pain, possible acute myocardial infarction; ECG, electrocardiogram; ER, patients evaluated in emergency room; CAD, coronary artery disease; UA, unstable angina.

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

| | | | Prediction of Clinical Complications | | | | | |
|---|---|---|---|---|---|---|---|---|
| Author | Patient Population | Total Pts | Criteria | Sens (%) | Spec (%) | PPV (%) | NPV (%) | End Points |
| Horowitz[83] 1982 | Possible AMI; prior MI excluded | 65 | SWMA | 100 | 53 | 28 | 100 | Death, recurrent MI, pump failure, serious dysrhythmias |
| Gibson[91] 1982 | Consecutive AMI | 68 | Remote asynergy | 79 | 61 | 34 | 92 | For development of cardiogenic shock |
| Horowitz[110] 1982 | Proved AMI | 43 | WM score >7 | 85 | 17 | 69 | 93 | Death, serious dysrhythmias, pump failure, severe heart failure |
| Nishimura[99] 1984 | Consecutive AMI | 61 | WM score index >2 | 80 | 90 | 89 | 82 | Death, serious dysrhythmias, pump failure |
| Jaarsma[98] 1988 | AMI Killip 1 or 2 | 77 | WM score >7 | 88 | 57 | 35 | 95 | For development of severe heart failure |
| Sabia[84] 1991 | Consecutive AMI | 30 | SWMA | 100 | 12 | 46 | 100 | Shock, serious dysrhythmias, angina leading to revascularization |
| Sabia[113] 1991 | Consecutive CP (in ER) | 171 | LV systolic dysfunction | 94 | 45 | 28 | 97 | *Early* nonfatal MI, serious dysrhythmias |
| Sabia[113] 1991 | Consecutive CP (in ER) | 171 | LV systolic dysfunction | 83 | 42 | 24 | 94 | *Late* nonfatal MI, death, revascularization |

Pts indicates patients; Sens, sensitivity; Spec, specificity; PPV, positive predictive value; NPV, negative predictive value; AMI, acute myocardial infarction; SWMA, segmental wall motion abnormalities; WM, wall motion; CP, chest pain, possible acute myocardial infarction; ER, patients evaluated in emergency room; LV, left venticular.

**Table 6. Evaluation of Myocardial Viability by Stress Echocardiography**

| Author | Patient Population | Test | Total Pts | Sens (%) | Spec (%) | PPV (%) | NPV (%) | Comments |
|---|---|---|---|---|---|---|---|---|
| **Early After Acute Myocardial Infarction (Stunning)** | | | | | | | | |
| Pierard[137] 1990 | AMI, postlysis | DSE[rightarrow] f/u TTE | 17 | 100 | 73 | 67 | 100 | Results similar to PET viability |
| Barilla[138] 1991 | Selected AMI, postlysis | DSE[rightarrow] f/u TTE | 21 | 100 | - | 100 | | No negative DSE results |
| Smart[136] 1993 | AMI, postlysis | DSE[rightarrow] f/u TTE | 51 | 86 | 90 | 86 | 90 | Responsive at low dose; biphasic results indicate residual stenosis |
| **Chronic Ischemic Dysfunction (Hibernation)** | | | | | | | | |
| Cigarroa[195] 1993 | Chronic MVD, LV dys | DSE[rightarrow] CABG[rightarrow] f/u TTE | 25 | 82 | 86 | 82 | 86 | Contractile reserve in 40% of akinetic segments |
| La Canna[197] 1994 | Chronic CAD, LV dys | DSE[rightarrow] CABG[rightarrow] f/u TTE | 33 | 87 | 82 | 90 | 77 | Analyzed by segment, not by pt |
| Afridi[196] 1995 | Chronic CAD, LV dys | DSE[rightarrow] PTCA[rightarrow] f/u TTE | 20 | 80 | 90 | 89 | 82 | Biphasic response=hibernating myocardium with residual stenosis |
| Perrone-Filardi[199] 1995 | Chronic CAD, LV dys | DSE[rightarrow] revasc[rightarrow] f/u TTE | 18 | 88 | 87 | 91 | 82 | CABG in 7, PTCA in 11; lower DSE sens for viability in akinetic vs hypokinetic segments |
| Arnese[198] 1995 | Chronic CAD, LV dys | DSE[rightarrow] CABG[rightarrow] f/u TTE | 38 | 74 | 96 | 85 | 93 | Thallium viability in 71 of 137 DSE-negative segments; postoperative improvement in only 10 of 71 |
| deFilippi[200] 1995 | Chronic CAD, LV dys | DSE[rightarrow] revasc[rightarrow] f/u TTE | 23 | 97 | 73 | 85 | 94 | Contractile reserve (DSE) predicts recovery better than perfusion (contrast echo) |

Pts indicates patients; Sens, sensitivity; Spec, specificity; PPV, likelihood of subsequent improvement in patients with evidence of viability; NPV, likelihood of lack of improvement in patients without viability; AMI, acute myocardial infarction; lysis, thrombolytic therapy; DSE, dobutamine stress echocardiography; f/u TTE, follow-up transthoracic echocardiography; PET, positron emission tomography; MVD, multivessel disease; LV dys, left ventricular dysfunction; CABG, coronary artery bypass graft; CAD, coronary artery disease; PTCA, percutaneous transluminal coronary angioplasty; revasc, revascularization.

**Table 7. Detection of Angiographically Documented Coronary Artery Disease by Stress Echocardiography: Exercise**

| Author | Total Pts | Sens (%) | Sens 1-VD (%) | Sens MVD (%) | Spec (%) | PPV (%) | NPV (%) | Comments |
|---|---|---|---|---|---|---|---|---|
| Armstrong[154] 1987 | 123 | 88 | 81 | 93 | 86 | 97 | 61 | TME, ≥ 50% stenosis; sens 98% if prior MI, 78% if no MI |
| Ryan[155] 1988 | 64 | 78 | 76 | 80 | 100 | 100 | 73 | TME, >50% stenosis |
| Sawada[152] 1989 | 57 | 86 | 88 | 82 | 86 | 86 | 86 | TME or UBE, ≥ 50% stenosis, all women |
| Labovitz[156] 1989 | 56 | 76 | - | - | 100 | 100 | 74 | TME; sens 61% if rest WMA excluded |
| Crouse[157] 1991 | 228 | 97 | 92 | 100 | 64 | 90 | 87 | TME, >50% stenosis |
| Pozzoli[158] 1991 | 75 | 71 | 61 | 94 | 96 | 97 | 64 | UBE, ≥ 50% stenosis |
| Galanti[159] 1991 | 53 | 93 | 93 | 92 | 96 | 96 | 93 | UBE, >70% stenosis |
| Marwick[160] 1992 | 150 | 84 | 79 | 96 | 86 | 95 | 63 | TME, >50% stenosis; normalcy rate=93% |
| Quinones[161] 1992 | 112 | 74 | 59 | 89 | 88 | 96 | 55 | TME, >50% stenosis |
| Salustri[162] 1992 | 44 | 87 | 87 | - | 85 | 93 | 75 | BE, >50% stenosis, all 1-VD |
| Amanullah[163] 1992 | 27 | 82 | - | - | 80 | 95 | 50 | UBE, >50% stenosis, unstable angina |
| Ryan[164] 1993 | 309 | 91 | 86 | 95 | 78 | 90 | 81 | UBE, ≥ 50% stenosis |
| Mertes[165] 1993 | 79 | 83 | - | - | 85 | 91 | 75 | SBE, >50% stenosis, after PTCA |
| Hoffman[166] 1993 | 66 | 80 | 79 | 81 | 88 | 95 | 58 | SBE, >50% stenosis |
| Cohen[167] 1993 | 52 | 78 | 63 | 90 | 87 | 94 | 48 | SBE, >70% stenosis |
| Hecht[168] 1993 | 180 | 93 | 84 | 100 | 86 | 96 | 79 | SBE, ≥ 50% stenosis |
| Roger[169] 1994 | 150 | 91 | - | 73 | - | - | - | TME, ≥ 50% stenosis |
| Marangelli[153] 1994 | 80 | 89 | 76 | 97 | 91 | 93 | 86 | TME, ≥ 70% stenosis |
| Beleslin[183] 1994 | 136 | 88 | 88 | 91 | 82 | 97 | 50 | TME, ≥ 50% stenosis |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Roger[170] 1995 | 127 | 88 | - | - | 72 | 93 | 60 | TME, ≥ 50% stenosis |
| Dagianti[184] 1995 | 60 | 76 | 70 | 80 | 94 | 90 | 85 | SBE, ≥ 50% stenosis |

Pts indicates patients; Sens, sensitivity; 1-VD, test results positive in patients with one-vessel disease; MVD, test results positive in patients with multivessel disease; Spec, specificity; PPV, positive predictive value; NPV, negative predictive value; TME, treadmill exercise; MI, myocardial infarction; >50%/70% stenosis, >50%/70% coronary luminal diameter narrowing as "significant" CAD; UBE, upright bicycle ergometry; WMA, wall motion abnormalities; BE, bicycle ergometry; SBE, supine bicycle ergometry; PTCA, percutaneous transluminal coronary angioplasty.

**Table 8. Detection of Angiographically Documented Coronary Artery Disease by Stress Echocardiography: Dobutamine**

| Author | Total Pts | Sens (%) | Sens 1-VD (%) | Sens MVD (%) | Spec (%) | PPV (%) | NPV (%) | Comments |
|---|---|---|---|---|---|---|---|---|
| Berthe[171] 1986 | 30 | 85 | - | 85 | 88 | 85 | 88 | Pts with MVD after AMI |
| Previtali[173] 1991 | 35 | 68 | 50 | 92 | 100 | 100 | 44 | 1 with prior MI; 70% stenosis |
| Sawada[174] 1991 | 55 | 89 | 81 | 100 | 85 | 91 | 81 | All with normal WM at rest; >50% stenosis |
| Sawada[174] 1991 | 41 | 81 | - | - | 87 | 91 | 72 | New WMA with abnormal WM at rest; >50% stenosis |
| Cohen[175] 1991 | 70 | 86 | 69 | 94 | 95 | 98 | 72 | 19 with prior MI; >70% stenosis |
| Salustri[172] 1992 | 46 | 79 | - | - | 78 | 85 | 70 | 15 with prior MI |
| Mazeika[176] 1992 | 50 | 67 | 50 | 75 | 93 | 96 | 52 | 13 with prior MI; >70% stenosis |
| Marcovitz[177] 1992 | 141 | 96 | 95 | 98 | 66 | 91 | 84 | 15 with prior MI; >50% stenosis |
| McNeill[151] 1992 | 80 | 70 | - | - | 88 | 89 | 67 | >70% stenosis |
| Segar[178] 1992 | 85 | 95 | - | - | 82 | 94 | 86 | >50% stenosis |
| Marwick[150] 1993 | 97 | 85 | 84 | 86 | 82 | 88 | 78 | No prior MI; >50% stenosis |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Marwick[179] 1993 | 217 | 72 | 66 | 77 | 83 | 89 | 61 | >50% stenosis |
| Hoffmann[166] 1993 | 64 | 79 | 78 | 81 | 81 | 93 | 57 | >50% stenosis |
| Forster[180] 1993 | 21 | 75 | 25 | 100 | 89 | 90 | 72 | >50% stenosis |
| Cohen[167] 1993 | 52 | 86 | 75 | 95 | 87 | 94 | 72 | >70% stenosis |
| Günalp[181] 1993 | 27 | 83 | 78 | 89 | 89 | 94 | 73 | No prior MI, >50% stenosis |
| Beleslin[183] 1994 | 136 | 82 | 82 | 82 | 77 | 96 | 38 | ≥ 50% stenosis |
| Pellikka[182] 1995 | 67 | 97 | - | - | 65 | - | - | >50% stenosis |
| Dagianti[184] 1995 | 60 | 72 | 60 | 80 | 97 | 95 | 83 | All with normal WM at rest; >50% stenosis |

Pts indicates patients; Sens, sensitivity; 1-VD, test results positive in patients with one-vessel disease; MVD, test results positive in patients with multivessel disease; Spec, specificity; PPV, positive predictive value; NPV, negative predictive value; AMI, acute myocardial infarction; WM, wall motion; >50%/70% stenosis, >50%/70% coronary luminal diameter narrowing as "significant" coronary artery disease; WMA, wall motion abnormality.

**Table 9. Prognostic Value of Stress Echocardiography**

| | | | | | Annualized Event Rate (%) | | | |
|---|---|---|---|---|---|---|---|---|
| **Author** | **Test** | **Patient Population** | **Total Pts** | **Avg f/u (mo)** | **Ischemic** | **Nonisch** | **NL** | **Comments** |
| **Early After Acute Myocardial Infarction** | | | | | | | | |
| Applegate[144] 1987 | TME | Post MI | 67 | 11 | 55 | 14 | | Death, MI, revascularization |
| Bolognese[145] 1992 | DIP | Post MI | 217 | 24 | 16 | 5 | | Death, MI, unstable angina; same for silent vs symptomatic ischemia |
| Picano[141] 1995 | DIP | Post MI | 1080 | 14 | 5.4 | 2.8 | | MI only event considered; fatal MI, 5 vs 0 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Quintana[146] 1995 | SBE | Post MI | 70 | 36 | 27 | 9 | | Death, MI, revascularization |
| **Chronic Ischemic Heart Disease** | | | | | | | | |
| Sawada[185] 1990 | TME | nl TME | 148 | 28 | - | - | 2 | Pretest probability of CAD, 30%; ECG isch, 19%; nondiagnostic, 28% |
| Krivokapich[186] 1993 | TME | CP, CAD | 360 | 12 | 34 | 9 | - | 59% normal |
| Mazeika[187] 1993 | DSE | CAD | 51 | 24 | 16 | 4 | | Pretest probability of CAD, 80%; revascularization excluded as a "soft" event |
| Afridi[196] 1994 | DSE | CP, CAD | 77 | 10 | 60 | 16 | 6 | CHF included as an event |
| Severi[188] 1994 | DIP | CP, no MI | 429 | 38 | 11 | 3 | | Worse prognosis if worse wall motion |
| Coletta[189] 1995 | DIP | CP, CAD | 268 | 16 | 18 | 1.4 | | Death or MI |
| Kamaran[192] 1995 | DSE | CP, CAD | 210 | 8 | 69 | 1 | | Cardiac death, MI considered "hard" events |
| Williams[190] 1996 | DSE | CAD, EF<40% | 108 | 16 | 33%* | 7% | | *Viable or ischemic |
| Marcovitz[193] 1996 | DSE | CAD | 272 | 15 | 12.8 | 4.5 | 1 | Cardiac death, MI considered "hard" events |

Pts indicates patients; f/u, follow-up; Nonisch, nonischemic; NL, no chest pain; TME, treadmill echocardiogram; MI, myocardial infarction; DIP, dipyridamole echocardiogram; SBE, supine bicycle ergometry; CAD, known or suspected coronary artery disease; DSE, dobutamine stress echocardiogram; ECG, electrocardiogram; CP, chest pain (suspected coronary artery disease); CHF, congestive heart failure; EF, ejection fraction.

Events include cardiac death, myocardial infarction, revascularization (in some series), and unstable angina requiring hospitalization (in some series).

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

| Event Patients* n=Population | (n) | % | Range (%)† | Control Patients* n=Population | (n) | % | Range (%)† |
|---|---|---|---|---|---|---|---|

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 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.

ºRange 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 |

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

*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.

# Tables 12 - 22

## 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.

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

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

TTE indicates transthoracic echocardiography; TEE, transesophageal 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.

## 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 |

PFO indicates patent foramen ovale; CVA/TIA, patients with documented cerebrovascular accident or transient ishemic 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 303, 307, 314, 317, 318.

## 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.

## 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.

## Table 18. Echocardiographic Predictors for Outcome of Elective Cardioversion

| Author | (n) | Study Design | Patient Group | Method | Parameters Evaluated | Results | Comments |
|---|---|---|---|---|---|---|---|

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Dittrich[330] | 85 | Retrospective | Variety of etiologies and coexisting cardiac problems Most pts in AF >5 y | M-mode and 2D | *M-mode*<br>LV diam, LVH (>11 mm), LVEDD, LVFS%<br><br>*2D*<br>LA long axis, RA long axis, LA area ($E_s$), RA area ($E_s$) | *Initial success*<br>No echo parameter predictive<br><br>*1-mo maintenance of NSR*<br>LA diam (M-mode) and RA long axis not predictive<br><br>LA long axis and LAA larger in pts staying in NSR (but great overlap)<br><br>*6-mo maintenance of NSR*<br>No echo parameter predictive | |
| Flugelman[336] | 40 | Retrospective | All had chronic AF, postoperative mitral valvotomy or MVR for rheumatic MS | 2D | LA diam LVFS% LV diam RV diam | *Success*<br>NSR at 3 mo<br><br>LA size larger in those with failure (*P* =.03) | No breakdown separating pts who initially failed conversion versus those who reverted within 3 mo. No useful cutoff was suggested or evaluated. |
| Dethy[331] | 50 | Prospective | All maintained NSR >24 h | 2D, M-mode | *M-mode*<br>LA diam<br><br>*Doppler parameters* based on E and A waves | *Maintenance of NSR at 6 mo*<br>LA diam >45<br>Sens=59%<br>Spec=44% PPV 66<br>LA diam>50<br>Sens=38% Spec=6%<br>PPV=63%<br><br>*Success vs failure*<br>LA size ns<br>A wave at 24 h<br>*P* =.12<br>Increase in A wave from 4-24 h<br>*P* =.003 | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Henry[333] | 37 | Retrospective | Pts had MV diagnosis or ASH. Many had multiple attempts at cardioversion. No pt had LA <45. | M-mode | LA diam | Only 25% maintained NSR to 6 mo<br><br>10% of pts with LA diam >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 <45. No information on initial success. Group not typical of usual mix of pts with atrial fibrillation. |
| Ewy[334] | 74 | Retrospective | Rheumatic heart disease, idiopathic AF | M-mode | LA diam | No pt with LA >6 cm and rheumatic heart disease or >5 in pts with idiopathic AF successfully converted, but 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[332] | 26 | Prospective | Rheumatic heart disease, idiopathic AF | M-mode | LA diam | *Success*<br>Maintenance of NSR for 1 mo LA diam smaller in pts with success vs failure *P* =.001. No pt with recurrence had LA <4.5. Success in 1 pt with LA >4.5. | Small number, but 4.5 appears to be a good cutoff in this pt group. |
| Halpern[335] | 21 | Prospective | Mixed group re coexisting heart disease. Cardioversion attempted only with procainamide. | M-mode | LA diam | *Initial success*<br>LA smaller in converters *P* <.005 All converters had LA <4 Only 1 nonconverter had LA <4 | Small number |

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

## 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° scoliosis
- Pectus carinatum or pectus excavatum requiring surgery
- Pes planus (demand displacent 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]

## Table 20. Echocardiographic Screening for Athletes

| Author | (n) | Methods | Results | Study Conclusion |
|---|---|---|---|---|
| Maron[357] | 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<br>3 mild septal hypertrophy (HCM vs athlete's heart?)<br>0 Marfan<br>0 definite HCM<br>0 any other CVD with risk for death or disease progression with athletic competition | Echocardiographic screening not justified |
| Lewis[356] | 265 (all but 3 black) | 265 collegiate athletes 2D and M-mode echocardiography | 30 (11%) MVP<br>1 small ASD<br>29 (11%) septal hypertrophy (HCM vs athlete's heart?) | Echocardiographic screening not justified |

| | | | | |
|---|---|---|---|---|
| Weidenbener[358] | 2997 | Limited: parasternal 2D long and short axis, estimated cost <$14 | 2% abnormal (64)<br>40 MVP<br>10 bicuspid AV<br>4 aortic dilatation<br>2 ventricular septal defect<br>2 dilated CS<br>1 AI<br>1 ASD<br>1 RV mass<br>1 septal hypertrophy | Can be performed inexpensively; however, "uncovered no abnormalities that precluded participation in athletic events," although "some conditions allowed only limited participation." |

ECG indicates electrocardiogram; 2D, two-dimensional; MVP, mitral valve prolapse; HCM, hypertrophic cardiomyopathy; CVD, cardiovascular disease; ASD, atrial septal defect; AV, aortic valve; CS, coronary sinus; AI, aortic insufficiency; RV, right ventricular.

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

| Author | 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%<br>Source of embolism 26%<br>Complication of CAD 10%<br>Miscellaneous 19%<br>Ventricular function 8%<br>Infective endocarditis 8% |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Oh[364] | 1990 retrospective | 51 | All | Suboptimal TTE reason for TEE | 59% | Multiple | 25--hemodynamic instability 49%<br><br>No. of pts<br>Infective endocarditis 10<br>Cardiac contusion 10<br>Heart donor 5<br>Aortic dissection, source of embolus, LV function, chest pain, other 10 |
| Font[365] | 1991 retrospective | 112 | All | 68% fair to poor image | *By TEE*<br>131 new lesions compared with 95 for TTE:<br>TTE/TEE=73% overall<br>equal for tamponade<br>detailed analysis of TTE/TEE frequency for each diagnosis | Multiple 56% postoperative cardiac or noncardiac surgery | RO vegetations 46%<br>RO valve dysfunction 43%<br>Assess ventricular function 19%<br>Source of embolus 12%<br>RO dissection 9%<br><br>Other (congenital heart disease, tamponade, LVOT obstruction, constriction) 14% |
| Foster[366] | 1992 retrospective | 83 | 34% had TTE | TEE with Doppler not available<br>TTE with Doppler complementary | 25 "new findings" in TEE pts who had TTE<br>19% cardiac surgery prompted by TEE | | Endocarditis 43%<br>Embolic source 14%<br>Mitral regurgitation 11%<br><br>Hypotension 11%<br>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<br><br>48 pts<br><br>Emergency department 32 pts | Aortic dissection 27<br><br>Hemodynamic instability 20 Embolic source 9<br><br>Evaluation of MR 7 Endocarditis 3 Other remainder |
| Khoury[368] | 1994 retrospective | 77 | All | Technically poor or inconclusive in 77 (100%), thus reason for TEE | Same<br><br>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<br><br>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%<br><br>In 59% a cause was found, resulting in urgent surgery in 21% | Multiple | Hemodynamic instability and inadequate TTE<br>No. of pts<br>53 global or regional LV function (34%)<br>31 severe valvular disease (20%)<br>22 endocarditis (14%)<br>12 suspected aortic dissection (8%)<br>11 shunt lesions (7%)<br>7 mass lesions (4%)<br>30 had 2 reasons |

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

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

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
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
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
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 h of placement
5. Aortic stenosis
9. Pulmonary atresia (Rastelli procedure)
   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).
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
11. Transposition of the great vessels
    Senning and Mustard procedure
    Sinus and atrioventricular nodal dysfunction
    Right ventricular (systemic ventricle) failure

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

6. Pulmonic valve stenosis
   Residual pulmonic valve stenosis
   Pulmonic valve regurgitation
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)
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)
   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
   Supravalvar pulmonic stenosis
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
13. Ebstein disease
   Tricuspid regurgitation, progressive
   Supraventricular tachycardias,
   Wolff-Parkinson-White syndrome
   Residual atrial shunts
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

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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.

# References


1. Fryback DG, Thornbury JR. The efficacy of diagnostic imaging. *Med Decis Making*. 1991;11:88-94.
2. 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.
3. Douglas PS. Justifying echocardiography: the role of outcomes research in evaluating a diagnostic test. *J Am Soc Echocardiogr.* 1996;9:577-81.
4. Eagle KA, Brundage BH, Chaitman BR, et al. ACC/AHA guidelines for perioperative cardiovascular evaluation for noncardiac surgery. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). *J Am Coll Cardiol.* 1996;27(4):910-48.
5. Cunnion RE, Natanson C. Echocardiography, pulmonary artery catheterization, and radionuclide cineangiography in septic shock. *Intensive Care Med.* 1994;20:535-7.
6. 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.
7. Smith MD. Evaluation of valvular regurgitation by Doppler echocardiography. *Cardiol Clin.* 1991;9:193-228.
8. Richards KL. Doppler echocardiography in the diagnosis and quantification of valvular heart disease. *Mod Concepts Cardiovasc Dis.* 1987;56:43-8.
9. 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.
10. Kilner PJ, Manzara CC, Mohiaddin RH, et al. Magnetic resonance jet velocity mapping in mitral and aortic valve stenosis. *Circulation.* 1993;87:1239-48.
11. 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.
12. Auerback ML. High tech in a low tech country. *West J Med.* 1993;159:93-4.
13. Mishra M, Chambers JB, Jackson G. Murmurs in pregnancy: an audit of echocardiography. *BMJ.* 1992;304:1413-4.
14. Northcote RJ, Knight PV, Ballantyne D. Systolic murmurs in pregnancy: value of echocardiographic assessment. *Clin Cardiol.* 1985;8:327-8.
15. 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.
16. Kumar R, Sinha N, Ahuja RC, Saran RK, Dwivedi SK, Suri A. Etiology of isolated mitral regurgitation: a clinico-echocardiographic study. *Indian Heart J*. 1993;45:173-8.
17. Xu M, McHaffie DJ. Nonspecific systolic murmurs: an audit of the clinical value of echocardiography. *N Z Med J* . 1993;106:54-6.
18. McKillop GM, Stewart DA, Burns JM, Ballantyne D. Doppler echocardiography in elderly patients with ejection systolic murmurs. *Postgrad Med J*. 1991;67:1059-61.
19. Smythe JF, Teixeira OH, Vlad P, Demers PP, Feldman W. Initial evaluation of heart murmurs: are laboratory tests necessary? *Pediatrics*. 1990;86:497-500.
20. 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.

21. Stamm RB, Martin RP. Quantification of pressure gradients across stenotic valves by Doppler ultrasound. *J Am Coll Cardiol.* 1983;2:707-18.
22. Hatle L, Angelsen B, Tromsdal A. Noninvasive assessment of atrioventricular pressure half-time by Doppler ultrasound. *Circulation.* 1979;60:1096-104.
23. 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.
24. Cheriex 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.
25. 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.
26. 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.
27. 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.
28. Perez JE, Ludbrook PA, Ahumada GG. Usefulness of Doppler echocardiography in detecting tricuspid valve stenosis. *Am J Cardiol.* 1985;55:601-3.
29. Otto CM, Pearlman AS. Doppler echocardiography in adults with symptomatic aortic stenosis. Diagnostic utility and cost-effectiveness. *Arch Intern Med.* 1988;148:2553-60.
30. 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.
31. 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.
32. 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.
33. 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.
34. Sahn DJ, Maciel BC. Physiological valvular regurgitation. Doppler echocardiography and the potential for iatrogenic heart disease. *Circulation.* 1988;78:1075-7.
35. Yoshida K, Yoshikawa J, Shakudo M, et al. Color Doppler evaluation of valvular regurgitation in normal subjects. *Circulation.* 1988;78:840-7.
36. Rahko PS. Prevalence of regurgitant murmurs in patients with valvular regurgitation detected by Doppler echocardiography. *Ann Intern Med.* 1989;111:466-72.
37. 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.
38. 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.

39. 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.
40. Grayburn PA, Fehske W, Omran H, Brickner ME, Luderitz B. Multiplane 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, Görge 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;93:730-36.
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 multiplane 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 echocardiology in the diagnosis of infective endocarditis [corrected] [published erratum appears in *Angiology* . 1991 Sep;42(9):765]. *Angiology*. 1991;42(7):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-V14.
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-I-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 Cardiogr.* 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 myocardial 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, Gheorghiade 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, Nemec 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, Mahmarian 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 in coronary artery disease. *Circulation.* 1991;83(suppl III):III-27-III-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 isonitrile 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 201Tl 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 doxorubicine 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, Wohlgelernter 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 I):I-303-I-310.

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. Chan RK, Johns JA, Calafiore P. Clinical implications of the morphological features of central pulmonary artery thromboemboli shown by transoesophageal echocardiography. *Br Heart J.* 1994;72:58-62.
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, Kligfield P, Hammond IW, Liebson PR, Campo E, Alonso DR, Laragh JH. Cost-effectiveness of echocardiography and electrocardiography for detection of left ventricular hypertrophy in patients with systemic hypertension. *Hypertension.* 1987;9(pt 2):II-69-II-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-II-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 ventrical 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, Grogin 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. Transoesophageal 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 echocardiography 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, Ellrodt 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(6):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. Weinstein JM, Meis CM, Balady GJ, Plehn JF. Role of echocardiography in the evaluation of syncope etiology. *Clin Res* . 1988;36:328A. Abstract.
344. Recchia D. Echocardiography has a low diagnostic yield in the evaluation of syncope of unclear etiology. *J Am Coll Cardiol.* 1994;(February)435A. Abstract.
345. Dhatreecharan S, Azar P, Werner MS, Homma S, Gillam LD. Diagnostic yield of echocardiography in patients with syncope--is current use justified? *J Am Coll Cardiol.* 1994;(February)307A. Abstract.
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 Paepe 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 cardiac 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* . 1994;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 Sep;76(3):609]. *Circulation.* 1987;75:1117-23.
431. Borow KM, Colan SD, Neumann A. Altered left ventricular mechanics in patients with valvular aortic stenosis and coarction 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;2069-73.
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. Roberson DA, Muhiudeen IA, Silverman NH, Turley K, Haas GS, Cahalan MK. Intraoperative transesophageal echocardiography of atrioventricular septal defect. *J Am Coll Cardiol.* 1991;18:537-45.
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-III-116.
493. Sahn DJ. Intraoperative applications of two-dimensional and contrast two-dimensional echocardiography for evaluation of congenital, acquired and coronary heart disease in open-chested humans during cardiac surgery. In: Rijsterborgh H, ed. *Echocardiology.* The Hague, Netherlands: Martinus Nijhoff; 1981:9-23.
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 screening 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 JI, 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.

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