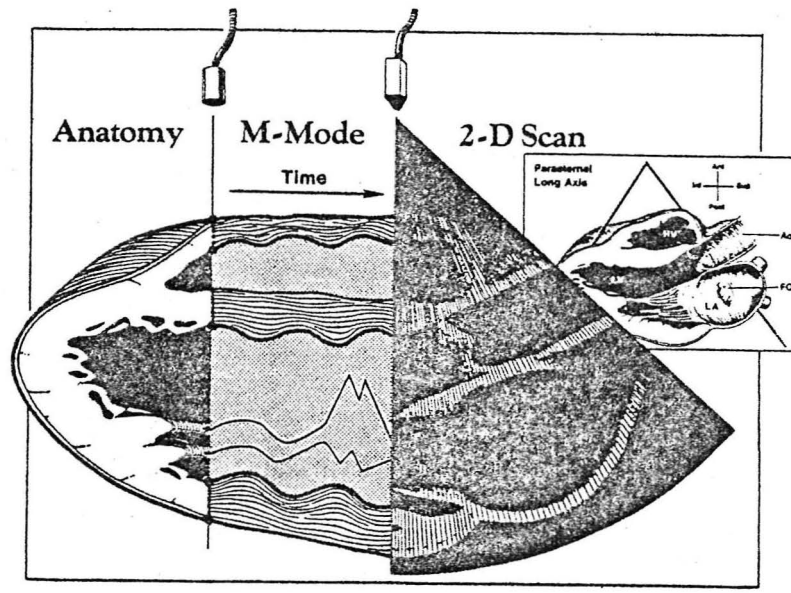
MEDICAL GRAND ROUNDS

June 30, 1983



A Mini-Course in Two-Dimensional Echocardiography

F. Andrew Gaffney, M.D.

Table of Contents

I INTRODUCTION

II HOW IMAGES ARE PRODUCED
- A. *General Ultrasound Principles*
- B. *Mechanical Scanners*
- C. *Phased Array Scanners*
- D. *Signal Processing*

III THE NORMAL EXAMINATION
- A. *Parasternal - Long Axis*
- B. *Parasternal - Short Axis*
- C. *Apical 4-Chamber*

IV CARDIOMYOPATHIES
- A. *Congestive Cardiomyopathies*
  - 1. Idiopathic congestive cardiomyopathy
- B. *Restrictive Cardiomyopathies*
- C. *Hypertrophic Cardiomyopathies*
  - 1. Symmetrical hypertrophy
  - 2. Asymmetrical hypertrophy
  - 3. Obstructive hypertrophy

V RIGHT HEART DISEASE
- A. *Right Heart Dilation - Pressure Versus Volume?*
- B. *Right Heart Valvular Disease*
  - 1. Tricuspid regurgitation
  - 2. Malignant carcinoid syndrome
- C. *Pulmonary Embolism*
- D. *Arrhythmogenic RV Dysplasia (Uhl's)*

VI VALVULAR HEART DISEASE
- A. *Rheumatic Heart Disease*
  - 1. The mitral valve
  - 2. The aortic valve
  - 3. Tricuspid valve
- B. *Infective Endocarditis*
  - 1. Prosthetic valve endocarditis
  - 2. Indications in bacteremia
- C. *Mitral Valve Prolapse*
- D. *Bicuspid Aortic Valve*

VII MITRAL ANNULAR CALCIFICATION

VIII CORONARY ARTERY DISEASE
- A. *Detection of Coronary Artery Disease*
  - 1. Left main coronary disease
  - 2. Kawasaki's disease
- B. *Detection of Wall Motion Abnormalities*
- C. *Complications of Coronary Artery Disease*
  - 1. Ischemic congestive heart failure
  - 2. Left ventricular thrombus
  - 3. Left ventricular aneurysms
  - 4. Left ventricular pseudoaneurysms
  - 5. Ventricular septal defects
  - 6. Right ventricular infarctions

IX PERICARDIAL DISEASE

X CONTRAST ECHOCARDIOGRAPHY

XI SUMMARY

I INTRODUCTION

A decade ago, the internist who could interpret electrocardiograms and chest X-rays was considered to have mastered cardiology's diagnostic technology. However, the introduction of a wide array of radiographic, scintigraphic and sonographic techniques has sometimes left the internist in a consumer's role with an almost total dependence on the cardiologist's and radiologist's interpretation of these newer diagnostic tests. In the case of M-Mode echocardiography, the lack of involvement by the internist is somewhat understandable. The images have only an indirect relationship to cardiac anatomy and physiology, so transfer of prior knowledge is not facilitated.

In 1976, two-dimensional echocardiography was introduced commercially in the U.S. and is currently available in almost every medium- or large-sized hospital. Two-dimensional echo offers substantial advantages over M-Mode echocardiography, both in terms of informational content as well as ease of interpretation (Popp et al., 1980). The latest generation of equipment provides real-time, high resolution images which are "anatomically correct" and are easily understood by the physician with a basic knowledge of cardiac anatomy and physiology. The test offers an excellent opportunity for the internist to "see" the patient's heart and make pathophysiological correlations with the history, physical examination and other diagnostic tests (Kotler et al., 1980; Kisslo, 1980). In addition, greater understanding on the part of the internist should produce more appropriate utilization of this non-invasive but expensive procedure (Reichek, 1982). The following pages are intended to provide the internist with a basic understanding of two-dimensional echocardiography, so that he or she will be able to order the test more appropriately and interpret its findings.

II HOW IMAGES ARE PRODUCED

A. *General Ultrasound Principles*

In simple terms, the echocardiography unit sends high frequency (1-5 MHz) sound into the body. As the sound strikes various cardiac structures, a portion is reflected or echoed back to the receiver. Its speed in the body is known (1560 m/sec), so the time it takes for sound to travel to a particular cardiac structure and return to the receiver allows determination of the structure's distance from the transmitter. To avoid overlap of returning and outgoing echoes, the sound waves are transmitted for only a few millionths of a second (microseconds). The next burst of sound is not transmitted until the echoes from the previous burst have been received.

Echocardiographs use the same device, the transducer, both to send and receive ultrasound. The transducer contains one or more crystals composed of a man-made ceramic material with piezo-electric properties (Figure 1).

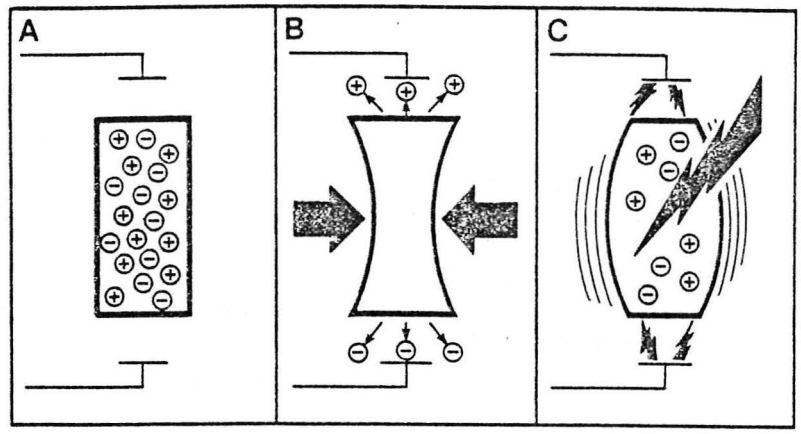
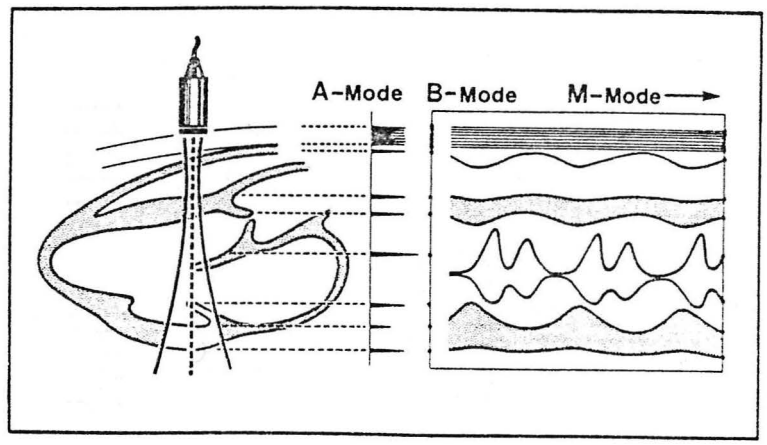


Fig. 1 Diagram illustrates the principles of piezoelectricity. *A*, A quiescent rectangular polar crystal is positioned between two electrodes. *B*, An external stress is applied, thereby deforming the crystal. This mechanical stress causes electrical charges bound within the crystal to shift to the surface, where they can be measured as a voltage. *C*, An electrical current is applied to the crystal. The interaction of this external electrical field with the charges in the ionic lattice of the crystal alters the crystal's shape. When the electric field is rapidly alternated, the crystal is thrown into a correspondingly rapid vibration, which produces a sound wave of like frequency.

If the crystal is excited by placing a voltage across it, vibration occurs and sound is created. Likewise, when the crystal is vibrated, electrons are moved and a voltage potential is created in direct relationship to the amplitude and frequency of the sound applied. The device changes sound to electricity and electricity to sound, hence its name "transducer." ("Magicube" flash cubes use a similar piezo-electric crystal material to generate the current needed to ignite the flash material in the cube.)

M-Mode (M = Motion) echocardiographs have a single crystal transducer which repeats its transmit-receive cycle about 1,000 times per second (1 KHz). If one is imaging a moving cardiac structure, its motion will be small relative to the interval of sampling by the M-Mode ultrasound beam, so a fairly accurate, high resolution representation of motion can be obtained. The motion is displayed "across time" by means of a strip chart recorder (Figure 2). The M-Mode provides information about where a given structure was at a given moment.



Two-dimensional echocardiographs provide information about cardiac structures and their motion in real-time, that is, as the action actually occurs. Unlike M-Mode which uses a single beam of sound and gives an "ice pick" or "soda straw" view of the heart, 2-D uses multiple beams of sound transmitted, more or less simultaneously, so a cross-sectional or two-dimensional image of the heart is created. There are two types of devices for transmitting multiple sound waves to obtain 2-D images - mechanical and phased array scanners.

B. *Mechanical Scanners*

Mechanical scanners have 1-4 crystals in the transducer. The crystals are mechanically moved in a circle or in an arc so that sound is transmitted through a 60-90° sector. The time required for the echoes to return to the transducer is translated by the instrument as a specific distance while directional information permits lateral localization of structures. The echoes from the 90-120 beams of sound (lines) are integrated and organized by a microprocessor and displayed on a television screen. Transmitting and receiving discrete beams through a 60-90° sector takes substantially longer than pulsing a single M-Mode beam (1 versus 120 individual beams needed for an image) so 2-D images are created about 30 times/sec rather than the 1,000/sec with M-Mode. This causes a substantial decrease in resolution (consider fast moving action recorded with a movie camera at 30 frames/sec versus 1,000 frames/sec). If the structure to be imaged is more than about 16 cm from the transducer, the "frame rate" is decreased even further and resolution decreases. Mechanical sector scanners give good images and are substantially less expensive than phased array units. However, because the transducer motion must be stopped to record M-Mode tracings, simultaneous M-Mode and 2-D cannot be done with mechanical scanners. For the same reasons simultaneous 2-D and doppler cannot be obtained with mechanical systems.

C. *Phased Array Scanners*

Phased array scanners use a completely different approach to direct sound beams throughout a sector to create an image (Figure 3).

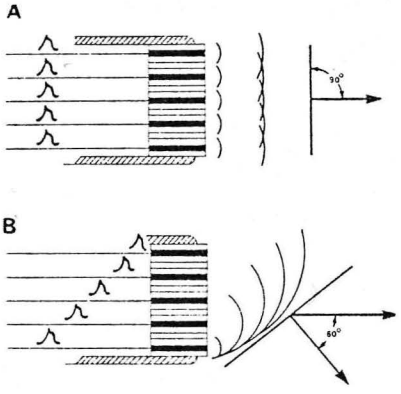


Fig. 3. Diagram illustrating the method of beam transmission and steering in the phased array format. *A*, A series of electrical pulses is depicted moving from left to right toward the transducer elements in the array. The pulses activate the elements, simultaneously producing a series of small wavelets. As these wavelets move from the transducer face, they summate to form a beam that propagates away from the transducer. *B*, The transducer elements are activated slightly out-of-phase. In this example, the upper transducer is activated first, producing an acoustic wavelet that propagates to the right and away from the array. Rapidly thereafter, the second element is excited, producing a second wavelet slightly behind the first. The sequence continues until the bottom element in the array is activated. In tissue, these five individual wavefronts summate to produce an acoustic beam that approximates in shape and direction the beam that would be produced by a transducer aimed in that direction.

Phased array transducers contain 32-64 crystals. When excited, these crystals transmit sound waves in a pattern much like the concentric waves caused by dropping a stone into a quiet pool of water. The waves merge so that a linear wave front is created. (The waves approaching the beach are an example of a wave front produced by merging multiple smaller waves.) If all crystals are fired simultaneously, the resulting wave front will travel straight out from the transducer. If the crystals are fired with a progressive delay so that those on the left are fired earlier and those on the right later, the resulting wave front will be directed to the right. By varying the delay, the wave front can be directed to any angle through a 90° sector. A microprocessor controls the delay in crystal firing to give approximately 120 "scan lines" in the 90° sector. The time required for the sound wave to cover the required distance is the same for mechanical or phased array systems so these systems also provide about 30 frames per second. To record simultaneous M-Mode and 2-D echoes, the phased array system selects a few of its many crystals for M-Mode while the rest are used for the 2-D image. Earlier phased array systems had terrible M-Mode quality, but that provided by current equipment is superb. The M-Mode beam is also steered electronically so 2-D echo equipment can image difficult-to-find structures, such as the pulmonary valve, more easily than M-Mode machines. The equipment is substantially more complex and correspondingly more expensive. Mechanical scanners cost $35-70,000 while phased array units are $65-95,000.

D. *Signal Processing*

Once the sound is received by the transducer, it is handled in similar ways by both mechanical and phased array systems (Figure 4).

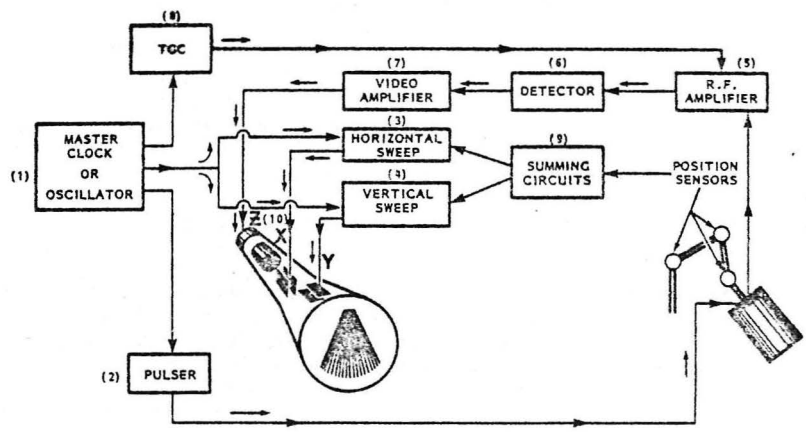


Fig. 4. Block diagram of a simple B-mode scanning system.

An internal clock provides timing, which is converted to distance information and displayed on the screen with an internally created depth scale. An electrocardiogram is also provided for timing cardiac events. Newer units will "gate" from the ECG to provide serial images selected from one or two times in the cardiac cycle, usually end-systole and end-diastole.

Sound is attenuated as it passes through the body, so echoes from structures near the transducer are stronger than those from more distant sites. To correct for this, some adjustment is made to attenuate the near echoes and enhance the distant echoes. This is called "Time Gain Compensation" or TGC. Some devices provide a series of slide potentiometers allowing one to attenuate or amplify echoes for individually multiple depth level, while other devices change near or far echoes in a more gross fashion and then allow a continuous, linear variation between near and far field echoes (a ramp).

All systems have some method for selectively enhancing or suppressing echoes based on their strength rather than location. A crude control, called "reject" simply eliminates all echoes below a certain strength. Computer processing also allows a variety of enhancement/attenuation schemes for emphasizing weak and strong echoes, weak only, strong only, strong more than weak, etc. An almost limitless number of transformation curves are available. Some units provide a set of controls to set these curves, while others have a computer "menu" of stored processing curves. Some equipment allows processing of the image before it is recorded on tape; other systems also allow reprocessing of previously recorded studies.

The assessment of image quality is highly subjective and a great source of consternation for equipment manufacturers who must design machines with wide capabilities to meet the varied and sometimes irrational demands of the users and purchasers of 2-D echoes. One can quantify resolution of a given system. In simple terms, resolution is the distance two echo-dense objects must be separated in order to appear as two separate targets in the echo image. In two-dimensional systems, the beam is really 3-dimensional so axial and lateral resolution must be considered separately. Axial resolution, for most systems, is 1-2 mm, while lateral resolution is 4-8 mm. The machine is quite accurate in measuring time and hence distance, but is not as accurate in determining direction, because the sound tends to diverge and beam width increases beyond the "focal zone.".

Resolution is also affected by the frequency of sound transmitted by the transducer. Low frequency sound penetrates well but has poor resolving power. Higher frequencies give substantially better images but at the expense of penetration. The practical result is that most adults are imaged with 2.5 MHz transducers, while thinner adults and children can be imaged with the 3.5-5.0 MHz systems since penetration is less of a problem.

Although technical or equipment factors are important, individual differences in anatomy have even greater effects on image quality. Sound attenuation in air is about 200 times greater than in muscle or blood. This is why a water-based

gel is used during echocardiography to provide an interface between the transducer and the body. If the lung is interposed between the transducer and the heart, as in obstructive lung disease, most of the sound will be absorbed and a poor image will result. Likewise extensive calcification (in the elderly) or scarring (post-thoractomy patients) attenuate sound and diminish image quality.

Finally, images are viewed on a television screen. Smaller screens give the appearance of a better quality image, and this has led manufacturers to install smaller and smaller viewing screens. The trade-off is a decreased ease of viewing, so most companies provide a 9 inch high resolution black and white monitor. Video images from patient studies are stored on video tape (usually 1/2") and can be replayed or reproduced "hard copy" by printing with a dry photocopy system. Substantial image degradation occurs when paper copies are made, so one must usually view the tape for interpretation. Photos of 2-D echo images include only half the information seen with a normal video image so most of the material presented today will be from video tapes and not still photographs. (For more technical information, the reader should review Weyman (1982) or Feigenbaum (1981) Textbook of Echocardiography.

III THE NORMAL EXAMINATION

An initially intimidating aspect of two-dimensional echocardiography is the almost limitless number of possible views. This potential can be highly advantageous, since the heart's position and architecture vary substantially from patient to patient. Unlike M-Mode echocardiography, 2-D echo does not require standardized patient positioning since one's view of the heart is easily determined from the image itself. However most of the time, a few standard views will provide all the necessary information (Figure 5).

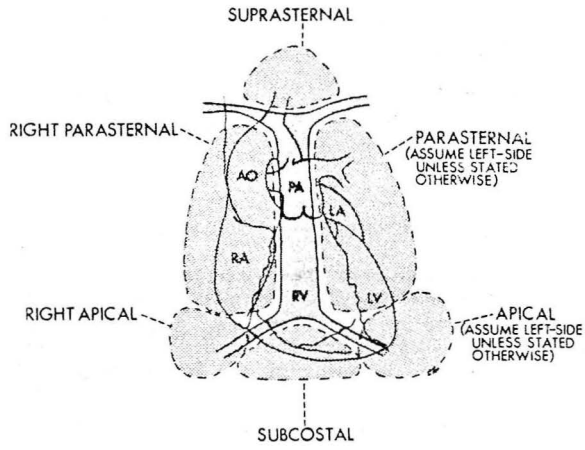
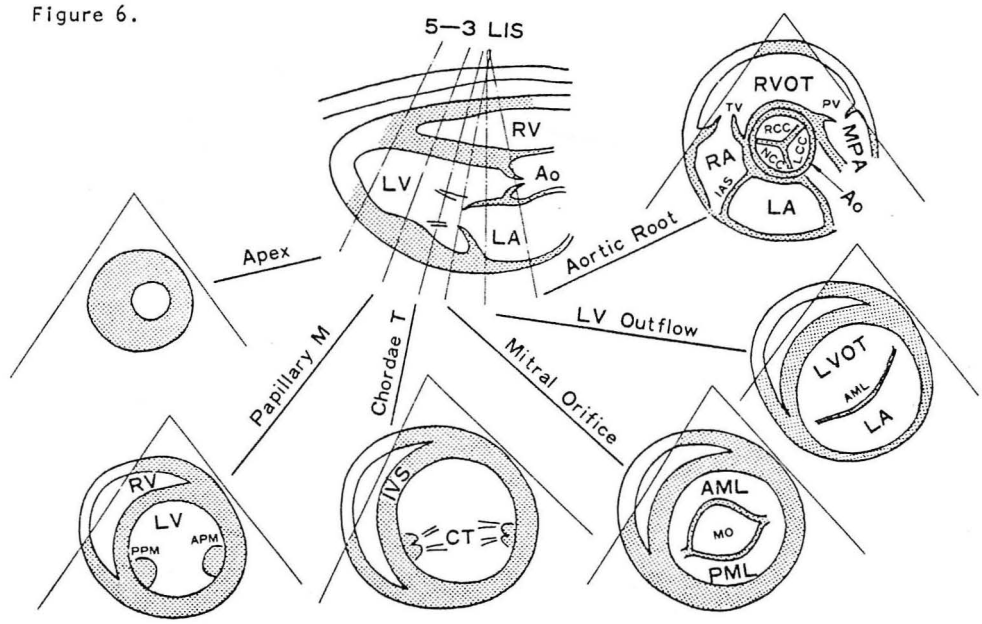


FIGURE 5. *Diagram indicating the nomenclature to describe the locations on the body from which echocardiographic studies can be obtained. AO = aorta; RA = right atrium; PA = pulmonary artery; RV = right ventricle; LA = left atrium; LV = left ventricle.*

The right sided structures are always displayed on the left side of the video screen as one would view a chest X-ray (Henry et al., 1980; Tajik et al., 1978 - superb pictures but 4-chamber views are non-standard, i.e. RV is not on left side of screen). The standard positions and views are shown in Figure 6. These will be described in brief.

Figure 6.



A. *Parasternal - Long Axis*

Parasternal long axis (top center) provides images of the mitral and aortic valves as well as dimensions of the right ventricle, left ventricle, left atrium and proximal aorta. It is very similar to the position and information obtained with a complete M-Mode sweep from the aorta through the left ventricle. The right atrium and left ventricular apex are not seen in this view.

B. *Parasternal - Short Axis*

The transducer is rotated 90° from the orientation producing the long axis view. The angle of viewing is then varied to provide cross-sectional

images through the heart from base to apex (Figure 6). Standard views (clockwise) would be through a) the aortic root, b) LV outflow tract, c) the mitral oriface, d) mitral chordae, e) papillary muscles, and, f) left ventricular apical region. This view shows the relationship of the aorta to the septum, mitral valve aperture and left ventricular wall motion. The view through the base of the heart is quite useful because it shows parts of the right and left atria, RV outflow tract, tricuspid, aortic and pulmonary valves and main pulmonary artery to its bifurcation.

C. *Apical 4-Chamber*

The apical 4-chamber view is obtained by placing the transducer at the apex and aiming toward the right shoulder (Figure 7a). Ventricular cross-sectional areas are maximized. The size of the four chambers, as well as the relative position of the mitral and tricuspid valves, can be appreciated. By angling the transducer superiorly, the round cross-sectional image of the aorta is also seen. This is sometimes called an Apical 5-chamber view. The transducer can be rotated 90° to give an apical 2-chamber view of either the right or left side of the heart (Figure 7b).

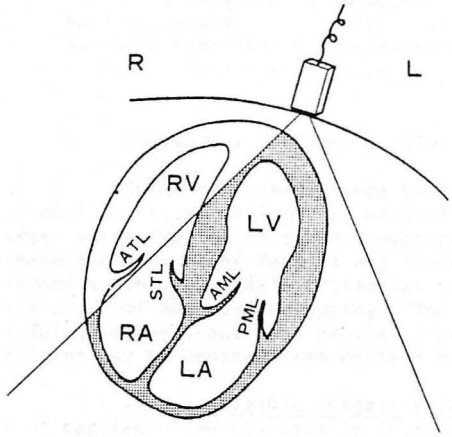


Figure 7a.

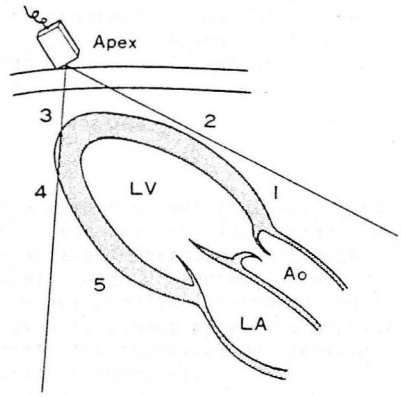


Figure 7b.

In pediatric cases and in patients with lung disease and/or a midline heart location, subcostal views should be obtained. The great vessels can be viewed from the suprasternal notch, but these are usually of poor quality and not often useful in adults. Non-standard views can be recognized as more experience is gained and structures accurately identified. The ability to obtain views from almost any angle is one of the major diagnostic advantages of 2-D echo.

IV CARDIOMYOPATHIES

Patients often present with an enlarged heart in association with a wide range of symptoms and electrocardiographic findings. The echocardiogram provides substantial information because it first allows separation of patients with pericardial effusions from those with true cardiac enlargement (DeMaria et al., 1980). The echo also provides precise information about which cardiac chambers are enlarged and to what extent their function has been impaired. Cardiomyopathies, the most common causes of cardiac enlargement, will be discussed in this section. The general classification scheme suggested by Goodwin in 1972 will be used. A general outline is given in Table 1.

Table 1.

	Congestive	Hypertrophic	Restrictive
Chambers Involved	All	LV (RV)	LV, LA (All)
Chamber Dimensions	Enlarged	Normal or Reduced	Normal or Reduced
Wall Thickness	Thin	Thick	Normal to Thick
Systolic Function	Decreased	Increased	Decreased
Diastolic Function	Decreased	Decreased	Decreased

A. *Congestive Cardiomyopathies*

Congestive cardiomyopathy is found in a number of settings including alcoholism, hypertension, myocarditis, following pregnancy, in families and after extensive, often silent, myocardial ischemia and infarction. Although these and many other factors are listed as "causes," with the exception of ischemia, they are really "associations" which do not provide information as to the cause of the cardiomyopathy. The 2-D echo usually cannot provide a precise etiology either, but does provide some help in that hypertensive and ischemic origins may be deduced from certain echocardiographic features.

1. Idiopathic congestive cardiomyopathy consists of dilation of all four cardiac chambers, usually in a predictably symmetrical pattern. Predominant involvement of only one side of the heart would suggest another diagnosis. Left and right ventricular function are usually decreased in a global fashion without evidence of segmental wall motion abnormalities or dyskinetic areas. Typically, LV function is more affected. Finding the opposite should raise the question of secondary right heart disease.

The cardiac valves should appear normal although the excursion of the mitral and aortic valves may be decreased and age-related thickening can be seen. The relatively slow sampling rate of the 2-D system in the setting of decreased cardiac output often causes the mitral valve to appear thickened when it is really normal. An M-Mode trace will show a normal thickness and answer the question.

A number of echo laboratories use an index called "E Point Septal Separation" (EPSS) to indicate the degree of left ventricular dysfunction (Massie et al., 1977; Lew et al., 1978; Pollick et al., 1982) (Figure 8).

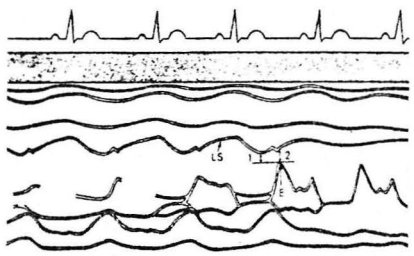


Fig. 8. Diagram of an M-mode echocardiogram demonstrating two ways of measuring the distance between the E point of the mitral valve and the left septal echo. E-point septal separation *number 1* represents the distance between the peak downward motion of the septum and the maximum upward excursion of the E point. E-point septal separation *number 2* represents the distance between the mitral valve and the septum at the time of the E point. LS = left side of the interventricular septum.

It is the distance from the septum to the point of maximal diastolic excursion of the mitral valve. The normal valve is less than 5 mm. Increases are reasonably well correlated with the extent of left ventricular dysfunction for several reasons. With congestive cardiomyopathies, both septal and mitral leaflet excursions are reduced, and the left ventricle becomes a more rounded or globular structure. Thus, a single numerical value reflects a number of different pathophysiological events and correlates well with global left ventricular function. It does not add any new information and is not reported routinely in our laboratory.

Some etiological information may be obtained from left ventricular free wall thickness and aortic root size. The left ventricle may be expected to hypertrophy as dilation occurs in an attempt to keep intramyocardial wall stress constant. The normal relationship of LV mass to volume is usually preserved but in idiopathic congestive cardiomyopathy, the mass:volume ratio is not normal and only minimal hypertrophy is found. The LV wall is usually less than 1.2 cm in thickness in congestive cardiomyopathy uncomplicated by systemic arterial hypertension. The aorta should be normal in diameter in congestive cardiomyopathies. The finding of left ventricular hypertrophy and aortic dilation provides strong evidence for long-standing hypertension and may be a clue to the etiology of the cardiomyopathy. Unfortunately, a large proportion of the

cardiomyopathy patients at Parkland have a history of hypertension and excessive alcoholic intake. Their hearts usually show some hypertensive changes (LV septum and posterior wall=1.2-1.5 cm), but not nearly to the extent seen in other hypertensive patients with only hypertensive cardiomyopathy.

Additional findings in patients with cardiomyopathy include the presence of small pericardial effusions and both right and left ventricular thrombi. The former are easily recognized and their contribution to the extent of the "cardiomegaly" can be determined. Ventricular thrombi in patients with congestive cardiomyopathy are said to be easily recognized (Reeder et al., 1981). It is probably more correct to say that typical findings of thrombus are common in these patients, but the actual diagnosis of clot is not easy. These clots are often small and are found within the extensive trabeculae which develop in dilated, poorly functioning hearts. The thrombi are usually not discrete and well-localized as in the post-infarction patient, so 2-D echo should not be considered a sensitive technique for detecting ventricular thrombi in congestive cardiomyopathy.

Assessment of left ventricular wall motion is discussed later, but a point should be made here with respect to congestive cardiomyopathy. Both systolic and diastolic ventricular function are important in the production of signs and symptoms of congestive heart failure. Changes in preload, afterload and contractility produced by vasodilators, diuretics, anti-hypertensive medications and digitalis may result in great improvement of ventricular function and/or volume. The echo may be used to assess these changes. However, an echo ordered after several days of aggressive treatment, may be nearly normal or within normal limits even though the patient was in florid pulmonary edema a few days earlier. Our laboratory is sometimes questioned about the lack of abnormal findings in patients in this situation. As obvious as it might seem, one must remember that the 2-D echo shows function as it is, not as it was.

B. *Restrictive Cardiomyopathies*

Restrictive cardiomyopathies have a multiplicity of causes and the 2-D echo exam can only suggest their presence and etiology. Except perhaps in the case of amyloid (Siqueira-Filho et al., 1981) it cannot provide definitive diagnostic information. There is usually normal or decreased chamber size with decreased left ventricular function both in systole and diastole. The LV walls are thickened and in the case of cardiac amyloid, may show an abnormal, glittery texture. The RV is also involved. It is reported that amyloid also involves the valves, papillary muscles and atrial walls, differentiating it from other infiltrative cardiomyopathies. In practice, this is probably not a very useful sign and the diagnosis must be made by cardiac biopsy or by finding amyloid in other non-cardiac sites. Hemochromatosis usually produces cardiac dilation (Buja and Roberts, 1971). This and its non-cardiac manifestations permit differentiation from the other infiltrative diseases.

A decreased ejection or shortening fraction suggests abnormal systolic function. Signs of impaired diastolic function include a dilated left atrium and decreased mitral valve motion, which reflect a chronically elevated LV end-diastolic pressure (Feigenbaum, 1981a). The absence of segmental wall motion abnormalities or thinning and a dilated aortic root help differentiate infiltrative cardiomyopathies from ischemic and hypertensive heart disease respectively. Likewise, the finding of LVH on 2-D echo with decreased electrocardiographic LV voltage would be strong evidence for an infiltrative/restrictive cardiomyopathy and is probably the most common clue for diagnosis at this institution.

C. *Hypertrophic Cardiomyopathies*

Hypertrophic cardiomyopathies comprise such a wide range of etiologies and pathophysiologies that they alone could easily be the subject of an entire Grand Rounds. A good approach is to determine the pattern of hypertrophy and the presence or absence of obstruction (Maron and Epstein, 1979). The variable patterns of hypertrophy and the dynamic nature of obstruction make this a difficult task but one for which 2-D echo is particularly well suited (Maron et al., 1981a and b; Silverman et al., 1982).

The sine-qua-non of hypertrophic cardiomyopathy is an abnormal mass:volume ratio. Small increases in LV thickness are to be expected in dilated hearts and do not indicate a primary hypertrophic process. Increases in LV wall thickness in athletes, especially those who engage in activities with a prominent isometric component should be considered normal (Longhurst et al., 1979). The "hypertrophy" involves the septum and LV posterior wall more or less symmetrically although the septum is normally 1-2 mm thicker. A left ventricular posterior wall thickness of $\geq$ 1.2 cm should suggest a non-physiological cause of hypertrophy.

1. Symmetrical hypertrophy is the most common form of symmetrical hypertrophic cardiomyopathy associated with systemic arterial hypertension. Even though this is called "symmetrical" hypertrophy, the septum is usually slightly thicker than the posterior wall (mean ratio of septum:LVPW = 1.1:1). Unfortunately, many hypertensive patients also have localized areas of hypertrophy, especially in the region of the septum where the anterior aorta attaches. Many of these patients have systolic ejection murmurs and dilated aortic roots with normal aortic valve leaflets. There is no evidence of obstruction (vide infra), although the murmur may suggest the presence of turbulence. LV function may be normal or decreased, but the high ejection fractions seen with obstructive hypertrophic cardiomyopathies are almost always absent. Left atrial dilation occurs frequently and is due to decreased LV compliance and a high LVEDP. Whether this plays a role in the frequent association of hypertension and atrial fibrillation is not known.

Differentiating hypertensive cardiomyopathies from familial hypertrophic cardiomyopathies can be extremely difficult. Attempts to obtain adequate historical information about the patients previous blood pressures should be made although this information is frequently inaccurate or unavailable. Many

hypertrophic cardiomyopathies are familial, so echocardiographic examination in younger family members may be helpful. Hypertension may also be familial, so blood pressures should be measured if family screening is done.

2. Asymmetrical hypertrophy was initially described as a single disease called Idiopathic Hypertrophic Subaortic Stenosis (IHSS) (Brock, 1957; Teare, 1958). It is inherited as an autosomal dominant trait and characterized by a septal to posterior LV wall thickness ratio of > 1.3, decreased septal motion, systolic anterior motion of the mitral valve and a dilated left atrium (Henry et al., 1973). Pathologically, disarray of myocardial fibers was found and was said to produce an abnormal, glittery appearace of the septum on two-dimensional echo. Unfortunately, subsequent studies have provided much new information, but probably an equal amount of confusion.

With 2-D echo studies, it became obvious that septal hypertrophy was not a uniform process and could involve any or all of the septum (Maron et al., 1981a; Yamaguchi et al., 1979). About two-thirds of the patients with asymmetrical septal hypertrophy show mid-septal enlargement while about 1/4 have thickening throughout the septum. Most patients show consistent thickening in a superior-inferior direction. Almost half the patients have hypertrophy of the LVPW. Clear-cut guidelines for separating asymmetrical and symmetrical forms do not exist because of this, but most authors would probably agree that septal:posterior wall diastolic thickness ratios of > 1.5 would be classified as "asymmetrical" and are more likely to represent a familial or primary form of hypertrophic cardiomyopathy.

Septal motion is usually reduced in patients with Asymmetrical Septal Hypertrophy (ASH), particularly when the hypertrophy is severe (> 1.8 cm). This is somewhat surprising when one considers that the ejection fractions in these patients are usually above normal unless lowered by drugs or congestive heart failure. The LV is usually normal in size although the left atrium is frequently dilated as a result of increased LV end-diastolic pressures, reduced LV compliance and mitral regurgitation that is commonly seen in patients with ASH. Impaired diastolic function also produces decreased mitral motion, i.e. pseudo mitral stenosis. Abnormally small LV end-systolic volumes may also produce mitral valve prolapse with a morphologically normal valve (Böcker et al., 1981). This has nothing to do with click-murmur syndrome and should be considered an artifact. It is not usually reported by our laboratory for that reason.

3. Obstructive hypertrophy is the presence or absence of left ventricular outflow tract obstruction and cannot be determined quantitatively by 2-D echo, but clues to its presence and severity are provided. The most reliable sign is that of notching of the aortic valve during systole (Figure 9). This is caused by dynamic LV outflow tract (LVOT) obstruction decreasing the rate of LV ejection which produces an incomplete closure of the aortic valve. This corresponds to the "spike and dome" carotid artery tracing and is highly specific for LVOT obstruction. Unfortunately, the sensitivity of the sign is not known and is probably not seen in all cases of obstruction, especially when only small gradients are present.

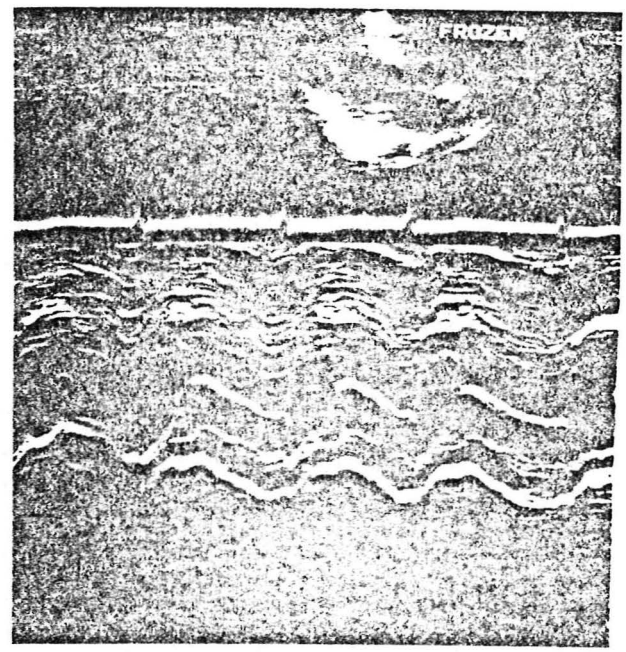


Figure 9 shows an M-Mode tracing from the aortic valve of a patient with septal hypertrophy and obstruction. Note the mid-systolic notching of the non-coronary leaflet in the second cardiac cycle.

A number of papers have suggested that systolic anterior motion of the mitral valve (SAM) is a reliable sign of LVOT obstruction (Henry et al., 1973; Rossen et al., 1974). Unfortunately, it is not specific for obstruction, and it is probably not the mitral leaflets which cause it. Two-dimensional studies show very clearly that the abnormal echoes recorded in the LVOT during systole are caused by the chordae tendineae in hypertrophic, hypercontractile left ventricle (Come et al., 1977). Obstruction is certainly more likely to occur in the presence of a very high ejection fraction and a small end-systolic LV volume, so the co-existence of SAM and obstruction is easily understood. One should consider SAM a marker of a high EF however and not of LV outflow tract obstruction.

The absence of valvular lesions helps to rule out other causes of LV hypertrophy. Examination of family members may be quite useful to detect unsuspected or silent cases of ASH. The aortic root should be normal, and enlargement would raise the question of whether systemic arterial hypertension may have been present. Maneuvers which reduce LV volume or increase its ejection fraction should be used to provoke signs of obstruction in patients with a hypertrophic cardiomyopathy.

V RIGHT HEART DISEASE

The right-sided cardiac chambers are difficult to examine by routine clinical methods including the physical examination, chest X-ray and electrocardiogram. For this reason, two-dimensional echo is especially useful in providing anatomical and functional information not easily obtained by other methods. Although chronic obstructive lung disease does create problems for echocardiography, the use of subcostal and apical 2-D views has lessened its impact.

A. *Right Heart Dilation - Pressure Versus Volume?*

In evaluation of the patient with RV dilation, one would want to know whether the left ventricle is involved as well, or if the disease seems to have affected the right heart only. A number of conditions lead to right ventricular dilation, but associated findings often provide useful diagnostic information.

Dilation of the right atrium (RA), ventricle (RV), and pulmonary artery would suggest either a significant left-to-right shunt and/or pulmonary hypertension. RV hypertrophy would favor the diagnosis of pulmonary hypertension. A large RV might mean Eisenmenger physiology. When RA pressure is substantially above that in the LA, the atrial septum can be seen bulging toward the LA in both systole and diastole. The pulmonary valve also has a characteristic systolic notching in pulmonary hypertension, although this is an inconsistent finding. It is virtually identical to the notching seen in aortic valve with IHSS (Figure 9). Dilated right heart structures and drop-out of atrial septal echoes and excessive motion of atrial septal mid-portion edges would suggest an ostium secundum atrial septal defect. If an intracardiac shunt is present and of sufficient magnitude to cause right heart enlargement, its site can usually be localized with contrast echocardiography.

Paradoxical septal motion is often discussed in association with RV enlargement, but unfortunately it occurs with both pressure and volume overload states and cannot be used to distinguish one from the other (Weyman et al., 1976). In both situations, the ventricular septum is displaced downward into the LV at end-diastole. With systole, isovolumetric contraction of the LV quickly overcomes the RV:LV pressure differential and the septum is pushed back toward the RV. This means that the septum moves "paradoxically" in early systole. It may be a useful sign in situations of acute increases in RV pressure such as pulmonary embolism or rupture of the aorta or septum or sinus of Valsalva into the RV.

B. *Right Heart Valvular Disease*

1. Tricuspid regurgitation. In most patients, clear 2-D echo views of the tricuspid and pulmonary valves are easily obtained. A vigorously contracting dilated RV with a large RA and normal PA would suggest tricuspid regurgitation (TR). Depending on etiology, the tricuspid valve itself may be normal or abnormal. The inferior vena cava (IVC) and hepatic veins are usually dilated

and even pulsatile. Contrast echocardiography is useful in identifying TR. An injection of contrast material into an arm vein will enter the heart via the superior vena cava and will not normally enter the IVC. Detection there, especially during ventricular systole, would be proof of TR. It is also possible to observe a to-and-fro pattern of motion of the contrast bubbles in TR (Veyrat et al., 1982).

2. Malignant carcinoid syndrome may produce substantial cardiac involvement that is easily recognized by 2-D echo (Howard et al., 1982). The tricuspid leaflets are shrunken and retracted without commissural fusion and signs of tricuspid regurgitation are present. The pulmonary valve may be similarly involved while the left sided valves will be normal. Carcinoid is unique in this respect and therefore easily diagnosed.

C. *Pulmonary Embolism*

Echocardiography is usually not useful in detecting pulmonary emboli but may detect acute pressure increases in the right ventricle (Winer et al., 1977; Arvan and DuBois, 1982). A sudden increase in RV size with paradoxical septal motion would indirectly suggest the diagnosis. The echo may also be useful in ruling out other causes of hypotension and low cardiac output, but most of the time, other tests will be more useful and time should not be wasted obtaining an echo acutely if the diagnosis of pulmonary embolism is likely.

D. *Arrhythmogenic RV Dysplasia (Uhl's)*

Parchment Right Ventricle, Uhl's Anomaly or most recently Arrhythmogenic RV Dysplasia all refer to the same disease characterized by RV dilation associated with a partial absence of RV myocardium (Baran et al., 1982; Gaffney et al., 1983). Children born with a total absence of myocardium die in infancy or early childhood, while patients with less severe disease survive into adulthood. The RV is massively dilated, poorly contracting, and thin walled. The RA is similarly dilated while the pulmonary artery is of normal size. There is diastolic opening of the pulmonary valve from atrial systole since the RV serves mainly as a passive conduit. The patients often have recurrent ventricular tachycardia with a characteristic "after potential" or "epsilon wave" on their electrocardiogram. The ventricular septum and moderator band are normal as are the cardiac valves. A normally positioned tricuspid valve rules out Ebstein's anomaly. The ability to recognize these patients non-invasively may permit more effective treatment of the associated life-threatening arrhythmias to prevent sudden death.

VI VALVULAR HEART DISEASE

Two-dimensional echo is unique in its ability to examine cardiac valve structure and motion. This is of great value for determining both the etiology and severity of valvular heart disease. Because various diseases tend to affect one or more valves in a consistent pattern, this section will discuss valvular heart disease from that perspective.

A. *Rheumatic Heart Disease*

Although the U.S. incidence of acute rheumatic fever and therefore valvular heart disease has been decreasing steadily the past two decades, the recent arrival of immigrants from Mexico and southeast Asia has brought substantial rheumatic heart disease. The history is often not clear, but calcification, scarring and commissural fusion provide reliable information about the presence of rheumatic valvular disease. The damage to leaflets and chordae may produce either stenotic or regurgitant lesions or a combination of both. The valves will be discussed in order of frequency of involvement.

1. The mitral valve typically shows thickening of the leaflets. The commissures may be fused. The anterior leaflet with its larger surface area pulls the posterior leaflet up during diastole, so that its motion is restricted and a bowed configuration is seen. Scarring and calcification may be seen in the leaflets, chordae or papillary muscles. Two-dimensional echo is an extremely sensitive method for detecting calcification, but differentiation from fibrosis is difficult and up to 1/3 of mitral valves said to be calcified on 2-D exam will not show the calcium on X-ray. This probably represents oversensitivity of 2-D echo and poor sensitivity of radiography (Zanolla et al., 1982). Minor degrees of thickening are also commonly seen with aging and in other conditions, but commissural fusion as well as involvement of other valves are strong evidence of rheumatic heart disease.

Associated findings indirectly point to a diagnosis predominant of regurgitation or stenosis. The latter typically produces an enlarged left atrium with a small, hypocontractile left ventricle and aortic root. Occasionally a clot may be seen in the LA. A parasternal, short-axis view at the tips of the mitral leaflets provides an estimate of mitral valve cross-sectional area (Nichol et al., 1977). Technical details are important, but the measurements from 2-D have been found to correlate well with estimates of valve area taken at catheterization or surgery (Figure 10).

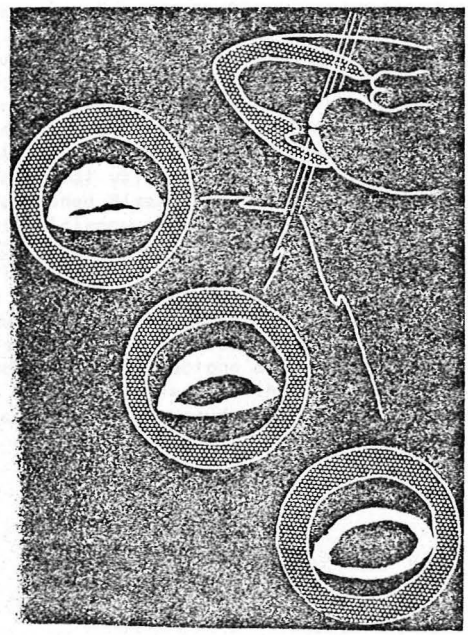


Figure 10.

If combined stenosis and regurgitation are present, 2-D echo may be the only non-surgical method of estimating valve size since the Gorlin formula (Gorlin and Gorlin, 1951) cannot be easily applied in combined lesions.

Mitral regurgitation cannot be diagnosed directly from echo, but an enlarged LA and LV with an increased or high normal LV ejection fraction and increased MV excursion would suggest regurgitation (Wann et al., 1977). It is usually possible to estimate the extent of regurgitation or stenosis in combined lesions by looking at leaflet motion and LA and LV size. Non-invasive estimates of LV volume and function can be used to assess severity and determine the timing of more invasive tests or treatments.

2. The aortic valve may show changes similar to those seen in rheumatic mitral disease, i.e. thickening of the leaflets, commissural fusion and variable amounts of calcification and fibrosis. Like the mitral valve, stenosis, regurgitation or a combination of the two lesions may be seen. A stenotic valve should be associated with concentric LV hypertrophy and a dilated aortic root. Estimates of severity are based on leaflet thickness and motion, but the degree of stenosis can be determined only in general categories since measurements of aortic valve area are less reliable than mitral valve area measurements (DeMaria et al., 1980; Godley et al., 1981). Problems in diagnosis occur in the elderly patient with calcification in the aortic valve and concentric LV hypertrophy. Hypertension in an elderly patient may cause similar findings, and leaflet thickening with annular calcification is common in this age group, so great attention must be paid to leaflet separation, thickness and the localization of calcification. The ability to place patients in diagnostic categories of mild, moderate or severe aortic stenosis on that basis is often adequate for clinical purposes, especially when the patients are elderly or the relationship of symptoms to aortic stenosis is not clear.

Aortic regurgitation, like mitral regurgitation, cannot be diagnosed directly but its presence can be inferred (Kotley et al., 1980). The LV will be dilated and show signs of volume overload with exaggerated septal and posterior wall motion and a high ejection fraction. Hypertrophy is common but the wall thickness is usually 1.3 cm or less. In combined lesions, the degree of LV dilation and hypertrophy reflect the amount of regurgitation or stenosis present, although associated disease of the mitral valve or systemic hypertension may produce similar findings. The mitral valve is commonly involved in rheumatic aortic valve disease so that combined disease is almost always rheumatic in etiology. However, aortic insufficiency produces fluttering of the mitral leaflets in diastole. The 2-D image shows thickened leaflets because the 30 Hz frame rate of the 2-D system is not adequate to resolve the fine flutter pattern of leaflet motion. Decreased LV compliance and elevated end-diastolic pressures produce an enlarged left atrium and reduced mitral valve motion. This combination looks remarkably like mitral stenosis and may lead to an incorrect diagnosis. Simultaneous high quality M-Mode and 2-D tracings of the mitral valve will identify flutter as the cause of apparent leaflet thickening, while normal posterior mitral leaflet motion identifies pseudo-mitral valve prolapse. Left

atrial diameter is usually less than 4.5 cm in this setting, but may be much larger with true mitral stenosis. Various M-Mode indices of LV size and volume have been suggested as criteria for aortic valve replacement, but these measurements are unreliable in large dilated LV's. Even though 2-D measurements are much better, rest measurements may not be sensitive enough to detect early signs of LV dysfunction (Huxley et al., 1983).

3. Tricuspid valve involvement is less common but pathologically similar to aortic and mitral rheumatic disease. Secondary tricuspid regurgitation is also a common problem in mitral rheumatic heart disease because pulmonary hypertension may produce a dilated RV and RA. Careful imaging of the tricuspid valve leaflets in this setting can be useful in distinguishing rheumatic tricuspid regurgitation from secondary, non-valvular causes. Many times, the presence of tricuspid involvement is unsuspected or underestimated because of the subtle nature of the physical findings and the inherent difficulties in examining the right heart. Contrast studies may be of benefit in evaluating TR (see Section X).

B. *Infective Endocarditis*

Infective endocarditis may affect any cardiac valve, but like rheumatic disease, it is virtually always found on either the mitral, aortic or tricuspid valves. Any leaflet may be involved and multi-leaflet or multi-valve disease is not rare. A vegetation appears as a bright, mobile, echo-dense structure attached to the valve leaflets or rarely to the chordae tendineae. The vegetations (2 mm - 4 cm) often move in a direction or speed different from the leaflet itself. The presence of vegetations allows confirmation of the diagnosis and provides localization of the involvement so clinical problems can be anticipated and appropriate surgical intervention provided when necessary.

Thickening of a leaflet in a bacteremic patient is not diagnostic of endocarditis, but does show the presence of an abnormal valve with the increased risk of infection. Occasionally, a flail leaflet may show a pattern of motion similar to that of a vegetation, but careful stop frame analysis will show leaflet destruction and allow one to discriminate a flail leaflet from a vegetation.

There is substantial controversy as to the meaning of vegetations detected by echocardiography. Originally, some investigators suggested that the mere presence of a vegetation was an indication for surgery. Subsequent studies of left-sided endocarditis have supported the concept that patients with vegetations are at greater risk for embolic and hemodynamic complications, but not every patient will require an operation (Stewart et al., 1980). Vegetations on the tricuspid valve seem to have similar prognostic significance in terms of embolic complications and prolonged course although hemodynamic complications are not a significant factor.

Serial echoes are not useful in assessing anti-bacterial therapy since vegetations may persist for months to years after the initial infection (Martin et al., 1980). Likewise, the absence of vegetations on 2-D echo does not rule

out endocarditis and normal exams may be seen in 1/3 to 1/2 of patients with clinically diagnosed bacterial endocarditis. If significant leaflet involvement is found, the echo is useful in assessing the extent of damage and its hemodynamic consequences and serial examinations should be done for that reason. A pre-discharge echo should be obtained to provide a baseline study, especially in drug abuse patients, since recurrences are likely and vegetations may persist for months to years.

In following patients with aortic valve endocarditis, a sign of hemodynamic compromise is that of "early mitral valve closure" (Figure 11).

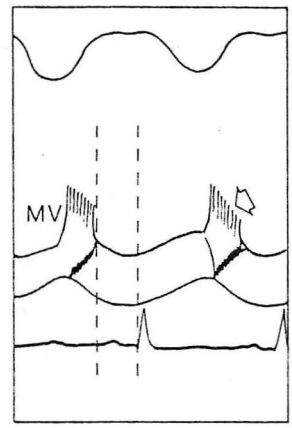


Figure 11.

The physiology underlying this finding is often misunderstood, so it will be discussed in detail. Normally, mitral leaflet motion consists of an initial opening in diastole during early rapid filling of the LV (see M-Mode trace on the cover). As flow declines, the mitral leaflets drift toward a closed position only to be reopened by flow associated with atrial systole. In acute aortic insufficiency, the LV has not yet dilated adequately to accommodate the volume load so the end-diastolic pressure (LVEDP) becomes quite high. It may be so high that atrial systole produces little or no additional movement of blood and the mitral leaflets fail to reopen. Thus the mitral valve is closed long before ventricular systole. If the heart rate is rapid (> 110-120) or if there is 1° heart block, the rapid filling phase and atrial systole become superimposed and give the appearance of early closure, regardless of the LVEDP or degree of aortic leak. (This is the same physiology that causes S_3 and S_4 gallops to merge and be heard as a summation gallop.)

Flail valve leaflets or rarely the complete absence of a leaflet are almost always accompanied by significant hemodynamic consequences which require surgical intervention. We have not seen a single patient with endocarditis and a flail leaflet survive without surgery. Most of these patients are severely ill on admission and require emergency valve replacement. The echo has been extremely useful in this situation and most of our patients have had surgery in this setting without catheterization. There are obvious risks to this and one should probably continue to catheterize such patients until substantial experience has been gained by the echocardiographer in this setting.

Mitral valve leaflets with vegetations will often show abnormal motion typical for mitral valve prolapse. This often leads to a diagnosis of MVP and myxomatous disease as the basis for the endocarditis. Unfortunately, a previously normal valve will also prolapse when a vegetation is attached on one of its leaflets. One therefore wonders in how many cases of MVP and endocarditis was the MVP the underlying cause and how many times was it the result.

1. Prosthetic valve endocarditis is extremely difficult to recognize because of differences in patterns of involvement as well as technical problems associated with imaging prosthetic valves. Often vegetations are not present. Ring dehiscence may occur and would be seen as excessive valve motion (Wang et al., 1982). A fluoroscopic exam would give similar information. Valve ring abcesses occur and in technically favorable circumstances may be identified on 2-D echo by the presence of a fluid-filled cyst involving the ventricular septum or bulging into the left atrium from the posterior aortic root (Nakamura et al., 1982). Unfortunately, by the time these abcesses are large enough to be recognized by 2-D echo, damage is severe and surgery often difficult or impossible. A routine post-operative echo in prosthetic valve patients is extremely useful for comparison purposes, but the cost effectiveness is not high.

2. Indications in bacteremia. A common problem in the Parkland echo laboratory is the number of requests of "Rule-out SBE." Our yield and that of others in a patient without positive blood cultures is 1% or less and clearly not high enough to justify using 2-D echo as a screening device (White et al., 1982). A possible exception would be the ICU patient with multiple catheters and persistent fevers despite broad spectrum antibiotics. Even in this group, the yield from 2-D echo is very low, but the lack of less expensive diagnostic alternatives may justify its use.

C. *Mitral Valve Prolapse*

The diagnosis of mitral valve prolapse can be considered either the boon or bane of echocardiography depending on one's point of view. Very large numbers of patients are referred to "rule-out" MVP. In practice, this is difficult because M-Mode diagnostic criteria are not applicable to 2-D echo studies. The M-Mode beam "sees" prolapse of the mitral leaflets in a direction parallel with the beam. Two-dimensional echo showing a cross-section of the mitral valve detects leaflet prolapse into the left atrium, i.e. in a direction perpendicular to that seen with M-Mode (Figure 12).

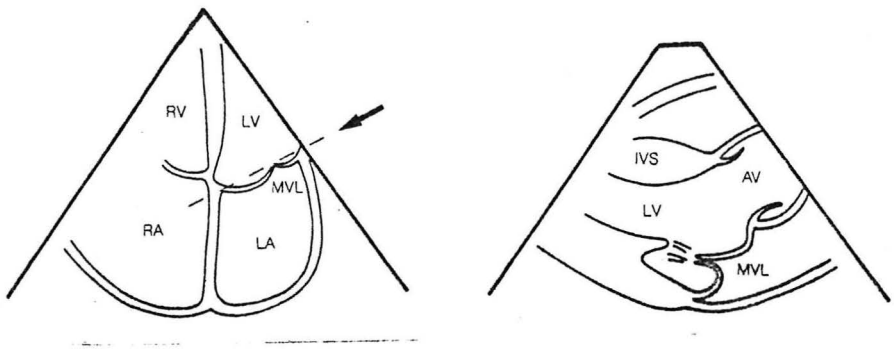


Figure 12. Schematic of apical 4-chamber and parasternal long axis views showing mitral valve prolapse of both leaflets.

Substantial overdiagnosis of MVP has become the rule. To provide better diagnostic definition in MVP, we examine not only valve motion but valve morphology as well.

Two-dimensional echoes in patients with clear-cut auscultatory and phonocardiographic evidence of MVP, reveal a wide spectrum of valve types. Some valves are large, thick, and redundant, and prolapse regardless of the view. Others are small, thin, normal-appearing valves which prolapse in mid- or late-systole only. In these latter patients, the prolapse is often intermittent and may be seen in only one view. The question then is whether MVP constitutes a single disease with a spectrum of valve findings or whether patients can be classified into identifiable categories based on mitral valve leaflet morphology.

To answer this question we performed *in vivo* echoes on patients with classical, persistent MVP, on survivors from families where a death occurred due to MVP, on patients undergoing mitral valve replacement for complications of MVP and *in vitro* on hearts of MVP victims of sudden death. We then reviewed 68 technically satisfactory 2-D echoes from 33 patients with classic auscultatory MVP and 35 normal healthy adults without known cardiac disease. For each echo, an arbitrary value of I-V was assigned to estimate mitral valve leaflet size: I = normal, II = equivocal, III = clearly enlarged, IV = even larger, and, V = largest ever seen. The presence or absence of MVP was also noted. To be considered prolapse, one or both mitral leaflets had to protrude behind an imaginary line through the mitral annulus. Both parasternal long axis and apical 4-chamber views were utilized. In addition, simple measurements of aortic, left atrial and mitral annular dimensions were made. There was some bias in grading valve size since the observers could tell whether the valve prolapsed or not. Otherwise all three readers were blinded to the diganosis and identity of each subject in the study. The results from this preliminary investigation are shown in Table 2.

Table 2.

	Valve Size I	II	III-V	MVP (+)	MV Annulus (mm)	Ao Root (mm)
MVP (N=33)	1	2	30	32	31.8±6.9	30.7±4.8
Normals (N=35)	33	2	0	1	28.5±4.2	30.9±5.1

Table 2 gives the results of 2-D echo evaluations in 33 MVP patients and 35 normal controls (Mean ± 1 SD). Measurements of mitral valve annulus and aortic root include only patients with large mitral valve leaflets, Sizes III-V, (N=30).

It appears that diagnostic criteria using motion and morphology can separate patients from controls with a high degree of sensitivity and specificity. The problem of diagnosis occurs in patients with morphologically normal valves but minor degrees of prolapse and an intermittent mid-systolic click or faint systolic murmur. Levine (1982) and Perloff (1982) have proposed that patients be divided into categories either of functional or "myxomatous" MVP. We believe that these 2-D criteria based on valve motion and morphology, permit classification of patients as either normal, functional MVP, or myxomatous MVP and provide guidance in terms of further work-up and treatment, if any, of symptomatic MVP patients. We are currently conducting studies to test this premise by looking at family members of both types of MVP patients to see if there is a genetic component of both types or only in myxomatous MVP.

MVP can occur in situations associated with myxomatous mitral leaflets or normal leaflets in the presence of a small LV volume. More than 3/4's of Marfan syndrome patients have large floppy mitral valves and MVP, but virtually all have a dilated aortic root as well and none of the symptoms of Barlow's disease. Enlarged mitral and aortic valves, a family history, the typical Marfan habitus, and other stigmata of the Marfan syndrome should be adequate clues for a correct diagnosis. In fact, a dilated aorta and MVP with a large floppy valve would be considered by this writer to be very strong evidence against MVP alone or Barlow's and would suggest a diagnosis of the Marfan syndrome or another connective tissue disorder such as Ehlers-Danlos (Jaffe et al., 1981). Atrial septal defects can produce MVP with a morphologically normal valve. The clue is that closure of the ASD eliminates the MVP. Some patients with idiopathic hypertrophic subaortic stenosis (IHSS) also have MVP with a morphologically normal valve. The prolapse occurs in the setting of a small LV and systolic volume and is relieved by maneuvers which increase LV volume. Recognition of these secondary causes of MVP is worthwhile since, in each case, the primary condition is more important than MVP itself.

D. *Bicuspid Aortic Valve*

Bicuspid aortic valve is one of the most common congenital cardiac anomalies (Roberts, 1970). It occurs in 1-2% of all births and in later life is associated with progressive stenosis due to calcification. Its identification

is useful in differentiating it from rheumatic heart disease and for determining the need for prophylaxis against bacterial endocarditis, the most common adult cause of death in these patients (Fenoglio et al., 1977). The sensitivity and specificity of diagnosis of this condition by 2-D echo has recently been reviewed in detail by Brandenburg et al. (1983).

These investigators retrospectively reviewed available 2-D echoes from 115 of 283 patients $\leq$ 50 years old, undergoing aortic valve replacement at the Mayo Clinic between June 1977 to June 1981. Chronic aortic regurgitation (AR) was present in 53, while 32 had aortic stenosis (AS). Seventeen had a history of infective endocarditis. Adequate surgical description or pathological material was available in all 115 cases. There were 50 bicuspid valves, 60 tricuspid aortic valves, 4 had unicommissural valves while 1 patient had a quadricuspid valve.

The number of cusps was determined by 2-D echo in 86 patients (75%). Inadequate images (13%) and heavily calcified valves (11%) produced a few uninterpretable echoes. The age restriction was important in keeping the figure at only 24%. Overall, sensitivity and specificity were 78 and 96% respectively, although in patients with AS the values were 67% and 80% respectively. Almost 1/3 of the studies in AS patients were "indeterminant."

The aorta, imaged cross-sectionally in a short axis-base view, appears to be a circle (Figure 13).

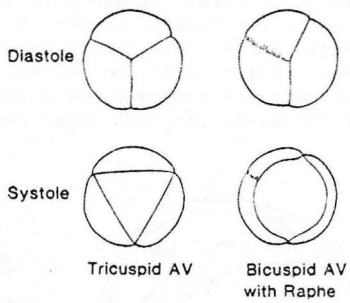


FIGURE 13 Diagram of parasternal short-axis scan of tricuspid (left) and bicuspid (right) aortic valves (AV) in diastole (top) and systole (bottom). Although diastolic appearance of both valves may be similar, viewing the opening pattern distinguishes a raphe from a commissure.

The three leaflets in diastole form a "Y". A bicuspid valve in diastole appears as a straight line across the aorta. Most bicuspid valves will be oriented with a nearly vertical closure line. If a median raphe is present, it will always be

in the anterior cusp. Care must be taken not to mistake this for a third cusp. Likewise, slight rotation of the transducer out of the short axis view can make a normal tricuspid aortic valve appear to be bicuspid. The "missing" leaflet should be diligently sought before the diagnosis of a bicuspid valve is made. Thickened and redundant leaflets are common in bicuspid valves and may be helpful signs.

These investigators also examined the eccentricity index as a method of differentiating tricuspid from bicuspid aortic valves. The normal aortic valve closes in the middle of the aorta while bicuspid aortic valves appear to close eccentrically when recorded by the M-Mode beam. The Eccentricity Index (E) (the aortic diameter divided by half the smaller distance from the leaflet to the aortic wall, (E = A ÷ 2a)) is normally 1.0 and if above 1.3-1.5 would suggest a bicuspid aortic valve (Nanda et al., 1974). The Mayo Clinic group found a wide scatter of values with a sensitivity and specificity of 55 and 71% respectively. A previous M-Mode study by Radford et al. (1976) showed a sensitivity of 75%. A sensitivity of 55% is more reflective of our experience at Parkland. In general, the Eccentricity Index is not a useful calculation except in obvious cases where it is not needed for diagnosis. Two-dimensional echo visualization is substantially better and offers the added benefit of being able to assess leaflet morphology as well as number.

VII MITRAL ANNULAR CALCIFICATION

A common finding in elderly patients is mitral annular calcification (MAC) (Geil, 1950; Simon and Liu, 1954). It is most common in women over age 60 but can be found in men, usually somewhat older, or in patients of either sex with end-stage renal disease or other disorders of calcium metabolism. These abnormal calcium deposits are located posteriorly in the mitral annulus itself or in the adjacent myocardium beneath the posterior mitral leaflet (D'Cruz et al., 1979). Their location may produce a dense echocardiograph pattern in the shape of a horseshoe or merely bright spots near the posterior leaflet of the mitral valve (Figure 14).

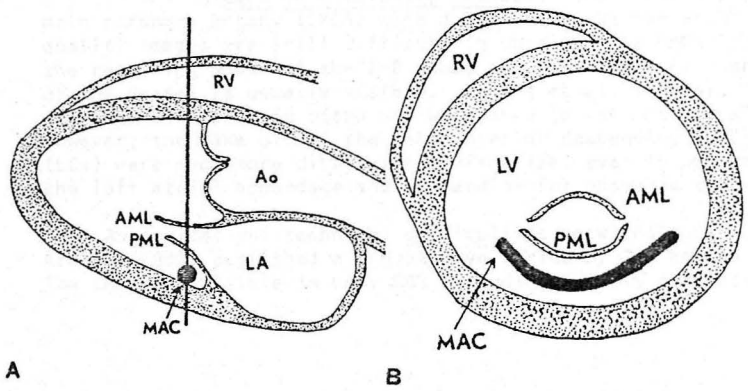


Fig. 14 Representation of location and shape of mitral annular calcification in long axis (A) and short axis (B) views. Mitral annular calcification indicated by solid parts of drawing. M-mode beam (straight line in A) intersects mitral annular calcification only at level of atrioventricular junction. Ao = aorta; AML = anterior mitral leaflet; LA = left atrium; MAC = mitral annular calcification; PML = posterior mitral leaflet; RV = right ventricle.

Extensive calcification can result in dysfunction of the mitral apparatus with mild to moderate mitral regurgitation. Invasion of the intraventricular septum has been associated with the development of conduction system defects and bundle branch block. There is less fibrous substance in the mitral ring anteriorly so less calcification is seen there. Several cases of endocarditis have been reported in patients with MAC (D'Cruz, 1982).

The cause or causes of MAC are not known, but the clinical presentation suggests it is a normal aging process, more advanced in women than men, and accelerated by abnormalities of calcium metabolism, such as those found in patients with ESRD. This writer believes that the location of MAC reflects points of chronic structural stress, worse in women with higher heart rates and smaller end-diastolic volumes. It is very common to see exaggerated motion of the posterior mitral annulus or posterior basal LV wall in women, and it is this area which develops MAC. Subtle age- and sex-related differences in calcium metabolism may explain the nearly five-fold increase in the prevalence of MAC in women compared with men.

MAC is of little significance except that it may sometimes produce a murmur or conduction system disease. It can be mistaken for mitral valve leaflet calcification on X-ray or 2-D echo, so an awareness of its characteristics is useful. As an asymptomatic finding, it should be considered a benign curiosity.

VIII CORONARY ARTERY DISEASE

Coronary artery disease patients comprise a large portion of both internal medicine and cardiology practices so it is only natural that 2-D echo has been applied extensively in this group. Some applications are genuinely useful (Willerson, 1979; Reeder, 1982) while others generally fall into the category of a "technique in search of a purpose." The following sections discuss this at length.

A. *Detection of Coronary Artery Disease*

1. Left main coronary disease. The first 2-D pictures of the left main coronary artery (LMCA) were published by Weyman et al. in 1976, but diagnostic quality images are still difficult to obtain. The LMCA is small, relative to the resolving power of the 2-D echo, and its course is such that only a portion of the vessel is usually visible. Rogers et al. (1980a), from the same group, examined 23 hearts *in vitro* and were able to see most or all of the LMCA in 70%. However, the take off of the left anterior descending (LAD) and left circumflex (LCx) were much more difficult to visualize, even in post-mortem hearts because the left atrial appendage and epicardial fat obscured the view.

Anatomical and technical difficulties notwithstanding, Chandraratna and Aranow (1980) published a prospective series of 2-D echoes from 123 patients. The LMCA was visible in only 60% including 9 of 15 patients with LMCA disease of

> 50% stenosis. All 9 patients had a narrowing detected prior to catheterization. Three patients with normal LMCA's were diagnosed as abnormal on 2-D echo. This is probably representative of most echocardiographers' experience and would suggest that ordinary 2-D echo is not very useful in detecting significant LMCA stenoses.

Rogers et al. (1980b) performed a further *in vivo* study with a simple signal processing technique which increased the brightness of high-intensity echoes caused by fibrosis and calcification. By doing so, they were able to detect calcification in the proximal left main coronary artery, but could not distinguish between significant or non-significant lesions.

At catheterization, almost half of the detected lesions were less than 50%. There was excellent agreement with the presence of calcium detected by fluoroscopy. For that reason, it would seem that cardiac fluoroscopy, especially with newer high resolution systems would offer the same diagnostic information on a more reliable basis.

2. Kawasaki's disease. Somewhat better results have been obtained in children with Mucocutaneous Lymph Node Syndrome or Kawasaki's Disease. This childhood disorder, first described in Japan in 1967 (Kawasaki et al., 1974), is characterized by fever, lymph node swelling, rash and in about 0.7% of the cases, coronary artery aneurysms which can produce myocardial infarctions and death. Both right and left coronary artery systems may be involved, alone or together, and the aneurysmal dilations in their proximal portions render them more likely to be detected. Many of the cases have been diagnosed prior to catheterization, so 2-D echo is probably a suitable non-invasive screening device (Hiraishi et al., 1979; Yoshida et al., 1982; Yoshikawa et al., 1979). There is probably a fairly low sensitivity but a relatively high specificity.

B. *Detection of Wall Motion Abnormalities*

Significant differences exist between the various diagnostic techniques in their ability to detect wall motion abnormalities. Angiography, whether accomplished by radiographic or scintigraphic techniques, provides silhouette images of the heart while 2-D echo gives a tomographic view. In all methods, multiple views must be taken to compensate for sampling errors. These views must then be related to precise anatomical locations in the heart. Because of differences in views, it is often difficult to make precise comparisons between 2-D echo and angiographic assessments of wall motion abnormalities.

To avoid the problems inherent in finding a suitable gold standard, Weiss et al. (1981) compared 2-D echo-detected wall motion abnormalities with autopsy findings in 2-D echo patients with heart disease. Their simple system utilized only parasternal long and short axis views and divided the heart into five regions. Wall motion was classified as normal, hypokinetic, or akinetic/dyskinetic. The hearts were examined in bread-loaf sections corresponding roughly to the tomographic views obtained by 2-D echo. Of the segments found abnormal at

autopsy, 90% were also abnormal on 2-D echo. Conversely, 46% of the segments examined by 2-D echo were abnormal, but had no pathology at autopsy. Thus, virtually all areas of transmural infarction will show akinesis/dyskinesis, but not all wall motion abnormalities are associated with any demonstrable pathology. Hypokinetic wall motion is very non-specific and may be seen with a subendocardial MI or histologically normal tissue. However many such areas of hypokinesis will be adjacent to an area of necrosis and probably represent mechanically or ischemically impaired but not infarcted tissue.

This clinicopathological study supported earlier work by Drs. Nixon, Narrahara and Smitherman of this institution (1980). They compared 2-D echo with technetium 99m pyrophosphate and thallium-201 scintigrams which provide information on the extent of myocardial infarction and perfusion abnormalities respectively (Figure 15).

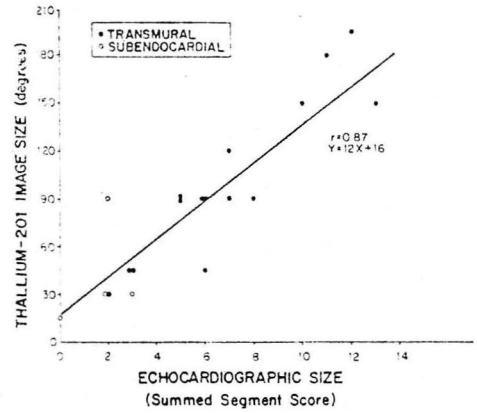


FIGURE 15. *Comparison between echocardiographically estimated summed segment scores and thallium-201 reperfusion scintigraphic image size in 19 patients.*

There was good agreement on infarct location in 26/29 cases, although thallium perfusion scintigraphy correlated much better with 2D echo than the PYP scans, r = 0.87 vs 0.74 respectively. The remaining variability is probably a result of wall motion abnormalities occurring next to ischemic or infarcted tissue. Similar findings in the setting of an acute MI have been reported by others (Visser et al., 1981; Gibson et al., 1982).

Clearly, better results will be obtained if one limits the study to patients with their first MI or patients with only transmural MI's, but this is not the usual CCU population. The technique for assessing wall motion can be fairly

simple, although substantial time is required to perform these analyses by 2-D echo (Parisi et al., 1981). Extensive wall motion abnormalities do have predictive value and may be useful in identifying high risk patients. Similar results in patients with unstable angina would support the use of 2-D echo in those patients as well (Nixon et al., 1982).

C. *Complications of Coronary Artery Disease*

1. Ischemic congestive heart failure can present a diagnostic problem in that the patients often have no history of anginal pain or infarction. The electrocardiogram may show only "poor R-wave progression" consistent with left ventricular hypertrophy and cardiomyopathy or old anterior infarction. The 2-D echo may provide useful diagnostic information in this setting. Segmental wall motion abnormalities localized to classical patterns of infarction are strong evidence for ischemic heart disease. If the cardiomyopathy is ischemic in origin, substantial areas of akinesis or dyskinesis may be seen with normal or increased wall motion in the basal portions of the heart.

2. Left ventricular thrombus may also be seen. This topic was extensively reviewed by Dr. J.V. Nixon in a recent Grand Rounds (September 2, 1982) so only a few salient points and key references will be mentioned (Assinger et al., 1981; Reeder et al., 1981; Stratton et al., 1982). The clots always form in an area of decreased wall motion; usually there is akinesis or dyskineses (i.e. paradoxical wall motion). Almost all are seen in the LV apex and may be recognized as an echo-dense mass protruding into the LV cavity in several different views. The clots, which are layered and not globular, blend into the LV wall contour and may be difficult to recognize. The myocardium is usually thin and of increased density due to scarring. A normal or increased wall thickness in a akinetic or dyskinetic segment would be presumptive evidence of LV clot. In the apical four chamber view, clots occur at the top of the sector image in an area of artefactually bright echoes. Gain settings and transducer position are very important if false positive and negative results are to be avoided. Two-dimensional echo has been said to be the best method of detection of LV clot associated with myocardial infarction, but its age or activity cannot be determined (Reeder et al., 1981). (Reeder had only a few cases to support this contention and although this writer personally agrees, there is little objective support for the statement.) Indium-111 labeled platelets have proven useful for showing active thrombus formation in the post-infarction patient, but the expense and availability of the technique have prevented its widespread use. Likewise, the significance of an LV clot demonstrated by 2-D echo is not known. There are case reports linking LV clot (Arvan and Plehn, 1982) to systemic emboli, but there are no studies which show definitively whether patients with LV clot are at increased risk of embolic events, or whether short-term anticoagulant therapy would prevent clots or their complications in susceptible individuals. The time course of development and resolution of these apical LV clots is also not known, although patients have been found with LV clot on admission with an acute MI and have developed clots while receiving full systemic heparinization (Friedman et al., 1982). Clearly, there are major difficulties associated with basing treatment decisions for LV thrombus on 2-D echo findings.

Nonetheless, echo laboratories continue to receive many requests for a 2-D echo to "rule out cardiac source of emboli." The yield of 2-D echo was carefully studied by Lovett et al. (1981) and Greenland et al. (1981) in the Annals of Internal Medicine. Their findings, discussed in an editorial by Larson et al. (1981), show very clearly that, in the absence of reasonable clinical suspicion of vegetations, myxoma, mitral valve prolapse, prosthetic valve thrombus, left atrial thrombus or left ventricular thrombus, the 2-D echo is not likely to be useful. They estimate that 40-50% of the $100 million spent annually for 2-D echoes in these patients would be saved if Lovett's and Greenland's recommendations were followed. The experience at Parkland would support these conclusions entirely. A careful history and cardiac exam would eliminate the need for a substantial portion of the requests currently being received.

3. Left ventricular aneurysms are thin walled dyskinetic segments of myocardial scar and are easily detected by 2-D echo (Baur et al., 1982). They can be a source of malignant arrhythmias or clots so their detection can be of value in some patients (vide infra).

4. Left ventricular pseudoaneurysms result when ventricular rupture occurs, but pericardial adhesions permit only a small localized leak of blood (Catherwood et al., 1980; Levy et al., 1981; Glover et al., 1981). With time, these pseudoaneurysms expand and produce cardiomegaly and congestive heart failure. They are prone to spontaneous rupture, which is a fatal complication. The true aneurysms have a mouth or opening the same width as the body of the aneurysm, while pseudoaneurysms have a narrow mouth usually less than half the diameter of the body of the pseudoaneurysm (Figure 16). They usually contain a large thrombus while true aneurysms are likely to contain smaller thrombi or none at all.

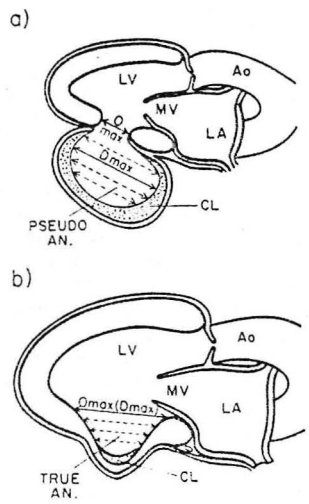


FIGURE 16 Schematic illustrations of the two types of left ventricular aneurysm. **a**, in a pseudoaneurysm (PSEUDO AN.) the maximal internal width of its neck or opening (O_{max}) is much smaller than the maximal parallel internal diameter (D_{max}) of the aneurysmal sac. **b**, in a true aneurysm (TRUE AN.) the maximal internal width of the mouth or opening (O_{max}) is usually equal to, or wider than, any other parallel internal diameter (• - - - - →) of the aneurysmal cavity. Therefore, in this example O_{max} represents the maximal parallel internal diameter (D_{max}). Ao = aorta; CL = clot; LA = left atrium; LV = left ventricle; MV = mitral valve.

5. Ventricular septal defects (VSD) and partial rupture of a papillary muscle may occur acutely following a myocardial infarction. Both conditions produce a loud systolic murmur and congestive heart failure or pulmonary edema. The VSD is usually visible with or without contrast while papillary muscle rupture produces a flail mitral leaflet or prolapse with an unattached papillary muscle head flying about the LV (Mintz et al., 1981; Erbel et al., 1981).

6. Right ventricular infarctions are difficult to recognize clinically although Croft et al. (1982) from this institution have shown the value of an RV precordial ECG. RV wall motion can be assessed by 2-D echo the same way as with an LV infarction. About 1/3 of the patients with an inferior MI will have RV involvement. D'Arcy and Nanda (1982) reported 10 patients with RV infarcts confirmed by autopsy (2), surgery (1) or ^{99}Tc-PYP scintigram (7). All had an akinetic diaphragmatic RV surface, 8 had RV dilation and 7 had abnormal septal motion. Contrast echocardiography was performed in 9/10 and confirmed the presence of tricuspid regurgitation.

In summary, 2-D echo's ability to visualize cardiac architecture and motion can be useful in estimating LV function, the extent of ischemic damage, and the presence of certain complications such as thrombus, aneurysm and pseudoaneurysm. It is not useful for examining the coronary arteries themselves in adults and is not a recommended or preferred way to diagnose the presence of ischemia or age of infarction. In institutions where nuclear cardiology is not well developed or readily accessible, it offers an alternative for scintigraphic assessment of wall motion and R and L ventricular size and function.

IX PERICARDIAL DISEASE

The pericardium is an echo-dense structure easily visualized by 2-D echo. Normally, the visceral and parietal layers or pericardium are opposed and moved in unison with the epicardium. Unfortunately, the normal density of the pericardium makes thickening difficult to detect. A doubling or tripling of the normal pericardial thickness is still at the limits of resolution for the 2-D echo. One should not depend on echo to diagnose pericardial thickening or fibrosis, even when substantial calcification is present.

Pericardial effusion is easily detected and was one of the first medical uses for echo (Feigenbaum et al., 1965). It is recognized as an echo free space between the epicardium and the pericardium with a loss of pericardial motion. The pericardium is attached to the great vessels anteriorly and to the pulmonary veins behind the left atrium. This means that pericardial effusions may surround both ventricles and the right atrium, but usually do not extend behind the left atrium. Larger pleural effusions do extend posteriorly, so that this location permits differentiation between the two types of effusion. Also, patients with large pleural effusions often have pericardial fluid as well. The intervening dense pericardial echoes and the failure of the pericardial fluid layer to extend behind the LA would be diagnostic of both pleural and pericardial effusions.

The 2-D echo is not as sensitive as the M-Mode in detecting small amounts of fluid, but the wider view of the 2-D echo offers several advantages. Loculated or localized fluid collections are more easily seen with 2-D as well as features such as fibrous strands, clots and tumor implants which provide a clue as to etiology. Neither M-Mode nor 2-D is particularly useful for quantitating the exact amount of fluid present. M-Mode formulae have been proposed but are subject to a wide range of errors (D'Cruz et al., 1977). It is probably sufficient to know if an effusion is present, if it is small, moderate or large, and if it has changed in size since the last echo or chest X-ray. These questions can be answered with a qualitative assessment and further quantitation is probably meaningless.

More important than diagnosing the presence of a pericardial effusion or its size is the problem of determining whether cardiac tamponade is present (Kronzon et al., 1983). Tamponade has traditionally been a clinically or hemodynamically diagnosed condition and echo could only determine whether a pericardiocentesis was likely to yield fluid or not. A number of abnormalities of chamber size and motion as well as valve excursion were noted but no clear cut relationship to tamponade was appreciated.

Shiina et al. (1979) published (in Japanese) observations on posterior displacement of the RV free wall in impending tamponade, but little work was done on this until this year. Weyman (1982) unaware of the report by Shiina, reported in his textbook of 2-D echo that he had seen patients with "a striking undulant motion of the anterior right ventricular wall in the region of the right ventricular outflow tract. This motion, on occasion, may be so pronounced that the right ventricular anterior wall may actually invaginate into the RV outflow tract during diastole. The significance of this phenomenon and its relationship to hemodynamic events, however remain unclear to date." This motion was only one of several abnormalities associated with large effusions and variably associated with electrical and mechanical alternans and tamponade (Gabor et al., 1971).

More recently, Armstrong et al. (1982), furthered the observation of Shiina in 91 patients including 17 with hemodynamically documented pericardial tamponade. They also found patients in whom the right ventricular free wall collapsed during diastolic filling - a time when the RV should be increasing in volume and dimensions. Thirteen of seventeen patients with tamponade had RV diastolic collapse. Wall motion reverted to normal after pericardiocentesis. They postulated that the tense pericardium became non-distensible and pericardial and RV pressures equalized during diastole. As a result, compression of the RV occurred. When pericardial pressure exceeds early diastolic RV pressure, the RV collapses, expanding only late, during atrial systole. Diastolic filling in the LV is less impeded due to LV wall stiffness and perhaps LV suction (Brecher GA, 1958). A differential effect in right and left ventricular compliance during acute cardiac tamponade in dogs has recently been demonstrated (Ditchey, 1981) and probably provides an excellent animal model for what has now been seen in the patients.

Although RV diastolic collapse appears to be a relatively reliable sign of impending tamponade, there are factors which affect its sensitivity and specificity. There is an assumption that the RV free wall is soft and easily compressed. If the patient has RVH or has had cardiac surgery with adhesions, serious tamponade may occur without diastolic collapse. Likewise, if the RV pressure is low due to decreased preload, one may see marked diastolic collapse in the absence of classical tamponade. Diastolic RV collapse is best considered a marker of equalization of RV diastolic and pericardial pressures. Most of the time, this will mean early embarrassment of RV filling by pericardial effusion, i.e. early tamponade. Its absence does not rule out tamponade, but in the setting of an otherwise normal RV, makes the diagnosis of tamponade very unlikely.

A combination of multiple echo demonstrated abnormalities of heart motion in tamponade associated with electrical and mechanical alternans provides a possible mechanism for these physical findings and are shown in Figures 17 and 18.

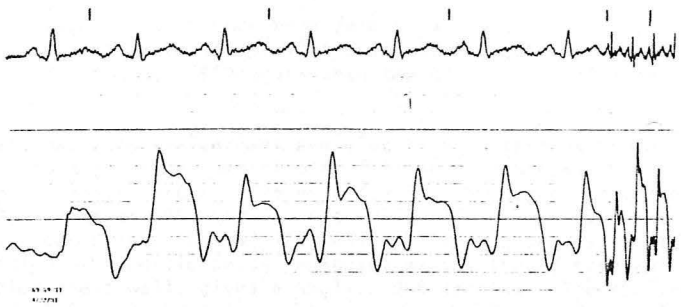


Figure 17. Pulmonary artery tracing from patient whose echo is shown in Figure 18. Note alternating pressure peaks. This was also present in his arterial pulse.

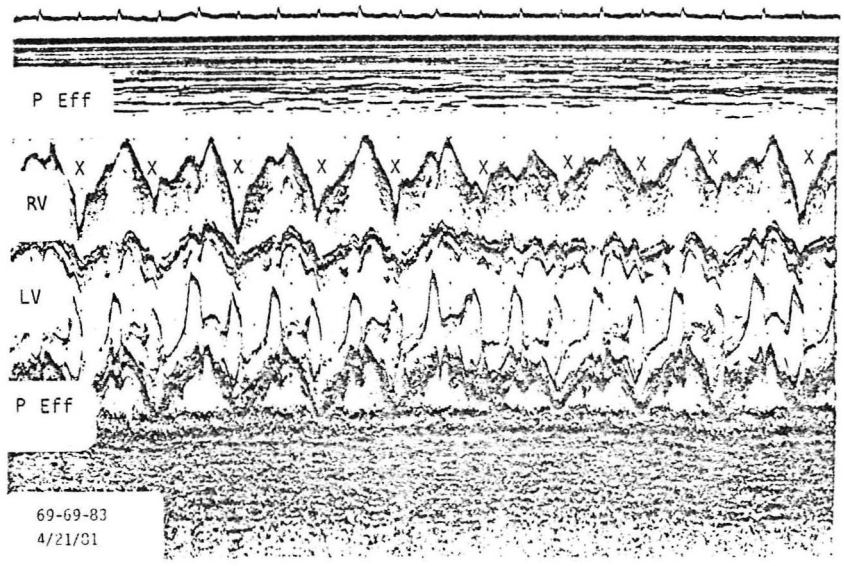


Figure 18. M-Mode echo demonstrating a large anterior and posterior pericardial effusion with electrical alternans. The smaller QRS occurs when the RV is most collapsed.

The patient has pulsus alternans and electrical alternans produced by pericardial tamponade. Right ventricular filling is enhanced by inspiratory augmentation of venous return. The expanded RV, now closer to the anterior chest wall, gives a larger QRS complex on the ECG. This relatively large RV stroke volume is ejected into the LV; increased LV filling causes RV diastolic collapse. With ejection, the aortic pulse is increased but the RV free wall, farther from the anterior chest wall, gives a smaller QRS complex. The smaller RV stroke volume underfills the LV on the next beat so the RV is allowed to fill more normally. The alternans can occur even when the patient has atrial fibrillation. Withdrawal of a small amount of fluid provides enough pericardial space for the RV to fill normally and the alternans disappears.

There is probably no better example than the 2-D echo findings in tamponade to show that echo can provide important physiological information, but that information must be interpreted carefully and integrated into the overall clinical picture if the correct diagnosis and treatment are to be chosen.

X CONTRAST ECHOCARDIOGRAPHY

Because of the large difference in attenuation of sound in air compared with water, gas bubbles in blood are highly visible on 2-D echoes. Gramiak first used microbubbles to perform contrast echocardiography in 1969. The microbubbles produce a dense opaque cloud which permits detection of flow, intracardiac shunts, endocardial surfaces and unusually located vessels or structures. The microbubbles are completely cleared by the pulmonary capillaries, so injection of contrast material into a peripheral vein will opacify only right-sided structures. The appearance of contrast in the left heart means a right-to-left cardiac shunt is present.

A number of agents are used to provide echocardiographic contrast, but reliability varies. Normal saline or 5% dextrose in water can be aerated by agitation. When injected, the microbubbles of air produce a contrast effect. These are obviously safe and inexpensive materials but the contrast is not dense and persists a few heart beats at the most. Indocyanine green, a harmless dye used to measure cardiac output, acts as a soap to stabilize dissolved gas bubbles. It provides better microbubble contrast than D5W or normal saline, but its bubbles also last only a few seconds and the material is expensive.

To provide dense, sustained opacification of the right heart and associated vessels, Wang Xinfong of the People's Republic of China suggested the use of hydrogen peroxide to produce intravenous microbubbles (Wang, 1979). He performed studies in several hundred cardiac patients with only minor complaints in 11 of 437 injections. We have performed studies with 0.2% H_2O_2, and other modifications to Wang's technique. Dilute H_2O_2 appears to be a superb contrast agent (Gaffney, 1983), although it is not recommended in patients with severe pulmonary hypertension or large right-to-left shunts.

With 2-D contrast echocardiography, one can reliably detect tricuspid and pulmonary regurgitation, as well as intracardiac shunts. Small right-to-left shunts, undetectable by green dye injections are easily found by 2-D echo. Atrial and ventricular septal defects are reliably found if the degree of shunting is significant or if right-to-left shunts of any size are present. The technique has been extremely useful in pediatric cardiology since it permits qualitative and semi-qualitative assessment of flow with a harmless peripheral venous injection. In many cases, cardiac catheterization can be avoided or shortened when contrast echocardiography is performed.

XI SUMMARY

In only seven years, two-dimensional echocardiography has become an important and widely utilized cardiac procedure. Its completely non-invasive nature makes it an excellent method for evaluating cardiac anatomy and physiology. When used properly, it often provides the clinical diagnosis or at least gives guidance in selecting the next more expensive or more invasive diagnostic procedure. Unfortunately, the benign nature of the 2-D echo has led to substantial over-utilization. The test is often ordered when new or useful information is not likely to be acquired or acted upon This test, like most others, is best used when specific diagnostic and therapeutic questions are being asked. A thorough understanding of a) the physiological information already available for a given patient, b) the information likely to be obtained from a 2-dimensional echo-cardiogram, and, c) the specific technical and methodological limitations of the procedure in a given patient should result in better care for the patient at a lower cost.

Two-dimensional echocardiography presents a unique opportunity for the physician to "see" the patient's heart and understand the physiological basis of the symptoms and physical findings. It is hoped that the material presented today will encourage the internist to take advantage of this opportunity.

REFERENCES

Arita M, Ueno Y, Masuyama Y: Detection of intracardiac thrombi in a case of cardiomyopathy by two dimensional echocardiography. Br Heart J 47:397-399, 1982.

Armstrong WF, Schilt BF, Halper DJ, Dillon JC, Feigenbaum H: Diastolic collapse of the right ventricle with cardiac tamponade: An echocardiographic study. Circulation 65:1491-1496, 1982.

Arvan S, DuBois B: M-mode and two-dimensional echocardiography of pulmonary emboli. JCU 10:177-179, 1982.

Arvan S, Plehn J: Embolization of a left ventricular mural thrombus: verification by two-dimensional echocardiography. Arch Intern Med 142:1952-1953, 1982.

Asinger RW, Mikell FL, Elsperger J, Hodges M: Incidence of left-ventricular thrombosis after acute transmural myocardial infarction. Serial evaluation by two-dimensional echocardiography. N Engl J Med 305:297-302, 1981.

Asinger RW, Mikell FL, Sharma B, Hodges M: Observations on detecting left ventricular thrombus with two dimensional echocardiography: emphasis on avoidance of false positive diagnoses. Am J Cardiol 47:145-156, 1981.

Baran A, Nanda NC, Falkoff M, Barold SS, Gallagher JJ: Two-dimensional echocardiographic detection of arrhythmogenic right ventricular dysplasia. Am Heart J 103:1066-1067, 1982.

Baur HR, Daniel JA, Nelson RR: Detection of left ventricular aneurysm on two dimensional echocardiography. Am J Cardiol 50:191-196, 1982.

Böcker K, Köhler E, Kuhn H, Körfer R: Holosystolic mitral valve prolapse and "SAM" after surgical treatment of hypertrophic obstructive cardiomyopathy (author's translation). Z Kardiol 70:403-405, 1981.

Brandenburg RO, Tajik AJ, Edwards WD, Reeder GS, Shub C, Seward JB: Accuracy of 2-dimensional echocardiographic diagnosis of congenitally bicuspid aortic valve: Echocardiographic-anatomic correlation in 115 patients. Am J Cardiol 51:1469-1473, 1983.

Brecher GA: Critical review of recent work on ventricular diastolic suction. Circ Res 6:554-566, 1958.

Brock RC: Functional obstruction of the left ventricle. Guy's Hosp Rep 106: 221-238, 1957.

Buja LM, Roberts WC: Iron in the heart: etiology and clinical significance. Am J Med 51:209-221, 1971.

Catherwood E, Mintz GS, Kotler MN, Parry WR, Segal BL: Two-dimensional echocardiographic recognition of left ventricular pseudoaneurysm. Circulation 62:294-303, 1980.

Chandraratna PAN, Aronow WS: Left main coronary arterial patency assessed with cross-sectional echocardiography. Am J Cardiol 46:91-94, 1980.

Come PC, Bulkley BH, Goodman ZD, Hutchins GM, Pitt B, Fortuin NJ: Hypercontractile cardiac states simulating hypertrophic cardiomyopathy. Circulation 55:901-908, 1977.

Croft C, Rude R, Willerson JT: Right ventricular infarction - changing concepts. Primary Cardiol (In Press)

D'Arcy B, Nanda NC: Two-dimensional echocardiographic features of right ventricular infarction. Circulation 65:167-173, 1982.

D'Cruz IA, Collison HK, Gerrardo L, Hensel P: Two-dimensional echocardiographic detection of staphylococcal vegetation attached to calcified mitral annulus. Am Heart J 103:295-298, 1982.

D'Cruz I, Panetta F, Cohen H, Glick G: Submitral calcification or sclerosis in elderly patients: M-Mode and two-dimensional echocardiography in "Mitral Annulus Calcification." Am J Cardiol 44:31-38, 1979.

D'Cruz I, Prabhu R, Cohen HC, Glick G: Potential pitfalls in quantitation of pericardial effusions by echocardiography. Br Heart J 39:529-535, 1977.

DeMaria AN, Bommer W, Joye J, Lee A, Bouteller J, Mason DT: Value and limitations of cross-sectional echocardiography of the aortic valve in the diagnosis and quantification of valvular aortic stenosis. Circulation 62:304-312, 1980.

DeMaria AN, Bommer W, Lee G, Mason DT: Value and limitations of two-dimensional echocardiography in assessment of cardiomyopathy. Am J Cardiol 46:1224-1231, 1980.

Ditchey R, Engler R, LeWinter M, Pavelec R, Bhargava V, Covell J, Moores W, Shabetai R: The role of the right heart in acute cardiac tamponade in dogs. Circ Res 48:701-711, 1981.

Erbel R, Schweizer P, Bardos P, Meyer J: Two-dimensional echocardiographic diagnosis of papillary muscle rupture. Chest 79:595-598, 1981.

Feigenbaum H: Echocardiography. Lea & Febiger, Philadelphia, Pa., 1981.

Feigenbaum H, Waldhausen JA, Hyde LP: Ultrasound diagnosis of pericardial effusion. JAMA 191:711-714, 1965.

Fenoglio JJ Jr, McAllister HA Jr, DeCastro CM, Davia JE, Cheitlin MD: Congenital bicuspid aortic valve after age 20. Am J Cardiol 39:164-169, 1977.

Friedman MJ, Carlson K, Marcus FI, Woolfenden JM: Clinical correlations in patients with acute myocardial infarction and left ventricular thrombus detected by two-dimensional echocardiography. Am J Med 72:894-898, 1982.

Friedman MJ, Sahn DJ, Goldman S, Eisner DR, Gittinger NC, Lederman FL, Puckette CM, Tiemann JJ: High predictive accuracy for detection of left main coronary artery disease by antilog signal processing of two-dimensional echocardiographic images. Am Heart J 103:194-201, 1982.

Gabor GE, Winsberg F, Bloom HS: Electrical and mechanical alternans in pericardial effusion. Chest 59:341-344, 1971.

Gaffney FA, Lin JC, Peshock RM, Bush L, Buja LM: Hydrogen peroxide contrast echocardiography. Am J Cardiol (In Press, September 1983)

Gaffney FA, Nicod P, Lin JC, Rude RE: Non-invasive recognition of the parchment right ventricle (Uhl's anomaly arrhythmogenic right ventricular dysplasia) syndrome. Clin Cardiol 6:235-242, 1983.

Gatewood RP Jr, Nanda NC: Differentiation of left ventricular pseudoaneurysm from true aneurysm with two dimensional echocardiography. Am J Cardiol 46:869-878, 1980.

Geill T: Calcification of the left annulus fibrosus (230 cases). Acta Med Scand Suppl 239:153-156, 1950.

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 49:1110-1119, 1982.

Glover MU, Hagan AD, Vieweg WV, Ceretto WJ: Pseudoaneurysm of the left ventricle diagnosed by two-dimensional echocardiography: case report. Milit Med 146:696-698, 1981.

Godley RW, Green D, Dillon JC, Rogers EW, Feigenbaum H, Weyman AE: Reliability of two-dimensional echocardiography in assessing the severity of valvular aortic stenosis. Chest 79:657-662, 1981.

Goodwin JR: Clarification of the cardiomyopathies. Mod Concepts Cardiovascular Dis 41:41-51, 1972.

Gorlin R, Gorlin SA: Hydraulic formula for calculation of area of the stenotic mitral valve, other cardiac valves and central circulatory shunts. Am Heart J 41:1-29, 1951.

Gramiak R, Shah PM, Kramer DH: Ultrasound cardiography: Contrast studies in anatomy and function. Radiology 92:939-948, 1969.

Greenland P, Knopman DS, Mikell FL, Asinger RW, Anderson DC, Good DC: Echocardiography in diagnostic assessment of stroke. Ann Intern Med 95:51-53, 1981.

Henry WL, Clark CE, Epstein SE: Asymmetrical septal hypertrophy (ASH) echocardiographic identification of the pathognomonic anatomic abnormality of IHSS. Circulation 47:225-233, 1973.

Henry WL, Clark CE, Glancy DL, Epstein SE: Echocardiographic measurement of the left ventricular outflow gradient in idiopathic hypertrophic subaortic stenosis. N Engl J Med 288:989-993, 1973.

Henry WL, DeMaria A, Gramiak R, King DL, Kisslo JA, Popp RL, Sahn DJ, Schiller NB, Tajik A, Teichholz LE, Weyman AE: Report of the American Society of Echocardiography committee on nomenclature and standards in two-dimensional echocardiography. Circulation 62:212-215, 1980.

Hiraishi S, Yashiro K, Kusano S: Non-invasive visualization of coronary artery aneurysm in infants and young children with mucocutaneous lymph node syndrome with two-dimensional echocardiography. Am J Cardiol 43:1220-1223, 1979.

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 103:814-822, 1982.

Horowitz RS, Morganroth J, Parrotto C, Chen CC, Soffer J, Pauletto FJ: Immediate diagnosis of acute myocardial infarction by two-dimensional echocardiography. Circulation 65:323-329, 1982.

Howard RJ, Drobac M, Rider WD, Keane TJ, Finlayson J, Silver MD, Wigle ED, Rakowski H: Carcinoid heart disease: diagnosis by two-dimensional echocardiography. Circulation 66:1059-1065, 1982.

Huxley RL, Gaffney FA, Corbett JR, Firth BG, Peshock R, Nicod P, Curry G, Lewis SE, Willerson JT: Early detection of left ventricular dysfunction in chronic aortic regurgitation as assessed by contrast angiography, echocardiography and rest and exercise scintigraphy. Am J Cardiol 15:1542-1550, 1983.

Jaffe AS, Geltman EM, Rodey GE, Uitto J: Mitral valve prolapse: a consistent manifestation of type IV Ehlers-Danlos syndrome. The pathogenetic role of the abnormal production of type III collagen. Circulation 64:121-125, 1981.

Kawasaki T, Kosaki F, Okawa S, Shigematsu I, Yanagawa H: A new infantile acute febrile mucocutaneous lymph node syndrome (MLNS) prevailing in Japan. Pediatrics 54:271-276, 1974.

Kisslo J: Two-dimensional echocardiography. Radiol Clin North Am 18:105-115, 1980.

Kotler MN, Mintz GS, Parry WR, Segal BL: M mode and two dimensional echocardiography in mitral and aortic regurgitation: pre- and postoperative evaluation of volume overload of the left ventricle. Am J Cardiol 46:1144-1152, 1980.

Kotler MN, Mintz GS, Parry WR, Segal BL: Two dimensional echocardiography in congenital heart disease. Am J Cardiol 46:1237-1246, 1980.

Kotler MN, Mintz GS, Segal BL, Parry WR: Clinical uses of two dimensional echocardiography. Am J Cardiol 45:1061-1082, 1980.

Kronzon I, Cohen ML, Winer HE: Contribution of echocardiography to the understanding of the pathophysiology of cardiac tamponade. J Am Coll Cardiol 1: 1180-1182, 1983.

Larson EB, Stratton JR, Pearlman AS: Selective use of two-dimensional echocardiography in stroke syndromes. (editorial) Ann Intern Med 95:112-114, 1981.

Levine HJ, Isner J, Salem DN: Primary vs secondary mitral valve prolapse: Clinical features and implications. Clin Cardiol 5:371-375, 1982.

Levy R, Rozanski A, Charuzi Y, Childs W, Waxman A, Corday E, Berman DS: Complementary roles of two-dimensional echocardiography and radionuclide ventriculography in ventricular pseudoaneurysm diagnosis. Am Heart J 102:1066-1069, 1981.

Lew W, Henning H, Schelbert H, Karliner JS: Assessment of mitral valve E point-septal separation as an index of left ventricular performance in patients with acute and previous myocardial infarction. Am J Cardiol 41:836-845, 1978.

Longhurst JC, Kelly AR, Gonyea WJ, Mitchell JH: Echocardiographic left ventricular masses in distance runners and weight lifters. J Appl Physiol 48:154-162, 1980.

Lovett JL, Sandok BA, Guiliani ER, Nasser FN: Two-dimensional echocardiography in patients with focal cerebral ischemia. Ann Intern Med 95:1-4, 1981.

Maron BJ, Epstein SE: Hypertrophic cardiomyopathy: A discussion of nomenclature. Am J Cardiol 43:1242-1244, 1979.

Maron BJ, Gottdiener JS, Bonow RO, Epstein SE: Hypertrophic cardiomyopathy with unusual locations of left ventricular hypertrophy undetectable by M-mode echocardiography. Identification by wide-angle two-dimensional echocardiography. Circulation 63:409-418, 1981a.

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 48:418-428, 1981b.

Martin RP, Meltzer RS, Chia BL, Stinson EB, Rakowski H, Popp RL: Clinical utility of two dimensional echocardiography in infective endocarditis. Am J Cardiol 46:379-385, 1980.

Massie BM, Schiller NB, Ratshin RA, Parmley WW: Mitral-septal separation: New echocardiographic index of left ventricular function. Am J Cardiol 39:1008-1016, 1977.

Mintz GS, Victor MF, Kotler MN, Parry WR, Segal BL: Two-dimensional echocardiographic identification of surgically correctable complications of acute myocardial infarction. Circulation 64:91-96, 1981.

Nakamura K, Suzuki S, Satomi G, Hayashi H, Hirosawa K: Detection of mitral ring abscess by two-dimensional echocardiography. Circulation 65:816-819, 1982.

Nanda NC, Gramiak R, Manning J, Mahoney EB, Lipchik EO, DeWeese JA: Echocardiographic recognition of the congenital bicuspid aortic valve. Circulation 49:870-875, 1974.

Nichol PM, Gilbert BW, Kisslo JA: Two-dimensional echocardiographic assessment of mitral stenosis. Circulation 55:120-128, 1977.

Nixon JV, Brown CN, Smitherman TC: Identification of transient and persistent segmental wall motion abnormalities in patients with unstable angina by two-dimensional echocardiography. Circulation 65:1497-1503, 1982.

Nixon JV, Narahara KA, Smitherman TC: Estimation of myocardial involvement in patients with acute myocardial infarction by two-dimensional echocardiography. Circulation 62:1248-1255, 1980.

Parisi AF, Moynihan PF, Folland ED, Feldman CL: Quantitative detection of regional left ventricular contraction abnormalities by two-dimensional echocardiography. II. Accuracy in coronary artery disease. Circulation 63:761-767, 1981.

Perloff JK: Evolving concepts of mitral valve prolapse. New Engl J Med (Editorial) 307:369-370, 1982.

Pollick C, Pittman M, Filly K, Fitzgerald PJ, Popp RL: Mitral and aortic valve orifice area in normal subjects and in patients with congestive cardiomyopathy: determination by two dimensional echocardiography. Am J Cardiol 49:1191-1196, 1982.

Popp RL, Rubenson DS, Tucker CR, French JW: Echocardiography: M-mode and two-dimensional methods. Ann Intern Med 93:844-856, 1980.

Radford DJ, Bloom KR, Izukawa R, Moes CAF, Rowe RD: Echocardiographic assessment of bicuspid aortic valves. Circulation 53:80-85, 1976.

Reeder GS, Seward JB, Tajik AJ: The role of two-dimensional echocardiography in coronary artery disease: a critical appraisal. Mayo Clin Proc 57:247-258, 1982.

Reeder GS, Tajik AJ, Seward JB: Left ventricular mural thrombus: two-dimensional echocardiographic diagnosis. Mayo Clin Proc 56:82-86, 1981.

Reichek N: Uses and abuses of two-dimensional echocardiography. Int J Cardiol 1:221-227, 1982.

Roberts WC: The congenitally bicuspid aortic valve: A study of 85 autopsy cases. Am J Cardiol 26:72-83, 1970.

Rogers EW, Feigenbaum H, Weyman AE, Godley RW, Vakili ST: Evaluation of left coronary artery anatomy *in vitro* by cross-sectional echocardiography. Circulation 62:782-787, 1980a.

Rogers EW, Feigenbaum H, Weyman AE, Godley RW, Johnston KW, Eggleton RC: Possible detection of atherosclerotic coronary calcification by two-dimensional echocardiography. Circulation 62:1046-1053, 1980b.

Rossen RM, Goodman DJ, Ingham RE, Popp RL: Echocardiographic criteria in the diagnosis of idiopathic hypertrophic aortic stenosis. Circulation 50:747-751, 1974.

Sahn DJ: Real-time two-dimensional echocardiography. J Pediatr 99:175-185, 1981.

Shiina S, Yaginuma T, Kondo K, Kawai N, Hosoda S: Echocardiographic evaluation of impending cardiac tamponade. J Cardiogr 9:555, 1979.

Silverman KJ, Hutchins GM, Weiss JL, Moore GW: Catenoidal shape of the interventricular septum in idiopathic hypertrophic subaortic stenosis: two dimensional echocardiographic confirmation. Am J Cardiol 49:27-32, 1982.

Simon MA, Liu SF: Calcification of the mitral valve annulus and its relationship to functional valve disturbances. Am Heart J 48:497-505, 1954.

Siqueira-Filho AG, Cunha CL, Tajik AJ, Seward JB, Schattenberg TT, Giuliani ER: M-mode and two-dimensional echocardiographic features in cardiac amyloidosis. Circulation 63:188-196, 1981.

Stewart JA, Silimperi D, Harris P, Wise NK, Fraker TD, Kisslo JA: Echocardiographic documentation of vegetative lesions in infective endocarditis: Clinical implications. Circulation 61:374-380, 1980.

Stratton JR, Lighty GW Jr, Pearlman AS, Ritchie JL: Detection of left ventricular thrombus by two-dimensional echocardiography: sensitivity, specificity, and causes of uncertainty. Circulation 66:156-166, 1982.

Tajik AJ, Seward JB, Hagler DJ, Mair DD, Lie JT: Two dimensional real-time ultrasonic imaging of the heart and great vessels. Mayo Clinc Proc 53:271-302, 1978.

Teare D: Asymmetrical hypertrophy of the heart in young adults. Br Heart J 20:1-8, 1958.

Veyrat C, Kalmanson D, Farjon M, Manin JP, Abitbol G: Non-invasive diagnosis and assessment of tricuspid regurgitation and stenosis using one and two dimensional echo-pulsed Doppler. Br Heart J 47:596-605, 1982.

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 47:1020-1025, 1981.

Wang R, Lee WT, Mok CK: Two-dimensional echocardiographic features of Björk-Shiley aortic prosthetic valve dehiscence. Thorax 37:540-541, 1982.

Wang X, Wang J, Huang Y, Cai C: Contrast echocardiography with hydrogen peroxide: I - Experimental study. Chinese Med J 92:595-599, 1979.

Wann LS, Feigenbaum H, Weyman AE, Dillon JC: Cross-sectional echocardiographic detection of rheumatic mitral regurgitation. Am J Cardiol 41:1258-1263, 1978.

Weiss JL, Bulkley BH, Hutchins GM, Mason SJ: Two-dimensional echocardiographic recognition of myocardial injury in man: comparison with postmortem studies. Circulation 63:401-408, 1981.

Weyman AE: Cross-sectional echocardiography. Lea & Febiger, Philadelphia, Pa., 1982.

Weyman AE, Feigenbaum H, Dillon JC, Johnston KW, Eggleton RC: Non-invasive visualization of the left main coronary artery by cross-sectional echocardiography. Circulation 54:169-174, 1976.

Weyman AE, Wann LS, Feigenbaum H, Dillon JC: Mechanism of abnormal septal motion in patients with right ventricular volume overload. Circulation 54:179-186, 1976.

White H, Wynne J: Clinical utility of echocardiography in suspected infective endocarditis. (Abstract) Circulation 66(Part II):103, 1982.

Willerson JT: Echocardiography after acute myocardial infarction. (Editorial) N Engl J Med 300:87-89, 1979.

Winer H, Kronzman I, Glassman E: Echocardiographic findings in severe paradoxical pulse due to pulmonary embolization. Am J Cardiol 40:808-810, 1977.

Yamaguchi H, Ishimura T, Nishiyama S: Hypertrophic non-obstructive cardiomyopathy with giant negative T waves (apical hypertrophy): Ventriculographic and echocardiographic features in 30 patients. Am J Cardiol 44:401-412, 1979.

Yoshida H, Maeda T, Funabashi T, Nakaya S, Takabatake S, Taniguchi N: Subcostal two-dimensional echocardiographic imaging of peripheral right coronary artery in Kawasaki disease. Circulation 65:956-961, 1982.

Yoshikawa J, Yanagihara K, Owaki T, Kato H, Takagi Y, Okumachi F, Fukaya T, Tomita Y, Baba K: Cross-sectional echocardiographic diagnosis of coronary artery aneurysms in patients with the mucocutaneous lymph node syndrome. Circulation 59:133-139, 1979.

Zanolla L, Marino P, Nicolosi GL, Peranzoni PF, Poppi A: Two-dimensional echocardiographic evaluation of mitral valve calcification. Sensitivity and specificity. Chest 82:154-157, 1982.