# ESSENTIALS OF ECHOCARDIOGRAPHY #2

## Diseases of the Heart Valves

Joseph A. Kisslo, MD
David B. Adams, RDCS
Graham J. Leech, MA

This unit covers the application of both M-mode and two-dimensional echocardiography in the investigation of acquire diseases of the heart valves, discussing the advantages and disadvantages of each technique. Subsequent units will discuss the applications of echocardiography in heart muscle disease and congenital disease.

## The Atrioventricular Valves

### Mitral Stenosis

In mitral stenosis, fusion of the commissures between the mitral valve leaflets causes obstruction to left ventricular inflow. The leaflets also become infiltrated with fibrous tissue, which produces shrinkage, thickening and immobility, particularly at the tips of the leaflets **(Fig. 1)**. Over a longer period, the fibrotic tissue may calcify and eventually the valve becomes a funnel-shaped structure that impedes blood flow as much by its rigidity as by actual stenosis of the orifice.

Chronic elevation of left atrial pressure in mitral stenosis usually causes the chamber to dilate, although the extent to which this occurs depends additionally on atrial wall stiffness. Elevated filling pressure also results in pulmonary venous hypertension, and hence a rise in pulmonary artery pressure. If severe, this leads eventually to right ventricular hypertrophy and the development of tricuspid regurgitation.

The pathological changes in the valve apparatus produce dramatic alterations to the echocardiographic images **(Fig. 2)**. On the M-mode recording, thickening of the leaflets and reduction in their mobility can be appreciated. Commissural fusion alters the motion of the posterior leaflet; instead of moving in the opposite direction to that of the anterior, it is pulled forward when the valve opens at the beginning of diastole. In the normal valve, rapid early diastolic filling of the left ventricle means that the valve can partially close in mid-diastole, and it then reopens during atrial systole.

This sequence produces the characteristic “M” shape of the recording, with the down slope of the “M” designated as the “E-F slope”. With mitral stenosis, however rapid filling is not possible, so the valve leaflets have to remain as widely separated as they can throughout diastole, and the “M” disappears. Another reason for a different pattern of leaflet motion is that atrial fibrillation is frequently present, and this eliminates the late diastolic reopening.

These changes are also apparent on two-dimensional recordings. **Fig. 3** shows commissural fusion causing the valve leaflets to assume a dome shape in diastole, with the anterior leaflet forming a characteristic "elbow" due to its tethering to the posterior leaflet. The hallmark of rheumatic mitral valve disease is the restriction of motion of the anterior mitral valve leaflet tip. Abrupt halting of the early diastolic opening motion coincides with the opening snap evident on auscultation.

Echocardiography is a very sensitive method for detecting mitral stenosis and simple inspection of the recordings **(Fig. 4)** can often differentiate between mild and very severe stenosis. Quantification of severity has, however, proved more difficult by M-mode. Early attempts measured the "E-F slope", which indicates the difference between early and mid-diastolic leaflet position, but these proved unsatisfactory because the E-F slope is influenced by factors such as stroke output and ventricular stiffness.

Traditional approaches for the calculation of mitral valve area involve cardiac catheterization. The value of cardiac catheterization methods for assessing a patient with mitral stenosis is questionable; the end-diastolic valve gradient is influenced by heart rate, stroke volume, and the presence of mitral aortic regurgitation, which also invalidate estimation of valve orifice area by the "Gorlin" formula.

Two-dimensional echocardiography is able to visualize the mitral orifice directly in diastole, thus providing information previously available only to the surgeon. Using the parasternal short-axis view, it is possible to differentiate between various orifice shapes that are indistinguishable by M-mode **(Fig. 5)**. Planimetry of a still-frame image **(Fig. 6)** allows the mitral valve orifice area to be measured. Great care is needed to ensure that the image selected is accurate. If the scan plane transects the valve obliquely, above or below the region of maximum constriction, the orifice will appear too large; if the wrong part of the cardiac cycle is used, it will appear too small. Other technical difficulties arise from limitations of lateral resolution of the instrument, and from multiple reverberations produced by valve calcification. In spite of these problems, in the hands of an experienced operator two-dimensional echocardiography has proved the best method available for preoperative estimation of mitral valve area. Thoughtful clinical uses of the two-dimensional echocardiographic data will obviate the need for cardiac catheterization in most patients.

In the setting of previous mitral valve commissurotomy, however, caution should be exercised. Such direct planimetric methods have not been shown to be as reliable in this setting.

Doppler echocardiographic methods have also been shown to be useful for estimation of mitral valve area using the pressure half-time method and for the estimation of mean mitral gradient. Such methods are particularly useful in a patient with previous commissurotomy and also serve to double-check the direct planimetric approaches.

Of course, with significant mitral stenosis, the left atrium will dilate. Such dilatation can be observed in any view of the left atrium **(Fig. 7)**. Estimation of left atrial size using echocardiography is very important. The normal left atrial size is below 4 cm in adults and dilates with chronic mitral stenosis, insufficiency, or atrial fibrillation. Patients with severe left atrial dilatation are usually very difficult to convert to normal sinus rhythm with cardioversion. The echocardiogram helps to select patients in which cardioversion might or might not be successful

## Mitral Annulus Calcification

In the elderly patient, calcium is sometimes present in the mitral valve annulus **(Fig. 8)** and it produces a dense band of echoes behind the posterior valve leaflet adjacent to the annulus. However, the leaflets themselves remain thin, and there is no commissural fusion, so they separate normally in diastole. Since the calcium absorbs, or reflects, much of the ultrasound energy an area of shadowing may be seen posterior to the calcific ring.

Mitral valve annular calcification can also be detected on M-mode. Due to lack of spatial information, however, the M-mode recordings are difficult to interpret except by experienced observers.

## Atrial Tumors and Clots

An uncommon but important condition is a left atrial tumor, most frequently a myxoma, which partially bocks the mitral orifice and can, therefore, be indistinguishable from mitral stenosis on clinical examination.

In most cases, M-mode recordings are diagnostic of this condition. As **Fig. 9** shows, the tumor produces an amorphous cloud of echoes that appears behind the mitral valve leaflet shortly after the valve opens. Depending on the mobility of the tumor, other recordings will show it in the left atrium during systole, and descending into the body of the left ventricle later in diastole. The anterior mitral leaflet is usually normal in thickness in this situation. The opening of the normal mitral leaflet in diastole, followed by the appearance of the mass lesion an instant later serves to differentiate the M-mode appearance of mitral stenosis and atrial myxoma.

The spatial presentation of echocardiographic data offered by two-dimensional echocardiography makes the diagnosis of left atrial myxoma quite easy in most cases **(Fig. 10)**. The size, mobility, and point of attachment of the tumor can be determined with some accuracy. Its motion exhibits a “rocking” action about the point of attachment, normally to the interatrial septum just behind the anterior mitral leaflet. Indeed, if it appears to be attached elsewhere it is more likely to be a malignant myosarcoma or lymphoma.

Additional two-dimensional short-axis and four-chamber views **(Fig. 11)** not only provide further graphic evidence of the tumor, but also greatly reduce the chance of accidentally missing the diagnosis because of small tumor size, lack of mobility, or abnormal attachment point. As a result, echocardiography obviates the need for invasive and dangerous techniques such as angiography.

Such left atrial tumors have, infrequently, been reported to the bilateral. The usual mechanism for extension into the right atrium is growth of the tumor through the fossa ovalis **(Fig. 12)**. Careful examination of both the left and right atria as well as the interatrial septum are required in such patients as it gives useful information to the surgeon in planning the surgical approach. Surgical removal of atrial myxoma requires resection of the atrial septum at the point of attachment to prevent regrowth of the myxoma **(Fig. 13)**.

Myxomas may occur in any cardiac chamber and on any cardiac valve. The second most common area of occurrence after the left atrium is the right atrium **(Fig. 14)**. As with left atrial

locations such tumors often prolapse into the valve orifice in diastole, impeding forward flow of blood through the heart **(Fig. 15)**. Wherever their location, the echocardiographic identification of such mass lesions is indication for surgical removal in suitable surgical candidates due to their propensity for obstruction to flow and the high rates of embolization of such tumors.

## Tricuspid Stenosis

Organic tricuspid stenosis is an unusual but important finding. The clinical signs can easily be masked by those of the mitral valve disease with which it is almost invariably associated. Because of the low pressures in the right side of the heart, significant tricuspid stenosis can produce transvalvular pressure gradients of 1mmHg or less, which are hard to detect by cardiac catheterization, even when suspected in advance.

Echocardiography, in contrast, is a very sensitive method for detecting tricuspid stenosis, and examination of the tricuspid valve should always be included in all patients, particularly in patients with rheumatic aortic or mitral valve disease. **Fig. 16** shows an M-mode scan from tricuspid to mitral valve in a patient with both mitral and tricuspid stenosis. Similar changes are evident in both valves, namely reduced mobility, leaflet thickening, and reduced or reversed motion of the posterior leaflet.

Clear two-dimensional visualization of the stenotic tricuspid orifice is not as easy as with the mitral valve. This is due to the lack of an appropriate window by which to access the plane of the open tricuspid valve. In fact, it is a rare patient where the severity of tricuspid stenosis may be planimetered. As with mitral stenosis, the severity of tricuspid stenosis may be estimated by Doppler methods and has been shown to be quite helpful in this regard.

The apical four-chamber view, however, will reveal tethering of the tricuspid leaflet tips in patients with tricuspid stenosis, **Fig. 17** shows fusion of both the tricuspid and mitral valve leaflet tips during diastole in tricuspid and mitral stenosis.

## Mitral Regurgitation

Without the use of Doppler, there are no specific signs of mitral regurgitation by either M-mode or two-dimensional echocardiography. Nevertheless, echocardiography can contribute significantly to the assessment of a patient with mitral regurgitation by indicating the hemodynamic severity and by determining the pathogenesis, both of which have important prognostic implications.

The primary echocardiographic features of mitral regurgitation are volume overloading of both the left atrium and the left ventricle. This results from the transfer of a proportion of the stroke volume back and forth between the two chambers. Wall thickness is normal (though by virtue of enlargement, wall mass increases). Left ventricular volume overload is recognized by an end-diastolic dimension greater than 5.5 cm, and hyperdynamic wall motion, most noticeable on the interventricular septum **(Fig. 18)**. Such profound changes noted by echocardiography require that the hemodynamic load from mitral regurgitation be severe.

It is axiomatic that severe mitral regurgitation must cause some enlargement of the left atrium and pulmonary veins, since they have to accommodate the regurgitant blood in addition to

normal left side inflow. Unfortunately, however, the proportion of the additional volume stored in the atrium and the veins varies, as does the relative duration of systole and diastole, so quantification of regurgitation from measurement of atrial size has not been useful. In cases of acute, severe mitral regurgitation, the left atrium is rarely noted to be enlarged even in the face of marked elevation of atrial pressures.

Changes in left atrial volume are reflected as motion of the aortic root. This is because the posterior atrial wall is firmly anchored to the lungs by the pulmonary veins. This forms one of the major supports of the whole heart, and the posterior atrial wall consequently moves very little. Expansion of the atrium therefore causes its anterior wall, which is echocardiographically indistinguishable from the posterior wall of the aortic root, to be pushed forward. Thus, vigorous motion of the aortic root indicates high left atrial stroke volume and is a feature of mitral regurgitation (though it is also seen in severe mitral regurgitation in the presence of abnormal ventricular function). Quantification of aortic root motion suffers from the same limitations as measurement of left atrial dimension.

Thus, without Doppler echocardiography, the severity of mitral regurgitation can only be deduced from the M-mode or two-dimensional echocardiographic recordings. Direct imaging of the valves, however, frequently reveals the etiology of the regurgitation and provides very useful information for planning surgical approaches. Now that primary valvular reconstruction, rather than replacement is possible, a surgeon experienced in interpretation of echocardiographic data may precisely identify disordered valvular anatomy and predict patient suitability for surgical valvuloplasty.

## Rheumatic Mitral Regurgitation

Some degree of regurgitation frequently accompanies rheumatic mitral stenosis. The pathological changes in the valve described earlier indicate the rheumatic etiology, but the presence of mild regurgitation usually cannot be detected by echocardiographic studies and it is best diagnosed by auscultation or by Doppler. However, extreme enlargement of the left atrium, especially if accompanied by a heavily calcified valve and enlargement of the left ventricle (which is normally small in pure stenosis, reflecting low cardiac output), should give strong suspicion of significant regurgitation.

When regurgitation is dominant, with little or no stenosis, the rheumatic changes in the mitral valve may be minimal and are frequently confined to a restriction of posterior leaflet motion, but without frank reversal of motion direction. In such cases, two-dimensional parasternal long-axis and apical four-chamber views are helpful for showing the shortened, immobile posterior leaflet, which is the cause of the incompetence.

## Mitral Valve Prolapse

The origin of the auscultatory finding of a mid-systolic click, often with a late-systolic murmur was, until recently, widely held to be extra-cardiac. Echocardiography has been responsible for showing that these findings are most commonly caused by prolapse of part of the mitral valve into the left atrium. The click occurs when the prolapsing motion is suddenly halted by the restraining chordae tendineae and is analogous to the opening snap of mitral stenosis. The murmur indicates the presence of mitral regurgitation.

Mitral valve prolapse usually occurs in a "floppy" mitral valve **(Fig. 19)** that has an abnormally large leaflet area and lengthened chordae tendineae due to abnormal collagen synthesis. It is histologically identical to the "myxomatous degeneration" associated with Marfan's disease. The incidence has been said to be as high as 20% in young females, though there is now independent pathological evidence from routine necropsies to support the view that mitral valve prolapse is present in some 3-5% of all adult females and 0.5% of adult males, the incidence rising with age in both sexes.

Mitral prolapse usually manifests either as a sudden posterior buckling **(Fig. 20)** associated with a mid-systolic click and/or late-systolic murmur **(Fig. 21)**, or a pan-systolic "hammock" pattern of the closed leaflets during systole **(Fig. 22)**. The nature and timing of the prolapse pattern is determined to a large degree by ventricular dimensions, and hence is influenced by stroke volume. Thus, for example, lowering stroke volume by standing a patient up reduces ventricular filling and, with a smaller chamber, the elongated chordae restrain the valve even less, allowing it to prolapse earlier. This can often turn a mid-systolic prolapse into the pan-systolic "hammock" type.

Although M-mode echocardiography, especially when aided by a simultaneous phonocardiogram, is a reliable method for detecting most cases of mitral prolapse, it does suffer some disadvantages. Firstly, it is not possible to distinguish an abnormal prolapse motion from the overall movement of the mitral annulus during systole. This shows a small degree of posterior displacement during the isovolumic contraction period, followed by a steady anterior movement. An arbitrary criterion has been proposed, which defines prolapse as a posterior displacement of more than 2 mm below a straight line joining the "C" and "D" points, but clearly the apparent incidence of the condition is greatly influenced by the value chosen. Minor degrees of prolapse can be detected best by M-mode, but false positives can be produced by incorrect ultrasound beam angulation (transducer too low on the chest) or by abnormal overall motion of the heart, as found in pericardial effusions.

Use of the two-dimensional parasternal long-axis view improves specificity because it shows the motion of the leaflets better in relation to that of the annulus **(Fig. 23)**. The mitral valve consists of two leaflets. The smaller, posterior of these comprises three "mini-cusps". Because the two papillary muscles are situated beneath the major commissures, the central "mini-cusp" of the posterior leaflet is less well supported by the chordae and the part most commonly affected by prolapse. It is clearly visualized by M-mode and two-dimensional echocardiography in the parasternal long-axis view. However, prolapse is sometimes confined to the lateral sections near the commissures, and can then easily be missed by M-mode. In such cases the two-dimensional examination, utilizing the apical four-chamber view, permits visualization of these areas. In this way, the use of the combined techniques provides superior sensitivity and specificity to that offered by either one alone.

## Bacterial Endocarditis

Bacterial endocarditis infection of the mitral valve can present in several ways, depending on the type of organism and the route by which the infection is carried. Primary infection is usually associated with a valve abnormality, (e.g., floppy or rheumatic valve). Secondary infection can arise as a result of a jet of blood from a leaking, infected aortic valve striking a normal mitral valve. In the former case, vegetations are most commonly present on either or both valve

leaflets, whereas in the latter, perforation of the anterior leaflet or erosion of the chordae tendineae are also found.

Vegetations can be detected by M-mode **(Fig. 24)**, but are much more readily seen by two-dimensional echocardiography. Using both long-axis and short-axis parasternal views, the location and approximate sizes of the masses can be determined **(Fig. 25)**.

Additional echocardiographic signs appropriate to the preexisting floppy or rheumatic mitral valve will be present. A possible source of confusion can arise from the redundant leaflet tissue and chordae of a floppy valve, which can give a "clubbed" appearance to the leaflet that is indistinguishable from that of a small vegetation. Caution must therefore be exercised in making the diagnosis of vegetations on a floppy valve. Thus, echocardiography can detect the presence of an abnormality, but knowledge of the clinical setting provides the observer with very useful information as to whether the abnormality is likely to be due to infective endocarditis.

The images of vegetations are very striking, and their violent motions often give the impression of imminent embolization. Even in the face of successful medical therapy these lesions rarely resolve quickly and may persist for years. Once the infection is controlled, however, the available evidence is that the mere presence of such masses does not indicate a need for surgical removal. When infection causes perforation of a mitral leaflet, and if there are no vegetations, the only echocardiographic sign may be rapid fluttering of the cusps during systole that is detectable on M-mode recordings.

Echocardiography is particularly useful for evaluating the complication of acute vegetative endocarditis such as valve ring abscess, leaflet rupture, or for determining the presence of secondary vegetations. **Fig. 26** shows a secondary vegetation due to mitral regurgitation on the posterior left atrial wall in a young girl with fever and multiple embolic strokes. Careful angulation of the interrogating beam revealed the mass to be in two parts **(Fig. 27)**. In this patient, Doppler echocardiography showed only mild mitral regurgitation. Because of the multiple embolic strokes, the vegetation was surgically removed and showed a morphology strikingly similar to that noted by two-dimensional echocardiography **(Fig. 28)**.

Careful echocardiographic examination in the clinical setting of endocarditis always requires complete evaluation for the presence of complications. **Fig. 29** demonstrates a necropsy specimen of an infected mitral valve with erosion and thrombus on the posterior wall of the left atrium where the regurgitant jet was likely to have been directed.

Infection of a tricuspid valve can also be primary, with vegetations on the valve or can be caused by a jet lesion from a ventricular septal defect that frequently ruptures chordae. Unlike the case of the mitral valve primary infection, tricuspid valve primary infection does not imply any abnormality of the valve itself. Vegetations are most clearly visualized in the apical four-chamber view or right ventricular inlet view. A patient with rupture of the tricuspid valve leaflet is seen in **Fig. 30** where the anterior leaflet of the tricuspid valve is seen to flail into the right atrium in systole. At surgery (viewed from over the patient's head and through the right atrium into the right ventricle) the flail tricuspid leaflet is seen held by a surgical instrument coming in from the right **(Fig. 31)**.

## Ischemia and Rupture of the Valve Support Apparatus

Mitral regurgitation can result from ischemic dysfunction of the papillary muscles. This sometimes manifests as a lengthening, or failure to shorten, of the valve support apparatus. From an echocardiographic viewpoint, ischemic mitral regurgitation is indistinguishable from mitral valve prolapse. More commonly, however, ischemia causes retraction of the papillary muscle, with the result that the leaflets fail to coapt adequately in systole.

Mitral regurgitation associated with a floppy mitral valve or papillary muscle ischemia is normally only mild. Echocardiographically there are few signs of papillary muscle dysfunction except for the presence of an underlying wall motion abnormality. However, if all support to a portion of the valve is lost due to fracture of some of the chordae **(Fig. 32)**, or if one of the papillary muscle heads ruptures following myocardial infarction **(Fig. 33)**, severe regurgitation usually results.

The echocardiographic findings vary according to the site and degree of damage to the valve apparatus. M-mode recordings generally show irregular or discordant motion of either or both valve leaflets **(Fig. 34)**. Additional echoes may be generated by fractured chordae that show as short lines randomly superimposed on the valve pattern. Echoes from parts of the valve leaflets may be seen in the left atrium as they are carried up into it during systole.

In such a setting, partial flail of one or both of the mitral valve leaflets is noted. Flail portions of the valve apparatus are much better appreciated on two-dimensional recordings, since they allow the abnormal motions to be followed for more of the cardiac cycle.

In the setting of acute myocardial infarction, the appearance of a new murmur suggests the presence of a ventricular septal defect or mitral regurgitation due to disordered mitral valve leaflets. The auscultatory findings in such patients may be quite confusing and V waves are present in both situations by pulmonary capillary wedge pressure recordings. Echocardiography is quite helpful in detecting and differentiating these potential complications. **Fig. 35** shows a flail posterior mitral valve leaflet in diastole that is seen to move entirely into the left atrium in systole **(Fig. 36)**.

## Functional Mitral Regurgitation

Gross enlargement of the left ventricle can dilate the mitral valve annulus to the point where the valve becomes incompetent. This is not easy to detect either clinically or echocardiographically, particularly since there is frequently concomitant tricuspid regurgitation. Because of the very poor left ventricular function, volume overload is not apparent, and any left atrial enlargement could be ascribed to chronic elevation of ventricular filling pressure. However, in patients with left ventricular failure who have considerable atrial enlargement, functional mitral regurgitation may be suspected.

## Tricuspid Regurgitation

As with the mitral valve, there are no specific signs of tricuspid regurgitation on echocardiographic recordings. With the widespread use of Doppler echocardiography it has been learned that small degrees of tricuspid regurgitation are quite common, being present in over 65% of otherwise normal individuals. Such patients have little, or no, evidence of murmur by auscultation.

When tricuspid insufficiency is severe, changes are noted echocardiographically. In the majority of these cases, most functional tricuspid regurgitation is secondary to pulmonary hypertension. Other causes include infective endocarditis and/or abnormalities of the valve itself, such as Ebstein's anomaly.

The primary echocardiographic manifestation of tricuspid regurgitation is volume overload of the right ventricle. The right ventricle becomes enlarged, and the direction of the interventricular septal motion appears to become reversed or "paradoxical". In fact, the left ventricle contracts normally, but superimposed on its motion is the greater movement of the hyperdynamic right ventricle **(Fig. 37)**.

Contrast echocardiography has been shown to help in the detection of tricuspid regurgitation if Doppler echocardiography is not available. When a bolus of liquid, usually sterile saline, is injected rapidly into a peripheral vein, microbubbles of gas are released. On a two-dimensional display, echoes from these microbubbles can be seen to pass back and forth across the tricuspid valve and also seen passing retrograde into the enlarged hepatic veins; they often continue for several minutes before they are eventually cleared into the pulmonary artery. M-mode recordings show that the contrast echoes first appear in the inferior vena cava during systole, and can be seen passing retrograde into the enlarged hepatic veins **(Fig. 38)**.

## The Semilunar Valves

## Aortic Valve Stenosis

Causes of left ventricular outflow obstruction include aortic valve stenosis and subvalvular and supravalvular obstructions. Subvalvular obstruction can be further divided into the discrete diaphragmatic or fibromuscular ring types, and obstruction secondary to septal hypertrophy caused by hypertrophic cardiomyopathy. Only primary valve leaflet pathology will be discussed here and the others dealt with in subsequent units.

Aortic valve stenosis can be present from birth due to malformation of the valve, or can develop in later life as a result of calcification either of a congenitally bicuspid valve **(Fig. 39)** or of a valve inflamed by rheumatic disease.

A congenitally stenotic valve may be grossly dysplastic, and in such cases its echocardiographic appearance is usually abnormal. **Fig. 40** demonstrates a short-axis of a bicuspid aortic valve in systole with a slit-like orifice.

Alternatively, the valve cusps may be thin, either with one commissure imperfectly formed, or consisting essentially of a diaphragm with a small hole through which the blood must pass. During ventricular ejection, such a valve is pushed upward to form a dome shape; while this may be apparent on a long-axis two-dimensional view, it cannot be appreciated by M-mode echocardiography, which frequently gives normal recordings in cases of severe stenosis. Even two-dimensional echocardiography can fail to detect any abnormality. The absence of echocardiographic evidence cannot, therefore, rule out the presence of congenital aortic stenosis.

Mild congenital defects of the aortic valve are common, occurring in about 1% of the population. In most cases, the valve is "bicuspid", either with two equal-sized cusps or with three cusps, two of which are fused together. The patient is usually asymptomatic and there may be little or no murmur. Typically, the M-mode diastolic closure line is eccentrically placed within the aortic lumen **(Fig. 41)**, and there are often multiple echoes in diastole, arising from corrugations on the cusp edges or from the raphe.

There is, however, an aortic ejection sound, generated as the valve is suddenly halted as it opens and forms the "dome" shape described above. High-speed M-mode echocardiograms recorded simultaneously with a phonocardiogram show the coincidence of the ejection sound with maximal valve opening and provide a reliable method for detecting bicuspid valves **(Fig. 42)**. In many cases, the valve itself appears abnormal. The doming action of the valve during systole can sometimes be seen on two-dimensional recordings, but it is not easy to obtain technically adequate images from the thin cusps **(Fig. 43)**.

In adult life, though rarely before the age of forty, some bicuspid valves calcify and become progressively more stenotic. Although histological examination can usually distinguish such cases from rheumatic aortic stenosis, the echocardiographic appearances of the valves are identical due to the severe fibrosis with calcification in both entities. In mild cases, fibrous thickening of the cusps is apparent, and their separation in systole is restricted. Here it may be possible to estimate the orifice area using the two-dimensional short-axis view. Assuming the lumen to be circular, the reduction in orifice diameter, which increases the gradient from 40 to 100 mmHg, is only 2mm. Bearing in mind the probable irregular shape of the hole, and the presence of reverberation echoes from the calcium, visualization of this is, at present, beyond the capability of current techniques.

Nevertheless, if two-dimensional echocardiography shows that a substantial orifice exists, there is unlikely to be severe stenosis. This can be very helpful, for example in eliminating aortic stenosis as the primary pathology in a patient who presents in a moribund state with a systolic murmur.

Two-dimensional echocardiography is also useful for detecting changes associated with chronic rheumatic or degenerative aortic stenosis. In such cases, the aortic cusps are immobile, or only partially mobile and are markedly thickened **(Fig. 44)**.

A rough correlation exists between the severity of calcification and the pressure gradient across the valve. Echocardiographic estimations of calcification are, however, not very reliable as reverberations from small regions of calcium tend to obscure the entire aorta, giving a misleading impression of the overall severity.

Echocardiography is a very precise method for assessing left ventricular hypertrophy, and this means that some helpful information about the effect of aortic stenosis on the myocardium can be obtained **(Fig. 45)**. M-mode recordings permit measurement of ventricular wall thickness and indication of their stiffness is obtained from the rate of diastolic filling. The relative dimensions of the ventricular cavity and wall thickness can also be used to estimate wall stress using the formula:

Wall Stress (S) = LVP x (r/h),

where LVP = peak LV pressure; r = cavity radius; h = mean wall thickness.

Thus, assuming the ventricle hypertrophies to maintain constant wall stress:

LVP – (constant) x (h/r)

If peak pressure can thus be estimated and systemic arterial pressure measured by a cuff around the brachial artery, it ought to be possible to determine the valve gradient. Several reports have suggested that this is the case, but not all researchers can produce adequate correlations to permit useful prediction in individual cases. Left ventricular hypertrophy may also be detected with two-dimensional echocardiography **(Fig. 46)**.

Doppler echocardiography provides the most reliable noninvasive means for determining aortic valve peak and mean gradients, as does estimating valve orifice area by the continuity equation.

The primary echocardiographic signs of aortic regurgitation are volume overload of the left ventricle combined with a rapid fluttering, either of the anterior mitral valve leaflet, or the septal endocardial, in the region of the left ventricular outflow tract, or both **(Fig. 47)**. Rarely, and only in patients with dilated ventricles, it may not be possible to detect this fluttering. Some detectable abnormality of the aortic root or valve is normally found combined with these signs. In cases of acute, severe regurgitation, premature closure of the mitral valve may be seen **(Fig. 48)** and is usually an ominous prognostic sign. This is caused by extremely high end-diastolic pressures in an uncompensated ventricle, and is associated with a loud mid-diastolic (Austin Flint) murmur.

## Aortic Regurgitation

If aortic regurgitation is the sole lesion, its severity may be gauged from the degree of left ventricular volume overload, but the duration and severity of the mitral or septal flutter are of no help in this regard.

It has long been appreciated that most patients tolerate even quite severe aortic regurgitation for many years, but that deterioration is rapid once left ventricular failure occurs. Thus, a major contribution of echocardiography to the management of aortic regurgitation is in determining the pathogenesis, and in providing a noninvasive method for monitoring left ventricular function.

As with other flow lesions, Doppler echocardiography is now the noninvasive standard for detecting the presence and severity of aortic regurgitation.

In rheumatic heart disease, the most common presentation after pure mitral stenosis is a combination of this with aortic regurgitation. The changes in the aortic valve can be minimal, causing only slight thickening of the cusp echoes. The aortic root diameter, measured at the level of the cusps, is normal. Evidence of the etiology comes from the mitral valve, which almost always shows some echocardiographic signs of rheumatic disease. The mitral valve abnormality may be confined to slight reduction in amplitude of the posterior leaflet motion without any clinical manifestations **(Fig. 49)**. If, on the other hand, the mitral valve is heavily calcified, any flutter caused by aortic regurgitation may be difficult to detect. Magnified views of the valve and interventricular septum, recorded by M-mode using higher paper speed and low gain settings, may help to demonstrate diastolic fluttering.

Where vegetations on the aortic valve result from the infection, they are best detected by using two-dimensional long-axis and short-axis views, shown in **Fig. 50**. Vegetations larger than 2-3 mm can usually be visualized clearly and their approximate size can be determined, along with the cusps to which they are attached.

Aortic valve endocarditis is sometimes complicated by the development of a mycotic aneurysm, which can rupture into one of the other cardiac chambers, or by the spread of the infection to the tricuspid valve. Although M-mode recordings may demonstrate the resulting hemodynamic changes, direct visualization of such defects is normally possible only by two-dimensional echocardiography **(Fig. 51)**.

In many cases, however, endocarditis infection of an aortic valve does not produce vegetations, but rather causes erosion of the cusp tissue that leads to rupture or perforation. The echocardiographic sign of such lesions is rapid fluttering of the aortic cusps during diastole, caused by a turbulent jet of blood passing through a defect in the otherwise closed valve.

Many infected aortic valves also appear to be bicuspid, and it is well known that any abnormality of the valve greatly increases its susceptibility to infection. But only about one percent of the population has a bicuspid valve, and roughly half of the patients requiring valve replacement for severe aortic regurgitation caused by endocarditis have a normal three-cusp configuration.

Not infrequently, the regurgitant jet of blood spreads the infection to the mitral valve, producing the signs described previously.

## Aortic Root Disease

Unlike the mitral valve, the aortic cusps have no supporting apparatus; they withstand the considerable diastolic pressure in the aorta by their shape, and by virtue of the fact that they rest against each other with some 30% of each cusp overlapping its neighbors. However, if the region of the aorta where the cusps are attached is dilated or distorted by disease, the cusps may fail adequately to withstand aortic diastolic pressure and regurgitation will occur.

**Fig. 52** shows a diagram of the aortic root. Between the aorta proper and the lower annulus, which forms part of the fibrous skeleton of the heart, is a tube comprised mainly of collagen, which bulges outward to form the sinuses of valsalva and to which the aortic cusps are attached. Dilation of the fibrous annulus is rare.

Dilation of the ascending aorta is commonly found in patients with aortic stenosis, but does not normally affect the valve cusps (otherwise almost all patients with aortic stenosis would have regurgitation). Dilation of the supra-aortic ridge at the point where the aorta meets the top of the collagen sleeve, however, pulls the cusps apart and reduces their mutual support **(Fig. 53)**. In this way, an enlargement in this area by as little as 2mm above the normal range can be sufficient to cause regurgitation severe enough to require valve replacement. Such dilation is readily detected by echocardiography, since it is at this level that the echoes from the cusps are routinely recorded.

Aortic root disease can be divided into those characterized by an inflammatory reaction, for example syphilis, Reiter's disease, ankylosing spondylitis, and giant cell aortitis, and those in which the elastic within the aorta is partially destroyed, as in Marfan's disease. In nearly all

cases, echocardiography shows a dilated aortic root, though in some cases of syphilis, valve incompetence results from distortion of the cusps rather than dilatation. It may also be possible to infer the presence of inflammation or calcium from dense aortic wall echoes. The cusps themselves often generate multiple echoes in diastole, similar to those frequently found with bicuspid valves **(Fig. 54)**. This is probably due to the thickening of the cusp edges caused by their rubbing against each other as they partially prolapse under the stress or aortic pressure. This "functional prolapse" should be distinguished from that of the mitral valve, and occasionally the aortic valve, in which the cusps themselves are abnormal.

One of the features of Marfan's disease is the destruction of the elastic in the aortic media, causing the root to dilate and the valve to become incompetent **(Fig. 55)**. However, many patients with aortic regurgitation have dilated aortic roots, biopsies from which are histologically identical to those of Marfan's disease but have none of the other clinical or echocardiographic stigmata of this condition. This "idiopathic root disease" is in fact the most common cause of severe aortic regurgitation in western countries where rheumatic fever is now rare. Interestingly, it is frequently found in patients with aortic regurgitation who also have bicuspid aortic valves.

## Aortic Dissection

In its most severe form, loss of structural strength in the aortic walls leads to aneurismal dilation and/or dissection of the intima. The dilation is at best documented by two-dimensional echocardiography, using the parasternal long-axis view to visualize the ascending aorta and the suprasternal position to see the aortic arch.

In cases where there is gross dilation at the valve level, the aortic cusps may show a pattern of early partial closure and fluttering indistinguishable from that characteristic of subvalvar aortic stenosis. This is probably caused by turbulent eddies of blood in the sinuses of valsalva produced by the high ejection velocity into the dilated aorta. With careful technique and good apparatus, high success has been reported in detecting dissection by two-dimensional echocardiography and in classifying it according to the DeBakey criteria.

The use of M-mode alone, however, leads to many false positives because of the difficulty in obtaining correct beam angulation and an inability to distinguish, for example, a true dissection from a double echo generated by the bulbous sinuses of valsalva or the transverse sinus of the pericardium posteriorly.

Even more productive is the use of transesophageal echocardiography where a small transducer, mounted on a flexible endoscope is inserted into the esophagus. Using this approach provides superior images of the ascending aorta and proximal transverse aortic arch in almost all patients. **Fig. 56** shows a short-axis transesophageal image of the proximal aorta where true and false lumens are easily visualized.

## Pulmonary Stenosis

In cases of mild pulmonary stenosis, no abnormality is detectable either by M-mode or two-dimensional echocardiography. With a more severe obstruction, it may be possible to detect right ventricular hypertrophy, but echocardiography is not a very sensitive method for diagnosing this. Another sign that has been reported, but in our experience is confined to patients with

severe obstruction, is an exaggerated “a-dip” on the pulmonary valve echocardiogram. This arises from the fact that the hypertrophied right atrium forcefully injects blood into an already full and stiff right ventricle during atrial systole. Pulmonary artery pressure is low, and so the sudden increase in right ventricular pressure is sufficient to partially open the pulmonary valve **(Fig. 57)**.

Echocardiography can assist phonocardiography in the diagnosis of pulmonary valve stenosis by confirming the origin of the early systolic ejection sound **(Fig. 58)**. A recording showing pulmonary valve closure may also help confirm the timing of the later pulmonary component of the second heart sound, although such recordings are routinely possible only in small children.

Now that Doppler echocardiography is available, it has been shown to be able to detect, and reliably quantitate, pulmonary stenosis.

## Pulmonary Regurgitation

Pulmonary regurgitation causes volume overloading of the right ventricle, indicated on the echocardiogram by enlargement of the right ventricle and reversal of septal motion, as described earlier for tricuspid regurgitation following surgery for pulmonary stenosis. The combination of residual right-sided hypertrophy with overfilling of the right ventricle due to regurgitation gives a very deep pulmonary valve a-dip, and may even open the valve fully before the onset of systole **(Fig. 59)**. Doppler, again, has been shown to be quite sensitive in detecting and quantitating the severity of pulmonic regurgitation.

## Pulmonary Hypertension

Pulmonary hypertension causes right ventricular hypertrophy, which may be coupled with volume overload due to associated pulmonary or tricuspid regurgitation. Where pulmonary hypertension develops as a result of a large left-to-right shunt, it reduces the volume of the shunt, eventually reversing it (Eisenmenger physiology). The echocardiographic signs thus reflect both the primary pathology and the degree of pulmonary hypertension.

In cases of severe pulmonary hypertension, the right ventricle compresses the left ventricular septum posteriorly, resulting in a characteristic “flattening” of the septum **(Fig. 60)**. The degree of flattening does not, however, correlate in a useful fashion with the severity of the pulmonary hypertension.

Elevation of pulmonary artery pressure reduces the amount by which right atrial systole causes the pulmonary valve leaflets to dome upward, and so the a-dip on the M-mode echo recording is reduced. Provided the patient is in sinus rhythm, absence of the a-dip is a fairly reliable sign of pulmonary hypertension.

Using high-speed M-mode recordings, it is possible to perform right ventricular systolic time interval studies and to measure, for example, pre-ejection period and ejection time. Pulmonary hypertension prolongs right ventricular isovolumic contraction time, and delays pulmonary valve opening at the onset of systole. The technique is difficult and measurements should be made at a constant point in the respiratory cycle to eliminate variations caused by changes in stroke volume. Provided that other causes of abnormal right ventricular dynamics such as right bundle

branch block and right ventricular failure can be eliminated, such methods permit quantitative estimation of pulmonary artery pressure.

Doppler echocardiography can detect the presence of pulmonary hypertension by a rapid time to peak of the systolic pulmonary waveform. Using tricuspid regurgitation, Doppler can also estimate peak right ventricular systolic pressure.

# Prosthetic Valves

## General Principles

Echocardiography is sometimes a useful tool for the management of patients who have one or more prosthetic valves. The aim of the echocardiogram study is to visualize the valve itself, along with its surrounding structures, and also to evaluate left ventricular function. There very high M-mode sampling rate generally makes it preferable to two-dimensional echocardiography for this purpose, although the latter can permit more rapid location of the valve and may assist in left ventricular studies.

When studying a prosthetic valve, it is necessary to direct the ultrasound beam in such a way that it detects motion of the prosthesis. Thus, for an aortic ball valve, either the apical or right supraclavicular transducer positions must be employed. Mitral ball prostheses are also best visualized from the apical region. Pivoting disk (e.g., Bjork-Shiley) and tissue xenograft valves can normally be seen from the parasternal position.

Because changes in echocardiograms may be so subtle with prosthetics, every patient having a valve replacement should be studied while still in the hospital, about two weeks after the operation. This provides a baseline against which to compare any later recordings. Since cardiac function in such patients remains abnormal, alterations in their hemodynamic status must be deduced from changes in the echocardiographic parameters, rather than their absolute values.

**Fig. 61** shows a diagram of the motion of a Starr-Edwards aortic prosthesis, as viewed from an apical transducer position, and **Fig. 62** is an actual recording. The echo-free space below the transducer reverberations represents the left ventricle. The first echo encountered from the prosthesis arises from the sewing ring (SR). Its motion is paralleled by the echo that arises from the top of the valve cage (C). Immediately below the sewing ring is the echo from the ventricular aspect of the ball (PB). A second echo (AB), having similar motion, is seen posteriorly to the cage; this actually arises from the top of the ball, which is inside the cage, but because ultrasound travels more slowly within the ball, this echo takes longer to return to the transducer and thus appears to the machine to have come from further away.

A similar analysis, taking into account the inverted position of the valve, applies to a mitral ball prosthesis when viewed from the apex **(Fig. 63)**. One important difference is that the pressure gradient across an aortic valve is high both in systole and diastole, so its movements are rapid and it stays fully open throughout systole and fully closed throughout diastole. A mitral valve, in contrast, has a high gradient across it is systole, but only a small gradient in diastole, and by the end of the filling period this should be nearly zero. Consequently, a correctly functioning mitral prosthesis is often seen to partially close toward the end of diastole **(Fig. 64)**.

A normal Bjork-Shiley disk aortic prosthesis is shown in **Fig. 65**. During diastole, the valve is closed and presents no target to the ultrasound beam from the parasternal position. When the valve opens in systole, it is encountered in the center of the aorta, and forms an intense echo, usually with multiple reverberations behind it **(Fig. 66, left)**. Similar recordings are obtained from a mitral disk prosthesis **(Fig. 66, right** – note the partial closure in the last diastole).

Tissue heterografts are normally mounted in a coronet-shaped frame or stent. They are aortic valves and so, in the aortic position, appear essentially normal, except for the heavy echoes from the stent. For use as a mitral replacement the valve is inverted, and thus the patient appears to have two aortic valves, one of which opens in systole, and the other in diastole **(Fig. 67)**.

## Differential Diagnosis of Prosthesis Malfunction

Malfunction of an artificial prosthesis is relatively rare nowadays. When it does occur, it is usually associated with rapid deterioration of the patient's condition.

The main signs of prosthetic valve obstruction are:

1. Delayed, reduced, incomplete, or intermittent valve opening or closing.

2. Multiple echoes around the valve, suggestive of organized thrombus or fibrous tissue impeding its motion. Other signs, such as ventricular hypertrophy, or reduced filling rate, are generally unhelpful unless they are definitely of recent appearance.

Failure of tissue prosthesis tends to be more gradual, involving development of mild regurgitation or stenosis secondary to calcification. The exception is infection, which can rapidly destroy the prosthesis. They may be seen as fluttering of the cusps during the closed phase, indicating perforation, or by the classical appearances of vegetations.

Partial dehiscence of a prosthetic valve, due to failure of its fixing sutures, produces gross incompetence. This is rare in the case of an aortic valve, but when it does occur it causes severe ventricular volume overload and high end-diastolic pressure evidenced by premature mitral valve closure. The most common cause of dehiscence is erosion of the surrounding tissue by infection and other signs indicating this may be present.

Dehiscence of a mitral valve is more common, and causes severe mitral regurgitation that can be difficult to detect clinically. Sometimes it is possible to obtain direct evidence of dehiscence **(Fig. 68)**. It should be suspected whenever the motion of the mitral stent appears unduly vigorous; in this respect, two-dimensional studies can be of assistance. Such dehiscence is readily detected by two-dimensional echocardiography. In addition, Doppler echocardiography has been shown to be helpful in detecting even small degrees of prosthetic valvular insufficiency and moderate degrees of stenosis.

## Septal Motion in Patients with Mitral Prostheses

Very useful information about mitral valve prosthesis function can be obtained from the analysis of septal motion. Immediately following surgery, septal motion is flat, or perhaps slightly reversed. The cause of this is not clear, but it is probably due to alteration of cardiac dynamics as

a result of opening the pericardium, and relative ischemia during cardiopulmonary bypass. Provided recovery is satisfactory, and in the absence of surgically induced conduction defects, septal motion essentially returns to normal within a year of surgery.

If the patient remains in chronic right heart failure, the associated tricuspid regurgitation produces right ventricular enlargement and reversed septal motion. Valve function, left ventricular cavity size, and posterior ventricular wall motion should all be normal.

When right heart failure is caused by mitral obstruction, the best evidence comes from the prosthesis motion. As described earlier, partial or hesitant opening or multiple echoes around the valve suggest buildup of tissue around the valve. Late diastolic partial closure is no longer seen (if it is present, stenosis is highly unlikely). Ventricular filling rate may be observed to be very slow.

Severe mitral regurgitation causes left ventricular volume overload, with vigorous, normal septal motion. Thus, a patient who, clinically, has evidence of right heart failure, but whose septal motion is normal, probably has significant left ventricular volume overload. If other causes can be eliminated (for example, ventricular septal defect or aortic regurgitation) the finding of normal septal motion in a patient with know tricuspid regurgitation is evidence of a mitral paraprosthetic leak.

Finally, when the right-sided failure is accompanied by left ventricular failure, the left ventricle will become dilated and hypokinetic, showing the appearances typical of cardiomyopathy.

# Diseases of the Heart Valves (Figure Legends)

*Fig. 1 Stenotic mitral valve viewed from the left atrium showing thickened leaflets and partial commissural fusion.*

*Fig. 2 M-mode recording of a normal mitral valve (left) and a stenotic mitral valve (right). Note the "M" shape described by the normal valve. The restricted mobility of the thickened anterior leaflet and the forward movement of the posterior leaflet in the stenotic valve are clearly seen.*

*Fig. 3 2-D parasternal left ventricular long-axis view of a stenotic mitral valve during diastole. Note the leaflet tip and left atrial enlargement typical of rheumatic mitral stenosis.*

*Fig. 4 M-mode recordings of mild (left) and severe (right) mitral stenosis. With severe stenosis the leaflets are calcified and show almost no movement.*

*Fig. 5 Diagram showing how 2-D echocardiograms (left and right) can differentiate between mitral orifices of various shapes that would appear identical on an M-mode recording (center)*

*Fig. 6 2-D parasternal short-axis view at the mitral valve orifice in a patient with rheumatic mitral stenosis. Note the smaller area and irregular shape of the stenotic valve orifice.*

*Fig. 7 2-D parasternal short-axis view at the aortic valve level during systole. The left atrial enlargement and bowing of the interatrial septum towards the right atrium is typical of high left atrial pressures in patients with mitral stenosis.*

*Fig. 8 2-D parasternal left ventricular long-axis view showing a calcified/fibrotic mitral annulus (arrowed).*

*Fig. 9 M-mode slow scan recording of a left atrial tumor. A dense cloud of echoes appears behind the anterior mitral valve leaflet shortly after diastolic opening.*

*Fig. 10 2-D parasternal left ventricular long-axis view in a patient with a left atrial myxoma.*

*Fig. 11 2-D apical four-chamber view showing a large left atrial myxoma in diastole.*

*Fig. 12 2-D parasternal short-axis view at the aortic valve level in a patient with a large left atrial myxoma. Note the tumor extension through the fossa ovalis into the right atrium.*

*Fig. 13 Pathological specimen of a left atrial myxoma after resection.*

*Fig. 14 2-D apical four-chamber view in a patient with a right atrial myxoma during systole.*

*Fig. 15 2-D apical four-chamber view of the same right atrial myxoma as is in Fig.14. With diastole the tumor moves through the tricuspid valve causing severe obstruction.*

*Fig. 16 M-mode scan in a case of mitral and tricuspid stenosis. Note the similar leaflet motions of the abnormal tricuspid (upper left) and mitral (lower right) valves.*

*Fig. 17 2-D apical four-chamber view in a patient with rheumatic mitral and tricuspid valvular stenosis.*

*Fig. 18 M-mode recording of the left ventricle in a case of mitral regurgitation. The left ventricular cavity is enlarged and wall motion hyperdynamic.*

*Fig. 19 Postmortem preparation showing the prolapse of the central scallop of the posterior mitral leaflet, viewed from the left atrium. Prolapse has been induced by filling the left ventricle with water to physiological pressure; a regurgitant jet (arrowed) is escaping from the base of the domed prolapsed section.*

*Fig. 20 M-mode recording of a floppy mitral valve showing sudden posterior buckling when prolapse occurs during late systole (arrowed).*

*Fig. 21 Magnified high-speed M-mode recording of the same case as Fig.20 with simultaneous phonocardiogram illustrating the relationship of the click (X) and later systolic murmur (SM) to the movement of the prolapsed valve.*

*Fig. 22 Magnified M-mode recording showing the "hammock" posterior movement associated with pan-systolic prolapse.*

*Fig. 23 2-D parasternal left ventricular long-axis view showing marked prolapse of the posterior mitral valve leaflet.*

*Fig. 24 M-mode recordings of a mitral valve showing echoes from vegetations attached to the anterior leaflet. In the magnified high speed view (right) the echoes from the vegetations are arrowed. The phonocardiogram shows a pan-systolic murmur and a diastolic filling murmur.*

*Fig. 25 2-D parasternal left ventricular long-axis view in a patient with bacterial endocarditis. Note the vegetative mass lesion of the mitral valve seen within the left atrium.*

*Fig. 26 2-D parasternal left ventricular long-axis view showing a secondary vegetative lesion on the free wall of the left atrium. The secondary lesion was due to a jet of mitral regurgitation striking the posterior left atrial wall.*

*Fig. 27 2-D parasternal left ventricular long-axis view with oblique angulation demonstrating two distinct parts of the vegetative lesion seen in Fig.26.*

*Fig. 28 Pathologic specimen of the vegetative lesion seen in the previous two figures.*

*Fig. 29 Pathological specimen at autopsy of an infected mitral valve showing erosion and thrombus on the posterior wall of the left atrium*

*Fig. 30 2-D parasternal right ventricular inflow view in a patient with a flail tricuspid valve leaflet.*

*Fig. 31 Photograph taken during surgery of a patient with a flail tricuspid leaflet. The flail leaflet is held by a surgical instrument coming in from the right.*

*Fig. 32 Ruptured chordae tendineae (arrowed).*

*Fig. 33 Ruptured papillary muscle.*

*Fig. 34 M-mode recording of a mitral valve showing the extreme posterior movement during systole and anterior movement during diastole (arrowed) of the flail posterior leaflet.*

*Fig. 35 2-D apical four-chamber view during diastole of a patient with flail posterior mitral valve leaflet.*

*Fig. 36 2-D apical four-chamber view in systole of the same patient in Figure 2.35. Note that the posterior mitral leaflet is now entirely within the left atrium.*

*Fig. 37 M-mode recording showing the reversed, or paradoxical systolic motion of the interventricular septum in a patient with tricuspid regurgitation.*

*Fig. 38 M-mode recording of the inferior vena cava (IVC) in a patient with tricuspid regurgitation. Following a peripheral venous injection of saline, microbubbles are seen to appear in the IVC after the QRS complex, in late systole.*

*Fig. 39 Specimen of a calcified bicuspid aortic valve.*

*Fig. 40 2-D parasternal short-axis view of the aorta in a patient with a bicuspid aortic valve. Note the slit-like orifice seen during systole*

*Fig. 41 M-mode recording of a bicuspid valve showing the eccentrically placed diastolic closure line (arrowed).*

*Fig. 42 Magnified high-speed M-mode recording of a bicuspid aortic valve with phonocardiogram showing the coincidence of the ejection sound (X) with maximal valve opening.*

*Fig. 43 2-D parasternal left ventricular long-axis view in a patient with a bicuspid aortic valve. Note the doming of the aortic valve leaflets during systole.*

*Fig. 44 2-D parasternal left ventricular long-axis view in a patient with calcified aortic stenosis. The aortic leaflets are markedly thickened and demonstrate reduced mobility during systole.*

*Fig. 45 M-mode recordings of a normal left ventricle (left) and in a case of left ventricular hypertrophy (right). Symmetrical thickening of the walls and reduced early diastolic filling rate are evident.*

*Fig. 46 2-D parasternal left ventricular short-axis view at the papillary muscle level in a patient with concentric left ventricular hypertrophy.*

*Fig. 47 M-mode recording showing the rapid fluttering (arrowed) of the anterior mitral valve leaflet and septal endocardium as a result of aortic regurgitation.*

*Fig. 48 M-mode recording with phonocardiogram showing presystolic closure of the mitral valve resulting from acute, severe aortic regurgitation. Mitral closure is silent and "X" indicates the ejection sound of a bicuspid aortic valve.*

*Fig. 49 M-mode recording showing anterior leaflet flutter and reduced posterior leaflet motion, indicating that the aortic regurgitation has rheumatic etiology.*

*Fig. 50 2-D parasternal left ventricular long-axis view during diastole. Note the aortic valve vegetation prolapsing into the left ventricular outflow tract.*

*Fig. 51 2-D parasternal left ventricular short-axis view of the aorta of the same patient as in Figure 2.50. The vegetative mass lesion is now seen to involve both the aortic and tricuspid valves.*

*Fig. 52 Diagram of the aortic root showing the manner in which the aortic cusps are suspended in the bulbous sleeve that forms the sinuses of Valsalva.*

*Fig. 53 Diagram illustrating the means by which dilation of the supra-aortic ridge pulls the aortic cusps apart and causes aortic regurgitation.*

*Fig. 54 M-mode recording of aortic root disease showing thickened aortic cusp edges resulting from their rubbing together as a result of partial prolapse.*

*Fig. 55 2-D parasternal left ventricular long-axis view in a patient with Marfan's disease. Note the marked dilatation of the aortic root.*

*Fig. 56 Short-axis transesophageal image of the proximal transverse aorta in a patient with aortic dissection. Note that both the true and false lumens are usually identified using this echocardiographic approach.*

*Fig. 57 M-mode recording of severely stenotic pulmonary valve showing its partial opening during right atrial contraction.*

*Fig. 58 Magnified, high-speed recording that demonstrates the coincidence of the early systolic ejection sound (X) with maximal pulmonary valve opening.*

*Fig. 59 High-speed M-mode recording showing premature opening of the pulmonary valve during inspiration (arrowed) causing the disappearance of the ejection sound (X).*

*Fig. 60 2-D parasternal left ventricular short-axis view at the papillary muscle level in a patient with pulmonary hypertension. Note the characteristic "flattening" of the interventricular septum.*

*Fig. 61 Diagram of the motion of a Starr-Edwards ball aortic prosthesis, as viewed from an apical transducer position.*

*Fig. 62 M-mode recording of a Starr-Edwards ball aortic prosthesis.*

*Fig. 63 Diagram of the motion of a Starr-Edwards ball mitral prosthesis viewed from the apex.*

*Fig. 64 M-mode recording of a ball mitral prosthesis viewed from the apex, showing partial closure toward end of diastole (arrowed).*

*Fig. 65 Diagram of the motion of a Bjork-Shiley disk aortic prosthesis viewed from the parasternal position.*

*Fig. 66 M-mode recordings of a Bjork-Shiley disk aortic prosthesis (left) and mitral prosthesis (right). Note the partial closure of the mitral prosthesis in late diastole.*

*Fig. 67 M-mode recording of a tissue mitral prosthesis. Echoes from the closed cusps are seen during systole.*

*Fig. 68 Diagram and M-mode recording of a partially dehisced prosthetic valve. As shown in the diagram and arrowed on the M-mode recording, the posterior part of the sewing ring is swinging freely to and fro.*