Clinical review

Basic transthoracic echocardiography

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Transthoracic echocardiography is one of the most commonly performed cardiac investigations. It can provide comprehensive information about cardiac structure and function, helping to establish a diagnosis and guide therapy, and is no longer the preserve of the specialist cardiology department. Examinations are frequently requested by doctors in other branches of medicine; they need to know what questions an echocardiogram can, and cannot, answer.

Echocardiography images are best viewed as moving pictures. These are shown on bmj.com, several with the motion of a full cardiac cycle. The website also contains brief explanations of the commonly used imaging modalities.

Summary points

- Assessment of left ventricular function is the commonest reason for requesting transthoracic echocardiography
- Echocardiography is an essential investigation whenever the underlying pathogenesis of heart failure is in doubt
- Echocardiography is the investigation of choice to define the cause of a heart murmur and to assess the severity of any underlying abnormality; it is used to determine the need for antibiotic prophylaxis, medical therapy, invasive investigation, or surgical intervention
- In patients with atrial fibrillation, echocardiography can identify underlying structural problems and guide treatment, especially the need for anticoagulation and the appropriateness of cardioversion

Sources and scope

As a brief review cannot encompass a field as vast as transthoracic echocardiography, we have focused on common clinical problems that can be investigated by echocardiography. Our primary sources were echocardiographic texts and clinical guidelines (see educational resources on bmj.com). We used Medline searches to update and supplement information on specific conditions.

Indications for echocardiography

Assessment of left ventricular function

The commonest reason for undertaking an echocardiogram is to assess left ventricular function.[1 2] In accordance with NICE (National Institute for Health and Clinical Excellence) guidelines,[3] this is often requested when patients have symptoms suggesting heart failure. Although clinical information and basic investigations will often identify patients with significant left ventricular systolic dysfunction,[4–6] echocardiography can quantify the severity and determine the underlying cause. Many patients with heart failure have concomitant or predominant diastolic dysfunction,[7 8] which may be more difficult to detect clinically but should be suspected in patients with evidence of heart failure but normal systolic function, particularly elderly people and those with hypertension.

Echocardiography is valuable in patients with known coronary heart disease, even those without overt heart failure. Some may have important systolic dysfunction and may benefit from treatments such as β blockers, angiotensin converting enzyme inhibitors, or, in selected cases, surgical revascularisation.[9–11]

In routine clinical practice, left ventricular systolic function is often assessed subjectively and graded as normal or mildly, moderately, or severely impaired. Several methods of describing left ventricular function are available. As they use formulas and assumptions, all are prone to error, particularly if there is ischaemic dysfunction (when regional abnormalities can corrupt estimates of global function). The most useful measurement is the ejection fraction, which is calculated automatically by measuring the difference in the volumes of the left ventricle in end-systole and end-diastole. The ejection fraction is the ratio between stroke volume and end-diastolic volume (the normal range lying between 50% and 70%).

Healthy myocardium thickens and contracts inwards in systole. In ischaemic myocardium, contraction may be diminished (hypokinesis) or, in extreme cases, absent (akinesis). Akinesis usually indicates infarcted tissue, which may also show discoordinate contraction and cause the myocardial wall to bulge outwards in systole (dyskinesis). The left ventricle can be divided into anatomically defined segments, and

P+ *Echocardiography images, explanations of common imaging modalities, additional educational resources, and resources for patients are on bmj.com*

abnormalities of motion can be assigned a score representing the severity of impaired contraction for each segment (fig 1).[12] This allows a cumulative "wall motion score index" to be calculated, giving an overall assessment of left ventricular systolic function.

Although coronary heart disease is the commonest cause of left ventricular systolic dysfunction, other mechanisms are possible, such as dilated cardiomyopathy or valvular dysfunction. In addition, some patients with heart failure will have predominant diastolic dysfunction or right ventricular impairment. Echocardiography is an essential investigation whenever the underlying pathogenesis of heart failure is in doubt. Failure to perform an echocardiogram may result in inappropriate treatment or potentially remediable causes being overlooked.

Murmur

Cardiac murmurs are caused by turbulence of blood. This may be due to valvular heart disease, increased flow across a normal valve, or shunts related to congenital or acquired defects. Echocardiography is the investigation of choice to define the aetiology and assess the severity of the underlying abnormality. In many situations echocardiographic features are used to determine the need for antibiotic prophylaxis, medical therapy, invasive investigation, or surgical intervention, to re-assess the response to treatment, and to monitor progression of an abnormality.

Many heart murmurs are benign. These are characterised by the absence of cardiovascular symptoms; short duration; low intensity; maximal at the left sternal edge; an ejection systolic pattern; a normal second heart sound; no evidence of left ventricular dilatation or hypertrophy; and the absence of other associated abnormalities. These features are common in high output states such as pregnancy and in children and adolescents. Such patients do not usually require an echocardiogram.[13] In contrast, a murmur in a patient with cardiorespiratory symptoms or clinical signs suggesting structural heart disease is a clear indication for echocardiography.

Aortic valve disease

Aortic stenosis is common and is usually related to degenerative change. Methods for quantifying the severity of stenosis include subjective visual assessment of valve opening and mobility, estimating the gradient across the valve using Doppler measurements, and determining the valve area using the continuity equation. The most frequently used figures are the mean and peak gradients across the aortic valve. To calculate the peak gradient, the velocity of blood is measured across the valve using continuous wave Doppler (fig 2). The pressure difference across a stenotic valve orifice can then be related to the change in velocity, using the simplified Bernoulli equation (pressure gradient in mm Hg equals 4 times the measured velocity squared: $\Delta P = 4[v^2]$). In most cases, mean gradient is a more reliable measurement. This is calculated by tracing around the Doppler waveform (fig 2).

Impairment of left ventricular function may result in a low gradient because of reduced stroke volume. Conversely, an increased stroke volume, as occurs with concomitant aortic regurgitation, may result in a high gradient. In these circumstances, the most accurate measurement of the severity of aortic stenosis is the valve area determined from the "continuity equation" (see educational resources on bmj.com).

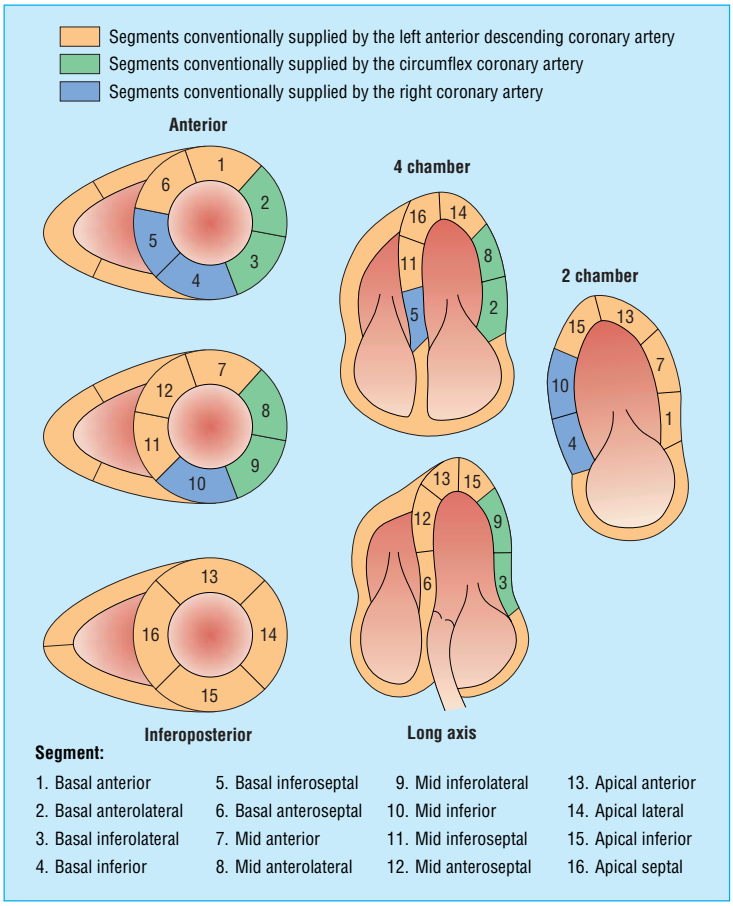

Fig 1 Segmental division of left ventricle for wall motion scoring, and nomenclature of segments. (The apical lateral segment may be supplied by the circumflex coronary artery, and when the right coronary artery is dominant it may also supply the mid-inferoseptal and apical inferior segments)

Aortic regurgitation is commonly due to degenerative disease, rheumatic disease, or dilatation of the aortic root. It may also follow endocarditis. The aetiology can usually be determined by two dimensional echocardiography. Aortic regurgitation is more

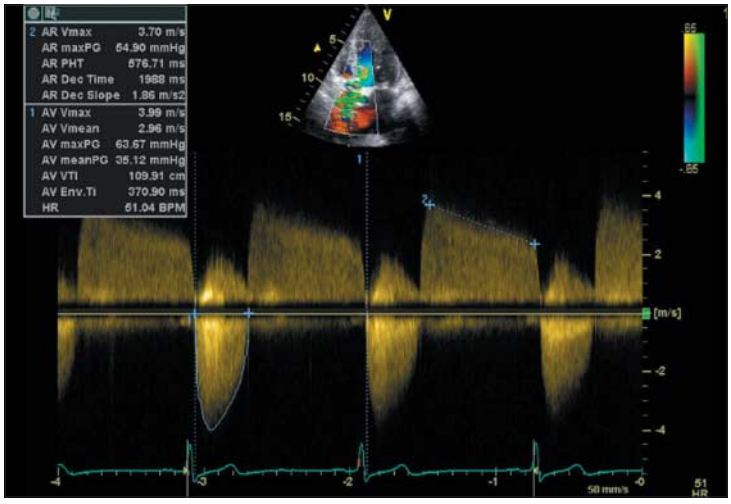

Fig 2 Continuous wave Doppler study of aortic valve (mixed aortic valve disease)

Fig 3 Colour flow mapping of mitral regurgitant (blue) jet in patient with mitral regurgitation. The jet area relative to the area of left atrium has been measured and suggests severe mitral regurgitation

difficult to assess than aortic stenosis and there is no criterion against which echocardiographic measurements can be easily validated. The simplest methods are based on visual assessments of the regurgitant jet, using colour flow mapping. These include the area of the jet relative to the area of the left ventricular outflow tract (LVOT), the width of the jet relative to the width of the LVOT, and the width of the jet at its narrowest point (just beyond the regurgitant orifice), termed the "vena contracta."[14] The rate at which the pressures in the ascending aorta and left ventricle equilibrate also indicates the severity of regurgitation. This can be assessed by measurement of the slope of the regurgitant continuous wave Doppler signal (fig 2) and the pressure half time (the time taken for the peak gradient to fall by 50%). The more severe the regurgitation, the quicker the pressures equalise and the shorter the pressure half time.

Echocardiography also has an important role in monitoring the progression of aortic regurgitation and identifying associated complications. In particular, echocardiography is used to observe changes in the dimensions of the left ventricle (especially the end-systolic diameter) and ejection fraction, and to detect, quantify, and monitor dilatation of the aortic root.[13]

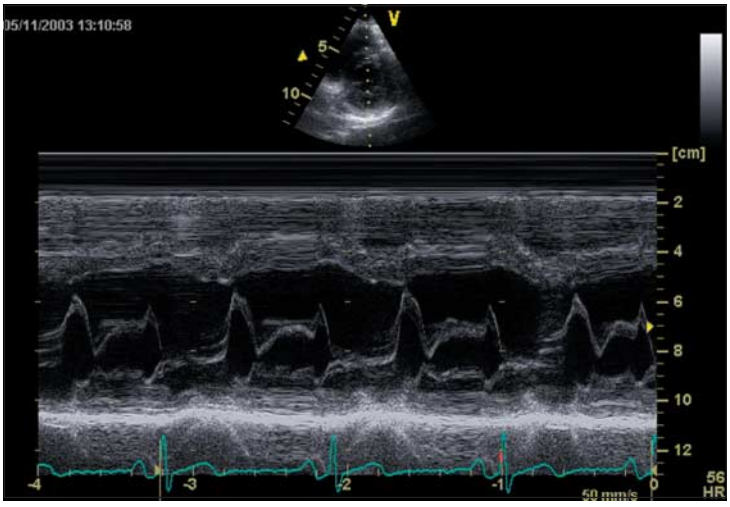

Fig 4 M-mode through mitral valve (normal)

Mitral valve disease

Two dimensional echocardiography can identify the mechanism of mitral regurgitation. This may be due to leaflet prolapse or degenerative disease of the valve or valvular apparatus, or both. Mitral regurgitation is common when the left ventricle is dilated: geometrical changes, including an increasingly spherical shape, result in annular dilatation, papillary muscle displacement, and tethering of the valve leaflets.

Methods for assessing the severity of mitral regurgitation are similar to those used to quantify aortic incompetence. The characteristics of the regurgitant jet on colour flow mapping (fig 3) provide important clues: more severe lesions cause broader jets filling more of the left atrium. The area of the regurgitant orifice and the volume of regurgitant blood can be estimated by using the proximal isovelocity surface area or continuity methods (see educational resources on bmj.com).

Mitral stenosis is almost exclusively caused by rheumatic heart disease and was the first valvular abnormality identified by echocardiography. M-mode scans can detect abnormal thickening and motion of the mitral valve leaflets (fig 4), and two dimensional echocardiography can assess the degree of valve opening. Doppler recordings determine the mean gradient across the valve and the pressure half time. The mitral valve orifice area (MVA) can then be calculated:

MVA (cm^2) = 220/pressure half time

For example, a valve with a pressure half time of 180 ms will have a calculated area of 220/180 = 1.2 cm^2. Valve area can also be assessed directly by planimetry or calculated using the proximal isovelocity surface area or continuity methods (see educational resources on bmj.com). The size of the left atrium and the right ventricular systolic pressure are also useful indicators of the severity of mitral valve stenosis. Right ventricular systolic pressure can be estimated by applying the simplified Bernoulli equation (see above) to the peak velocity of tricuspid regurgitation and adding this to the approximated right atrial pressure, estimated from the jugular venous pulsation or the calibre of the inferior vena cava, imaged from the subcostal window.

Echocardiography can also be used to determine whether the mitral valve is suitable for balloon valvuloplasty.[15] In general, valves which are very immobile or heavily calcified, have extensive thickening of the valve leaflets or chordae, or have severe associated mitral regurgitation will be unsuitable for percutaneous intervention.

Atrial fibrillation

Atrial fibrillation is common.[16] In some people, particularly those under 50 years, there may be no associated abnormalities of cardiac structure. In others it is related to valvular heart disease, coronary heart disease, diastolic dysfunction, or cardiomyopathy. Echocardiography can differentiate the underlying problem and guide treatment, in particular, the need for anticoagulation and suitability for cardioversion. It is usually best to control the heart rate before carrying out an echocardiogram.

Although echocardiographic findings are not the sole determinants of a patient's suitability for cardioversion, echocardiography can determine which patients with atrial fibrillation are likely to remain in sinus rhythm after cardioversion. Patients with major structural abnormalities, important left ventricular systolic dysfunction, or a left atrial diameter ≥4.5 cm are unlikely to maintain sinus rhythm.[13 17]

Stroke or transient ischaemic attack

Up to a fifth of ischaemic neurological events may be due to a cardioembolic source[13]; others, classified as cryptogenic, may also have a cardiac aetiology. The likelihood of a cardioembolic event depends on the clinical circumstances and, in particular, the probability of primary vascular disease and the presence of underlying cardiac disease. Younger patients have lower prevalence of significant atherosclerosis, and stroke is relatively more likely to have a cardioembolic aetiology. Events in multiple cerebrovascular territories makes a cardiac source more likely.

Echocardiography rarely shows a direct source of emboli, such as intracardiac thrombus, vegetations, or myxoma (fig 5). More commonly, it shows abnormalities which predispose to embolisation, such as mitral valve disease or a left ventricular aneurysm. A patent foramen ovale may be associated with paradoxical embolism when venous thrombus crosses into the arterial circulation. This can be shown by the rapid intravenous injection of agitated saline. This opacifies the right heart with microbubbles. With a patent foramen ovale, some of these bubbles may pass across to the left heart, particularly when the right-to-left shunt is accentuated by the Valsalva manoeuvre.

If the cardiovascular examination, electrocardiography, and chest x ray are all normal a cardiac source of embolisation is unlikely, particularly in elderly people,[18] and an echocardiogram is rarely warranted. In younger patients (often arbitrarily defined as <45 years[13]), the likelihood of atherosclerotic disease is lower and the risks of a cardiac source are higher. If a cardiac source is suspected and the transthoracic echocardiogram is equivocal, patients should undergo a transoesophageal scan.

A cardiac source of embolism should be suspected in cases of peripheral embolism, as cardiac emboli may also travel to peripheral vascular beds. Embolic occlusion of a large peripheral vessel makes a cardiac source more likely, as few other sites can give rise to thrombotic masses of this size. Indications for an echocardiogram are similar to those in patients with a cerebral event.

Screening echocardiograms

In some circumstances echocardiography is an appropriate screening test, even in the absence of cardiovascular symptoms. First degree relatives of patients with hypertrophic cardiomyopathy should have echocardiography, as this condition is commonly transmitted as an autosomal dominant trait.[19] First degree relatives of patients with idiopathic dilated cardiomyopathy should also undergo echocardiographic screening.[20]

Patients with borderline hypertension and equivocal results on electrocardiography may require an echocardiogram to determine the presence of left ventricular hypertrophy, as this may influence the decision to treat (in many cases, 24 hour ambulatory blood pressure monitoring is more useful). Conversely, in patients with an unequivocally raised blood pressure, specific electrocardiographic changes, or other evidence of end-organ damage an echocardiogram is usually superfluous. The exception is a young patient with severe hypertension, in whom aortic coarctation should be excluded.

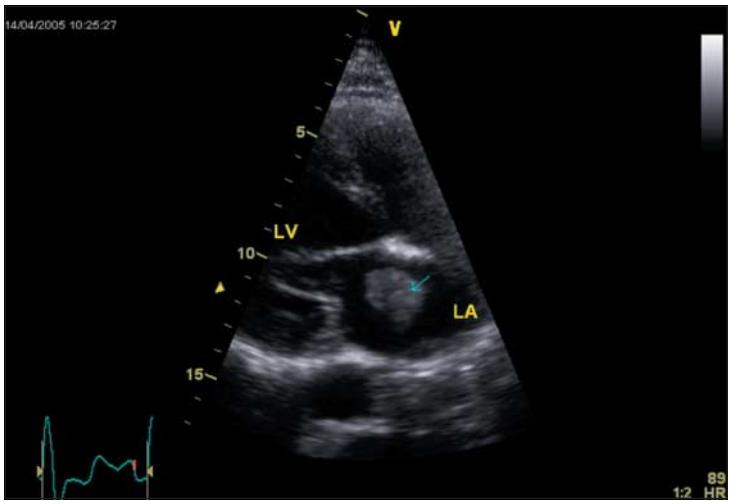

Fig 5 Apical four chamber view showing left atrial myxoma (arrow)

Echocardiography is also indicated in patients with collagen diseases, such as Marfan's syndrome, which are associated with a high incidence of cardiovascular abnormalities such as mitral valve prolapse or dilatation of the aortic root.[21] When the aortic root is dilated, careful echocardiographic or magnetic resonance imaging follow up is required as progressive and severe enlargement may occur and need surgical intervention.

Specialised techniques and recent advances in echocardiography

An increasing number of specialised echocardiographic techniques are available, including transoesophageal echocardiography, stress echocardiography, contrast echocardiography, three dimensional echocardiography, Doppler tissue imaging, and strain and strain rate imaging. Such advances have been paralleled by great technological improvements in echocardiographic equipment, including digitisation of images (improving their quality and the ability to store, compare, and transfer pictures), more advanced software packages (simplifying the analysis of data), and better quality images. With the expansion of traditional roles and the development of newer techniques there seems little doubt that the demands for echocardiography will continue to increase. It is important that services are developed accordingly and that clinicians from all specialties remain informed as to the potential uses of this versatile clinical tool.

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A memorable patient

… or two

As a young junior partner in a general practice in southern England in the 1970s, I was about to go out on a Saturday morning in the middle of February to make an unusually large number of home visits, a result of the winter flu outbreak that was putting enormous pressure on GPs and hospitals alike. The details of almost every visit simply read: "Very high temperature."

As I left the surgery, complaining at the length of the list, my wise senior partner (I had been his trainee) simply smiled benignly and said, "The trick today, David, will be to pick out the ones who have malaria and only spend two minutes on the ones with flu." (He had previously spent some time in Africa.) His flippant remark was not well received.

That afternoon, I visited one of those on my list—a young man lying on top of his bedclothes in a white towelling dressing gown, which contrasted strongly with his very sun-tanned skin that was quite out of place in an English winter. I asked where he had got his tan. He replied that he had just come back from Africa.

The duty haematologist at the local hospital clearly thought I was completely mad in asking him to screen for malarial parasites, but agreed to do so in view of the patient's history. Not long after receiving the specimens, he telephoned me. The patient did indeed have malaria, and the house physician—after initially announcing that there was a bed shortage and querying whether a young man with a fever really needed to come in—soon agreed to admit my patient, happy that a diagnosis was already in the bag.

Later in the evening, another young man telephoned to ask for a home visit: he ached all over and had high fever. In a rash moment, I said, "No problem, I will pop round and see you. It's either flu or malaria—there's a lot of that about this weekend."

The silence at the other end of the line—which I initially interpreted as the patient not appreciating my particular brand of humour—was soon followed by the anxious reply, "I have just come back from Africa; you don't really think it's malaria, do you?"

The duty haematologist at the local hospital thought I was joking when I requested yet more urgent tests for malaria, and I was as surprised as he was when I had to admit my second falciparum malaria of the weekend. The houseman had the two patients in two adjacent beds on his ward, and both made a good recovery. My reputation as a good diagnostician certainly grew—but how different it might have been if my wise former trainer had not thrown me his humorous challenge as I went out on my calls.

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We welcome articles up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake,* or any other piece conveying instruction, pathos, or humour. Please submit the article on http://submit.bmj.com Permission is needed from the patient or a relative if an identifiable patient is referred to. We also welcome contributions for "Endpieces," consisting of quotations of up to 80 words (but most are considerably shorter) from any source, ancient or modern, which have appealed to the reader.