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.

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. In accordance with NICE (National Institute for Health and Clinical Excellence) guidelines, 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, echocardiography can quantify the severity and determine the underlying cause. Many patients with heart failure have concomitant or predominant diastolic dysfunction, 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.

In patients with atrial fibrillation, echocardiography can identify underlying structural problems and guide treatment, especially the need for anticoagulation and the appropriateness of cardioversion.
abnormalities of motion can be assigned a score representing the severity of impaired contraction for each segment (fig 1). 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 removable 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. 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).

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

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.

**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:

$$\text{MVA (cm}^2\text{)} = \frac{220}{\text{pressure half time}}$$

For example, a valve with a pressure half time of 180 ms will have a calculated area of $\frac{220}{180} = 1.2 \text{ cm}^2$. 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. 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. 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.\(^*\)\(^{15,17}\)

**Stroke or transient ischaemic attack**

Up to a fifth of ischaemic neurological events may be due to a cardioembolic source,\(^*\)\(^{16}\) 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,\(^*\) and an echocardiogram is rarely warranted. In younger patients (often arbitrarily defined as < 45 years),\(^*\) 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.\(^*\) First degree relatives of patients with idiopathic dilated cardiomyopathy should also undergo echocardiographic screening.\(^*\)

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.

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.\(^*\) 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.

**Contributors:** PB had the idea for this article. GH did the literature search. GH and PB both wrote the article, and are both guarantors.

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Ethical approval was not required for this work.


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 laughingly 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 trainee had not thrown me his humorous challenge as I went out on my calls.

David A Churchill IT manager and retired general practitioner, Parklands Surgery, Chichester (wudenu@waitrose.com)

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 a thread of 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.
Tetraparesis associated with colchicine is probably due to inhibition by verapamil of the P-glycoprotein efflux pump in the blood-brain barrier

Uwe Tröger, Hartmut Lins, Jean-M Scherrmann, Claus-Werner Wallesch, Stefanie M Bode-Böger

An 83 year old man had an acute attack of gout. He treated himself with colchicine drops (2 mg in two days) and received didofenac because of continuous pain. Concurrently he had muscle weakness in his limbs. Four days later he became immobile and was transferred to hospital with flaccid tetraparesis (British Medical Research Council grade II-III). He had no signs of infection, hepatic or renal impairments, or stroke. Laboratory values were normal except a brief increase of creatine kinase to 1288.2 IU/l. Repeated nerve conduction studies did not show any relevant pathology, including normal terminal latencies and F wave latencies. Electromyography subsequently showed signs of lower motor neurone lesion. Muscle biopsy showed one isolated vacuole that could not be related to a colchicine induced myoneuropathy. Analysis of cerebrospinal fluid was unremarkable and indicated only a slight disturbance in the blood-cerebrospinal fluid barrier (protein 548 mg/l). An atypical Guillain-Barré syndrome was clinically diagnosed.

He was also taking 120 mg/day of slow release verapamil continuously for tachyarrhythmia, a sick sinus syndrome of the heart basically controlled by a pace maker, furosemide, acetylsalicylic acid, ambroxol, and theophylline. A tetraparesis due to neuromyopathy induced by colchicines was diagnosed because of concentration-time curves in serum and cerebrospinal fluid.

The diagnosis was revised to neuromyopathy induced by colchicine when excessive colchicine concentrations in serum as well as in cerebrospinal fluid were determined retrospectively using a radioimmunoassay. Although the serum concentration decreased slightly it remained constant in the cerebrospinal fluid in the following days (figure). The calculated half life in serum was increased eightfold compared with a dose and age matched reference population (272 hours v 34 hours). The cerebrospinal fluid to serum ratio of about 50% was much higher than normal (less than 10%). At the follow-up on day 40 he had recovered incompletely but colchicine was not detectable in serum.

Typical features of colchicine induced myoneuropathy such as high cumulative doses, long term treatment, or renal insufficiency were not found in our case. Verapamil is an inhibitor of CYP3A and a potent inhibitor (with norverapamil threefold stronger) of the P-glycoprotein transporter acting as a blood-brain barrier drug efflux pump. An increase of colchicine uptake was seen in a rat’s brain as well as in a rat’s plasma by verapamil up to 4.5-fold and 1.65-fold, respectively. These results indicate a dominant responsibility of the P-glycoprotein inhibition for the colchicine accumulation in cerebrospinal fluid. Colchicine is a substrate for CYP3A4 in the liver. Its inhibition might be responsible for increased cerebrospinal fluid concentrations.

Colchicine related tetraparesis is most likely due to a pharmacokinetic interaction in the human brain with verapamil and norverapamil. The Drug Commission of the German Medical Profession is not aware of any other cases of this drug interaction.

**Corrections and clarifications**

*Basic transthoracic echocardiography* The authors of this Clinical Review article, Graham S Hills and Peter Bloomfield, have alerted us to an error in their caption to figure 5 (BMJ 2005;330:1492-6, 18 Jun). It should read: “Modified parasternal long axis view showing left atrial myxoma.”

*The Law and Ethics of Medical Research: International Bioethics and Human Rights* In this book review by Berna Arda we mistakenly ascribed the wrong sex to the book’s author, Aurora Plomer, referring to her as “he” rather than “she” (BMJ 2003;326:298, 30 Jul).

*Radiation from CT and perfusion scanning in pregnancy* A mistyped abbreviation led to an error in a unit in this letter by J Valmai Cook and John Kyriou (BMJ 2005;331:350, 6 Aug). In the second paragraph, in relation to low dose technetium-99m perfusion lung scans, we should have said 50 megabecquerels (not millibecquerels). Also, we spelt out CPA as computer tomography for pulmonary angiography, whereas the procedure is more usually referred to as computed tomography pulmonary angiography.