Cardiac imaging in valvular heart disease

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ABSTRACT. The aim of this article is to provide a perspective on the relative importance and contribution of different imaging modalities in patients with valvular heart disease. Valvular heart disease is increasing in prevalence across Europe, at a time when the clinical ability of physicians to diagnose and assess severity is declining. Increasing reliance is placed on echocardiography, which is the mainstay of cardiac imaging in valvular heart disease. This article outlines the techniques used in this context and their limitations, identifying areas in which dynamic imaging with cardiovascular magnetic resonance and multislice CT are expanding.

Received 6 May 2010
Revised 16 January 2011
Accepted 19 January 2011

DOI: 10.1259/bjr/54030257

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Radiology

Valvular heart disease (VHD) is common and increases with age. In the past, VHD was typically caused by rheumatic heart disease, which remains a significant public health burden in developing countries. In industrialised nations, however, rheumatic disease has fallen substantially and VHD is now mainly degenerative in origin [1]. As a result of the link between degenerative VHD, older age and increasing life expectancy, there is a progressive increase in the prevalence of valve disease across Europe and the USA [2]. In the USA, the national prevalence of VHD is 2.5% after adjustment for age and sex, but using moderate or severe mitral regurgitation as an example, the numbers affected are expected to double by 2030 [3]. Currently, aortic stenosis (AS; 33.9%) is the most common form of VHD in Europe, followed by mitral regurgitation (MR; 24.8%), aortic regurgitation (AR; 10.4%) and mitral stenosis (MS; 9.5%). Native right-sided valve disease is much less common (1.2%). A significant proportion of the patients (28.1%) seen at any one time in hospital medical or surgical departments, or in medical outpatient clinics, will have had a percutaneous balloon commissurotomy, valve repair or valve replacement [1].

The detection of VHD in either hospital or community series is not a benign imaging observation but has profound consequences for those affected, with increased mortality and morbidity despite the availability of corrective surgical and percutaneous interventions. The Euro Heart Survey found that many patients present late with severe VHD; it also emphasised the importance of early detection as the key to improving prognosis, as patients are often denied intervention despite guideline-based indications because of advancing age [1]. The lower detection rate of VHD in the community compared with that in population studies suggests that a considerable proportion of patients with significant VHD are not diagnosed and do not present to medical services [2]. Many of the clinical signs of VHD taught in medical

schools relate to findings described many years ago in young patients presenting with rheumatic heart disease; these are no longer relevant to the older patients presenting with degenerative VHD today. Clinical examination is not a reliable guide to diagnosis or severity [4, 5]. This gap in the clinical diagnosis of VHD and the late presentation of many with severe disease emphasises the importance of quantitative, high-quality cardiac imaging. At present, echocardiography is the only method with sufficient availability to estimate the frequency and severity of VHD in the large populations who require assessment. Echocardiography continues to be the mainstay for diagnosis, assessment of severity and estimation of prognosis in VHD, despite advances in other modalities including cardiovascular magnetic resonance (CMR) and electrocardiogram (ECG)-gated multislice CT.

This review article discusses the role of currently available imaging modalities in the assessment of VHD. Each valve lesion is addressed in a separate section according to the prevalence of disease, with an additional section on prosthetic valves. Congenital valve lesions will not be discussed in this article, as another article in this issue covers this topic.

Aortic stenosis

AS is the most common indication for valve replacement in Europe and North America, and it is becoming increasingly prevalent as the population ages. Degenerative thickening of the aortic valve (AV) is present in 25% of people over the age of 65 years, and 3% of people over the age of 75 years have severe AS [1, 6–8]. The second most common cause of AS is a congenital bicuspid valve, which occurs with an overall prevalence of 0.5–1.0%. Rheumatic AS has become much less common in industrialised nations, although it remains the most common cause of valvular heart disease worldwide. Severe AS remains asymptomatic for many years and the risk of death in the absence of symptoms is less than 1% per year. Once symptoms appear, however, mortality rises sharply, with 10% dying in the first 6 months [9]. Clinical examination is unreliable in the

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diagnosis and assessment of severity, and transthoracic echocardiography (TTE) is the mainstay of diagnosis [4].

Diagnosis of AS requires good-quality two-dimensional echocardiography (2DE) to report on number of leaflets, pattern of leaflet thickening (more than 5 mm), mobility (restricted) and extent of calcification (mild/moderate/severe) [10] (Figure 1a). Heavy calcification is associated with rapid disease progression [11]. Grading of stenosis is based on Doppler assessment, with the minimum data set comprising maximal velocity ($V_{\max}$ m s⁻¹), mean gradient (mmHg) and aortic valve area (AVA) (cm²) [12, 13]. The AVA is a measurement of the physiological area of the aortic valve orifice. It is usually calculated with Doppler by using the continuity equation, which states that the flow in one area must equal the flow in a second area if there are no shunts in between. In practical terms, the flow from the left ventricular outflow tract (LVOT) is compared with the flow at the level of the aortic valve. Flow through the LVOT (LV stroke volume) is calculated by measuring the LVOT diameter (cm), calculating the LVOT area (cm²) assuming a circular orifice ($2\pi d^2/4$), and multiplying that value by the LVOT velocity time integral (VTI) (cm). The LVOT VTI is the area under the curve obtained from a spectral trace of velocity against a time axis using pulsed-wave Doppler. The AVA (cm²) is calculated by dividing the LV stroke volume by the AV VTI (cm) measured on the spectral Doppler display using continuous-wave Doppler (Figure 1b). Doppler during TTE is usually sufficient in the assessment of AS, but the different methods for grading AS are not always concordant [14]. Measurement of the AVA by planimetry, which is direct tracing of the aortic orifice at its point of maximal opening in mid-systole, is rarely feasible by TTE. Transoesophageal echocardiography (TOE) can be useful when transthoracic visualisation is poor and allows valve planimetry. Advances in real-time

three-dimensional echocardiography (RT3DE) have increased the accuracy of AV planimetry, largely because it overcomes the problem of ensuring that a measurement at the tips of the valve leaflets is not off-axis and therefore an over-estimate [15]. Direct measurement of stroke volume by RT3DE of the left ventricle (LV) may replace the continuity method of calculation of LV stroke volume using the left ventricular outflow tract area [16].

Recommendations for categorisation of severity have been published in major recent European guidelines, although there is a small difference with US recommendations in the use of mean gradient [10] (Table 1). Assessment of LV dimensions and LV function is a vital part of imaging the patient with AS, not only because surgery is indicated in patients who are asymptomatic but have impaired LV function (LV ejection fraction less than 50%) but also because care must be taken to identify patients with low-velocity, low-gradient severe AS when flow has been reduced by LV impairment [17]. Assessment must also take account of coexisting mitral regurgitation, which reduces the stroke volume ejected through the AV, and to mitral stenosis, which reduces LV pre-load. Severe AS is also associated with pulmonary hypertension and RV failure, which increases peri-operative risk [18].

Cardiac angiography is used as part of the work-up to surgery to diagnose coexisting coronary disease, but the stenosed AV is now almost never crossed owing to the 7% risk of stroke, pulmonary oedema and death [19].

The use of cardiac magnetic resonance (CMR) imaging in the assessment of VHD is increasing, largely because of its capability to measure LV size, mass and function accurately and reproducibly [20]. CMR is of most use in tracking asymptomatic patients with severe AS, looking for a fall in LVEF that would precipitate referral for surgery. When results from echocardiography are equivocal, CMR can help by estimating AVA either using the

Figure 1. (a) Transthoracic echocardiogram showing a trileaflet aortic valve (AV) with thickened leaflets and severe calcification. The patient had moderate aortic stenosis (AS) but progressed to valve replacement within 18 months of this scan. The image is annotated to show the left atrium (LA), right atrium (RA) and the right ventricle (RV). Also indicated is the non-coronary cusp (NCC), the left coronary cusp (LCC) and right coronary cusp (RCC) of the AV. (b) Continuous wave Doppler trace demonstrating maximal velocity, mean gradient and aortic valve area. Note that maximal velocity and mean gradient are consistent with moderate AS, whereas the AV area (AVA) suggests severe AS. Discrepancy in grading the severity of AS between lower than expected maximum velocity and mean gradient relative to a smaller valve area is common. This discrepancy may be due to coexisting left ventricle impairment, MR or mitral stenosis. It also occurs in older women with marked left ventricular hypertrophy who have low stroke volumes owing to small chamber size but severe AS. PG, peak gradient.

Table 1. Classification of the severity of left-sided valve disease in adults by echocardiography

Indicator	Mild	Aortic stenosis	
		Moderate	Severe
Jet velocity (m s^{-1})	<3.0	3.0–4.0	>4.0
Mean gradient (mmHg)	<25	25–40	>40
Valve area (cm^2)	>1.5	1.0–1.5	<1.0
Indicator	Mild	Mitral regurgitation	
		Moderate	Severe
Colour Doppler jet area	<20% LA area	20–40%	>40% LA area, large central MR jet
Vena contracta width (cm)	<0.3	0.3–0.69	≥ 0.70
Regurgitant volume (ml beat^{-1})	<30	30–59	≥ 60
Regurgitant fraction (%)	<30	30–49	≥ 50
Regurgitant orifice area (cm^2)	<0.20	0.20–0.39	≥ 0.40
Indicator	Mild	Aortic regurgitation	
		Moderate	Severe
Colour Doppler jet width	Jet width <25% LVOT	25–65%	Jet width >65% LVOT
Vena contracta width (cm)	<0.3	0.3–0.6	>0.6
Regurgitant volume (ml beat^{-1})	<30	30–59	≥ 60
Regurgitant fraction (%)	<30	30–49	≥ 50
Regurgitant orifice area (cm^2)	<0.10	0.10–0.29	≥ 0.30
Indicator	Mild	Mitral stenosis	
		Moderate	Severe
Mean gradient (mmHg)	<5	5–10.0	>10
Pulmonary artery systolic pressure (mmHg)	<30	30–50	>50
Valve area (cm^2)	>1.5	1.0–1.5	<1.0

LA, left atrium; LVOT, left ventricular outflow tract; MR, magnetic resonance.

Adapted from [64].

continuity equation [21] or more directly from the flow volume and VTI sampled from a single acquisition at the valve level [22]. Phase contrast velocity-encoded measurement of velocity and VTI tends to underestimate the severity of AS by about 10% compared with

Figure 2. Steady-state free-precession gradient cine with segmented k-space imaging at 1.5T of the aortic valve (AV) from a 76-year-old male requiring AV surgery at the time of coronary artery revascularisation. In this case, there is relatively little calcification and the valve orifice area (highlighted) can be measured as long as the planimetry is performed at the tips of the valve after piloting in two perpendicular planes.

TTE as a result of spatial and temporal averaging [23]. Alternatively, CMR planimetry can measure the anatomical AVA on the basis of direct visualisation of the valve orifice using steady-state free precession (SSFP) sequences (Figure 2). Planimetry, however, is a less than optimal approach in patients with calcific AS because leaflet calcification and jet turbulence often make accurate visualisation of the true orifice difficult, and because of the complex three-dimensional (3D) shape of the stenotic orifice.

Multislice cardiac CT may be useful in quantifying valve calcification and AVA [24, 25]. At present, it would appear that CT overestimates AVA compared with TTE [26]. CT is developing a particularly prominent role in planning transcatheter AV implantation, where it can deliver accurate measurement of the annulus, the angulation between LV apex and the aortic root, and extent of calcification [27]. In particular, aortic CT is vital in the planning of access routes for transcatheter AV implants, which may be delivered through the femoral arteries or the apex of the LV. Transfemoral access is perhaps the preferred route but requires adequate sizing to permit the delivery of the bulky stented AV (Figure 3).

Mitral regurgitation

Degenerative MR (owing to primary myxomatous disease, primary flail leaflets or annular calcification) is

Figure 3. Volume-rendered peripheral CT required prior to transcatheter aortic valve implantation via the transfemoral route, which is recognised to have a lower complication rate than the transapical approach. The transfemoral route requires a 22- or 24-French sheath and an appropriate minimum luminal diameter of the femoral and iliac vessels (8 mm for the 26-mm Edwards SAPIEN valve and 7.5 mm for the 23-mm Edwards SAPIEN valve). These diameters are likely to fall with the advent of smaller delivery catheters.

the most common aetiology in Europe, but ischaemic and functional MR are increasing in frequency [1]. A major aim of imaging in MR is the detection of valve degeneration that is amenable to repair rather than prosthetic mitral valve replacement (MVR). Repair has gained popularity as confidence in the technique has increased and because of an accumulation of data showing the durability and lower mortality and morbidity rates of repairs compared with replacements [28, 29]. Currently, almost 50% of patients in registries in Europe and the USA, and up to 90% in experienced centres, undergo repair [1]. Degenerative MR is usually related to mitral valve (MV) prolapse (excessive systolic motion of the leaflets more than 2 mm behind the plane of the annulus) and is the most repairable form.

TTE and TOE are the main methods for assessment of MR. The aims of imaging are to define the cause of MR (ischaemic or non-ischaemic), its mechanism (using the Carpentier classification), the degree of calcification and the localisation of lesion (using either the Carpentier or Duran nomenclature for scallops) [30]. TOE provides better imaging quality than TTE but provides incremental clinically meaningful information only when TTE is suboptimal or when complex, calcified or endocarditic lesions are suspected [31] (Figure 4a). Semi-quantitative visual assessment of the severity of the MR jet using colour flow Doppler is no longer used because of major limitations. Current methods focus on quantitative measurement of the vena contracta (VC (mm)) and flow convergence analysis, which calculates proximal isovelocity surface area (PISA) by using colour flow

Doppler to measure the effective regurgitant orifice area (EROA (mm²)) [32, 33]. The vena contracta is the narrowest portion of a regurgitant jet downstream of the regurgitant orifice. It is slightly smaller than the anatomical regurgitant orifice because of boundary effects and reflects a physiological orifice area. Accurate measurement requires visualisation of all three components that are present in all regurgitant jets: the area of proximal flow convergence (on the LV side of the MV in MR), the vena contracta (on the atrial side of the MV) and the area of jet expansion. Flow convergence analysis measuring PISA is a quantitative method that is based on the principle of conservation of mass. As blood flow converges towards a regurgitant orifice, it forms concentric isovelocity shells that are approximately hemispherical and are of decreasing surface area and increasing velocity. The flow in each of these hemispheres is the same as that crossing the orifice. Colour flow Doppler is used to image one of these hemispheres by setting a specific Nyquist limit or aliasing velocity. By setting the aliasing velocity to obtain a hemisphere, the flow rate (Q) through the regurgitant orifice is calculated as the product of the surface area of the hemisphere ($2\pi r^2$) and the aliasing velocity (V_a) ($Q = 2\pi r^2 \times V_a$). This flow rate across the PISA is equal to the flow rate at the regurgitant orifice. Assuming that the maximal PISA occurs at the peak regurgitant orifice, the maximal EROA is obtained by dividing the flow rate by the peak velocity of the regurgitant jet obtained by colour wave Doppler ($EROA = Q / \text{peak orifice velocity}$). The regurgitant volume is estimated as follows: regurgitant volume (ml) = EROA (cm²) / VTI (cm) of the regurgitant jet VTI (Figure 4b; Table 1). Measurement of left atrial (LA) size, LV dimensions and LVEF is important. Once LVEF falls below 60% or the end-systolic diameter is greater than 40–45 mm, patients are considered to have overt LV dysfunction, which is a clear indication for MV surgery. Increased LA volume (above 40 ml m⁻²) reflects volume overload and is associated with a worse outcome [34].

Parallel to the advancement of valve reconstruction surgery, 3D TTE produces reliable data for the comprehensive assessment of MR, with accuracy similar to that provided by 2D TOE [35]. Spatial localisation of pathological structures, including the commissural zones and the subvalvular apparatus, is improved by reconstructive 3D echocardiography, either by 3D TTE or by 3D TOE [36–38]. In fact, evaluation of MR is one of the first clinically established indications for 3DE (Figure 4c).

Left ventriculography and right-heart cardiac catheterisation are rarely needed in the assessment of MR [3]. Coronary angiography is done routinely in patients aged over 45 years.

CMR SSFP sequences provide information on the anatomy of the MV, although gradient echo cine pulse sequences are more sensitive for the localisation and sizing of regurgitant jets. Semi-quantitative assessments of MR jets by visual sizing or depth of penetration of MR into the LA have the same limitations as colour flow Doppler and should be avoided. Assessment of severity requires the calculation of regurgitant volume (RVol) by calculating the differences between RV and LV volumes, either by cine assessment of ventricular volumes or by velocity mapping and flow quantification in the pulmonary artery and aorta. In the absence of cardiac disease, RV and LV stroke volumes are equal, so

Figure 4. (a) Mid-oesophageal long axis view of the mitral valve (MV) made using transoesophageal echocardiography demonstrating a P2 scallop prolapse. The image is annotated to show the left atrium (LA), left ventricle (LV) and aortic root (AoR). There is elevation of the P2 scallop of the posterior mitral leaflet above the A2 scallop of the anterior mitral leaflet, rising above the line of the annulus. This type of lesion has the best long-term results from valve repair. (b) Colour flow Doppler of the MV in the same position as in (a) showing eccentric mitral regurgitation due to P2 prolapse, with the proximal isovelocity surface area on the left ventricular side of the valve measured during calculation of the effective regurgitant orifice of the MV. Also shown is the vena contracta on the left atrial side and the so-called zone of jet expansion. (c) Three-dimensional transoesophageal echocardiography of the MV with image reconstruction to show the left atrial side of the valve, the side of the valve seen by the cardiothoracic surgeon during MV repair. The view shows the MV annulus (Ann), the left atrial appendage (LAA) and an arrow indicating the P2 scallop of the posterior mitral leaflet above the plane of the valve and annulus.

that (assuming only the MV is affected and no intra-cardiac shunt is present) the difference in stroke volumes reflects the volume of MR. An alternative approach for measuring MR is to calculate total LV stroke volume with SSFP imaging and forward stroke volume in the aorta using phase-contrast velocity flow mapping. The difference between these values represents the MR volume [39–41] (Figure 5). It has been known for some time that through-plane phase-contrast velocity flow mapping can be used to assess MR volume when performed at the level of the MV annulus [42], although there are problems with this approach both due to movement of the mitral annulus during the cardiac cycle relative to the flow map and due to the problem of background phase-offset errors [43]. These techniques are validated but their incremental diagnostic role is not known and their use tends to be limited to those with equivocal results on echocardiography.

Multislice CT has similar accuracy to CMR and echocardiography in the quantitative analysis of MR using ventricular volumes, although again its incremental diagnostic role is not known. Once again, CT is forging a particular role in pre-operative planning for percutaneous coronary sinus-based MV repair [44].

Aortic regurgitation

The incidence of AR increases with age, typically peaking in the fourth to sixth decades of life. Moderate–severe AR has a prevalence of 0.5% and may be caused by disruption of the leaflets (degenerative, congenital bicuspid or rheumatic), dilatation of the root, or a combination of these [1, 2, 6]. AR creates not only volume overload but also an increase in afterload and therefore increased systolic wall stress, which often causes significant eccentric hypertrophy. Eccentric hypertrophy

Figure 5. (a) Steady-state free precession (SSFP) gradient cine in the horizontal long axis demonstrating the prolapse of the posterior mitral leaflet (PML) behind the plane of the anterior mitral leaflet (AML). (b) SSFP gradient cine in the vertical long axis demonstrating the jet of magnetic resonance (MR) directed anteriorly. It is important to note that the visual estimation of severity on SSFP is not accurate, particularly in very eccentric regurgitation (as in this case) when the volume of the jet appears small because of the Coandă effect. In this case, the stroke volume of the left ventricle (LV) calculated from the series of SSFP cines performed along the true short axis of the LV is 158 ml, with the stroke volume ejected into the aorta calculated to be 98 ml from the phase-encoded velocity map above the aortic valve (AV). The volume of MR is then calculated to be 60 ml and the regurgitant fraction: stroke volume (SVol) LV (158 ml) SVol AV (98 ml)/SVol LV (158 ml) $\times 100\%$ = 39%.

(as opposed to asymmetric hypertrophy) is defined by a relative wall thickness less than 0.42 as calculated using the formula: $2 \times \text{posterior wall thickness} / \text{LV internal dimension in diastole}$. Surgical correction of AR reduces afterload and therefore usually improves LVEF, whereas MVR increases afterload and usually worsens LVEF.

TTE is the key examination in the diagnosis, establishment of cause and assessment of severity of AR. Qualitative assessment using colour flow Doppler has high sensitivity and specificity for diagnosis. Validated methods for quantification of AR severity using colour flow Doppler include jet width relative to LV outflow tract diameter (%) and vena contracta (mm) (Figure 6). The vena contracta for AR is the narrowest portion of the regurgitant jet downstream on the LV side and has the same requirements as for MR. Quantitative assessment of regurgitation is feasible. Doppler estimations of mitral (or pulmonary) and aortic stroke volumes can be made using the continuity equation (the difference in stroke volume estimates and the regurgitant volume); and flow convergence can be analysed by measuring PISA using the same formula as for MR. These methods calculate regurgitation volume or fraction and EROA (mm^2), although both methods are less well established in AR than in MR as considerable sources of error exist in AR [33]. Continuous wave Doppler can be used to measure the regurgitant flow velocity of the AR jet, which reflects the diastolic pressure gradient between the aorta and the LV. The rate of deceleration and the derived pressure half-time correspond to the rate of equalisation of these pressures. With increasing AR severity, aortic diastolic pressure decreases more rapidly, the late diastolic jet velocity becomes lower, and pressure half-time (ms) becomes shorter. Other Doppler parameters include duration of diastolic flow reversal in the descending aorta, with more severe AR

associated with flow back towards the AV throughout diastole [45]. Most echocardiographers use a combination of these parameters to assess severity owing to the limitations of each measurement (Table 1). Measurement of the aorta at the annulus, sinuses of Valsalva, sinotubular junction and ascending aorta indexed for body surface area (BSA) are particularly important in AR. 2D and 3D TOE may be performed to better define the anatomy of the valve and ascending aorta, especially when valve-sparing intervention is considered.

Patients may remain asymptomatic with severe AR for many years, but an increase in LV dimensions (end-systolic dimension $>55\text{ mm}$) or fall in LVEF ($<55\%$) is a primary indication for surgery. Tracking patients with AR over many years to identify LV enlargement or LV dysfunction is perhaps the most important part of follow-up and, as a consequence, there has been a long history of replacing 2DE with other, more reproducible imaging techniques. Rest and exercise nuclear ventriculography is established in follow-up of AR [46]. 2DE has been supplanted by RT3DE for tracking LV volumes and function, as RT3DE eliminates the error in 2DE resulting from foreshortening of the LV and removes the need for geometric modelling. Multiple studies have confirmed RT3DE's improved agreement with reference techniques for LV volumes and function, including CMR and radio-nuclide ventriculography [47]. The reproducibility of RT3DE in determining LV volumes and function is similar to that of CMR, but RT3DE is not yet widely available and its use may still be limited by a poor acoustic window. CMR is often particularly useful in AR as it can combine reproducible measurement of LV volumes and function, measurement of aortic dimensions and calculation of regurgitant volume and fraction [48] (Figure 7). The avoidance of the radiation exposure inherent in nuclear

Figure 6. Transoesophageal echocardiogram taken in the mid-oesophageal three-chamber view showing the aortic root (AoR), left ventricle (LV) and left atrium (LA). The vena contracta of the aortic regurgitant jet is the narrowest portion of the jet on the ventricular side of the valve. This reflects the physiological regurgitant orifice and is a sensitive marker of severity.

ventriculography and CT makes CMR even more attractive in patients with AR, who often require longer follow-up than those with other forms of VHD.

Mitral stenosis

The prevalence of MS has decreased greatly in developed countries because the main aetiology is rheumatic (85%) and other causes are rare [1]. The main mechanism of rheumatic MS is commissural fusion, with other anatomical lesions being leaflet thickening, chordal shortening and fusion, and finally superimposed calcification [49]. The main lesion of degenerative MS is annular calcification, which has few or no haemodynamic consequences when isolated.

TTE is the key diagnostic tool in MS. First, MV planimetry is performed by direct tracing of the valve orifice in mid-diastole from the parasternal short axis

Figure 7. Steady-state free precession gradient cine imaging of the aortic valve in the perpendicular long axis view of the left ventricular outflow tract, showing the left ventricle (LV), aortic valve, aortic regurgitant (AR) jet and aortic root (AoR). In this case, the regurgitant fraction calculated from LV and right ventricle volumes, and by phase-encoded velocity mapping, was 48% (consistent with moderate AR).

view; this is the reference method for assessment of severity [MVA (cm^2)]. Severity of MS is also quantified by continuous wave Doppler of the diastolic pressure gradient using the Bernoulli equation to calculate mean gradient (mmHg) and pressure half-time, defined as the time interval (ms) between the maximum mitral gradient in early diastole and the time point when the gradient is half the maximum initial value (Table 1). Maximal velocity or gradient is ignored in the assessment of MS as it is governed by LA compliance and LV compliance. Second, morphological evaluation of the MV is important because of its implications for choice of intervention. The aim is to identify MS that is amenable to percutaneous balloon commissurotomy, and a variety of scoring systems are available that can be used to assess suitability and outcome [50] (Table 2).

TOE can be helpful if TTE provides suboptimal information on valve morphology and degree of coexisting MR. The main advantage of TOE is its much higher sensitivity in detecting LA thrombus, particularly in the LA appendage, which cannot be visualised routinely in adults on TTE. RT3DE provides genuine advantages in the assessment of MS by planimetry, optimising the positioning of the measurement plane and, therefore, improving reproducibility [51, 52] (Figure 8).

Cardiac catheterisation is restricted to cases of MS in which the results from echocardiography are equivocal. Right-heart catheterisation is useful in the presence of pulmonary hypertension, specifically to assess pulmonary vascular resistance.

CMR can be used to perform planimetry of the mitral orifice and pressure half-time assessment, although it tends to overestimate MVA when compared with TTE and cardiac catheterisation [53]. The reproducibility of CMR planimetry is acceptable [53], although use of CMR is hampered by the common association of MS with atrial fibrillation and with signal void caused by calcification. CT reliably detects MV calcification [54], and may have accuracy similar to that of echocardiography in assessing severity, although the case series analysed to date are small [55].

Right-sided valvular heart disease

Native right-sided valvular heart disease occurs only infrequently, with a prevalence of 1.2% [1]. TTE is the first-line cardiac imaging modality for right-sided VHD, specifically for structural assessment of valve leaflets (Figure 9). Pathological tricuspid regurgitation (TR) is usually functional owing to annular dilatation, which may arise because of LV failure, RV pressure and/or volume overload, or chamber dilatation. The tricuspid valve (TV) annulus is very dynamic and changes markedly with different loading conditions; even during the normal cardiac cycle, there is a 30% reduction in annular area in atrial systole [56]. This means that the degree of TR is very variable and heavily dependent on intravascular volume status, RV loading and RV function. Semi-quantitative evaluation of TR severity by TTE is based on the vena contracta width, dilatation of the inferior vena cava and reduction or reversal of systolic blood flow in the hepatic veins [33, 57]. CMR may be used to calculate TR RVols and fraction in the same way as for MR, but, more so than

Table 2. Wilkins' mitral valve morphology scoring for mitral stenosis

Grade	Mobility	Subvalvular thickening	Leaflet thickening	Calcification
1	Highly mobile with only leaflet tips restricted	Minimal thickening just below the mitral leaflets	Leaflets near normal in thickness (4–5 mm)	A single area of increased echo brightness
2	Leaflet mid and base portions have normal mobility	Thickening of chordal structures extending to one-third of the chordal length	Mid-leaflets normal, considerable thickening of margins (5–8 mm)	Scattered areas of brightness confined to leaflet margins
3	Valve continues to move forward in diastole, mainly from the base	Thickening extended to distal third of the chords	Thickening extending through the entire leaflet (5–8 mm)	Brightness extending into the mid-portions of the leaflets
4	No or minimal forward movement of the leaflets in diastole	Extension thickening and shortening of all chordal structures extending down to the papillary muscles	Considerable thickening of all leaflet tissue (>8 mm)	Extensive brightness throughout much of the leaflet tissue

Adapted from Wilkins et al [50].

in any other VHD, serial imaging of TR must take into account the clinical status of the patient. CMR is the reference method for assessment of RV volumes and function, although the technique remains challenging [58] (Figure 10). ECG-gated CT provides similar volumetric assessment, albeit at a lower temporal resolution than that provided by CMR.

By contrast, tricuspid stenosis (TS) is almost exclusively of rheumatic origin, occurring only rarely in isolation without left-sided valve lesions. As TS is also usually accompanied by TR, the transvalvular gradient is clinically more relevant for the assessment of severity and for decision-making than the actual stenotic valve area. Moreover, planimetry of the valve area is extremely difficult and even 3DE methods are not widely validated. Current methods for quantification include maximal velocity ($>2\text{ m s}^{-1}$), pressure half-time ($>190\text{ ms}$) and mean gradient ($>5\text{ mmHg}$) [12]. There is variation in TV flow with inspiration, so it is common for measurements to be taken at end-expiration. CMR phase-mapping is affected by annular plane displacement and is much less accurate in narrow, eccentric jets.

The pulmonary valve is the valve least likely to be affected by acquired heart disease. Indeed, almost all cases of pulmonary valve disease are congenital in origin.

Prosthetic heart valves

Patients who have undergone previous valve surgery represent an important proportion of patients with VHD, the large majority (82%) having undergone valve replacement [1]. History and clinical examination can alert physicians to significant prosthetic valve (PV) dysfunction, but cardiac imaging is required to confirm or refute this possibility. Echocardiography is the method of choice, but imaging by any modality is more technically demanding than that in native valve assessment. First, almost all replacement valves are obstructive compared with native valves, with the degree of obstruction varying according to their size and design. The problem in imaging is then to separate the expected "normal" haemodynamics of a PV from mild pathological obstruction and from patient-prosthesis mismatch

Figure 8. Three-dimensional echocardiogram using perpendicular views at 90° displayed in the green and red squares to define the tips of the mitral valve (MV) in mid-diastole at the point of maximal excursion of the leaflets. This point was identified by scrolling through images acquired at an average of $20\text{--}30\text{ frames s}^{-1}$. From these two planes, an en-face view of the MV is displayed in the blue square and the area of the MV orifice is traced; in this case, the area is 1.3 cm^2 , consistent with moderate mitral stenosis.

Figure 9. Transthoracic echocardiogram performed from the apical acoustic window to show the four-chamber view, with the left atrium (LA), left ventricle (LV), right atrium (RA) and right ventricle (RV) displayed. The image on the left is a standard two-dimensional image, that on the right is the simultaneously displayed colour Doppler, with yellow indicating flow towards the probe and blue flow away from the valve. In the RA, a jet of severe tricuspid regurgitation occupies most of the chamber. This jet has a wide vena contracta caused by thickening and fixation of the septal and anterosuperior leaflets of the tricuspid valve (TV), which shows thickened and restricted TV leaflets with severe tricuspid regurgitation caused by carcinoid heart disease. ASup, anterosuperior; Sep, septal.

(PPM), when the valve implanted is too small for the patient's requirements. Second, almost all replacement valves have trivial or mild transvalvular regurgitation (so-called "washing jets"), which varies according to the PV design and which must be distinguished from pathological regurgitation. Third, imaging may be complicated by artefacts and shielding from the PV. Any request for imaging of a PV should be rejected unless it contains information on the type and size of PV implanted and the date of replacement, together with some basic information on the patient, such as BSA, as this enables the imager to compare the velocities and gradients found with those expected from published tables of normal values [59]. In particular, baseline assessment using the imaging modality to be used during follow-up should be performed at 6–12 weeks post surgery for all PV replacements. The results then obtained are used to compare all subsequent changes in PV and ventricular function. PVs are imaged from multiple views to obtain information on occluder motion, thickening or calcification, and abnormal motion of the sewing ring (Figure 11). TTE imaging of occluder motion is often inadequate because of artefacts, and TOE is needed in most cases of suspected prosthetic dysfunction, thrombosis or endocarditis [60]. The minimum data set for PV assessment includes maximal velocity ($V_{\max} \text{ m s}^{-1}$), mean gradient (mmHg) and valve area [AVA (cm^2)], together with comment on regurgitation.

Cardiac catheterisation has a limited role in the assessment of PV dysfunction, although cinefluoroscopy is useful in evaluating the disc mobility of mechanical valves. Prospective studies have indicated that although Doppler echocardiography allows the measurement of gradients and areas, as well as the semi-quantification of

regurgitation, fluoroscopy is superior to echocardiography in identifying disc motion [61]. Cinefluoroscopy is rapid and easy to perform and provides valuable information not only in cases where mechanical PV obstruction is suspected but also when occluder motion is restricted, leading to transvalvar regurgitation. Cinefluoroscopy, however, has little value in bioprosthetic valves and is largely being replaced by ECG-gated multislice CT, which not only can identify stuck mechanical

Figure 10. Steady-state free precession gradient cine imaging in the horizontal long axis view, demonstrating displacement of the septal leaflet of the tricuspid valve (arrow) towards the apex of the right ventricle (RV) as a result of the failure of the tricuspid valve tissue to delaminate. This results in atrialisation of the RV and enlargement of the right atrium (RA), typical of Ebstein's anomaly. ASup, anterosuperior; Sep, septal.

Figure 11. Transoesophageal images of a bileaflet mechanical mitral valve prosthesis. In (a), both leaflets of the valve are closed; in (b), one leaflet opens while the other intermittently sticks shut on two-dimensional images, leading to (c) eccentric forward flow through only one opening valve on colour flow Doppler. (d) Cinefluoroscopy demonstrating the outline of one leaflet open away from the plane of the sewing ring, with the other stuck in-plane.

valves but also can quantify calcification and delineate pannus in bioprosthetic valves [62] (Figure 12). Systematic comparison of CT with echocardiography is limited as yet but is likely to develop significantly. Cardiac MRI is safe for all commercially available prosthetic heart valves, including the earliest inserted

such as the Björk–Shiley valve, but is less extensively used because of artefact in mechanical valves [63]. In fact, almost all sternal wires, clips, stents, occlusion devices and prosthetic valves within the chest are safe in a 1.5 T magnet, and the only problem within the magnet is interference with image quality resulting from local image

Figure 12. 64-slice electrocardiogram-gated CT of a 25mm Caromedics (Sorin Group, Milan, Italy) bileaflet mechanical mitral valve replacement with leaflets (a) open and (b) closed. Parallel alignment of valve leaflets should be seen in the open position.

artefact. On the other hand, no patient with a pacemaker or implanted defibrillator should approach the magnet, although newer MR-compatible pacemakers are available.

Conclusion

Cardiac imaging continues to develop at a pace in the assessment of VHD. Echocardiography remains at the forefront of assessment, with an emphasis on the use of 3D techniques to improve the reliability of data collected during long-term follow-up. There is increasing use of anatomical and dynamic imaging with CMR and multislice CT to provide supplementary diagnostic information, replacing echocardiography when limitations arise from loss of acoustic window. Future directions include increased use of peri-operative echocardiography during percutaneous valve interventions, replacing the use of fluoroscopy; increasing use of CMR for catheter and device guidance to expedite the planning and performance of percutaneous and surgical interventions in VHD; and replacement of cardiac catheterisation prior to surgical intervention for VHD by multislice CT.

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