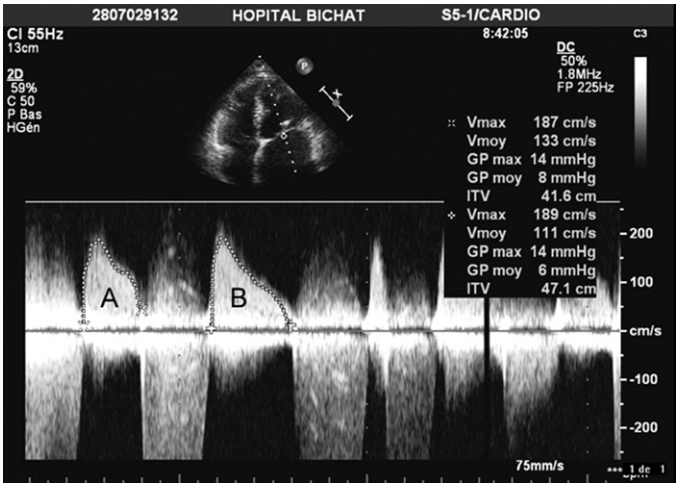

Figure 7 Determination of mean mitral gradient from Doppler diastolic mitral flow in a patient with severe mitral stenosis in atrial fibrillation. Mean gradient varies according to the length of diastole: it is 8 mmHg during a short diastole (**A**) and 6 mmHg during a longer diastole (**B**).

flow waveforms on the display screen. Mean gradient is the relevant haemodynamic finding (Figure 7). Maximal gradient is of little interest as it derives from peak mitral velocity, which is influenced by left atrial compliance and LV diastolic function.[45]

Heart rate at which gradients are measured should always be reported. In patients with atrial fibrillation, mean gradi ent should be calculated as the average of five cycles with the least variation of R–R intervals and as close as possible to normal heart rate.

Mitral gradient, although reliably assessed by Doppler, is not the best marker of the severity of MS since it is dependent on the mitral valve area (MVA) as well as a number of other factors that influence transmitral flow rate, the most important being heart rate, cardiac output, and associated MR.[46] However, the consistency between mean gradient and other echocardiographic findings should be checked, in particular in patients with poor quality of other variables (especially planimetry of valve area) or when such variables may be affected by additional conditions [i.e. pressure half-time ($T_{1/2}$) in the presence of LV diastolic dysfunction; see below]. In addition, mean mitral gradient has its own prognostic value, in particular following balloon mitral commissurotomy.

B.1.2. MVA Planimetry (Level 1 Recommendation). Theoretically, planimetry using 2D echocardiography of the mitral orifice has the advantage of being a direct measurement of MVA and, unlike other methods, does not involve any hypothesis regarding flow conditions, cardiac chamber compliance, or associated valvular lesions. In practice, planimetry has been shown to have the best correlation with anatomical valve area as assessed on explanted valves.[47] For these reasons, planimetry is considered as the reference measurement of MVA.[1,2]

Planimetry measurement is obtained by direct tracing of the mitral orifice, including opened commissures, if applicable, on a parasternal short-axis view. Careful scanning from the apex to the base of the LV is required to ensure that the CSA is measured at the leaflet tips. The measurement plane should be perpendicular to the mitral orifice, which has an elliptical shape (Figure 8).

Gain setting should be just sufficient to visualize the whole contour of the mitral orifice. Excessive gain setting may cause underestimation of valve area, in particular when leaflet tips are dense or calcified. Image magnification, using the zoom mode, is useful to better delineate the contour of the mitral orifice. The correlation data on planimetry was performed with fundamental imaging and it is unclear whether the use of harmonic imaging improves planimetry measurement.

The optimal timing of the cardiac cycle to measure planimetry is mid-diastole. This is best performed using the cineloop mode on a frozen image.

It is recommended to perform several different measurements, in particular in patients with atrial fibrillation and in those who have incomplete commissural fusion (moderate MS or after commissurotomy), in whom anatomical valve area may be subject to slight changes according to flow conditions.

Although its accuracy justifies systematic attempts to perform planimetry of MS, it may not be feasible even by experienced echocardiographers when there is a poor acoustic window or severe distortion of valve anatomy, in particular with severe valve calcifications of the leaflet tips. Although the percentage of patients in whom planimetry is not feasible has been reported as low as 5%, this number highly depends on the patient population.[48] The above-mentioned problems are more frequent in the elderly who represent a significant proportion of patients with MS now in industrialized countries.[49]

Another potential limitation is that the performance of planimetry requires technical expertise. Not all echocardiographers have the opportunity to gain the appropriate experience because of the low prevalence of MS in industrialized countries. The measurement plane must be optimally positioned on the mitral orifice. Recent reports suggested that real-time 3D echo and 3D-guided biplane imaging is useful in optimizing the positioning of the measurement plane and, therefore, improving reproducibility.[50,51] It also improves the accuracy of planimetry measurement when performed by less experienced echocardiographers.[52]

In the particular case of degenerative MS, planimetry is difficult and mostly not reliable because of the orifice geometry and calcification present.

B.1.3. Pressure half-time (Level 1 Recommendation). $T_{1/2}$ is defined as the time interval in milliseconds between the maximum mitral gradient in early diastole and the time point where the gradient is half the maximum initial value. The decline of the velocity of diastolic transmitral blood flow is inversely proportional to valve area (cm^2), and MVA is derived using the empirical formula:[53]

$$MVA = 220/T_{1/2}$$

$T_{1/2}$ is obtained by tracing the deceleration slope of the E-wave on Doppler spectral display of transmitral flow and valve area is automatically calculated by the integrated software of currently used echo machines (Figure 9). The Doppler signal used is the same as for the measurement of mitral gradient. As for gradient tracing, attention should be paid to the quality of the contour of the Doppler flow, in particular the deceleration slope. The deceleration slope is sometimes bimodal, the decline of mitral flow velocity being more rapid in early diastole than during the following part of the E-wave. In these cases, it is recommended that the deceleration slope in mid-diastole rather than the early deceleration slope be traced (Figure 10).[54] In the rare patients with a concave shape of the tracing, $T_{1/2}$ measurement may not be feasible. In patients with atrial fibrillation, tracing should avoid mitral flow from short diastoles and average different cardiac cycles.

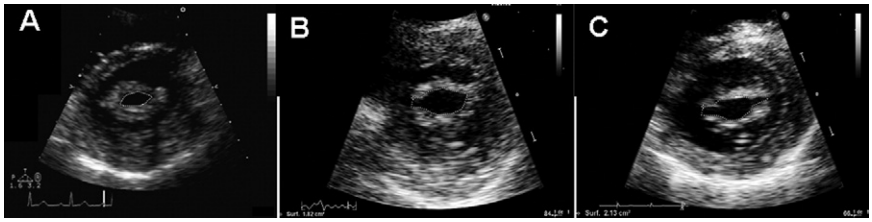

Figure 8 Planimetry of the mitral orifice. Transthoracic echocardiography, parasternal short-axis view. (**A**) Mitral stenosis. Both commissures are fused. Valve area is 1.17 cm^2. (**B**) Unicommissural opening after balloon mitral commissurotomy. The postero-medial commissure is opened. Valve area is 1.82 cm^2. (**C**) Bicommissural opening after balloon mitral commissurotomy. Valve area is 2.13 cm^2.

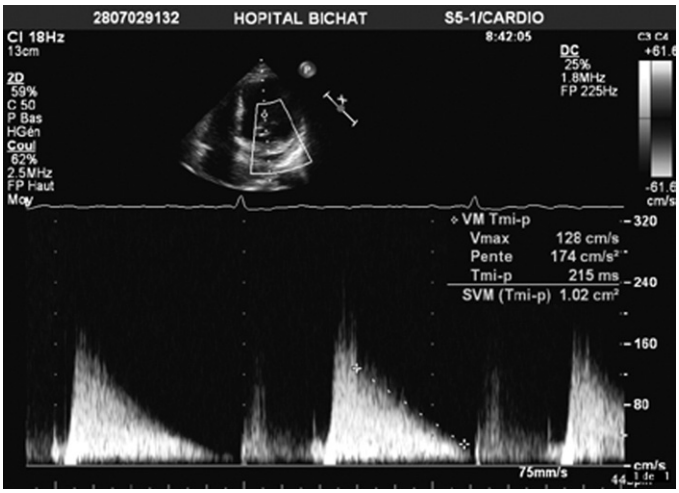

Figure 9 Estimation of mitral valve area using the pressure half-time method in a patient with mitral stenosis in atrial fibrillation. Valve area is 1.02 cm^2.

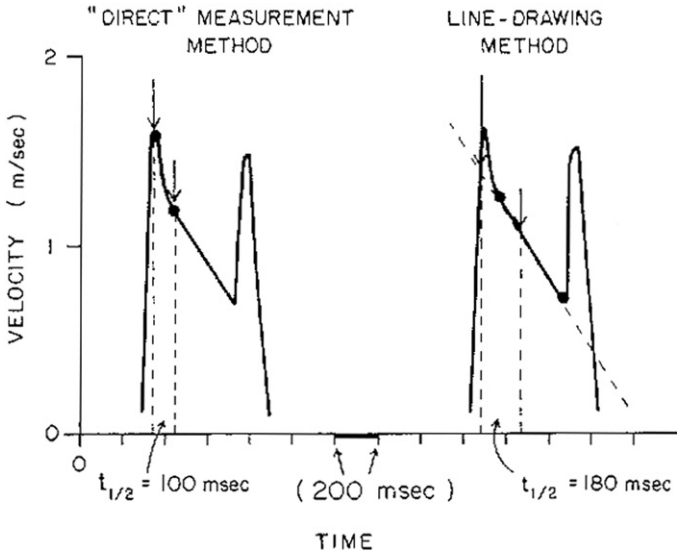

Figure 10 Determination of Doppler pressure half-time ($T_{1/2}$) with a bimodal, non-linear decreasing slope of the E-wave. The deceleration slope should not be traced from the early part (left), but using the extrapolation of the linear mid-portion of the mitral velocity profile (right). (Reproduced from Gonzalez *et al.*[54]).

The $T_{1/2}$ method is widely used because it is easy to perform, but its limitations should be kept in mind since different factors influence the relationship between $T_{1/2}$ and MVA.

The relationship between the decrease of mean gradient and MVA has been described and empirically validated using initially catheterization data and then Doppler data. However, fluid dynamics principles applied to simulations using mathematical models and in vitro modelling of transmitral valve flow consistently showed that LV diastolic filling rate, which is reflected by the deceleration slope of the E-wave, depends on MVA but also on mitral pressure gradient in early diastole, left atrial compliance, and LV diastolic func tion (relaxation and compliance).[53,55] The empirically deter mined constant of 220 is in fact proportional to the product of net compliance, i.e. the combined compliance of left atrium and LV, and the square root of maximum transmitral gradient in a model that does not take into account active relaxation of LV.[56] The increase in mean gradient is frequently compensated by a decreased compliance, and this may explain the rather good correlation between $T_{1/2}$ and other measurements of MVA in most series.

However, there are individual variations, in particular when gradient and compliance are subject to important and abrupt changes. This situation occurs immediately after balloon mitral commissurotomy where there may be important discrepancies between the decrease in mitral gradient and the increase in net compliance.[56] Outside the context of intervention, rapid decrease of mitral velocity flow, i.e. short $T_{1/2}$ can be observed despite severe MS in patients who have a particularly low left atrial compliance.[57] $T_{1/2}$ is also shortened in patients who have associated severe AR. The role of impaired LV diastolic function is more difficult to assess because of complex and competing interactions between active relaxation and compliance as regards their impact on diastolic transmitral flow.[58] Early diastolic deceleration time is prolonged when LV relaxation is impaired, while it tends to be shortened in case of decreased LV compliance.[59] Impaired LV diastolic function is a likely explanation of the lower reliability of $T_{1/2}$ to assess MVA in the elderly.[60] This concerns patients with rheumatic MS and, even more, patients with degenerative calcific MS which is a disease of the elderly often associated with AS and hypertension and, thus, impaired diastolic function. Hence, the use of $T_{1/2}$ in degenerative calcific MS may be unreliable and should be avoided.

B.1.4. Continuity equation (Level 2 Recommendation). As in the estimation of AVA, the continuity equation is based on the conservation of mass, stating in this case that the filling volume of diastolic mitral flow is equal to aortic SV.

$$\mathrm{MVA} = \pi\left(\frac{D^2}{4}\right)\left(\frac{\mathrm{VTI}_{\mathrm{Aortic}}}{\mathrm{VTI}_{\mathrm{mitral}}}\right)$$

where D is the diameter of the LVOT (in cm) and VTI is in cm.[61]

Table 5 Assessment of mitral valve anatomy according to the Wilkins score[64]

Grade	Mobility	Thickening	Calcification	Subvalvular Thickening
1	Highly mobile valve with only leaflet tips restricted	Leaflets near normal in thickness (4–5 mm)	A single area of increased echo brightness	Minimal thickening just below the mitral leaflets
2	Leaflet mid and base portions have normal mobility	Midleaflets normal, considerable thickening of margins (5–8 mm)	Scattered areas of brightness confined to leaflet margins	Thickening of chordal structures extending to one-third of the chordal length
3	Valve continues to move forward in diastole, mainly from the base	Thickening extending through the entire leaflet (5–8 mm)	Brightness extending into the mid-portions of the leaflets	Thickening extended to distal third of the chords
4	No or minimal forward movement of the leaflets in diastole	Considerable thickening of all leaflet tissue (>8–10 mm)	Extensive brightness throughout much of the leaflet tissue	Extensive thickening and shortening of all chordal structures extending down to the papillary muscles

The total score is the sum of the four items and ranges between 4 and 16.

Stroke volume can also be estimated from the pulmonary artery; however, this is rarely performed in practice because of limited acoustic windows.

The accuracy and reproducibility of the continuity equation for assessing MVA are hampered by the number of measurements increasing the impact of errors of measurements. The continuity equation cannot be used in cases of atrial fibrillation or associated significant MR or AR.

B.1.5. Proximal isovelocity surface area method (Level 2 Recommendation). The proximal isovelocity surface area method is based on the hemispherical shape of the convergence of diastolic mitral flow on the atrial side of the mitral valve, as shown by colour Doppler. It enables mitral volume flow to be assessed and, thus, to determine MVA by dividing mitral volume flow by the maximum velocity of diastolic mitral flow as assessed by CWD.

$$\text{MVA} = \pi(r^2)(V_{\text{aliasing}})/\text{Peak } V_{\text{mitral}} \cdot \alpha/180^0$$

where r is the radius of the convergence hemisphere (in cm), V_{aliasing} is the aliasing velocity (in cm/s), peak V_{Mitral} the peak CWD velocity of mitral inflow (in cm/s), and a is the opening angle of mitral leaflets relative to flow direction.[62]

This method can be used in the presence of significant MR. However, it is technically demanding and requires multiple measurements. Its accuracy is impacted upon by uncertainties in the measurement of the radius of the convergence hemisphere, and the opening angle.

The use of colour M-mode improves its accuracy, enabling simultaneous measurement of flow and velocity.[62]

B.1.6. Other indices of severity. Mitral valve resistance (Level 3 Recommendation) is defined as the ratio of mean mitral gradient to transmitral diastolic flow rate, which is calculated by dividing SV by diastolic filling period. Mitral valve resistance is an alternative measurement of the severity of MS, which has been argued to be less dependent on flow conditions. This is, however, not the case. Mitral valve resistance correlates well with pulmonary artery pressure; however, it has not been shown to have an additional value for assessing the severity of MS as compared with valve area.[63]

The estimation of pulmonary artery pressure, using Doppler estimation of the systolic gradient between right ventricle (RV) and right atrium, reflects the consequences of MS rather than its severity itself. Although it is advised to check its consistency with mean gradient and valve area, there may be a wide range of pulmonary artery pressure for a given valve area.[1,2] Nevertheless, pulmonary artery pressure is critical for clinical decision-making and it is therefore very important to provide this measurement.

B.2. Other echocardiographic factors in the evaluation of mitral stenosis *B.2.1. Valve anatomy.* Evaluation of anatomy is a major component of echocardiographic assessment of MS because of its implications on the choice of adequate intervention.

Commissural fusion is assessed from the short-axis parasternal view used for planimetry. The degree of commissural fusion is estimated by echo scanning of the valve. However, commissural anatomy may be difficult to assess, in particular in patients with severe valve deformity. Commissures are better visualized using real-time 3D echocardiography.[52]

Commissural fusion is an important feature to distinguish rheumatic from degenerative MS and to check the consistency of severity measurements. Complete fusion of both commissures generally indicates severe MS. On the other hand, the lack of commissural fusion does not exclude significant MS in degenerative aetiologies or even rheumatic MS, where restenosis after previous commissurotomy may be related to valve rigidity with persistent commissural opening.

Echocardiographic examination also evaluates leaflet thickening and mobility in long-axis parasternal view. Chordal shortening and thickening are assessed using long-axis parasternal and apical views. Increased echo brightness suggests calcification, which is best confirmed by fluoroscopic examination. The report should also mention the homogeneity of impairment of valve anatomy, in particular with regards to commissural areas in parasternal short-axis view.

Impairment of mitral anatomy is expressed in scores combining different components of mitral apparatus or using an overall assessment of valve anatomy[49,64,65] (Tables 5 and 6). Other scores have been developed, in particular taking into account the location of valve thickening or calcification in relation to commissures; however, they have not been validated in large series. No score has been definitely proven to be superior to another and all have a limited predictive value of the results of balloon mitral commissurotomy, which depends on other clinical and echocardiographic findings.[64]

Table 6 Assessment of mitral valve anatomy according to the Cormier score[48]

Echocardiographic group	Mitral valve anatomy
Group 1	Pliable non-calcified anterior mitral leaflet and mild subvalvular disease (i.e. thin chordae $\geq$10 mm long)
Group 2	Pliable non-calcified anterior mitral leaflet and severe subvalvular disease (i.e. thickened chordae $<$10 mm long)
Group 3	Calcification of mitral valve of any extent, as assessed by fluoroscopy, whatever the state of subvalvular apparatus

Thus, the echocardiographic report should include a comprehensive description of valve anatomy and not summarize it using a score alone.

B.2.2. Associated lesions. The quantitation of left atrial enlargement favours 2D echocardiography enabling left atrial area or volume to be evaluated. Standard time-motion measurement lacks accuracy because enlargement does not follow a spherical pattern in most cases. Left atrial spontaneous contrast as assessed by TEE is a better predictor of the thromboembolic risk than left atrial size.[66] Transoesophageal echocardiography has a much higher sensitivity than the transthoracic approach to diagnose left atrial thrombus, in particular when located in the left atrial appendage.

Associated MR has important implications for the choice of intervention. Quantitation should combine semi-quantitative and quantitative measurements and be particularly careful for regurgitation of intermediate severity since more than mild regurgitation is a relative contraindication for balloon mitral commissurotomy.[1,2,41] The mechanism of rheumatic MR is restriction of leaflet motion, except after balloon mitral commissurotomy, where leaflet tearing is frequent. The analysis of the mechanism of MR is important in patients presenting with moderate-to-severe regurgitation after balloon mitral commissurotomy. Besides quantitation, a traumatic mechanism is an incentive to consider surgery more frequently than in case of central and/or commissural regurgitation due to valve stiffness without leaflet tear. The presence of MR does not alter the validity of the quantitation of MS, except for the continuity-equation valve area.

Other valve diseases are frequently associated with rheumatic MS. The severity of AS may be underestimated because decreased SV due to MS reduces aortic gradient, thereby highlighting the need for the estimation of AVA. In cases of severe AR, the $T_{1/2}$ method for assessment of MS is not valid.

The analysis of the tricuspid valve should look for signs of involvement of the rheumatic process. More frequently, associated tricuspid disease is functional tricuspid regurgitation (TR). Methods for quantitating TR are not well established and highly sensitive to loading conditions. A diameter of the tricuspid annulus 40 mm seems to be more reliable than quantitation of regurgitation to predict the risk of severe late TR after mitral surgery.[2,67]

B.3. Stress echocardiography (Level 2 Recommendation) Exercise echocardiography enables mean mitral gradient and systolic pulmonary artery pressure to be assessed during effort. Semi-supine exercise echocardiography is now preferred to post-exercise echocardiography as it allows for the monitoring of gradient and pulmonary pressure at each step of increasing workload. Haemodynamic changes at effort are highly variable for a given degree of stenosis. Exercise echocardiography is useful in patients whose symptoms are equivocal or discordant with the severity of MS.[1,2] However, thresholds of mitral gradient and pulmonary artery pressure, as stated in guidelines to consider intervention in asymptomatic patients, rely on low levels of evidence.[1] Estimations of SV and atrioventricular compliance are used for research purposes but have no current clinical application. Dobutamine stress echocardiography has been shown to have prognostic value but is a less physiological approach than exercise echocardiography.[68,69]

C. How to Grade Mitral Stenosis

Routine evaluation of MS severity should combine measurements of mean gradient and valve area using planimetry and the $T_{1/2}$ method (Tables 7 and 8). In case of discrepancy, the result of planimetry is the reference measurement, except with poor acoustic windows. Assessment of valve area using continuity equation or the proximal isovelocity surface method is not recommended for routine use but may be useful in certain patients when standard measurements are inconclusive.

Associated MR should be accurately quantitated, in particular when moderate or severe. When the severity of both stenosis and regurgitation is balanced, indications for interventions rely more on the consequences of combined stenosis and regurgitation, as assessed by exercise tolerance and mean gradient, than any single individual index of severity of stenosis or regurgitation.[2] Intervention may be considered when moderate stenosis and moderate regurgita tion are combined in symptomatic patients.

Consequences of MS include the quantitation of left atrial size and the estimation of systolic pulmonary artery pressure. The description of valve anatomy is summarized by an echocardiographic score. Rather than to advise the use of a particular scoring system, it is more appropriate that the echocardiographer uses a method that is familiar and includes in the report a detailed description of the impair ment of leaflets and subvalvular apparatus, as well as the degree of commissural fusion.

Assessment of other valvular diseases should be particularly careful when intervention is considered. This is particularly true for the quantitation of AS and tricuspid annular enlargement.

Transthoracic echocardiography enables complete evaluation of MS to be performed in most cases. Transoesophageal echocardiography is recommended only when the transthoracic approach is of poor quality, or to detect left atrial thrombosis before balloon mitral commissurotomy or follow ing a thromboembolic event.[1,2] The use of cardiac catheterization to assess the severity of MS should be restricted to the rare cases where echocardiography is inconclusive or discordant with clinical findings, keeping in mind that the validity of the Gorlin formula is questionable in case of low output or immediately after balloon mitral commissurotomy.[1,2,70] Right-heart catheterization remains, however, the only investigation enabling pulmonary vascular resistance to be assessed, which may be useful in the case of severe pulmonary hypertension.

The normal MVA is 4.0–5.0 cm^2. An MVA area of $>$1.5 cm^2 usually does not produce symptoms. As the severity of stenosis increases, cardiac output becomes subnormal at rest and fails to increase during exercise. This is the main reason for considering MS significant when MVA is $<$1.5 cm^2 (Table 9).[1,2] Indexing on body-surface area is useful to take into account body size. However, no

Table 7 Recommendations for data recording and measurement in routine use for mitral stenosis quantitation

Data element	Recording	Measurement
Planimetry	– 2D parasternal short-axis view	– contour of the inner mitral orifice
	– determine the smallest orifice by scanning from apex to base	– include commissures when opened
	– positioning of measurement plan can be oriented by 3D echo	– in mid-diastole (use cine-loop)
	– lowest gain setting to visualize the whole mitral orifice	– average measurements if atrial fibrillation
Mitral flow	– continuous-wave Doppler	– mean gradient from the traced contour of the diastolic mitral flow
	– apical windows often suitable (optimize intercept angle)	– pressure half-time from the descending slope of the E-wave (mid-diastole slope if not linear)
	– adjust gain setting to obtain well-defined flow contour	– average measurements if atrial fibrillation
Systolic pulmonary artery pressure	– continuous-wave Doppler	– maximum velocity of tricuspid regurgitant flow
	– multiple acoustic windows to optimize intercept angle	– estimation of right atrial pressure according to inferior vena cava diameter
Valve anatomy	– parasternal short-axis view	– valve thickness (maximum and heterogeneity) – commissural fusion – extension and location of localized bright zones (fibrous nodules or calcification)
	– parasternal long-axis view	– valve thickness – extension of calcification – valve pliability – subvalvular apparatus (chordal thickening, fusion, or shortening)
	– apical two-chamber view	– subvalvular apparatus (chordal thickening, fusion, or shortening)
		Detail each component and summarize in a score

threshold of indexed valve area is validated and indexing on body-surface area overestimates the severity of valve stenosis in obese patients.

Ideally, the severity assessment of rheumatic MS should rely mostly on valve area because of the multiple factors influencing other measurements, in particular mean gradient and systolic pulmonary artery pressure. This justifies attempts to estimate MVA using the above-mentioned methods even in patients with severe valve deformity. The values of mean gradient and systolic pulmonary artery pressure are only supportive signs and cannot be considered as surrogate markers of the severity of MS. Abnormal values suggest moderate to severe stenosis. However, normal resting values of pulmonary artery pressure may be observed even in severe MS. In degenerative MS, mean gradient can be used as a marker of severity given the limitations of planimetry and $T_{1/2}$.

Stenosis severity is important, although it is only one of the numerous patient characteristics involved in decision-making for intervention, as detailed in guidelines.[1,2] Intervention is not considered in patients with MS and MVA >1.5 cm^2, unless in symptomatic patients of large body size. When MVA is <1.5 cm^2, the decision to intervene is based on the consequences of valve stenosis (symptoms, atrial fibrillation, pulmonary artery pressure) and the suitability of the patient for balloon mitral commissurotomy. Exercise testing is recommended in patients with MVA, <1.5 cm^2 who claim to be asymptomatic or with doubtful symptoms.

The impact of echocardiographic findings on the prognosis of MS has mainly been studied after balloon mitral commissurotomy. Multivariate analyses performed in studies reporting a follow-up of at least 10 years identified valve anatomy as a strong predictive factor of event-free survival.[71–74] Indices of the severity of MS or its haemodynamic consequences immediately after balloon commissurotomy are also predictors of event-free survival, whether it is MVA,[70,73] mean gradient,[70,72] and left atrial or pulmonary artery pressure.[72,73] The degree of MR following balloon mitral commissurotomy and baseline patient characteristics such as age, functional class, and cardiac rhythm are also strong predictors of long-term results of balloon mitral commissurotomy.[71–73]

Large studies of natural history and of results of surgical commissurotomy predate the current echocardiographic practice and thus do not enable the prognostic value of echocardiographic findings to be assessed.

IV. TRICUSPID STENOSIS

A. Causes and Anatomic Presentation

Tricuspid stenosis (TS) is currently the least common of the valvular stenosis lesions given the low incidence of rheumatic heart disease. In regions where rheumatic heart disease is still prevalent, TS is rarely an isolated disorder; more often, it is accompanied by MS. Other causes of TS include carcinoid syndrome (always combined with TR which

Table 8 Approaches to evaluation of mitral stenosis

Measurement	Units	Formula / Method	Concept	Advantages	Disadvantages
Valve area					
- planimetry by 2D echo	cm^2	tracing mitral orifice using 2D echo	direct measurement of anatomic MVA	- accuracy - independence from other factors	- experience required - not always feasible (poor acoustic window, severe valve calcification)
- pressure half-time	cm^2	$220 / T_{1/2}$	rate of decrease of transmitral flow is inversely proportional to MVA	easy to obtain	dependence on other factors (AR, LA compliance, LV diastolic function…)
- continuity equation	cm^2	$MVA = (CSA_{LVOT})(VTI_{Aortic}) / VTI_{Mitral}$	volume flows through mitral and aortic orifices are equal	independence from flow conditions	- multiple measurements (sources of errors) - not valid if significant AR or MR
- PISA	cm^2	$MVA = \pi(r^2)(V_{aliasing}) / \text{peak } V_{Mitral} \cdot \alpha/180°$	MVA assessed by dividing mitral volume flow by the maximum velocity of diastolic mitral flow	independence from flow conditions	technically difficult
Mean gradient	mm Hg	$\Delta P = \Sigma 4v^2 / N$	pressure gradient calculated from velocity using the Bernoulli equation	easy to obtain	dependent on heart rate and flow conditions
Systolic pulmonary artery pressure	mm Hg	$sPAP = 4v^2_{Tricuspid}$ + RA pressure	addition of RA pressure and maximum gradient between RV and RA	obtained in most patients with MS	- arbitrary estimation of RA pressure - no estimation of pulmonary vascular resistance
Mean gradient and systolic pulmonary artery pressure at exercise	mm Hg	$\Delta P = \Sigma 4v^2 / N$ $sPAP = 4v^2_{Tricuspid}$ + RA pressure	assessment of gradient and sPAP for increasing workload	incremental value in assessment of tolerance	- experience required - lack of validation for decision-making
Valve resistance	dyne. $sec^{-1} \cdot cm^{-5}$	$Mvres = \frac{P_{Mitral}}{(CSA_{LVOT})(VTI_{Aortic})} / DFT$	resistance to flow caused by MS	initially suggested to be less flow-dependent, but not confirmed	no prognostic value no clear threshold for severity no additional value vs. valve area

Level of recommendations: (1) appropriate in all patients (yellow); (2) reasonable when additional information is needed in selected patients (green); and (3) not recommended (blue).
AR, Aortic regurgitation; CSA, cross-sectional area; DFT, diastolic filling time; LA, left atrium; LV, left ventricle; LVOT, left ventricular outflow tract; MR, mitral regurgitation; MS, mitral stenosis; MVA, mitral valve area; MV_{res}, mitral valve resistance; ΔP, gradient; sPAP, systolic pulmonary artery pressure; r, the radius of the convergence hemisphere; RA, right atrium; RV, right ventricle; $T_{1/2}$, pressure half-time; v, velocity; VTI, velocity time integral; N, number of instantaneous measurements.

Table 9 Recommendations for classification of mitral stenosis severity

	Mild	Moderate	Severe
Specific findings			
Valve area (cm^2)	>1.5	1.0–1.5	<1.0
Supportive findings			
Mean gradient (mmHg)[a]	<5	5–10	>10
Pulmonary artery pressure (mmHg)	<30	30–50	>50

[a]At heart rates between 60 and 80 bpm and in sinus rhythm.

is commonly predominant),[75] rare congenital malformations,[76–79] valvular or pacemaker endocarditis and pacemaker-induced adhesions,[80–82] lupus valvulitis,[83] and mechanical obstruction by benign or malignant tumors.[84–87] Most commonly, TS is accompanied by regurgitation so that the higher flows through the valve further increase the transvalvular gradient and contribute to a greater elevation of right atrial pressures.[88]

As with all valve lesions, the initial evaluation starts with an anatomical assessment of the valve by 2D echocardiography using multiple windows such as parasternal right ventricular inflow, parasternal short axis, apical four-chamber and subcostal four-chamber. One looks for valve thickening and/or calcification, restricted mobility with diastolic doming, reduced leaflet separation at peak opening, and right atrial enlargement (Figure 11).[89] In carcinoid syndrome, one sees severe immobility of the leaflets, described as a 'frozen' appearance (Figure 12). Echocardiography also allows for the detection of valve obstruction by atrial tumours, metastatic lesions, or giant vegetations. Three-dimensional echocardiography can provide better anatomical detail of the relation of the three leaflets to each other and assessment of the orifice area.[90] Using colour flow Doppler one can appreciate narrowing of the diastolic inflow jet, higher velocities that produce mosaic colour dispersion, and associated valve regurgitation.

B. How to Assess Tricuspid Stenosis

The evaluation of stenosis severity is primarily done using the haemodynamic information provided by CWD. Although there are reports of quantification of orifice area by 3D echocardiography, the methodology is neither standardized nor sufficiently validated to be recommended as a method of choice. The tricuspid inflow velocity is best recorded from either a low parasternal

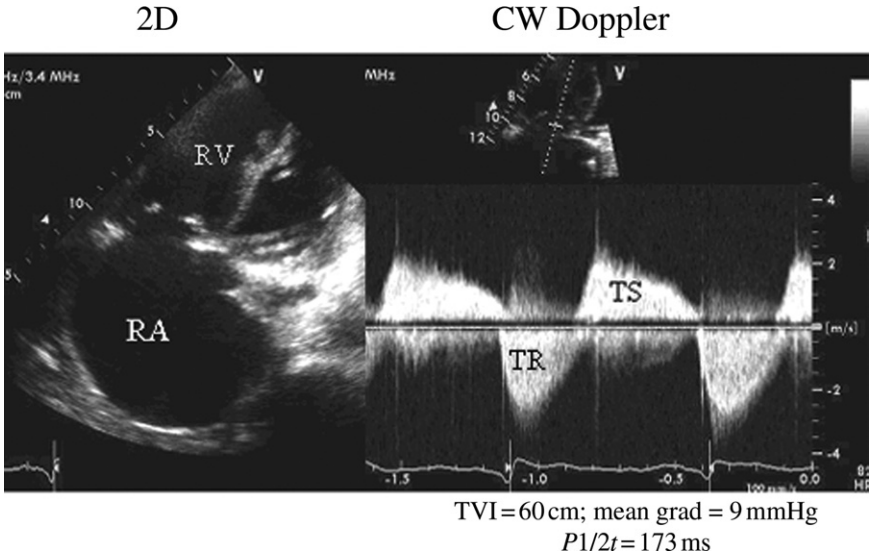

Figure 11 The left panel illustrates a 2D echocardiographic image of a stenotic tricuspid valve obtained in a modified apical four-chamber view during diastole. Note the thickening and diastolic doming of the valve, and the marked enlargement of the right atrium (RA). The right panel shows a CW Doppler recording through the tricuspid valve. Note the elevated peak diastolic velocity of 2 m/s and the systolic tricuspid regurgitation (TR) recording. The diastolic time–velocity integral (TVI), mean gradient (Grad), and pressure half-time ($T_{1/2}$) values are listed.

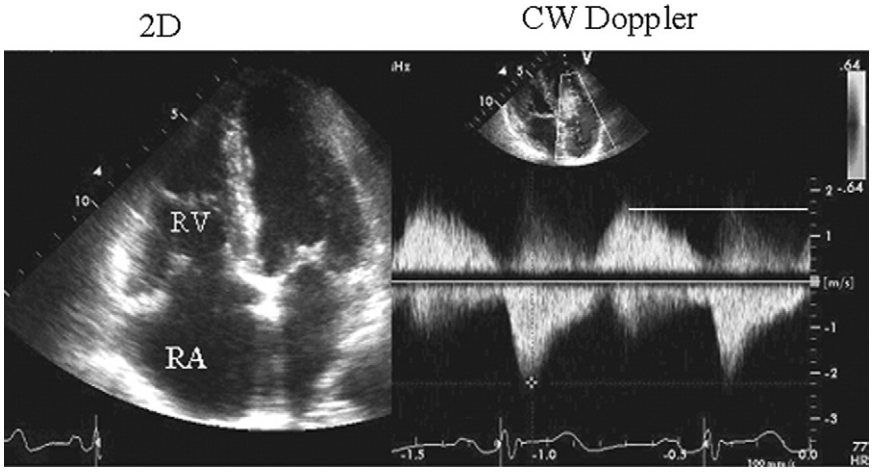

Figure 12 The left panel illustrates a 2D echocardiographic image of a tricuspid valve in a patient with carcinoid syndrome, obtained in an apical four-chamber view during systole. Note the thickening and opened appearance of the valve. The right panel shows a continuous-wave Doppler recording through the tricuspid valve. Note an elevated peak diastolic velocity of 1.6 m/s and the systolic TR recording.

right ventricular inflow view or from the apical four-chamber view. For measurement purposes, all recording should be made at sweep speed of 100 mm/s.[90] Because tricuspid inflow velocities are affected by respiration, all measurements taken must be averaged throughout the respiratory cycle or recorded at end-expiratory apnea. In patients with atrial fibrillation, measurements from a minimum of five cardiac cycles should be averaged. Whenever possible, it is best to assess the severity of TS at heart rates $<$100 bpm, preferably between 70 and 80 bpm. As with MS, faster heart rates make it impossible to appreciate the deceleration time (or pressure half-time).

The hallmark of a stenotic valve is an increase in transvalvular velocity recorded by CWD (Figures 11 and 12). Peak inflow velocity through a normal tricuspid valve rarely exceeds 0.7 m/s. Tricuspid inflow is normally accentuated during inspiration; consequently, with TS, it is common to record peak velocities $>$1.0m/s that may approach 2 m/s during inspiration. As a general rule, the mean pressure gradient derived using the $4v^2$ equation is lower in tricuspid than in MS, usually ranging between 2 and 10 mmHg, and averaging around 5 mmHg. Higher gradients may be seen with combined stenosis and regurgitation.[91–93]

The primary consequence of TS is elevation of right atrial pressure and development of right-sided congestion.Because of the frequent presence of TR, the transvalvular gradient is clinically more relevant for assessment of severity and decision-making than the actual stenotic valve area. In addition, because anatomical valve orifice area is difficult to measure (not withstanding future developments in 3D), and TR is so frequently present, the typical CWD methods for valve

Table 10 Findings indicative of haemodynamically significant tricuspid stenosis

Specific findings	
Mean pressure gradient	≥5 mmHg
Inflow time–velocity integral	>60 cm
$T_{1/2}$	≥190 ms
Valve area by continuity equation[a]	≤1 cm^{2a}
Supportive findings	
Enlarged right atrium ≥moderate	
Dilated inferior vena cava	

[a]Stroke volume derived from left or right ventricular outflow. In the presence of more than mild TR, the derived valve area will be underestimated. Nevertheless, a value ≤1 cm^2 implies a significant haemodynamic burden imposed by the combined lesion.

area determination are not very accurate. The pressure half-time method ($T_{1/2}$) has been applied in a manner analogous to MS. Some authors have used the same constant of 220, while others have proposed a constant of 190 with valve area determined as: $190/T_{1/2}$.[93] Although validation studies with TS are less than those with MS, valve area by the $T_{1/2}$ method may be less accurate than in MS. This is probably due to differences in atrioventricular compliance between the right and left side, and the influence of right ventricular relaxation, respiration, and TR on the pressure half-time. However, as a general rule, a longer $T_{1/2}$ implies a greater TS severity with values >190 frequently associated with significant (or critical) stenosis.

In theory, the continuity equation should provide a robust method for determining the effective valve area as SV divided by the tricuspid inflow VTI as recorded with CWD.[94] The main limitation of the method is obtaining an accurate measurement of the inflow volume passing through the tricuspid valve. In the absence of significant TR, one can use the SV obtained from either the left or right ventricular outflow; a valve area of ≤1 cm^2 is considered indicative of severe TS. However, as severity of TR increases, valve area is progressively underestimated by this method. Nevertheless, a value ≤1 cm^2, although it is not accounting for the additional regurgitant volume, may still be indicative of a significant hemodynamic burden induced by the combined lesion.

C. How to Grade Tricuspid Stenosis

From a clinical standpoint, the importance of an accurate assessment of TS is to be able to recognize patients with haemodynamically significant stenosis in whom a surgical- or catheter-based procedure may be necessary to relieve symptoms of right-sided failure. In the presence of anatomic evidence by 2D echo of TS, the findings listed in Table 10 are consistent with significant stenosis with or without regurgitation.

V. PULMONIC STENOSIS

Echocardiography plays a major role in the assessment and management of pulmonary valve stenosis.[95] It is useful in detecting the site of the stenosis, quantifying severity, determining the cause of the stenosis, and is essential in determining an appropriate management strategy.[96] Ancillary findings with pulmonary stenosis such as right ventricular hypertrophy may also be detected and assessed. Although the majority of pulmonary stenosis is valvular, narrowing of the right ventricular outflow tract (RVOT) below the valve from concurrent right ventricular hypertrophy may occur as may narrowing of the pulmonary artery sinotubular junction above the valve.

A. Causes and Anatomic Presentation

Pulmonary stenosis is almost always congenital in origin. The normal pulmonary valve is trileaflet. The congenitally stenotic valve may be trileaflet, bicuspid, unicuspid, or dysplastic.[97]

Acquired stenosis of the pulmonary valve is very uncommon. Rheumatic pulmonary stenosis is rare even when the valve is affected by the rheumatic process.[98] Carcinoid disease is the commonest cause of acquired pulmonary valve disease (combined stenosis and regurgitation with usually predominant regurgitation) and this may be sufficiently severe to require prosthetic replacement. Various tumors may compress the RV outflow tract leading to functional pulmonary stenosis. These tumors may arise from within the heart or associated vasculature or be external to the heart and compress from without.[99,100] Pulmonary valve stenosis may also occur as part of more complex congenital lesions such as tetralogy of Fallot, complete atrioventricular canal, double outlet RV, and univentricular heart. Peripheral pulmonary artery stenosis may co-exist with valvular pulmonary stenosis such as in Noonan's syndrome and Williams syndrome.

Stenosis below (proximal to) the pulmonary valve may result from a number of causes, both congenital and acquired. Congenital ventricular septal defect (VSD) may also be associated with RV outflow tract obstruction secondary to development of obstructive midcavitary or infundibular muscle bundles (double chamber RV) or in rare cases as a result of the jet lesion produced by the VSD in this area. Severe right ventricular hypertrophy of any cause but in some cases caused by valvular pulmonary stenosis itself may be responsible for narrowing of the infundibular area below the pulmonary valve. Iatrogenic causes include prior surgery or intervention on this area. Other causes include hypertrophic or infiltrative processes such as hypertrophic obstructive cardiomyopathy or glycogen storage disorders and compression from a tumour or vascular structure.

Stenosis of the pulmonary artery above the valve (distal to the valve) may occur in the main pulmonary trunk at the bifurcation, or more distally in the branch vessels. In rare instances, a membrane just above the valve may cause stenosis. Pulmonary artery stenosis may occur as an isolated finding without other malformations.

B. How to Grade Pulmonary Stenosis

Pulmonic stenosis severity Quantitative assessment of pulmonary stenosis severity is based mainly on the transpulmonary pressure gradient. Calculation of pulmonic valve area by planimetry is not possible since the required image plane is in general not available. Continuity equation or proximal isovelocity surface area method, although feasible in principle, has not been validated in pulmonary stenosis and is rarely performed.

B.1.1. Pressure gradient. The estimation of the systolic pressure gradient is derived from the transpulmonary velocity flow curve using the simplified Bernoulli equation $\Delta P = 4v^2$. This estimation is reliable, as shown by the good correlation with invasive measurement using cardiac catheterization.[101] Continuous-wave Doppler is used to assess the severity when even mild stenosis is present. It is important to line up the Doppler sample volume parallel to the flow with the aid of colour flow mapping where appropriate. In adults, this is usually most readily performed from a parasternal short-axis view but in children and in some adults the highest gradients may be found from

Table 11 Grading of pulmonary stenosis

	Mild	Moderate	Severe
Peak velocity (m/s)	<3	3–4	>4
Peak gradient (mmHg)	<36	36–64	>64

the subcostal window. A modified apical five-chamber view may also be used where the transducer is angled clockwise to bring in the RV outflow tract. Ideally, the highest velocity in multiple views should be used for the determination.[102,103]

In most instances of valvular pulmonary stenosis, the modified Bernoulli equation works well and there is no need to account for the proximal velocity as this is usually, 1 m/s. There are exceptions to this, however. In the setting of subvalvular or infundibular stenosis and pulmonary stenosis as part of a congenital syndrome or as a result of RV hypertrophy, the presence of two stenoses in series may make it impossible to ascertain precisely the individual contribution of each. In addition, such stenoses in series may cause significant PR resulting in a higher Doppler gradient compared with the net pressure drop across both stenoses.[104] Pulsed-wave Doppler may be useful to detect the sites of varying levels of obstruction in the outflow tract and in lesser degrees of obstruction may allow a full evaluation of it. Muscular infundibular obstruction is frequently characterized by a late peaking systolic jet that appears 'dagger shaped', reflecting the dynamic nature of the obstruction; this pattern can be useful is separating dynamic muscular obstruction from fixed valvular obstruction, where the peak velocity is generated early in systole.

In certain situations, TEE may allow a more accurate assessment of the pulmonary valve and RVOT. The pulmonary valve may be identified from a mid-esophageal window at varying transducer positions from 50 to 90, anterior to the aortic valve. The RVOT is often well seen in this view. It is in general impossible to line up CW to accurately ascertain maximal flow velocity. Other windows in which the pulmonary outflow tract may be interrogated include the deep transgastric view in which by appropriate torquing of the transducer, the RV inflow and outflow may be appreciated in a single image. This view can allow accurate alignment of the Doppler beam with the area of subvalvar/valvular stenosis through the RV outflow tract.

In pulmonary valve stenosis, the pressure gradient across the valve is used to ascertain severity of the lesion more so than in left-sided valve conditions due in part to the difficulty in obtaining an accurate assessment of pulmonary valve area. The following definitions of severity have been defined in the 2006 American College of Cardiology/American Heart Association (ACC/AHA) guidelines on the management of valvular heart disease:[1]

Severe stenosis (Table 11): a peak jet velocity >4 m/s (peak gradient >64 mmHg) Moderate stenosis: peak jet velocity of 3–4 m/s (peak gradient 36–64 mmHg)
Mild stenosis: peak jet velocity is: <3 m/s (peak gradient less than 36 mmHg).

In determining the need for intervention, no specific Doppler gradients have been agreed on.

Severity of pulmonary stenosis using Doppler gradients has been based on catheterization data with demonstration of reasonable correlation between instantaneous peak Doppler gradients and peak-to-peak gradients obtained by catheterization. Typically though, Doppler peak gradients tend to be higher than peak-to-peak catheterization gradients.[102] Doppler mean gradient has been shown in one study to correlate better with peak-to-peak catheterization gradient but is not commonly used.[105]

B.1.2. Other indices of severity. A useful index of severity is to determine the RV systolic pressure in patients with pulmonary stenosis from the tricuspid regurgitant velocity and the addition of an estimate of right atrial pressure. The pulmonary artery systolic pressure should be RV systolic pressure − pulmonary valve pressure gradient. In settings where there are multiple stenoses in the RV outflow tract or in the more peripheral pulmonary tree (sometimes associated with valvular pulmonary stenosis), the failure of the measured pulmonary valve gradient to account for much of the RV systolic pressure may be a clue for the presence of alternative stenoses.

B.1.3. Valve anatomy. Evaluation of anatomy is important in defining where the stenosis is maximal, as discussed above. Valve morphology is often evident especially the thin mobile leaflets seen with the dome-shaped valve. Dysplastic leaflets move little and are rarely associated with the post-stenotic dilatation common in dome-shaped leaflets. Calcification of the valve is relatively rare so the valve appearance does not play a huge role in decisions for balloon valvuloplasty. However, the size of the pulmonary annulus should be measured in order to define the optimal balloon size for successful dilatation of the valve.[106]

B.1.4. Associated lesions. Pulmonic stenosis especially when severe may be associated with right ventricular hypertrophy, eventually right ventricular enlargement, and right atrial enlargement. Given the unusual shape of the RV and its proximity to the chest wall, accurate estimation of RV hypertrophy and enlargement may be difficult. The parasternal long-axis and subcostal long-axis views are often best in assessing RV hypertrophy. The normal thickness of the RV is ~2–3 mm but given the difficulties in estimating thickness, a thickness of >5mm is usually considered abnormal. RV enlargement is typically assessed in the apical or subcostal four-chamber view.[107–109]

As described above, pulmonary stenosis may form part of other syndromes or may be associated with other congenital lesions. Dilatation of the pulmonary artery beyond the valve is common and is due to weakness in the arterial wall in a manner analogous to bicuspid aortic valve and is not necessarily commensurate with the degree of obstruction. Detection of other lesions such as infundibular stenosis, VSD, or tetralogy of Fallot is all important in the assessment of these patients.

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