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POSITION STATEMENT

2024 CSANZ Position Statement on Indications, Assessment and Monitoring of Structural and Valvular Heart Disease With Transthoracic Echocardiography in Adults

Adrian Chong, MBBS, BPharm, FRACP [a], Tony Stanton, MBChB, PhD, FRACP [b], Andrew Taylor, MBBS, PhD, FRACP [c], David Prior, MBBS, PhD, FRACP [d], Andre La Gerche, MBBS, PhD, FRACP [e], Bonita Anderson, DMU (Cardiac), MAppSc (MedUltrasound), ACS [f], Gregory Scalia, MBBS, MMedSc, FRACP [g], Jennifer Cooke, MBBS, FRACP [h], Arun Dahiya, MBBS, FRACP [i], Andrew To, MBChB, FRACP [j], Mark Davis, MBChB, PhD, FRACP [k], Philip Mottram, MBBS, PhD, FRACP [l], Stuart Moir, MBBS, PhD, FRACP [l], David Playford, MBBS, PhD, FRACP [m], Devan Mahadavan, MBBS, PhD, FRACP [n], Liza Thomas, MBBS, PhD, FRACP [o], Sudhir Wahi, MBBS, MD, FRACP [p,*]

[a]Department of Cardiology, Princess Alexandra Hospital, Mater Hospital Brisbane, University of Queensland, Brisbane, Qld, Australia
[b]Sunshine Coast University Hospital, School of Health University of Sunshine Coast, School of Medicine and Dentistry Griffith University, Birtinya, Qld, Australia
[c]Department of Cardiology, Royal Melbourne Hospital, Alfred Hospital, Melbourne, Vic, Australia
[d]Albury Wodonga Health, Albury, NSW, Australia
[e]St Vincent's Hospital, Baker Heart and Diabetes Institute, University of Melbourne, Melbourne, Vic, Australia
[f]Cardiac Sciences Unit, The Prince Charles Hospital, Queensland University of Technology, Brisbane, Qld, Australia
[g]The Prince Charles Hospital, University of Queensland, Brisbane, Qld, Australia
[h]Department of Cardiology, Eastern Health, Monash University, Melbourne, Vic, Australia
[i]Department of Cardiology, Princess Alexandra Hospital, Logan Hospital, Griffith University, Brisbane, Qld, Australia
[j]Department of Cardiology, Health New Zealand Waitemata, Auckland, New Zealand
[k]Waikato Hospital, Hamilton, New Zealand
[l]Victorian Heart Institute, Monash University, Melbourne, Vic, Australia
[m]The University of Notre Dame, Fremantle, WA, Australia
[n]Department of Cardiology, Queen Elizabeth Hospital, Lyell McEwin Hospital, Adelaide, SA, Australia
[o]Department of Cardiology, Westmead Hospital, Westmead Clinical School University of Sydney, South West Clinical School University of New South Wales, Sydney, NSW, Australia
[p]Department of Cardiology, Princess Alexandra Hospital, University of Queensland, Brisbane, Qld, Australia

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*Corresponding author at: Assoc. Prof. Sudhir Wahi, Director of Cardiology, Director of Echocardiography, Department of Cardiology, Princess Alexandra Hospital, University of Queensland, Brisbane, Qld 4102, Australia; Email: s.wahi@uq.edu.au

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Transthoracic echocardiography (TTE) is the most widely available and utilised imaging modality for the screening, diagnosis, and serial monitoring of all abnormalities related to cardiac structure or function. The primary objectives of this document are to provide (1) a guiding framework for treating clinicians of the acceptable indications for the initial and serial TTE assessments of the commonly encountered cardiovascular conditions in adults, and (2) the minimum required standard for TTE examinations and reporting for imaging service providers. The main areas covered within this Position Statement pertain to the TTE assessment of the left and right ventricles, valvular heart diseases, pericardial diseases, aortic diseases, infective endocarditis, cardiac masses, pulmonary hypertension, and cardiovascular diseases associated with cancer treatments or cardio-oncology. Facilitating the optimal use and performance of high quality TTEs will prevent the over or under-utilisation of this resource and unnecessary downstream testing due to suboptimal or incomplete studies.

Keywords Transthoracic echocardiography • Indications • Comprehensive • Serial • Surveillance

Introduction

Transthoracic echocardiography (TTE) is the primary imaging modality for the comprehensive screening, diagnosis, and serial monitoring of all abnormalities related to cardiac structure or function. An excessive increase in the use of TTE, due to its widespread availability and open accessibility, has led to significant concerns regarding inappropriate usage and cost-ineffectiveness. With the rising burden of cardiovascular disease in the ageing population, and the competing interests of increasing demands for diagnostic imaging and health budgetary constraints, the utility of echocardiography must be rationalised and ideally be evidence-based. An appropriate diagnostic imaging study has been previously defined as "one in which the expected incremental information, combined with clinical judgement, exceeds the expected negative consequences by a sufficiently wide margin for a specific indication that the procedure is generally considered acceptable care and a reasonable approach for the indication" [1,2]. The primary objectives of this document are to provide (1) a guiding framework for treating clinicians of the acceptable indications for the initial and serial TTE assessments of the most commonly encountered cardiovascular conditions in adults, and (2) the minimum required standard for TTE examinations and reporting for imaging service providers.

This CSANZ Position Statement represents the expert opinions of the writing committee, and where available, incorporates recommendations from available published evidence and contemporary clinical guidelines. While the listed cardiovascular conditions are not comprehensive, the primary goal was to include the major clinical indications or cardiac diseases which use echocardiography in the initial diagnosis and for longitudinal surveillance. The main areas covered within this Position Statement pertain to the TTE assessment of:

1. The left ventricle (LV)
2. The right ventricle (RV)
3. Aortic valve (AV) diseases
4. Mitral valve (MV) diseases
5. Right-sided valvular diseases
6. Pericardial diseases
7. Aortic diseases
8. Infective endocarditis (IE)
9. Cardiac masses
10. Pulmonary hypertension (PHTN)
11. Cardio-oncology

It is the consensus of the writing committee that the allocated examination time for a comprehensive routine TTE should be 45 minutes, depending on the indication(s) for the study and complexity of the findings. This recommended time is for image acquisition and measurement performance only and does not include the examination preparation (i.e., explanation of the procedure, obtaining patient consent, blood pressure measurements, and patient set-up), the write-up of preliminary findings, or the cleaning and disinfecting of equipment between patients. Longer examination times (>45 minutes) may be required for complex cases while shorter times (<30 minutes) may be adequate for focussed studies, such as the serial evaluation of pericardial effusions. Attempting to perform a comprehensive TTE in <30 minutes will lead to a significant reduction in scan quality, measurement inaccuracy, sonographer injuries, and unnecessary repeat examinations.

While a summary of the TTE requirements for a thorough examination and key parameters have been included, a comprehensive review of the echocardiographic assessment for each cardiovascular condition is beyond the scope of this Position Statement. Readers are encouraged to review relevant societal guidelines with respect to methodology, advantages, and limitations, as required. Of note, the TTE evaluation of patients with adult congenital heart disease has been excluded, as these patients should ideally be assessed in tertiary or specialised centres with the appropriate level of expertise.

The Key Recommendations made in this Position Statement are summarised in Box 1.

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Box 1. Key Recommendations.

1. Left ventricle (LV)

- An initial transthoracic echocardiogram (TTE) is recommended for investigation of acute haemodynamic instability, as well as symptoms or signs of cardiac failure or ischaemia, arrhythmias or electrocardiographic (ECG) abnormalities, and hypertension.
- Repeat TTE is recommended if there is a change or deterioration in the patient's clinical status or following optimisation of guideline-directed medical therapy in heart failure with reduced ejection fraction to decide on further therapies (e.g., cardiac resynchronisation therapy [CRT] or an implantable cardioverter–defibrillator [ICD]).
- TTE surveillance in stable heart failure to detect subclinical changes prior to overt clinical deterioration may be considered every 2 years, if the patient is a candidate for further escalation of therapy.
- TTE is appropriate for monitoring of asymptomatic patients with genetic cardiomyopathies (e.g., hypertrophic cardiomyopathy, Fabry disease) for ongoing risk stratification and periodic interval screening of first-degree relatives.
- Routine repeat TTE surveillance is not indicated in post-acute coronary syndrome, stable coronary artery disease, or hypertensive patients without resultant/residual LV dysfunction, in the absence of clinical change.
- Minimum requirements for echocardiographic evaluation will depend on the underlying condition but must include quantitative assessments of LV size, systolic function (including a numerical LV ejection fraction [LVEF]), wall thickness, diastolic parameters, left atrial volume index (LAVI), valvular function, and estimates of right atrial and pulmonary pressures. Ultrasound enhancing agents (UEA) should be used to enhance LV endocardial border delineation when required.
- The routine use of 3D (for LV volumes and LVEF) and deformation imaging (LV global longitudinal strain [GLS]) are strongly encouraged where possible.

2. Right ventricle (RV)

- An initial TTE is recommended for investigation of symptoms or signs of right heart failure, suspected pulmonary hypertension (PHTN), following a pulmonary embolism (PE), or suspected arrhythmogenic cardiomyopathy (AC).
- Repeat TTE for serial assessment is recommended if there is a change or deterioration in the patient's clinical status, for ongoing evaluation of PHTN during treatment with pulmonary vasodilator therapy, post-PE for monitoring of RV function and development of chronic thrombo-embolic PHTN, and ongoing screening for an AC-phenotypic expression in genetically predisposed or affected individuals and their first-degree relatives.
- Minimum requirements for echocardiographic evaluation must include qualitative and quantitative assessments of RV size and systolic function, right-sided valvular disease, and pulmonary pressures in accordance with contemporary guidelines.

3. Aortic valve (AV) diseases, including AV replacements (AVR)

- TTE is recommended to establish the diagnosis in patients with symptoms or signs of AV disease (aortic stenosis, aortic regurgitation, and/or bicuspid aortic valves).
- TTE is recommended for re-evaluation in patients with established AV disease who present with new or changed symptoms or physical examination findings, and prior to or during pregnancy.
- Surveillance interval for repeat TTEs in asymptomatic patients with known AV disease will depend on the stage or severity of the valvular lesion(s).
- A baseline "fingerprint" TTE is recommended post-surgical AVR (SAVR) followed by routine annual surveillance after 5 years for bioprosthetic SAVRs, or earlier if there is change in the patient's clinical status, or suspected SAVR dysfunction.
- A baseline TTE is recommended following a transcatheter AVR (TAVR) at 30 days and annually thereafter, but interval assessment should be tailored to the individual patient depending on comorbidities and echocardiographic findings.
- Repeat TTE is recommended within 3–6 months after initial diagnosis of structural valve degeneration in bioprosthetic SAVRs or TAVRs to assess for rapid progression.
- No routine surveillance is recommended for mechanical AVRs following a "normal" baseline post-operative TTE, in the absence of clinical change or other associated cardiac pathology requiring ongoing monitoring.
- Minimum requirements for echocardiographic evaluation of native AV disease or post-AVR must include qualitative, semi-quantitative, and quantitative assessments of valvular function and secondary effects on underlying ventricular function in accordance with contemporary guidelines.

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Box 1. (continued).

4. Mitral valve (MV) diseases, including MV replacements (MVR) and repairs (MVr)

- TTE is recommended to establish the diagnosis in patients with symptoms or signs of MV disease (mitral regurgitation or stenosis).
- TTE is recommended for re-evaluation in patients with established MV disease who present with new or changed symptoms or physical examination findings (including new onset atrial fibrillation), and prior to or during pregnancy.
- TTE is recommended for pre-procedural decision making prior to planned MV interventions.
- Surveillance interval for repeat TTEs in asymptomatic patients with known MV disease will depend on the stage or severity of the valvular lesion(s).
- A baseline "fingerprint" TTE is recommended following a MVr and MVR (surgical or transcatheter).
- Routine surveillance TTE is recommended annually beginning at 5 years post-surgical bioprosthetic MVR, or earlier if there is a change in the patient's clinical status, or suspected dysfunction.
- Routine surveillance TTE is recommended every 3 years after surgical MVr, or earlier if there is a change in the patient's clinical status, or suspected dysfunction.
- No routine surveillance is recommended for mechanical MVRs following a "normal" baseline post-operative TTE, in the absence of clinical change or other associated cardiac pathology requiring ongoing monitoring.
- More frequent routine surveillance is required for transcatheter MVr (including edge-to-edge leaflet repair, direct annuloplasty systems, and artificial chords) and transcatheter MVR.
- Minimum requirements for echocardiographic evaluation of native MV disease, post-MVr or MVR must include qualitative, semiquantitative, and quantitative assessments of valvular function, secondary effects on underlying ventricular function, and pulmonary pressures in accordance with contemporary guidelines.

5. Right-sided valvular diseases

- TTE is recommended to establish the diagnosis in patients with symptoms or signs of right-sided valvular disease (tricuspid or pulmonic stenosis or regurgitation).
- TTE is recommended for re-evaluation in patients with established right-sided valvular disease who present with new or changed symptoms or physical examination findings.
- Surveillance interval for repeat TTEs in asymptomatic patients with known right-sided valvular disease will depend on risk factors (e.g., previous corrective surgery for congenital heart disease) and stage or severity of the valvular lesion(s).
- Minimum requirements for echocardiographic evaluation of right-sided valvular diseases must include qualitative, semiquantitative, and quantitative assessments of valvular function, secondary effects on underlying ventricular function, and pulmonary pressures in accordance with contemporary guidelines.

6. Pericardial diseases

- An initial TTE is recommended for investigation of symptoms or signs of suspected pericardial diseases including acute or recurrent pericarditis, pericardial effusions, constrictive pericarditis, pericardial tumour or cyst, and congenital absence of the pericardium.
- Repeat TTE for serial assessment is recommend if there is a clinical change or deterioration in the patient's clinical status and for ongoing monitoring of "concerning" pericardial effusions.
- Repeat TTE is recommended within 3 months to assess the efficacy of therapy in medically managed transient constriction or effusive–constrictive pericarditis.
- TTE is recommended for procedural guidance for diagnostic or therapeutic pericardiocentesis.
- Repeat TTE is recommended immediately post-pericardiocentesis if the patient has ongoing symptoms or a large amount of fluid was drained, or within 24–48 hours to re-assess for any residual effusion and its haemodynamic effect. Timing and frequency of subsequent TTE assessments will depend on the patient's clinical status, as determined by the treating physician.
- Repeat TTE is recommended within 24–48 hours post-pericardiectomy for haemodynamic assessment and to identify any immediate post-operative complications. Timing and frequency of subsequent TTE assessments to evaluate the efficacy of surgery (pericardial thickening or recurrence of constrictive physiology) and monitor for changes in cardiac function will depend on the patient's clinical status, as determined by the treating physician.
- Minimum requirements for echocardiographic evaluation must include qualitative and quantitative assessments of the pericardial pathology (e.g., size, location, and character of the pericardial effusion, and pericardial thickening), haemodynamic consequences (e.g., evidence of raised intrapericardial pressure or other features of tamponade/constrictive physiology), and underlying ventricular and valvular function. Simultaneous imaging with a respirometer (slow sweep speed between 25–50mm/s) should be performed for assessment of enhanced ventricular interdependence.

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Box 1. (continued).

7. Aortic diseases

- TTE is recommended for assessment of suspected acute aortic syndromes, congenital anomalies (e.g., aortic coarctation), and screening for thoracic aortic aneurysms in patients with conditions associated with aortopathies (e.g., bicuspid aortic valves, genetic syndromes, or non-syndromic familial thoracic aortic diseases).
- Repeat TTE for serial assessment of thoracic aortic aneurysms is recommended if there is a change in clinical symptoms or examination findings, to determine rate of expansion, and/or timing of intervention when the treatment threshold is reached (surveillance interval for repeat TTEs in asymptomatic patients with thoracic aortic aneurysms will depend on the size of the aorta).
- Surveillance TTE is recommended following thoracic aortic intervention depending on the underlying pathology, type of repair, and presence of concomitant valvular lesions.
- Minimum requirements for echocardiographic evaluation must include measurements of landmark aortic dimensions and comprehensive assessment of any associated valvular (and secondary ventricular) abnormalities or dysfunction.

8. Infective endocarditis (IE)

- TTE is recommended to assist in the diagnosis of IE in the appropriate clinical setting.
- Repeat TTE may be performed in patients with suspected IE and a negative initial study for further assessment in 5–7 days if the clinical suspicion of IE remains high.
- Transoesophageal echocardiography (TOE) should be performed in patients with a positive TTE (if clinically appropriate) for confirmation, exclusion of local complications, or to assist with diagnosis in the setting of intracardiac prosthesis/devices or an equivocal TTE (i.e., poor images, more than mild valvular regurgitation or thickening).
- Repeat TTE and/or TOE is recommended if a new complication of IE is suspected (e.g., new murmur, embolism, persisting fever, heart failure, abscess, or atrioventricular block).
- Repeat TTE is indicated at completion of treatment (antibiotic ± surgery) for evaluation of the "new" baseline.
- Minimum requirements for echocardiographic evaluation must include qualitative and quantitative assessments of the vegetation(s) (e.g., size, location, mobility), any associated valvular dysfunction or local complications, and underlying ventricular function.

9. Cardiac masses

- TTE is recommended for assessment of a known or suspected cardiac mass based on clinical presentation (e.g., systemic emboli) or when incidentally detected by an alternate imaging modality.
- Serial TTE may be appropriate during conservative management to monitor for interval progression (that would influence treatment decisions) or if there is a change in clinical status.
- Repeat TTE is indicated following definitive intervention to serve as a baseline for future comparison or to monitor for recurrence.
- Minimum requirements for echocardiographic evaluation must include qualitative and quantitative assessments of the mass (or masses) (e.g., size, location, attachment, involvement of adjacent structures), potential haemodynamic consequences (e.g., functional valvular obstruction or regurgitation), risk of embolic complications, and underlying ventricular and valvular function.
- UEAs may be beneficial in patients with suboptimal image quality to delineate the attachment of the mass, and/or for assessment of vascularity.

10. Pulmonary hypertension (PHTN)

- An initial TTE is recommended for evaluation of patients with symptoms or signs of suspected PTN or screening of patients with conditions associated with an increased likelihood of PHTN (e.g., connective tissue diseases).
- Repeat TTE for serial assessment of PHTN is required to monitor therapeutic response to disease-specific treatments (i.e., Pharmaceutical Benefits Scheme prescribing requirement).
- Surveillance interval for repeat TTE for monitoring of PHTN will be determined by the World Health Organization clinical class or group, stage of therapy, clinical and/or disease stability, and need for follow-up of co-existent ventricular or valvular dysfunction.
- Minimum requirements for echocardiographic evaluation must include measurement of the right ventricular systolic pressure (RVSP [including the use of agitated saline or UEAs for tricuspid regurgitation Doppler signal enhancement and surrogate measures of pulmonary pressures or vascular resistance]), and qualitative/quantitative assessments of both right/left-sided ventricular and valvular anatomy/function.

11. Cardio-oncology

- An initial TTE is recommended prior to initiation of systemic chemotherapy or radiotherapy for baseline assessment and risk stratification.
- Repeat TTE is indicated during cancer therapy for regular serial assessment (surveillance intervals determined by the specific anticancer regimen), if there is a clinical change or deterioration, and after conclusion of treatment to allow detection of early or late cancer therapy-related cardiac dysfunction and initiation of cardioprotective therapy.
- Minimum requirements for echocardiographic evaluation must include quantitative assessments of LV size, systolic function (including three-dimensional left ventricular ejection fraction [3D LVEF] and global longitudinal strain [GLS] where possible), diastolic parameters, valvular function, and RVSP.

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1. The Left Ventricle

Assessment of left ventricular (LV) size and function is perhaps the most fundamental component of the TTE examination. While an evaluation of LV size and function is performed in every TTE, there are a number of conditions where this is the principal indication for the imaging request. In these instances, serial scanning plays an important role in ongoing patient management in terms of assessing response to treatment, guiding disease-specific therapy, and re-evaluation of patients with clinical deterioration. As there is a broad variety of disease processes that may lead to left (and/or right) ventricular dysfunction, there is no specific format for image or data acquisition during serial TTE studies applicable to all indications. Rather than a "one size fits all" approach, it is recommended that in addition to the basic TTE examination, additional echocardiographic parameters should be acquired according to the indication for serial imaging. It must be emphasised that every effort should be made to report quantitative measurements (i.e., numerical LV ejection fraction [LVEF]) in addition to qualitative statements during the assessment of LV systolic function.

1.1. Recommendations for Performance of TTE for Assessment of the LV

In most instances, the indication for the initial TTE will be guided by symptoms and clinical examination.

While clinical assessment will remain important, TTE can improve diagnostic accuracy. The main symptoms which warrant investigation of the LV are shortness of breath, chest pain, palpitations, pre-syncope, syncope, and peripheral oedema. These symptoms, whether acute or chronic, may be the first manifestation of cardiovascular and/or respiratory diseases, anaemia, or a host of other conditions. Clinical signs warranting a baseline TTE include examination findings of heart failure, tachy/brady-arrhythmias, hypertension/hypotension or other features of haemodynamic instability, as well as electrocardiographic (ECG) findings of LV hypertrophy, left bundle branch block, arrhythmias (e.g., atrial fibrillation (AF) or frequent ventricular ectopy/tachycardia) or signs of acute or chronic ischaemia [3]. The need for, and frequency of, serial examinations will in turn depend on the findings of the initial TTE.

In asymptomatic patients without concerning clinical signs, TTE would be recommended for the screening of first-degree relatives of those with an inherited cardiomyopathy (e.g., hypertrophic cardiomyopathy), initial evaluation of those with a known systemic or acquired disease that could be associated with structural cardiac abnormalities (e.g., haemochromatosis), or prior to exposure to medications (including chemotherapeutic agents) or radiation therapy known to cause LV dysfunction and heart failure. TTE would also be useful to guide further investigation and management in patients at risk of heart failure (e.g., those with hypertension, diabetes, and/or vascular disease) with elevated serum natriuretic peptides in the absence of symptoms or previously established cardiac disease [4]. Finally, an initial TTE is necessary for the comprehensive evaluation of cardiac structure and function in a potential cardiac transplant donor.

1.2. Recommendations for Serial TTE Assessment of the LV

1.2.1. Heart failure

Heart failure is defined as a complex clinical syndrome consisting of both typical symptoms and signs that are generally exertional (but may also occur at rest), secondary to a structural abnormality of the heart leading to reduced cardiac function [5]. The updated contemporary universal definition of heart failure now extends this to include additional corroboration by increased serum natriuretic peptide levels and/or objective evidence of cardiogenic pulmonary or systemic congestion by means of imaging or invasive haemodynamic assessments [6]. TTE is essential for the initial patient assessment to diagnose heart failure, identification of an aetiology especially for conditions where specific disease-modifying therapies exist, as well as the ongoing monitoring of disease activity. Using an LVEF threshold of 50%, patients are primarily classified into the categories of heart failure with reduced ejection fraction (HFrEF) or with preserved ejection fraction (HFpEF). Further subclassification based on aetiology (e.g., ischaemic heart disease) may be performed but an exhaustive description is beyond the scope of this document.

Heart Failure With Reduced Ejection Fraction (HFrEF)

Subsequent to the initial diagnosis of HFrEF serial imaging is an important component of ongoing management. While the Australian heart failure guidelines do not mandate intervals for echocardiographic examinations, both the American Heart Association (AHA) and the European Society of Cardiology (ESC) suggest that following the initial diagnosis a repeat TTE should be performed within 3–6 months of optimisation of heart failure medications to assess response to therapy [4,5,7]. The accurate assessment of LV systolic function is particularly important in those patients where cardiac resynchronisation therapy (CRT) or an implantable cardioverter–defibrillator (ICD) is clinically applicable. Current Australian and international guidelines recommend an LVEF threshold of $\leq$35% for consideration of these devices [5].

A repeat TTE is also indicated when there has been a deterioration in the patient's clinical condition, such as a significant reduction in exercise capacity or an admission to hospital with decompensated heart failure. In instances where there have been substantial changes to the patient's medical therapy (including the cessation or initiation of new drugs or other therapies), a repeat TTE may be performed to assess treatment response.

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While the 2022 AHA/American College of Cardiology (ACC)/Heart Failure Society of America (HFSA) guideline states that "in the absence of clinical status change, treatment interventions that might have had a significant effect on cardiac function, or candidacy for invasive procedures or device therapy, routine repeat assessment of LV function is not indicated" (Class 3: No benefit; Level of Evidence: C [Expert Opinion]), many clinicians would advocate routine re-evaluation of LV function in stable heart failure patients for detection of subclinical changes to pre-emptively intervene prior to overt clinical deterioration [4]. As there is no clinical data supporting this approach, the writing committee would recommend that any such TTE surveillance in this cohort should occur no more frequently than every 2 years, in the absence of a significant clinical change.

Imaging requirements. As some therapeutic interventions depend on specific thresholds (e.g., LVEF ≤35% for consideration of CRT or a primary prevention ICD) it is paramount that any assessment of LV systolic function be provided in a numerical value as well as in a descriptive format. LVEF should be measured objectively, either by the two-dimensional (2D) biplane method of disks summation (modified Simpson's rule) as a minimum, or preferably with three-dimensional (3D) echocardiographic quantification. When LV endocardial definition is suboptimal, intravenous ultrasound enhancing agents (UEA) should be used [8]. In instances where a visual estimation of LVEF is performed, this should also be expressed numerically rather than with the descriptive terms of normal function or of mild, moderate, or severe dysfunction. LV size should be quantitatively assessed using the LV end diastolic volume (LVEDV) or dimension, indexed to body surface area (BSA). The use of the Teichholz or Quinones methods for calculating LVEF from LV linear dimensions is no longer recommended [9]. Measures of deformation (i.e. 2D speckle-tracking echocardiography [STE] global longitudinal strain [GLS]) and the aforementioned 3D imaging are strongly encouraged, where possible, and have been recommended in international expert consensus statements since 2017 [10].

Objective evaluation of diastolic function and assessment of cardiac filling pressures should also be provided to the treating clinician. As a minimum, pulsed-wave Doppler (PWD) of the mitral inflow (E and A velocities), mitral annular tissue Doppler e' velocities, LV E/e' ratio, tricuspid regurgitation (TR) peak velocity (TR V_{max}), and left atrial volume index (LAVI) must be acquired to diagnose and grade the degree of LV diastolic dysfunction [11]. In the absence of significant valvular disease or chronic arrhythmias, an increased LAVI may be an indicator of chronically elevated left atrial (LA) pressure. In addition, the size and degree of respiratory variation of the inferior vena cava (IVC) and LV E/e', as estimates of right atrial (RA) and LA pressures respectively, should be routinely assessed and reported [12]. Finally, an evaluation of the pulmonary artery systolic pressure (PASP) and any significant deterioration in valvular disease (e.g., functional mitral regurgitation [MR]) should be quantified. PASP is directly estimated using the right ventricular systolic pressure (RVSP) calculated by the modified Bernoulli equation (RVSP = (4 x TR V_{max}^2) + RA pressure), in the absence of pulmonic stenosis or right ventricular outflow tract (RVOT) obstruction.

Heart Failure With Preserved Ejection Fraction (HFpEF)

As therapeutic options for HFpEF are much more limited in comparison to HFrEF, the role of serial TTEs in patients with HFpEF is less well-established. As by definition the LVEF is "normal" (or low-normal) in HFpEF, repeat imaging in this condition is more focussed on the assessment of functional parameters of diastolic function, pulmonary hypertension, and secondary right ventricular (RV) failure, instead of the LVEF. Furthermore, in contrast to patients with HFrEF, there is no randomised data supporting implantation of CRT or ICD devices. Whilst there is no specific guideline indication for repeating a TTE 3-6 months following optimisation of medical therapy in patients with HFpEF, it would be reasonable to adopt a similar longitudinal surveillance approach to that of HFrEF.

Additional imaging requirements. The recommendations with respect to the use of quantitative or semiquantitative measures for ventricular size and function as described above for HFrEF also apply to serial TTE examinations in patients with HFpEF. Assessment of RA and LA filling pressure remains essential. Additionally, as LV hypertrophy (LVH) is extremely common in patients with HFpEF, the assessment of wall thickness is recommended [13]. LV mass (LVM) with indexation for BSA (LVMI) may then be calculated using the linear measurement technique, with gender-specific cut-offs available for reference [9]. However, assessment of LVM by this method is heavily dependent on geometrical assumptions and is less reliable in the presence of asymmetrical hypertrophy or LV dilatation. LVM can be more accurately measured by 3D echocardiography or cardiac magnetic resonance imaging (CMRI) [14]. The use of LV GLS is also strongly encouraged as this is often found to be abnormal in HFpEF patients [11]. Given the prognostic value of LA dilation in HFpEF the LAVI should also be routinely measured.

1.2.2. Infiltrative and inflammatory cardiomyopathies

Numerous systemic conditions may involve the heart leading to systolic and/or diastolic dysfunction. In addition to assisting with the initial diagnosis of cardiac involvement, TTE monitoring is an important component of patient follow-up. In these instances, the focus of the TTE examination will relate primarily to the manner and extent to which the disease process expresses itself in the heart. For example, advanced cardiac sarcoidosis typically results in a

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HFrEF phenotype and, therefore, interval TTE assessment should proceed along the same lines as recommended for patients with reduced LVEF. Conversely, amyloid cardiomyopathy most commonly results in HFpEF and, as such, serial imaging should be considered on that basis. However, this distinction between phenotypes is not absolute as some sarcoid patients may present with diastolic rather than systolic heart failure, and amyloid cardiomyopathy may progress to HFrEF in advanced stages. The focus and frequency of echocardiographic examinations in these patients may therefore change over time depending on disease activity and degree of LV dysfunction. While not available in all practices, the use of more advanced measures of LV function, such as GLS, is applicable in these settings and recommended for clinical use where possible [15,16].

Acute myocarditis has a broad spectrum of clinical phenotypes, ranging from severe LV systolic dysfunction with cardiogenic shock, to an acute coronary syndrome (ACS) mimic with normal echocardiographic findings. TTE is an essential investigation in the diagnosis of myocarditis. However, the role of subsequent imaging is not well established as many patients recover completely from their acute illness. It would be reasonable to repeat a TTE in 3–6 months following an episode of acute myocarditis to ensure no relapse has occurred but the need for ongoing serial echocardiography beyond this timepoint in patients who have fully recovered cardiac function and remain clinically well is probably not justified. One exception would be biopsy-proven giant cell myocarditis, where relapses are frequent even in the rare instances following recovery of LV function, and ongoing monitoring with TTE is critical. Although there are no guidelines for the frequency of echocardiographic follow-up of this rare condition, an interval of 6–12 months would seem justifiable given the natural history of this disease.

1.2.3. Hypertrophic cardiomyopathy (HCM)

HCM is a progressive lifelong disease with changes to both systolic and diastolic function as well as LVM. Given the variability in clinical phenotype, serial TTE monitoring in asymptomatic patients with HCM is warranted every 1–2 years, when there are new events or changes in clinical status, and for ongoing risk stratification [17,18]. Periodic interval screening of first-degree relatives of affected individuals is recommended and the frequency of TTE reassessment is age-dependent (i.e., age of diagnosis in the proband and age of the first degree relative) [18,19]. Repeat TTE also has a role in the assessment of response to medical, percutaneous, and/or surgical intervention of left ventricular outflow tract (LVOT) obstruction in hypertrophic obstructive cardiomyopathy (HOCM) [20].

Additional imaging requirements. In addition to routine evaluation of LV/RV size and systolic function, wall thickness, co-existent valvular pathology, and atrial sizes, comprehensive assessment of diastolic filling pressures and estimation of the PASP should be performed. The LVOT gradient should also be measured at rest and with the Valsalva manoeuvre when indicated. LA diameter measured by 2D (or M-mode [motion-based] if adequate alignment) in the parasternal long axis view and maximal LV wall thickness must be reported, as these parameters are components of the ESC HCM Risk Calculator for Sudden Cardiac Death which is often used to guide ICD implantation [21].

1.2.4. Hypertensive heart disease

The LV is one of the main targets of end-organ damage in hypertension. TTE assessment, including the evaluation of LVM/LVMI, can provide important prognostic information for hypertensive patients. There is, however, no evidence for serial imaging in the absence of clinical suspicion for hypertensive heart disease, nor is there justification for repeat TTE examinations to monitor response to antihypertensive therapy [1,14]. However, repeat TTE should be performed if there is a change in the patient's clinical status, or may be considered with poor control of blood pressure [22].

Additional imaging requirements. In addition to routine evaluation of LV/RV size and systolic function, LVM, co-existent valvular pathology, and atrial sizes, comprehensive assessment of diastolic filling pressures and estimation of the PASP should be performed. Given its role in risk stratification the maximal LV wall thickness should be clearly documented.

1.2.5. Cardiac transplantation

While outcomes after heart transplantation continue to improve, survival remains limited by acute and chronic allograft rejection as well as the development of coronary vasculopathy, both of which may be detected by TTE [23]. Allograft rejection is usually asymptomatic, rapid in onset, and bears a poor prognosis. Regular surveillance of heart transplant recipients is mandatory, particularly in the first-year post-transplantation [24]. TTE should be performed immediately post-operatively to identify surgical complications or signs of early allograft rejection, and to document baseline data of the transplanted heart. A pre-discharge TTE is also recommended for the same indications. In the first 12 months post-transplantation, at least one TTE every 3 months should be performed to complement endomyocardial biopsies [24]. In the second year, a TTE every 6 months followed by annual studies beyond 2 years in asymptomatic patients is recommended, while maintaining a low threshold for more frequent assessments, as clinically indicated. However, as protocols at heart transplant centres vary, the appropriate timing of TTE following cardiac transplantation will depend on local institutional guidelines.

Additional imaging requirements. The use of deformation imaging (e.g., GLS and RV free wall longitudinal strain) is recommended given their superior ability to detect early myocardial dysfunction in comparison to standard measures such as LVEF [24]. A baseline TTE assessment including these advanced parameters will allow detection of interval change on follow-up studies which may signal rejection.

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1.2.6. Coronary artery disease

After an ACS, imaging with TTE should be performed routinely to evaluate infarct size and ventricular function. An urgent TTE is indicated if there is a suspected complication of an acute myocardial infarction (e.g., ventricular septal defect [VSD], ischaemic MR, free wall rupture, or LV thrombus), post-percutaneous coronary intervention (e.g., coronary artery or LV perforation), or following surgical revascularisation (e.g., acute graft dysfunction) [25,26]. After percutaneous coronary intervention or surgical revascularisation, a repeat study should be considered to document baseline LV function prior to discharge, and to guide implementation of appropriate medical therapy.

There is no indication for routine re-assessment of LV function in stable post-ACS patients without resultant/residual LV dysfunction or in patients with stable coronary artery disease. However, any clinical deterioration should trigger re-evaluation, specifically to identify progressive LV dysfunction or late mechanical complications [22].

Additional imaging requirements. In addition to LV size and function, the extent of myocardial damage and/or severity of valvular dysfunction should be assessed. The use of LV regional wall motion plots is encouraged for visual clarity [27]. In ST-elevation myocardial infarctions (STEMI), mechanical complications such as a VSD or acute ischaemic MR should also be excluded. Contrast TTE using UEAs may be helpful in the assessment of a suspected contained free wall rupture or to exclude an apical thrombus.

1.3. Requirements for Comprehensive TTE Assessment of the LV

Key TTE parameters for the assessment of LV size and function are summarised in Table 1.

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Table 1 Key echocardiographic parameters for assessment of the LV.

Condition	Recommendations	Parameters
Heart failure with reduced ejection fraction (HFrEF)	Initial diagnosis, clinical deterioration, or significant change in therapy, and/or post-intervention 3–6 months following optimisation of medical therapy Device therapy • Evaluation after appropriate time interval following revascularisation and/or optimal medical therapy to determine candidacy for ICD/CRT and/or to decide optimal device selection. • To determine candidacy for/optimisation of ventricular assist device settings. Routine monitoring in stable patients (no more frequently than every 2 years without significant clinical change)	Quantitative measurement of LV size and function • Minimum dataset should include LV volumes and LVEF using 2D biplane Simpson's method. • Where possible, 3D LV volumes and LVEF and GLS should be performed. • Use of UEAs where LV endocardial definition is suboptimal. Measures of diastolic function • Minimum dataset should include mitral valve inflow Dopplers, mitral annular e' velocities, E/e' ratio, TR V_{max}, and LAVI. PASP (RVSP) Assessment of functional mitral regurgitation if present
Heart Failure with preserved Ejection Fraction (HFpEF)	Initial diagnosis, clinical deterioration, or significant change in therapy, and/or post-intervention Routine monitoring in stable patients (no more frequently than every 2 years without significant clinical change)	As for HFrEF LV wall thickness LAVI LV GLS is recommended
Infiltrative and inflammatory cardiomyopathy	Initial diagnosis, clinical deterioration, or significant change in therapy, and/or post-intervention 3-6 months following recovery from episode of acute myocarditis Routine monitoring in stable patients (no more frequently than every 2 years without significant clinical change)	As for HFrEF or HFpEF, according to phenotype LV GLS is recommended
Hypertrophic cardiomyopathy (HCM)	Initial diagnosis, clinical deterioration, or significant change in therapy, and/or post-intervention Routine monitoring in stable patients every 1–2 years Periodic interval screening of first-degree relatives of affected individuals, as recommended	As for HFpEF Maximal LV wall thickness LVOT gradient at rest and with Valsalva LA diameter (2D or M-mode)
Hypertensive heart disease	Initial diagnosis No routine indication for repeat follow-up studies after initial diagnosis in the absence of significant change in clinical status but may be considered in patients with poorly controlled blood pressure	As for HFpEF Maximal LV wall thickness LVM/LVMI
Cardiac transplantation	Assessment of potential donor heart pre-transplant Assessment of allograft in early post-operative phase Assessment of allograft pre-discharge to establish baseline Serial monitoring according to institutional protocol	As for HFrEF
Coronary artery disease	Initial evaluation of suspected ACS in the emergency setting when the ECG is inconclusive Baseline assessment post-ACS intervention Suspected complication of acute myocardial ischaemia/infarction Suspected complication of percutaneous or surgical revascularisation No indication for serial monitoring in stable post-ACS patients in absence of residual LV dysfunction unless clinical deterioration	As for HFrEF Extent of myocardial damage (use of LV regional wall motion plots should be considered) Valvular function Exclusion of mechanical complications and LV thrombus (including use of UEAs as required)

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Table 1. (continued).

Condition	Recommendations	Parameters
Screening of asymptomatic first-degree relatives for heritable conditions	Frequency of clinical screening depends on the primary cardiac condition and age of the affected/unaffected family member(s)	As for HFrEF, HFpEF, or HCM according to phenotype

Abbreviations: 2D, two-dimensional; 3D, three-dimensional; LV, left ventricular; LVEF, left ventricular ejection fraction; LVOT, left ventricular outflow tract; LA, left atrial; ACS, acute coronary syndrome; ECG, electrocardiography; UEA, ultrasound enhancing agents; M-mode, motion mode; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure; LVM, left ventricular mass; LVMI, left ventricular mass with indexation for body surface area; LAVI, left atrial volume index; GLS, global longitudinal strain.

2. The Right Ventricle

Abnormalities of right ventricular (RV) structure and function may occur in isolation but are most commonly seen in combination with other cardiopulmonary abnormalities such as left heart disease, valvular dysfunction, and pulmonary hypertension. In all these settings, abnormalities of RV function are of additional prognostic significance. It is therefore important to comprehensively assess the RV in every TTE to diagnose and quantify all abnormalities. As for the LV, there are certain conditions in which echocardiographic evaluation of the RV is the principal indication for the imaging request.

2.1. Recommendations for Performance of TTE for Assessment of the RV

2.1.1. Right heart failure (RHF)

Heart failure can occur due to dysfunction of either or both ventricles, resulting from an inability to match the contractile work requirements against the metabolic demand and load. Cardiac work requirements increase with exercise and thus symptoms of heart failure often manifest during exertion. A simple construct of heart failure may only consider the LV and its failure to meet output demands resulting in pulmonary congestion. However, it is important to recognise that the 'upstream' effect results in increasing pulmonary artery pressures and demand on the right side of the heart. Cardiac output is only as good as the worst ventricle. Consequently, if RV function is diminished such that it is unable to meet the rising output demand and/or compensate for the increased afterload due to left heart failure, the RV then becomes the limiting constraint of overall cardiac function. Ongoing RV volume/pressure loading will further result in RA dilatation and dysfunction. TR may also be exacerbated by ventriculo–atrial and/or annular dilation resulting in worsening RHF. Unsurprisingly, RV function has now become a dominant prognostic index in many forms of heart failure [28,29].

The clinical syndrome most frequently associated with RHF refers to the peripheral venous congestion and oedema resulting from inadequate right heart pump function. Raised jugular venous pressure, hepatic congestion, and peripheral oedema are important clinical indications warranting baseline TTE assessment of the RV. It is important to note, however, there is substantial individual variability in the degree to which elevated venous pressures are tolerated. The need for, and frequency of, serial examinations will in turn depend on the findings of the initial TTE.

2.1.2. Suspected pulmonary hypertension (PHTN)

TTE is recommended as a first-line screening tool for all patients in whom PHTN is suspected [1,3,30]. Post-capillary PHTN is a common complication of left heart disease. The rarer pre-capillary PHTN should be suspected in patients with systemic sclerosis, previous pulmonary thromboembolism, or uncommonly in familial diseases. Recognition and diagnosis of pre-capillary PHTN is important due to the availability of specific pulmonary vasodilator therapies. PHTN may also be suspected based upon abnormalities in respiratory function testing (reduced diffusion capacity), cardiopulmonary exercise testing (reduced ventilatory efficiency), or incidental imaging findings of pulmonary artery dilatation or RV enlargement. It also needs to be considered as an important differential diagnosis in the breathless patient following exclusion of other common causes. The TTE study should carefully assess the RV, RA, tricuspid valve, and provide an estimation of right-sided filling pressures and the PASP (RVSP).

2.1.3. Pulmonary embolism (PE)

RV dysfunction is a frequent and prognostically significant complication of PE. In the acute setting, haemodynamic instability is caused by the sudden increase in afterload as a result of pulmonary vascular obstruction, in combination with the secondary hypoxia. Severe RV dysfunction may ensue and is a critical factor in the decision-making process for use of advanced therapies such as thrombolysis, clot retrieval, or surgery. This scenario also includes the critically ill patient with unexplained hypotension where an acute PE with RV failure may be the underlying aetiology. Urgent TTE is therefore recommended as part of the assessment of acute

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PE with associated haemodynamic instability, for immediate risk stratification, and to guide therapeutic decisions [31]. Assessment of RV function is also important in the subacute phase as up to 10% of patients may develop long-term pulmonary vascular disease resulting in chronic thromboembolic PHTN (CTEPH) for which effective therapies are available.

2.1.4. Suspected arrhythmogenic cardiomyopathy (AC)

Previously termed arrhythmogenic right ventricular cardiomyopathy, AC may be suspected in individuals with a suggestive family history, symptoms of palpitations or syncope, when accompanied by ECG or other electrophysiological abnormalities. The diagnosis is reached by combining structural, histological, electrical, and genetic features, with TTE potentially providing major or minor diagnostic imaging task force criteria [32,33]. Echocardiographic abnormalities range from relatively subtle focal structural alterations through to marked RV dilation with global or regional dysfunction. Abnormalities of the LV may also be present. Advanced echocardiographic techniques, such as evaluation of regional strain patterns, may assist with early diagnosis. TTE is often used in combination with CMRI which can provide incremental assessment of myocardial inflammation and scar. Stress echocardiography may also be a useful tool to identify subclinical involvement of the right heart in an arrhythmogenic process. Failure to augment RV contractility can be considered abnormal and may be associated with ventricular arrhythmias [34].

2.2. Recommendations for Serial TTE Assessment of the RV

2.2.1. Right heart failure

TTE is indicated in the assessment of a patient with known primary or secondary RV dysfunction in whom there is a decline in clinical state or new/worsening symptoms (progressive exertional or resting dyspnoea, peripheral venous congestion, or oedema). An accurate baseline TTE assessment of the RV facilitates serial examinations by allowing identification of significant interval change. The frequency of repeat TTE examinations will be defined by the time course and severity of the change in the patient's status or imaging findings, at the treating clinician's discretion, although management decisions will be primarily driven by clinical assessment rather than the echocardiographic appearance.

2.2.2. Pulmonary hypertension

In cases of pulmonary arterial hypertension, particularly during treatment with pulmonary arterial vasodilators, repeat TTE for ongoing risk assessment is recommended routinely every 6–12 months, 3–6 months after a change in therapy, or if there is clinical deterioration [30].

2.2.3. Post-pulmonary embolism

In the setting of significant RV dysfunction or PHTN following an acute PE, repeat TTE evaluation to document normalisation of RV function or pulmonary pressures is recommended after 3 months [31]. Further serial examinations may be performed if these remain abnormal. In cases of ongoing exercise intolerance post-PE, repeat assessment of the right heart and pulmonary pressures is recommended to exclude CTEPH [31].

2.2.4. Arrhythmogenic cardiomyopathy

Screening for AC is indicated in patients with an established genetic predisposition or in first-degree family members of affected individuals. Screening for phenotypic expression may be repeated at a 12–24 month interval, depending on the patient's age and presence of symptoms. First-degree relatives require serial TTE assessments every 1–3 years from 10–12 years of age as part of their cardiac evaluation [33].

2.3. Requirements for Comprehensive TTE Assessment of the RV

The 2015 American Society of Echocardiography (ASE) and European Association of Cardiovascular Imaging (EACVI) Recommendations for Cardiac Chamber Quantification and a recent state-of-the-art review by Jones et al. provide detailed technical discussions describing the morphological and functional examination of the right heart [9,35]. Key TTE parameters for the assessment of RV size and function are summarised in Table 2.

RV assessment should include a qualitative assessment of chamber size and wall motion in the parasternal long/short-axis, apical, and subcostal views. As for the LV, quantification of RV size may be performed using linear dimensions, 2D areas, or 3D volumes. Linear dimensions can be useful to highlight asymmetry or focal enlargement (such as the RVOT or even pulmonary artery dilation). RV areas can provide an excellent and simple metric of chamber size. They should be carefully measured with rotation of the probe to maximise RV area in an apical 4-chamber view (i.e., RV-focussed view). Given the complex geometry of the RV and limitations of standard 2D imaging, 3D echocardiography, where available, has the potential to provide a more accurate assessment of RV volumes (although remains underestimated compared with CMRI) [36]. This can provide a useful reference, in comparison to LV volumes, where various cardiac imaging modalities have been used to determine that RV:LV volume ratios of <1.27 can be considered normal [37].

RV systolic function may be objectively assessed by the tricuspid annular plane systolic excursion (TAPSE), tissue Doppler-derived tricuspid lateral annular systolic velocity (RV S′), fractional area change (FAC), and strain. Although TAPSE and RV S′ are easily obtainable and reproducible measures of RV longitudinal function, there may be technical limitations affecting their accuracy. The curvature of the RV free wall can present difficulties with alignment of the ultrasound beam. Pivoting of the dilated RV around the LV may lead to a false assumption of normal longitudinal function. Thus, attention to the technical aspects of these measurements is of great importance. 2D STE addresses some of these challenges and likely represents a better

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Table 2 Key echocardiographic parameters for assessment of the RV.

Condition	Recommendation	Parameters
Right heart failure	Initial diagnosis, clinical deterioration, or significant change in therapy Serial assessment should be no more frequent than every 2 years without significant clinical change	Quantitative measurement of RV size and systolic function • Minimum dataset should include RV basal dimension, RVOT proximal dimension, TAPSE, RV S ', and FAC • Where possible 3D RV volumes and RVEF and free wall strain should be performed PASP (RVSP) Assessment of functional tricuspid regurgitation if present
Pulmonary hypertension	Initial diagnosis, clinical deterioration, or 3-6 months after significant change in therapy Serial evaluation may be required for ongoing disease-specific treatments (as per PHTN section)	As for right heart failure
Post-pulmonary embolism	Initial diagnosis 3 months following diagnosis if there is RV dysfunction at baseline Serial evaluation after 6 months if there is persistent RV dysfunction	As for right heart failure
Suspected arrhythmogenic cardiomyopathy	Initial diagnosis, clinical deterioration, or significant change in therapy and/or post-intervention Routine monitoring in stable patients every 1-2 years Periodic interval screening of first-degree relatives of affected individuals as recommended	As for right heart failure • Specific attention to be given to assessment for presence of regional RV akinesia/dyskinesia/aneurysm and measurements of the RVOT dimensions and RV FAC as part of the Task Force diagnostic criteria [32]

Abbreviations: RV, right ventricular; RVOT, right ventricular outflow tract; RV S ', tissue Doppler tricuspid lateral annular systolic excursion velocity; FAC, fractional area change; TAPSE, tricuspid annular plane systolic excursion; RVEF, right ventricular ejection fraction; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure; 3D, three-dimensional; PTHN, pulmonary hypertension.

measure of longitudinal deformation (strain and strain rate) and function. The measurement of RV end-diastolic (RVAd) and RV end-systolic (RVAs) areas allow the calculation of the FAC (RVFAC (%) = [RVAd-RVAs]/RVAd x 100) as an estimate of global RV systolic function. RV ejection fraction (RVEF) by 3D echocardiography is also a robust measure of systolic function [36].

The RV is highly sensitive to afterload, and haemodynamic evaluation of the loading conditions is critical. RVSP is used as a surrogate for the PASP in the absence of pulmonic stenosis or RVOT obstruction and should be assessed where technically possible in all patients, especially when the primary issue is right heart function. Estimation of the RA pressure based on the size and collapsibility of the IVC should also be performed, as required, for the calculation of the RVSP [12]. When estimation of the PASP using the TR V_{max} method is suboptimal (e.g., insufficient TR, poorly aligned or incomplete continuous-wave Doppler (CWD) TR signal, severe TR, or severe RV systolic dysfunction), a short pulmonary artery acceleration time (<90 ms) or presence of notching in the RVOT PWD signal profile is suggestive of the presence of PHTN [38]. A number of clinical and echocardiographic parameters have been used as surrogates for pulmonary vascular resistance (PVR), and to differentiate the presence of pre-capillary from post-capillary PHTN. Although parameters such as the echocardiographic pulmonary to left atrial ratio (ePLAR) have been examined in a variety of clinical scenarios and do have some clinical utility, most measures appear to have only moderate diagnostic accuracy [39,40]. In patients with a suboptimal TR CWD envelope, the use of agitated saline contrast or UEAs may be considered for enhancement of the spectral signal [38,41,42].

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3. Aortic Valve Diseases

All patients with suspected or known valvular heart disease should undergo a comprehensive TTE assessment to confirm the clinical findings, establish the diagnosis, and determine the lesion mechanism and severity [43–47]. Defining the aetiology, quantifying the degree of valvular stenosis or regurgitation, and assessing ventricular function and presence of cardiac chamber remodelling allows clarification of the "stage" of disease for risk stratification, and ultimately guiding decision-making for timing of intervention (AHA/ACC stage A—at risk; stage B—asymptomatic mild–moderate or progressive disease; stage C—asymptomatic severe disease; stage D—symptomatic severe disease). TTE is well-established as the primary imaging modality for the diagnosis and longitudinal surveillance of valvular heart diseases.

3.1. Aortic Stenosis (AS)

Degenerative calcification, as a result of atherosclerotic-like leaflet thickening, stiffening, and calcification, is the most common aetiology of valvular aortic stenosis (AS) [48–50]. Congenitally bicuspid aortic valves (BAVs; 0.5%–2.0% of the general population) may also undergo similar remodelling resulting in the development of AS in younger patients 5–10 years earlier [51–53]. BAVs are also at increased risk of other complications including concomitant aortic regurgitation, endocarditis, and aortopathies. Rheumatic aortic valve disease, usually with accompanying mitral valve involvement, remains relatively rare in developed countries.

3.1.1. Recommendations for performance of TTE for assessment of AS [43,45,46,54]

1. To establish the diagnosis in patients with symptoms or signs of AS or a BAV (including determination of the aetiology, severity, and haemodynamic consequences).
2. Re-evaluation of known AS in patients with a significant change in clinical status.
3. Before or during pregnancy in patients with known or suspected AS or congenital aortic valve abnormalities (to determine the severity and haemodynamic consequences of the AS).

Of note the incidental finding of aortic valve (AV) calcification on computerised tomography (CT) of the chest has also been regarded as an indication for performing a TTE to exclude or define severity of AS [45].

3.1.2. Recommendations for serial TTE assessment of AS [1,22,45–47,54]

1. In patients with known AS, the frequency of a repeat TTE examination is based on symptoms, severity of AS, stability of findings on serial assessments, and the haemodynamic consequences. Repeat TTE should be performed when there are new or changed symptoms or physical examination findings.
2. In asymptomatic patients with severe AS, TTE assessment every 6–12 months is recommended (increased frequency may be considered with progressive decline in LV systolic function on serial imaging despite LVEF remaining in the "normal" range).
3. In moderate AS, TTE assessment every 1–2 years is recommended (increased frequency when significant leaflet calcification is present).
4. In mild AS, TTE assessment every 3–5 years is recommended (≥ 3 years with no change in symptoms or signs).
5. Re-assessment during pregnancy—once or twice during pregnancy for mild AS, every trimester for moderate AS, and every 1–2 months for severe AS.
6. In patients with a BAV—every 3–5 years with no valvular dysfunction and every 2 years with valve thickening and mild AS.

3.1.3. Requirements for comprehensive TTE assessment of AS

The primary role of echocardiography in the assessment of AS is to determine the aetiology and haemodynamic severity of the stenosis. TTE is the preferred diagnostic investigation for AS and invasive haemodynamic assessment is only recommended when echocardiographic findings are inconclusive or discordant with clinical findings. Key TTE parameters for assessment of valvular AS are summarised in Table 3.

Degenerative calcification and rheumatic injury are the two most common aetiologies of AS, with the former mostly prevalent in industrialised countries and the latter in the developing world [50]. 2D TTE imaging is required to define valvular morphology and pathognomonic characteristics of the underlying aetiology and identify other associated valvular (e.g., rheumatic mitral valve disease) or aortic (e.g. aortopathy with BAVs) pathologies. Qualitative or semi-quantitative 2D TTE is able to assess the degree of restriction of cusp mobility/excursion and leaflet calcification. Direct measurement of the geometric orifice area by 2D (or 3D) planimetry may be useful as a surrogate measure of stenosis severity.

Haemodynamic severity of AS is based upon calculation of the AV area (AVA) by the Doppler-derived continuity equation and assessment of the AV peak velocity (AV V_{max}) and mean pressure gradient (mPG) by CWD interrogation (Figure A) [46,47,50]. AVA is the primary parameter for the diagnosis of AS severity. An AVA of $<1.0cm^2$ (and/or AVA indexed for BSA $<0.6cm^2/m^2$) is an independent predictor of mortality and is the universally accepted threshold for AV intervention in the symptomatic patient. A major weakness in the evaluation of the AVA is the LVOT diameter (LVOTd) measurement by 2D TTE, which is often underestimated, resulting in a mathematically squared error in the continuity equation. In addition to an AVA of $<1.0cm^2$, the findings of an AV V_{max} ≥4.0m/s and mPG ≥40mmHg are also consistent with severe AS. In order to prevent underestimation of the AV V_{max} and mPG, it is critical to ensure that the CWD insonation angle is optimally aligned with the stenotic jet. Interrogation from multiple imaging windows with the

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Table 3 Key echocardiographic parameters for assessment of AS.

Clinical	Height, weight, body surface area (BSA)
	Heart rate and rhythm
	Blood pressure
2D/M-mode	Aortic valve (AV) morphology (number of cusps, mobility, thickness, calcification)
	Left ventricular outflow tract (LVOT) diameter
	LV/RV dimensions (including LVH)
	LV/RV systolic function parameters (LVEF, RV TAPSE, RV FAC)
	LAVI
	Other valvular involvement (rheumatic valve disease)
	Aorta (as per aortic diseases section)
Spectral Doppler	AV peak velocity (AV V_{max}; mandatory interrogation with the non-imaging pulsed echo Doppler flow velocity meter [PEDOF] probe)
	AV mean gradient
	AV velocity time integral (VTI)
	LVOT peak velocity and VTI
	Dimensionless severity index (DSI; VTI ratio)
	AV area (AVA) (continuity)
	AVA indexed for BSA
	Stroke volume indexed for BSA (SVI)
	LV diastolic function parameters (mitral valve inflow Dopplers, mitral annular e' velocities, E/e' ratio, and TR V_{max})
	PASP (RVSP)
	RV systolic function parameters (RV S')
Colour Doppler	Aortic regurgitation severity
	Mitral regurgitation severity
	Tricuspid regurgitation severity
Advanced (if available and technically feasible)	Pressure recovery
	LV GLS

Abbreviations: 2D, two-dimensional; M-mode, motion mode; TAPSE, tricuspid annular plane systolic excursion; LV, left ventricular; LVEF left ventricular ejection fraction; LVH, left ventricular hypertrophy; RV, right ventricular; RV S ', tissue Doppler tricuspid lateral annular systolic excursion velocity; FAC, fractional area change; LAVI, left atrial volume index; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure; GLS, global longitudinal strain.

pulsed echo Doppler flow velocity meter (PEDOF) probe (non-imaging CWD transducer with a high signal-noise ratio and smaller "footprint") is mandatory. In clinical practice, discordance between TTE parameters is commonly encountered. The dimensionless severity index (DSI; ratio of the LVOT and AV peak velocities or velocity time integrals [VTI]) can be used as a safeguard to highlight potential technical mistakes. A DSI of <0.25 is in keeping with severe AS, in parallel with a reduction of the AVA to <25% of normal, and 4-fold increase in the transvalvular velocity [55]. It must be emphasised that the meticulous performance of these measurements and early recognition of errors are necessary to avoid misclassification of AS severity.

As AVA, Doppler velocities, and pressure gradients are all flow-dependent variables, concurrent evaluation of the patient's flow-state (by the calculated stroke volume [SV] indexed for BSA [SVI] or flow rate) must be performed and reported. Furthermore, co-existent systemic hypertension may also result in underestimation of the AV V_{max} and mPG, and re-assessment must be undertaken when the patient is normotensive [46,47,50]. Additional considerations, such as pressure recovery corrections and assessment of systolic timing intervals, may also be required to rationalise the primary data and avoid misdiagnosis [56]. Accurate grading of AS severity is crucial to guide therapeutic decisions and should be performed using an integrated multiparametric approach as recommended in the societal guidelines [47,50]. When the subgroups of classical or paradoxical low-flow low-gradient AS are encountered, following exclusion of measurement errors or other confounders, further investigation with low-dose dobutamine stress echocardiography or assessment of AV calcification by cardiac computed tomography (CCT) may be required to corroborate AS severity.

The LV is subject to increased afterload in the setting of AS, resulting in elevated wall stress, myocardial remodelling, myocyte apoptosis, and ultimately replacement fibrosis. Therefore, in addition to quantification of LV size, wall thickness (and mass), the routine echocardiographic assessment of AS must include a comprehensive evaluation of LV systolic function. While an LVEF of <50% is the recommended threshold for intervention in asymptomatic patients, a relative LVEF deterioration in the absence of an alternate cause may also be a reasonable indication to consider aortic valve replacement (AVR), due to the observed increased mortality even above that cut-off [46,47,57–59]. Subclinical LV systolic dysfunction (i.e., preserved LVEF) can be recognised earlier and with better sensitivity by an impairment in LV GLS. A reduction in GLS has been demonstrated to be associated with worsening of AS severity and with increased risk for symptom development, need for AVR, and poorer survival in asymptomatic severe AS patients [60–62]. Risk stratification with GLS may potentially identify those who could benefit from closer surveillance or earlier intervention [63]. Untreated severe AS will eventually result in further "extra-valvular" cardiac damage beyond the LV (i.e., LA dilatation, MR, AF, PHTN, TR, and RV dysfunction) with incremental mortality at each stage, in both asymptomatic and symptomatic patients, and must therefore also be evaluated as part of the comprehensive TTE assessment [64–66].

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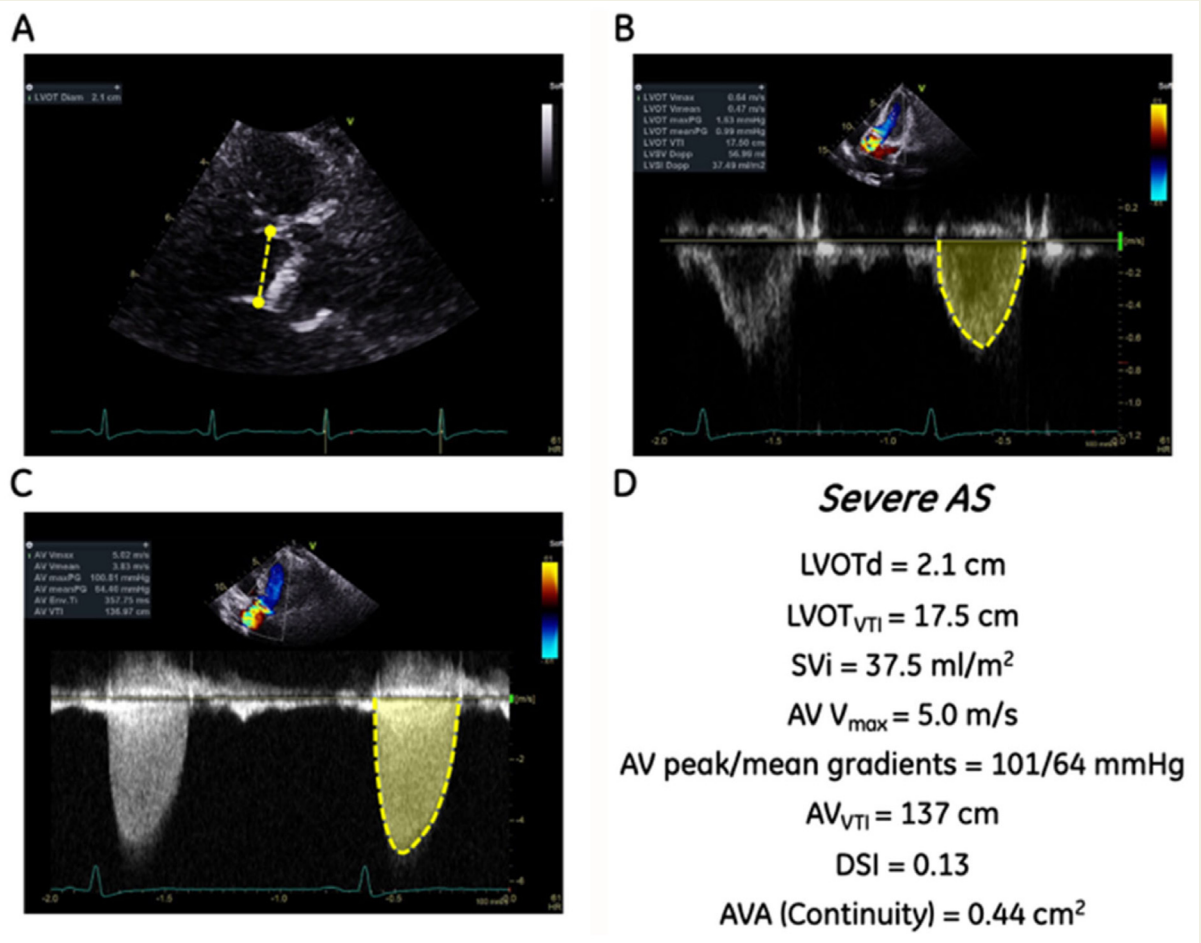

Figure A Primary haemodynamic parameters for assessment of aortic stenosis (AS) severity. A. LVOT diameter (LVOTd) measured in the parasternal long-axis view at the level of the aortic annulus in mid-systole. B. $LVOT_{VTI}$ measurement traced using the pulsed-wave Doppler signal acquired from the apical 5-chamber view. C. Continuous-wave Doppler of the aortic stenosis jet demonstrating measurements of the AV peak velocity (AV V_{max}) and tracing of the velocity curve to derive the peak/mean gradients and AV_{VTI}. D. Calculation of the dimensionless severity index (DSI = Ratio of the $LVOT_{VTI}$: AV_{VTI}) and aortic valve area by the continuity equation (AVA = 0.785 x $LVOTd^2$ x $LVOT_{VTI}$ / AV_{VTI}) consistent with severe AS. Abbreviations: LVOT, left ventricular outflow tract; LVOTd, left ventricular outflow tract diameter; VTI, velocity time integral; SVi, stroke volume index; AV, aortic valve; V_{max}, peak velocity.

3.2. Aortic Regurgitation (AR)

AR is caused by AV cusp malcoaptation due to primary leaflet abnormalities (e.g., acquired [calcific degeneration, endocarditis, rheumatic disease, radiation- or drug-induced valvulopathy] vs congenital [uni-, bi- or quadricuspid valves, or related to an underlying VSD]) or secondary to aortic root pathology (e.g., acquired [idiopathic dilatation, hypertension, aortitis, autoimmune conditions, dissection, trauma] vs congenital [annuloaortic ectasia or connective tissue disease]), or a combination of both [43,47,67,68].

AR can be acute or chronic. Acute severe AR is commonly due to aortic dissection, endocarditis, or chest trauma. It usually results in haemodynamic compromise, pulmonary oedema, and cardiogenic shock, requiring urgent surgical intervention. TTE and/or transoesophageal echocardiography (TOE) is indicated for immediate assessment. The remainder of this section will focus primarily on chronic AR.

3.2.1. Recommendations for the performance of TTE for assessment of AR [43,46,47,54]

1. To establish the diagnosis in patients with symptoms or signs of AR (to determine aetiology, severity, and haemodynamic consequences [including baseline evaluation of LV size and systolic function, RV size and systolic function, and aortic root shape/size]).
2. Re-evaluation of known AR in patients with a significant change in clinical status.
3. Assessment of AR and degree of aortic dilatation in patients with BAVs or enlarged aortic roots.

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4. Before pregnancy in patients with known AR (to determine AR severity and LV dimensions and systolic function).

3.2.2. Recommendations for serial TTE assessment of AR

A comprehensive overview of the pathophysiology and natural history of AR is beyond the scope of this document. In chronic severe AR, the LV is subjected to both volume and pressure overload as a direct consequence of the regurgitant volume and systolic hypertension. Compensatory eccentric hypertrophy develops, resulting in LV dilatation and increased compliance, augmenting preload, and maintaining a "normal" LVEF without elevation in LV filling pressures. In response to rising wall stress and afterload, concentric LVH also occurs, and eventually the asymptomatic "compensated" state transitions to a symptomatic "decompensated" phase with overt LV systolic dysfunction and progressive chamber dilatation. Symptomatic patients with angina or heart failure have associated mortality rates of >10% and >20% per year, respectively, and asymptomatic patients with underlying LV systolic dysfunction have been reported to develop symptoms at a rate of >25% per year [43]. Therefore, chronic severe AR with symptomatic heart failure (or angina) or asymptomatic LV systolic dysfunction (LVEF ≤50% [ESC/ European Association for Cardio-thoracic Surgery {EACTS}] or LVEF ≤55% [ACC/AHA]) are universally accepted indications for aortic valve surgery [46,47]. However, asymptomatic patients with significant LV dilation (LV end-diastolic diameter (LVEDD) ≥70mm or LV end-systolic diameter (LVESD) >50mm) and "normal" LV systolic function also have a 10%–19% per year risk of developing symptoms, LV dysfunction, or death, supporting the need for closer TTE surveillance as chamber enlargement first develops, and to exclude rapid progression [69]. Intervention to improve prognosis and/or post-surgical outcomes should be considered when there is imaging evidence of LV remodelling (LV end systolic diameter (LVESD) >50mm (or indexed for BSA >25mm/m^2)) in the absence of symptoms. The updated ACC/AHA guidelines have recently modified and expanded the indication for consideration of early aortic valve surgery in asymptomatic severe AR patients with low surgical risk to include those with LVEF >55% and progressive LV dilatation to LVEDD >65mm or decline in LVEF to 55%–60% on ≥ 3 serial assessments [46]. The latest ESC/EACTS guidelines have also now also added an indication to consider intervention when surgical risk is low in patients with asymptomatic severe AR if the LVEF is 50%–55% or LVESD >20mm/m^2 [47].

Indications for repeat TTE assessment of AR [1,22,45–47,54]

1. In patients with known AR, repeat TTE should be performed when there are new or changed symptoms or physical examination findings.
2. In asymptomatic severe AR, annual or biannual assessment is recommended (repeat 6 months after the initial TTE to exclude subacute progression and then annually if no change in clinical symptoms or signs; increased frequency may be considered when LV dilatation is present or approaches the threshold for intervention, or progressive decline in LV systolic function on serial imaging despite LVEF remaining in the "normal" range).
3. In moderate AR, routine surveillance every 1–2 years is recommended.
4. In mild AR, routine surveillance every 3–5 years is recommended (extended interval if demonstrated stability over 10–15 years).
5. Re-assessment during pregnancy—once or twice during pregnancy for mild AR and every trimester for severe AR.
6. In patients with a BAV—every 3-5 years with >mild AR (no AS).

3.2.3. Requirements for comprehensive TTE assessment of AR

TTE assessment of AR must ideally characterise the aetiology, mechanism, and severity, and define LV systolic function and presence of remodelling (LV dilatation and/or hypertrophy, which is usually absent or minimal in acute AR) [68]. This fundamental evaluation will determine if surgical indication thresholds have already been met or periodic interval surveillance with clinical and imaging follow-up is more appropriate. Key parameters for the assessment of chronic AR are summarised in Table 4.

2D imaging of the AV complex (valve leaflets and aortic root) is required to identify the native anatomy, valve morphology, and underlying mechanism of the AR. Accurate functional classification of AR using a Carpentier-type approach—Type I, normal cusp motion with aortic dilatation (Ia-c) or cusp perforation (Id); Type II, cusp prolapse; Type III, cusp restriction—is essential to guide the surgical approach, where valve-sparing surgery or valve repair may be preferred over valvular replacement depending on the available expertise [47,68]. 2D TTE assessment of the thoracic aorta is also mandatory to delineate the presence of any associated aortopathy or aneurysmal dilatation, where concomitant aortic surgery may also be indicated [46,47].

No single parameter is able to accurately grade AR severity due to technical, geometrical, or haemodynamic limitations, and assumptions or variations inherent within each measurement. Therefore, the haemodynamic severity of AR is determined via an integrated approach, including qualitative, semiquantitative, and quantitative Doppler assessments of the regurgitant jet, presence of diastolic flow reversal in the distal aorta, and 2D linear or volumetric measurements of LV size and systolic function. Detailed information regarding the methodology, advantages, and pitfalls of these parameters are described in the 2022 EACVI/ESC council of valvular heart disease position paper and the 2017 ASE Recommendations for Non-invasive Evaluation of Native Valvular Regurgitation and 2015 ASE/EACVI Recommendations for Cardiac Chamber Quantification guidelines [9,68,70].

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Table 4 Key echocardiographic parameters for assessment of chronic AR.

Clinical	Height, weight, body surface area (BSA)
	Heart rate and rhythm
	Blood pressure
2D/M-mode	Aortic valve morphology (leaflets)
	Aortic root (as per aortic diseases section)
	LV size (LVEDD, LVEDV, LVESD and LVESV [indexed for BSA])
	LVEF
	LVOT and mitral annular diameters
Spectral Doppler	AR signal density
	AR pressure half-time
	Diastolic flow reversal (proximal descending and abdominal aorta)
	LVOT and mitral annular VTIs
Colour Doppler	AR jet width (or area) in the LVOT
	Vena contracta width (VCW)
	FCR and corresponding Nyquist velocity
Quantification (as required/feasible)	Regurgitant volume (RVol)
	Regurgitant fraction (RF)
	Effective regurgitant orifice area (EROA)
Advanced (if available and technically feasible)	LV GLS
	3D LVEDV/LVESV and LVEF

Abbreviations: AR, aortic regurgitation; 2D, two-dimensional; 3D, three-dimensional; LV, left ventricular; FCR, flow convergence radius; GLS, global longitudinal strain; LVEDD, left ventricular end-diastolic diameter; LVEDV, left ventricular end-diastolic volume; LVESD, left ventricular end-systolic diameter LVESV, left ventricular end-systolic volume; LVEF, left ventricular ejection fraction; LVOT, left ventricular outflow tract.

Qualitative and semiquantitative colour Doppler imaging (CDI) parameters include the vena contracta width (VCW), flow convergence radius (FCR), and the ratio of the width (and/or area) of a central jet relative to that of the LVOT (Figure B). Spectral Doppler parameters include PWD of the descending thoracic and abdominal aorta and CWD of the AR jet. Specific indicators of mild AR include a VCW <0.3cm, absent or small FCR at a Nyquist limit of 50–70cm/s, central jet width <25% of the LVOT, incomplete or faint AR CWD signal, pressure half-time (PHT) >500ms, and normal LV size [68,70]. In addition to the 2D finding of a clearly abnormal valve such as a flail leaflet, Doppler parameters of severe AR include a VCW >0.6cm, large flow convergence, central jet width ≥65% of the LVOT, holodiastolic flow reversal in the abdominal aorta, PHT <200ms, and LV dilatation without an alternative explanation. When ≥4 of these specific criteria are respectively concordant, the probability of mild or severe AR is high.

AR quantification is recommended when grading of severity is not definitive based on the initial semiquantitative and qualitative Doppler assessment and, if feasible, to be accurately performed on the primary images acquired. Quantitative measurements include calculation of the effective regurgitant orifice area (EROA), regurgitant volume (RVol), and the regurgitant fraction (RF). These parameters can be measured by the proximal isovelocity surface area (PISA) and/or volumetric methods (Figure C). An EROA ≥0.3cm^2, RVol ≥60mL, or RF ≥50% are the specified cut-offs for severe AR, while an EROA <0.1cm^2, RVol <30mL, or RF <30% would indicate mild AR. The EROA ≥0.3cm^2 and RVol ≥60mL thresholds have been previously demonstrated to predict poorer cardiac prognosis in patients with asymptomatic severe AR and preserved LVEF [71,72].

It should be highlighted that the current echocardiographic linear LV dimensions (as measured by 2D and/or M-mode) used to define the intervention thresholds for chronic severe AR are based on historical standards and outcome data. However, LVESD may under-recognise the degree of LV dilatation in response to haemodynamically significant AR in some patients due to the complex geometrical changes that occur with remodelling (i.e., LVESD may remain below the 25mm/m^2 cut-off despite significant chamber enlargement by LVESV indexed for BSA [LVESVI] criteria) [73]. An LVESVI ≥45mL/m^2 as measured by the 2D biplane Simpson's method has been associated with higher cardiac event rates and is better at predicting increased mortality in chronic severe AR in comparison to traditional LVESD thresholds [71,73,74]. TTE evaluation of AR must therefore include a comprehensive LV assessment and, where required and/or available, UEAs to enhance endocardial border definition (when suboptimal), or 3D echocardiography (to overcome 2D foreshortening) for more accurate and reproducible measurement of LV volumes [9]. Additional measurements of GLS as a marker of subclinical LV systolic dysfunction may also have an incremental risk stratification role in asymptomatic chronic significant AR and preserved LVEF [75]. LV GLS > −19.5% has been observed to be associated with increased mortality in asymptomatic patients with ≥3+ AR, LVEF ≥50%, and LVESD <25mm/m^2. When TTE parameters of AR assessment remain inconclusive, discordant, or inconsistent with the clinical presentation, further imaging by TOE or quantification by CMRI may be considered if clinically indicated.

3.3. Prosthetic Aortic Valve Replacements (AVR)

Surgical AVR (SAVR) remains the standard of care for the majority of aortic valve interventions due to severe AS or AR. Whether a bioprosthetic or mechanical SAVR is implanted remains a shared decision balancing the patient's age/preference, procedural risks, suitability for anticoagulation (bleeding vs thromboembolism), and expected prosthesis performance/durability, with the potential need for re-intervention (surgical or transcatheter) due to

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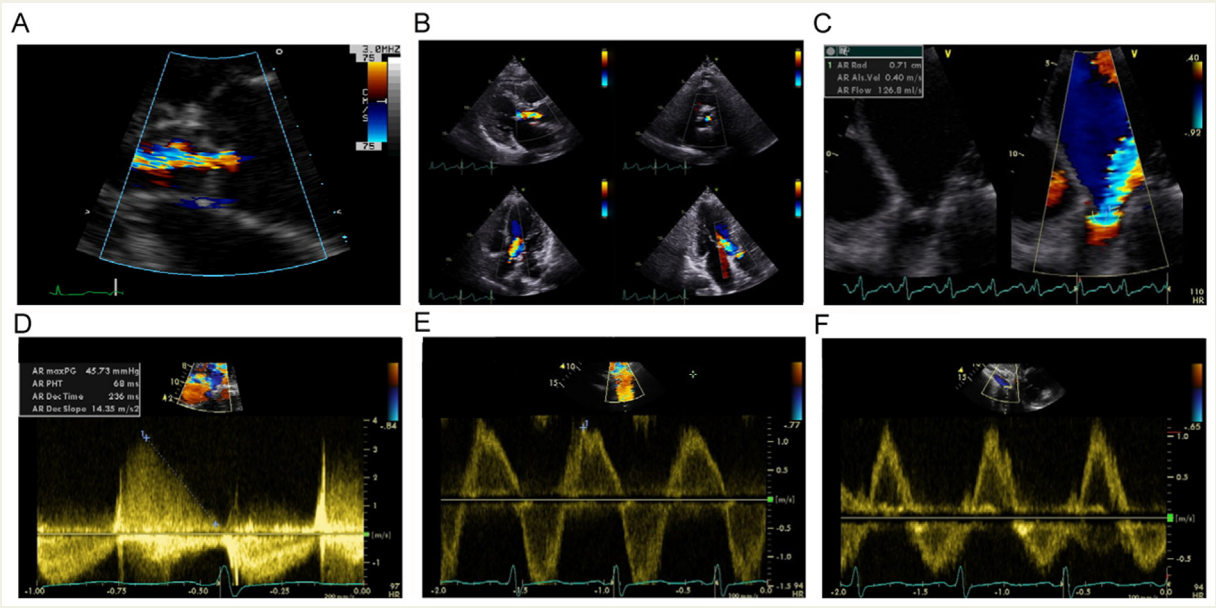

Figure B Semiquantitative and qualitative methods for assessing AR severity. A. Vena contracta width. B. Colour jet width and area from multiple views. C. Flow convergence radius D. Continuous-wave Doppler of the AR jet. E. Pulsed-wave Doppler of the descending thoracic aorta. F. Pulsed-wave Doppler of the abdominal aorta.
Abbreviation: AR, aortic regurgitation.

structural valve degeneration (SVD) or dysfunction. AVR essentially replaces severe native AV disease with that of risks of prosthetic valve dysfunction or complications (SVD, non-SVD, thrombosis, or infective endocarditis). Long-term durability of SAVRs is dependent on both the type of bioprosthesis and patient factors. Risk factors for earlier and increased risk of bioprosthetic SAVR deterioration include a younger age at time of implantation (<60 years of age), smoking, hypertension, diabetes, end-stage renal failure, hyperparathyroidism, systemic inflammatory disease, and patient-prosthesis mismatch with elevated initial transvalvular gradients [46,76,77].

Transcatheter AVR (TAVR) is currently approved in Australia for the treatment of symptomatic severe AS in patients deemed by a Heart (Valve) Team consensus as being high-surgical risk or inoperable candidates. Multiple self- or balloon-expandable platforms are available with evolving designs and emerging longitudinal data on haemodynamic performance and durability [78]. Routine TTEs post-TAVR must include comprehensive assessments of haemodynamic function of the prosthesis, transvalvular/paravalvular AR, LV/RV function, concomitant native valvular dysfunction, pulmonary pressures, and monitoring for early or late complications (e.g., valve migration, root rupture, progressive or new AR/AS, leaflet calcification or thrombosis) [78–82]. Regular TTE imaging follow-up is also a requirement for TAVR registries allowing prospective data collection useful for establishing "normal" echocardiographic parameters and assessment of valve durability [83].

3.3.1. Recommendations for performance of TTE post-SAVR [22,45–47,76,77,84–87]

1. For baseline haemodynamic assessment ("fingerprint") within 4–6 weeks post-surgery (if the initial TTE is performed prior to hospital discharge following SAVR, re-assessment can be considered at 4–6 weeks when optimal imaging windows are more accessible, patient haemodynamics are more stable, and peri-operative LV systolic dysfunction has resolved).
2. Repeat TTE for routine surveillance at 5 years post-bioprosthetic SAVR.
3. Repeat TTE for routine annual surveillance post-bioprosthetic SAVR in asymptomatic patients (variation exists between current AHA/ACC and ESC guidelines with regards to the starting time point):
   a. Beginning 10 years after implantation for bioprosthesis with well-established durability.
   b. Beginning 5 years after implantation for patients with bioprosthetic SAVR implanted <60 years of age or other risk factors for SVD as described above.
   c. Annually from implantation for "new" prostheses designs without proven durability data.
4. Routine annual TTE surveillance is recommended immediately post-Ross procedure or AV repair.
5. Repeat TTE is recommended with any new or changed clinical symptoms or suspected prosthesis dysfunction or complication.

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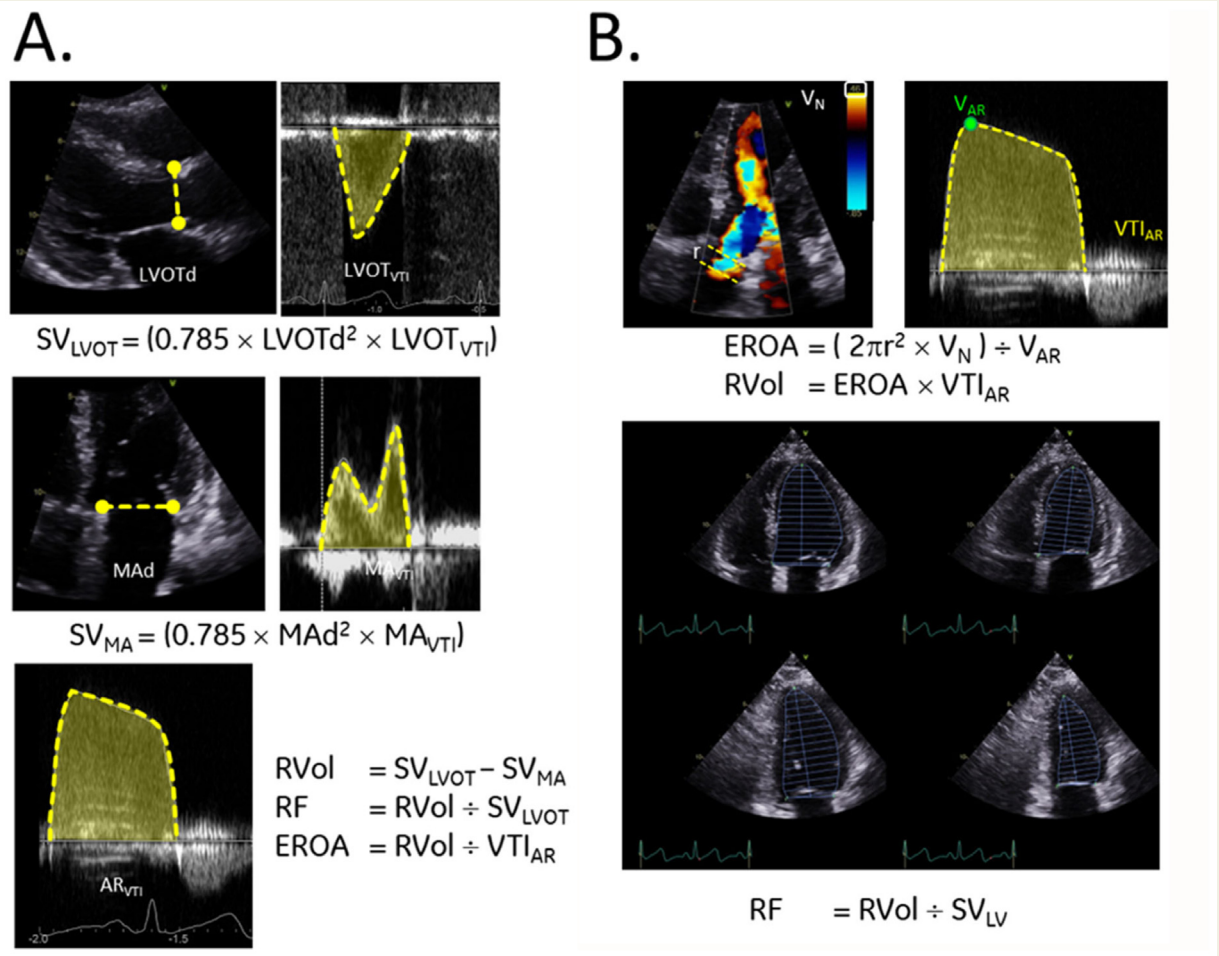

Figure C Quantitative methods for assessing AR severity. A. Volumetric method: the regurgitant volume (RVol), regurgitant fraction (RF) and the effective regurgitant orifice area (EROA) are derived from the LVOT diameter (LVOTd), LVOT velocity time integral ($LVOT_{VTI}$), the mitral annular diameter (MAd), the mitral annular VTI (MA_{VTI}), and the VTI of the AR CWD signal (VTI_{AR}). B. PISA method: the EROA and RVol are derived from the PISA radius (r), the colour Nyquist limit (V_N), the peak AR velocity (V_{AR}), and the AR VTI (VTI_{AR}). The RF can be derived from the RVol and the total LV stroke volume (SV_{LV}) which is the difference between the biplane left ventricular end diastolic (Bp LVEDV) and end systolic (Bp LVESV) volumes.
Abbreviations: AR, aortic regurgitation; LVOT, left ventricular outflow tract; LV, left ventricular; VTI, velocity time integral; CWD, continuous-wave Doppler; PISA, proximal isovelocity surface area.

6. Repeat TTE within 3–6 months after initial diagnosis of SVD post-bioprosthetic SAVR to assess for rapid progression.

An initial TTE is recommended in all patients after prosthetic valve replacement for evaluation of valve haemodynamics [46,85,86]. This baseline assessment provides a reference point which can then be compared with future studies, such that the patient serves as his/her own longitudinal control for detection of progressive prosthetic valve dysfunction. The recommended timing of this post-operative baseline study is variable. Ideally, a comprehensive baseline TTE should be obtained when the chest wound has healed, chest wall oedema has resolved, LV systolic function has recovered, and when the patient is more mobile and less tender. Therefore, this baseline study may be performed at the first post-operative visit which is typically 4–6 weeks following surgery. Serial clinical and TTE assessment post-AVR are aimed at early detection and monitoring for prosthesis, ventricular, or other valvular dysfunction, and to help guide medical optimisation or need for re-do intervention. There has been more recent focus on standardisation of definitions and outcomes of SVD as proposed by the Valve-in-Valve International Data (VIVID) group with a resultant shift in the recommendations towards earlier TTE surveillance for TAVRs post-implantation and bioprosthetic SAVRs [88,89]. No routine surveillance is recommended for mechanical prostheses with normal function on the baseline TTE, in the absence of clinical change or other associated cardiac or aortic pathology requiring ongoing imaging

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follow-up (e.g., LV systolic dysfunction or aortic dilatation) [44,45].

3.3.2. Recommendations for performance of TTEs post-TAVR [45–47,77,88,89]

1. Immediately (prior to discharge) following TAVR implantation, 30 days, and then annually thereafter.
2. Repeat TTE would be recommended with any new or changed clinical symptoms or suspected prosthesis dysfunction or complication.
3. Repeat TTE within 3–6 months after initial diagnosis of SVD post-TAVR to assess for rapid progression.

Although the recommendations above serve as a general guide to timing for surveillance post-AVR, the specific interval between serial TTE assessments must be tailored to the individual by the treating clinician based upon the patient's clinical status, comorbidities, and baseline/follow-up echocardiographic findings (e.g., new or persistent post-operative LV systolic dysfunction or evidence of SVD that will require increased frequency of monitoring by imaging up to 3–6 monthly depending on lesion severity or stability).

3.3.3. Requirements for comprehensive TTE assessment of AVRs

A comprehensive review of echocardiographic SAVR or TAVR evaluation is beyond the scope of this document but has been detailed in the relevant societal guidelines, consensus statements, and expert reviews [20,78,80,81,85,86,88]. Key TTE parameters for assessment of AVRs are briefly discussed and summarised in Table 5.

Comprehensive TTE assessment of the AVR requires interrogation from multiple on- and off-axis views to overcome acoustic shadowing and accurately evaluate the structural appearance of the prosthesis (leaflet or occluder morphology/mobility), seating/stability/integrity of the sewing ring for SAVRs, or additionally stent position/expansion/apposition for TAVRs, calcification or other abnormalities (e.g., vegetation, thrombus, or pannus) associated with any components of the AVR, and to identify the presence/origin/location/severity of any AR [20,85,86]. The fundamental haemodynamic assessment of any AVR includes measurements of the peak transvalvular velocity (AVR V_{max}), AVR mPG, calculation of the Doppler velocity index (DVI) and effective orifice area (EOA)/effective orifice area indexed for BSA(IEOA), and the acceleration (AT)/ejection (ET) times and AT : ET ratio (Figure D). AVR transvalvular Doppler velocities, pressure gradients, and the EOA are dependent on the prosthesis size but also on the underlying flow-state and LV systolic function, warranting the same considerations at time of echocardiographic assessment as previously discussed with native valvular AS. Where available, these parameters should be referenced against the normative values for the individual prosthesis model and size, and comparison to the baseline study or serial post-AVR TTE assessments would be useful if prosthetic valve dysfunction is suspected. Prosthesis stenosis should be considered when AVR V_{max} is ≥3m/s, mPG ≥20mmHg, and DVI <0.30 (<0.35 for TAVRs), or an interval increase in the mPG by >10mmHg with corresponding decrease in the DVI or EOA by >25% [79,85,86,89]. "Significant" AVR stenosis is supported by an AVR V_{max} ≥4m/s, mPG ≥35mmHg, DVI <0.25, EOA <0.8cm^2, and an increase in the mPG ≥20mmHg during follow-up assessment (mPG >40mmHg, DVI <0.20 for an LVOTd >2.5cm, and EOA <1.0cm^2 [if the patient's BSA was ≥1.6m^2] were updated proposed cut-offs for severe bioprosthetic SAVR or TAVR stenosis by the VIVID group in the context of SVD) [85,86,89]. In addition to the parameters above, when elevated transvalvular velocities or gradients are encountered, AVR stenosis may be further differentiated from patient–prosthesis mismatch or a high-flow state by the presence of a rounded CWD jet contour as indicated by an AT>100ms and AT : ET ratio >0.37 [85,86,89,90].

The integrative echocardiographic approach to the assessment of chronic pathological prosthetic AR in SAVRs shares similar principles to that of native aortic valves. Initial 2D appearances supporting the presence of abnormal transvalvular AR include immobility of an occluder (mechanical AVR) or leaflet thickening/calcification/prolapse/flail (bioprosthetic AVR) [85,86]. Any evidence of dehiscence or "rocking" of the prosthesis will be associated with paravalvular AR. Semiquantitative and qualitative Doppler parameters for grading severity of prosthetic AR include the VCW, jet width : LVOT ratio, PHT, density of the AR CWD signal, and presence of holodiastolic flow reversal in the descending thoracic and abdominal aorta. Quantification by EROA, RVol, and RF may also be considered with the same methodology, if feasible. However, the evaluation of paravalvular AR is more challenging, particularly post-TAVRs where defects may be multiple with irregular and eccentric jets, requiring more detailed interrogation and caveats imposed on the aforementioned conventional Doppler parameters [81]. Additional semiquantitative CDI parameters for the assessment of paravalvular AR severity include the circumferential extent of the jet origin (length at the neck) relative to the total prosthesis circumference and the vena contracta area (VCA; measured by 2D or 3D) on the short-axis view. The same cut-offs for circumferential extent are applicable to both SAVR and TAVRs with <10%, 10%–29%, and ≥30% corresponding to mild, moderate, and severe paravalvular AR, respectively [81,86]. VCAs of <0.10cm^2, 0.10–0.29cm^2, and ≥0.30cm^2 indicate mild, moderate, and severe paravalvular AR post-TAVR [81]. Persistent or progressive LV dilatation post-AVR, in the absence of an alternative cause, should also mandate careful exclusion of significant prosthetic regurgitation, including further imaging with TOE and/or CMRI as required.

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Table 5 Key echocardiographic parameters for assessment of an AVR [81,85,86,89].

Clinical	Date of valve replacement
	Type and size of the prosthetic valve
	Normal Doppler echocardiographic values for the prosthetic valve (where available)
	Height, weight, body surface area (BSA)
	Symptoms and related clinical findings
	Heart rate and rhythm
	Blood pressure
2D	Opening and closing motion of the prosthesis (leaflets for bioprosthesis and occluders for mechanical prostheses)
	Presence of prosthetic leaflet calcification or abnormal echodensity attached to various components of the prosthesis
	Sewing ring seating, stability, and integrity (SAVR)
	Stent position, apposition/expansion, stability, and integrity (TAVR)
	LVOT diameter
Spectral Doppler	AVR peak velocity (AVR V_{max})
	AVR mean gradient
	AVR velocity time integral (VTI)
	LVOT VTI
	Doppler velocity index (DVI)
	Effective orifice area (EOA) by continuity
	EOA indexed for BSA (IEOA)
	Shape or contour of the CWD signal
	Acceleration (AT) and ejection (ET) times and AT : ET ratio
Colour Doppler	If AR present, determine: • Normal or pathological • Transvalvular or paravalvular • Severity (assess as for native AR, with the addition of the circumferential extent of the AR colour jet expressed as a % of the sewing circumference and vena contracta area [VCA] for paravalvular AR)
Other imaging data	LV/RV dimensions
	LV/RV systolic function parameters (LVEF, RV TAPSE, RV FAC)
	LVH
	Aorta (as per aortic diseases section)
	Co-existent valvular disease
	PASP (RVSP)
Previous post-operative studies, when available	Comparison of above parameters in suspected prosthetic valvular dysfunction

Abbreviations: AVR, aortic valve replacement; 2D, two dimensional; TAPSE, tricuspid annular plane systolic excursion; SAVR, surgical aortic valve replacement; TAVR, transcatheter aortic valve replacement; LVOT, left ventricular outflow tract; CWD, continuous-wave Doppler; AR, aortic regurgitation; LV, left ventricular; RV, right ventricular; FAC, fractional area change; LVH, left ventricular hypertrophy; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure.

4. Mitral Valve Diseases

4.1. Mitral Stenosis (MS)

Valvular mitral stenosis (MS) can be caused by an acquired deformation of the valve secondary to rheumatic heart disease, thoracic irradiation, or various systemic diseases such as Fabry disease, mucopolysaccharidosis, endomyocardial fibrosis, systemic lupus erythematosus, and rheumatoid arthritis [46,91,92]. Rarely, MS may occur as a result of congenital malformations of the valve. Non-rheumatic calcific MS may also be a result of extensive mitral annular calcification secondary to degenerative changes and/or chronic renal disease [93].

4.1.1. Recommendations for performance of TTE for assessment of MS [46,47,54,94]

1. To establish the diagnosis in patients with symptoms or signs of MS (including determination of the aetiology, severity, and haemodynamic consequences).
2. Re-evaluation of known MS in patients with a significant change in clinical status.
3. Pre-procedural decision-making for valvular intervention.

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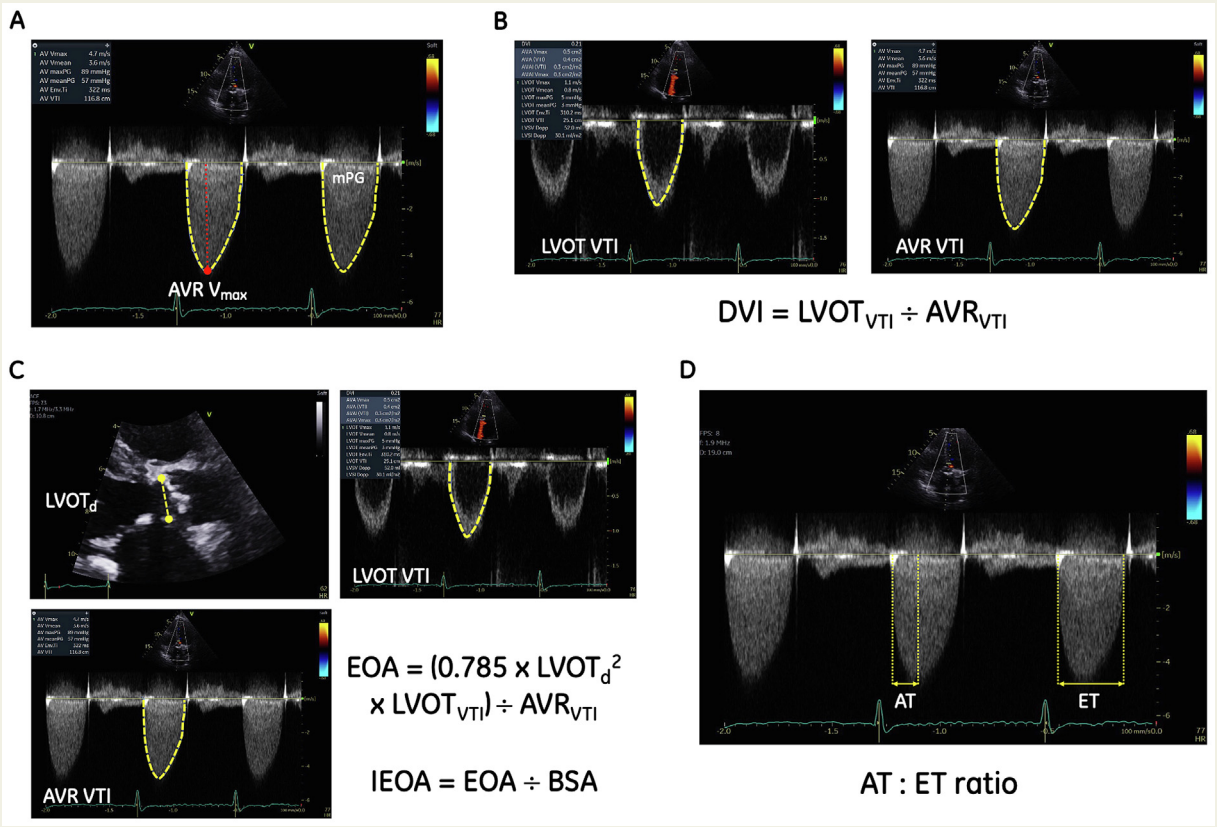

Figure D Haemodynamic assessment of an AVR. A. From the continuous-wave Doppler profile across the AVR, the peak velocity (AVR V_{max}), and mean pressure (mPG) gradient are measured. B. The Doppler velocity index (DVI) is derived from the LVOT velocity time integral ($LVOT_{VTI}$) and the AVR velocity time integral (AVR_{VTI}). C. The effective orifice area (EOA) is derived from the LVOT diameter (LVOTd), $LVOT_{VTI}$, and the AVR_{VTI}, and the indexed EOA (IEOA) is derived from the EOA and body surface area (BSA). D. The acceleration (AT) and ejection (ET) times are measured from the continuous-wave Doppler profile across the AVR for calculation of the AT : ET ratio.
Abbreviations: AVR, aortic valve replacement; LVOT, left ventricular outflow tract.

4. Before or during pregnancy in patients with known or suspected MS (to determine the severity and haemodynamic consequence of the MS).

4.1.2. Recommendations for serial TTE assessment of MS [1,22,46,47,54,95]

1. In patients with known MS, the frequency of a repeat TTE examination is based on symptoms, the type and severity of MS, suspected rate of progression, and the haemodynamic consequences. Repeat TTE should be performed when there are new or changed symptoms or physical examination findings.
2. In asymptomatic patients with severe MS (mitral valve area [MVA]≤1.5cm^2), annual TTE assessment is recommended.
3. In patients with moderate or progressive MS, routine surveillance TTE is recommended every 2–3 years.
4. Re-assessment during pregnancy—every trimester and prior to delivery for mild MS, and every 1–2 months for moderate–severe MS, depending on haemodynamic tolerance.

4.1.3. Requirements for comprehensive TTE assessment of MS

The primary role of echocardiography in the assessment of MS is to determine the aetiology and haemodynamic severity of the stenosis. TTE is considered the gold standard for the non-invasive assessment of MS. In addition, TTE (and TOE) can be used to calculate the Wilkins score to determine suitability for, and likelihood of success of, percutaneous balloon mitral valvuloplasty (PBMV). Key TTE parameters for the assessment of chronic MS are summarised in Table 6.

The aetiology of MS is based upon the 2D appearance of the valve. Rheumatic mitral valve (MV) disease is characterised by diastolic doming of the leaflets due to commissural fusion. In planning for PBMV for valvular MS, images depicting the anatomical appearance of the MV apparatus should be acquired for assessment of leaflet mobility/thickness/calcification (including commissural calcification), and degree of subvalvular involvement.

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Table 6 Key echocardiographic parameters for assessment of MS.

Clinical	Height, weight, body surface area (BSA) Heart rate and rhythm Blood pressure
2D/M-mode	Mitral valve (MV) morphology (leaflets, annulus, and supporting apparatus) MV Area (2D planimetry) LAVI LV/RV dimensions LV/RV systolic function parameters (LVEF, RV TAPSE, RV FAC)
Spectral Doppler	MVA (pressure half-time, continuity, or proximal isovelocity surface area [PISA]) MV mean gradient (Heart rate noted) PASP (RVSP) RV systolic function parameters (RV S′)
Colour Doppler	MR severity TR severity
Other imaging data	Co-existent valvular disease (suggesting possible rheumatic involvement)
Advanced (if available and technically feasible)	3D MVA RV free wall longitudinal strain

Abbreviations: LAVI, left atrial volume index; LV, left ventricular; RV, right ventricular; TAPSE, tricuspid annular plane systolic excursion; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure; RV S′, tissue Doppler-derived tricuspid lateral annular systolic velocity; MR, mitral regurgitation; TR, tricuspid regurgitation; 2D, two-dimensional; 3D, three-dimensional; MVA, mitral valve area.

Careful evaluation of the aortic and tricuspid valves for evidence of rheumatic involvement should also be routinely performed [94].

Haemodynamic severity of MS is best characterised by the MVA. A MVA of $\leq$1.5cm^2 is regarded as severe MS [46,47,94,96]. MVA can be estimated via 2D or 3D planimetry, diastolic pressure half-time (PHT), SV (continuity) method, or the PISA technique (Figure E). The preferred approach is by planimetry, as this best correlates with the anatomical MVA (MVA by 3D planimetry is more accurate than 2D) [97]. Where suboptimal image quality prohibits accurate planimetry, MVA can be calculated from the Doppler-based methods, as listed above. However, these are dependent upon many variables such as loading conditions, heart rate/rhythm, LA/LV compliance, haemodynamic assumptions, co-existing valve disease, and technical factors [46,47,94,96]. For example, the MVA derived from the PHT is inaccurate in the setting of tachycardia, AF, non-linear Doppler velocity curves, pregnancy, or with changes in LA/LV compliance [98]. Furthermore, as degenerative MS is typically a disease of the elderly often associated with AS, hypertension, and decreased LV compliance, the MVA may be overestimated via the PHT method [99]. MVA calculated by the continuity method using the LVOT SV and MV VTI is only feasible in the absence of significant AR or MR. RVOT SV can be substituted in the presence of the latter. Alternatively, when there is significant MR or changes to LA/LV compliance, MVA can be estimated using the PISA method [98]. Additional haemodynamic parameters to support MS severity include the transmitral mPG and the RVSP, as these parameters reflect the haemodynamic consequences of MS and have prognostic value [46]. However, the mPG is highly dependent on transvalvular flow and the diastolic filling period (mPG may be disproportionately elevated with co-existent MR or at higher heart rates) [46,94,96].

MS results in LA dilatation/dysfunction, post-capillary PHTN with secondary RV dilatation/dysfunction, and worsening of functional TR. Therefore, the TTE assessment of MS must include a comprehensive evaluation of left and right heart chamber sizes and function, and estimation of pulmonary pressures as detailed in the previous sections and societal guidelines [9]. Concomitant MR and TR should be assessed in accordance with the published ASE recommendations [68].

4.2. Mitral Regurgitation (MR)

The mitral valve complex is composed of several components, all of which need to function in unison to ensure competency of the valve. Therefore, disorders or dysfunction of one or more of these components may lead to MR. Examples include annular dilatation/calcification, leaflet abnormalities/infection, chordal elongation/maldevelopment/rupture, and papillary muscle dysfunction/malalignment/rupture.

Based on the aetiology and mechanism, MR is classified as primary (organic or degenerative) or secondary (functional). Primary MR occurs when there is a structural alteration of one or more components of the mitral valve complex. Secondary MR occurs when there is a structurally normal mitral valve with insufficient coaptation due to cardiac chamber remodelling, with or without annular dilatation. Furthermore, via the Carpentier classification, MR can be defined according to mitral leaflet motion - Type I, normal leaflet motion with MR secondary to annular dilatation or leaflet perforation; Type II, excessive leaflet motion (e.g., mitral valve prolapse or flail); Type III, restricted leaflet motion in systole and diastole (IIIa; e.g., rheumatic) or in systole only (IIIb; e.g., ischemic/dilated cardiomyopathy) [100].

MR can also be acute or chronic. Acute MR may occur due to papillary muscle rupture following an acute STEMI, spontaneous chordal rupture in patients with mitral valve prolapse (MVP), leaflet tear or perforation due to chest

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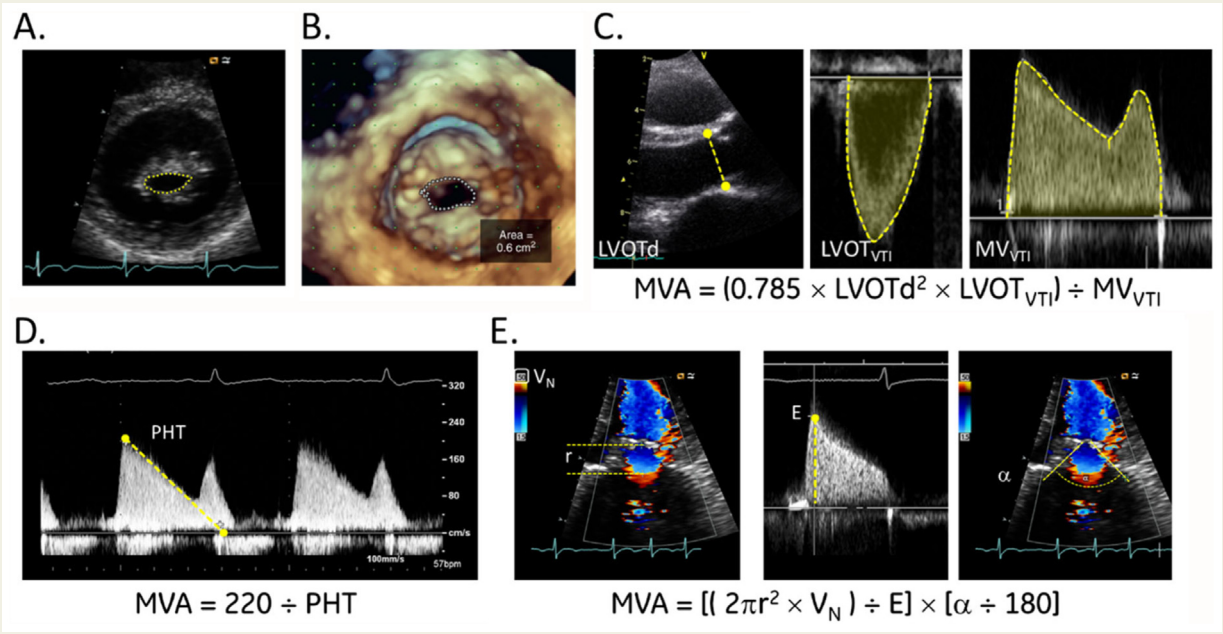

Figure E Methods for measuring the MVA. A. 2D planimetry. B. 3D planimetry. C. Continuity method: derived from the LVOT diameter (LVOTd), LVOT velocity time integral ($LVOT_{VTI}$), and the mitral valve velocity time integral (MV_{VTI}). D. PHT method. E. PISA method: derived from the PISA radius (r), the colour Nyquist limit (V_N), the peak early diastolic velocity (E), +/- the angle of flow convergence (α).
Abbreviations: MVA, mitral valve area; 2D, two-dimensional; 3D, three-dimensional; LVOT, left ventricular outflow tract; PHT, pressure half-time; PISA, proximal isovelocity surface area.

trauma, leaflet perforation or chordal rupture secondary to infective endocarditis, systolic anterior motion of the mitral valve in Takotsubo cardiomyopathy, or iatrogenic balloon-induced trauma during percutaneous balloon mitral valvotomy [46,101]. More recently developed therapeutic approaches, such as TAVR and intracardiac left ventricular assist devices, may also result in acute MR due to chordal rupture as a result of device misplacement or leaflet tethering and/or chordal entanglement of guidewires or catheters [101,102]. The remainder of this section will focus primarily on chronic MR.

4.2.1. Recommendations for performance of TTE for assessment of MR [2,22,47,54,84,103]

1. To establish the diagnosis in patients with symptoms or signs of MR and/or MVP to determine aetiology, severity, and haemodynamic consequences, including baseline evaluation of LV size and systolic function (±regional wall motion abnormalities [RWMA], RV size and systolic function, LA size, PASP).
2. Re-evaluation of known MR in patients with a significant change in clinical status.
3. New onset of AF.
4. Pre-procedural decision-making for valvular repair.
5. Before pregnancy in patients with known MR (to determine MR severity and LV dimensions and systolic function).

For primary MR, the baseline TTE is important for the evaluation of LV/RV size and systolic function, LA size, estimation of the PASP, and determining the mechanism and severity of the MR [46]. For secondary MR, an initial study is useful for establishing the aetiology and extent of LV RWMAs, global LV function, MR severity, and degree of PHTN [46]. Importantly, the initial TTE also serves as a baseline for future comparisons.

4.2.2. Recommendations for serial TTE assessment of MR

A comprehensive overview of the pathophysiology and natural history of MR is beyond the scope of this document. Importantly, an LVEF <60% in patients with significant chronic MR implies underlying impaired LV systolic function (LVEF of 60% is considered the lower limit of normal in these patients). An LVEF ≤60% or an LVESD ≥40mm portends a poor long-term prognosis and persistent post-operative LV systolic dysfunction [46,47,84,103]. The accompanying LA dilatation may lead to the development of atrial arrhythmias and the elevated LA/LV filling pressures can progress to post-capillary PHTN. New onset AF, LAVI ≥60mL/m^2, and PASP approaching 50mmHg are associated with a worse prognosis in primary MR [47,103,104]. In secondary MR, LV size and systolic function are less useful in determining prognosis, as the LV remodelling is the cause rather than the effect [103]. Any grade of MR is associated with increased mortality in ischaemic or dilated cardiomyopathies [103,105]. Predictors of adverse outcomes following percutaneous edge-to-edge MVr include RV systolic

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dysfunction (TAPSE≤14mm or RV S′ <9.5cm/s), PASP >50mmHg, LVEF <44%, SV <50mL, marked LV dilatation (LVEDV >216mL, LVESV >110mL), and severe TR [106–108].

Indications for repeat TTE assessment of MR [2,22,45–47,54]

1. In patients with known MR, repeat TTE should be performed when there are new or changed symptoms or physical examination findings.
2. In asymptomatic severe MR with preserved LV systolic function (LVEF >60%), annual or biannual assessment is recommended (6-monthly assessment as LVEF and LVESD approach threshold values or significant interval change; with emphasis on assessment of LVEF, LVESD, and PASP).
3. In asymptomatic moderate primary MR with preserved LV systolic function (LVEF >60%), routine surveillance every 1–2 years is recommended.
4. Routine surveillance every 3–5 years for mild MR.
5. Re-assessment during pregnancy—once or twice during pregnancy for mild MR and every trimester for severe MR.

4.2.3. Requirements for comprehensive TTE assessment of MR

TTE is crucial in establishing the aetiology, mechanism, severity, and haemodynamic consequences of MR, guiding patient management and surgical/interventional planning, as well as predicting procedural success and prognosis. MR severity is dynamic and the patient's blood pressure and heart rate/rhythm at time of the TTE assessment must be recorded. Key TTE parameters for the assessment of chronic MR are summarised in the Table 7.

The aetiology and mechanism of MR is based upon a careful inspection of the 2D appearance of the valve and its supporting apparatus. This should also include a distinction between primary and secondary MR, and a categorisation of leaflet motion based on the Carpentier classification as previously mentioned. 3D echocardiography is especially useful in MVP, as this modality can provide detailed views of the mitral valve scallops.

With the exception of severe anatomic lesions (e.g., flail leaflet, ruptured papillary muscle, severe leaflet retraction, or a large perforation), there is no single echocardiographic parameter that is sufficient to quantify MR severity [68,103]. Therefore, the haemodynamic severity of MR is determined via an integrated approach, including LV and LA chamber sizes, and semiquantitative and quantitative parameters. Detailed information regarding the methodology, advantages, and pitfalls of these parameters are described in the 2022 EACVI/ESC council of valvular heart disease position paper and the 2017 ASE Recommendations for Non-invasive Evaluation of Native Valvular Regurgitation and 2015 ASE/EACVI Recommendations for Cardiac Chamber Quantification guidelines [9,68,70].

Table 7 Key echocardiographic parameters for assessment of chronic MR.

Clinical	Height, weight, body surface area (BSA)
	Heart rate and rhythm
	Blood pressure
2D/M-mode	Mitral valve (MV) morphology (leaflets, annulus, and supporting apparatus)
	LV size (LVEDD, LVEDV, LVESD and LVESV [indexed for BSA])
	LVEF
	Presence or absence of LV regional wall motion abnormalities
	LVOT and mitral annular diameters
	LAVI
	RV dimensions
	RV systolic function parameters (TAPSE, FAC)
Spectral Doppler	Transmitral inflow
	Pulmonary venous flow
	MR signal density, timing, and shape
	LVOT and mitral annular velocity time integrals (VTIs)
	PASP (RVSP)
	RV systolic function parameters (RV S′)
Colour Doppler	MR jet area
	Vena contracta width (VCW)
	FCR and corresponding Nyquist velocity
	TR severity
Quantification (as required/ feasible)	Regurgitant volume (RVol)
	Regurgitant fraction (RF)
	Effective regurgitant orifice area (EROA)
Advanced (if available and technically feasible)	3D mitral valve morphology (especially for MVP)
	3D vena contracta area (VCA)
	LV GLS
	3D LVEDV/LVESV and LVEF where possible
	RV free wall longitudinal strain
	LA strain
Co-existent MS or for edge-to-edge clip planning	3D MV area (MVA)
	MV mean gradient (Heart rate noted)

Abbreviations: MR, mitral regurgitation; LV, left ventricular; LVEDD, left ventricular end diastolic diameter; LVEDV, left ventricular end-diastolic volume; LVESD, left ventricular end-systolic diameter; LVESV, left ventricular end-systolic volume; LVEF, left ventricular ejection fraction; LVOT, left ventricular outflow tract; LAVI, left atrial volume index; TAPSE, tricuspid annular plane systolic excursion; FAC, fractional area change; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure; RV S′, tissue Doppler-derived tricuspid lateral annular systolic velocity; FCR, flow convergence radius; TR, tricuspid regurgitation; MVP, mitral valve prolapse; GLS, global longitudinal strain; RV, right ventricular; LA, left atrium/atrial; 3D, three-dimensional.

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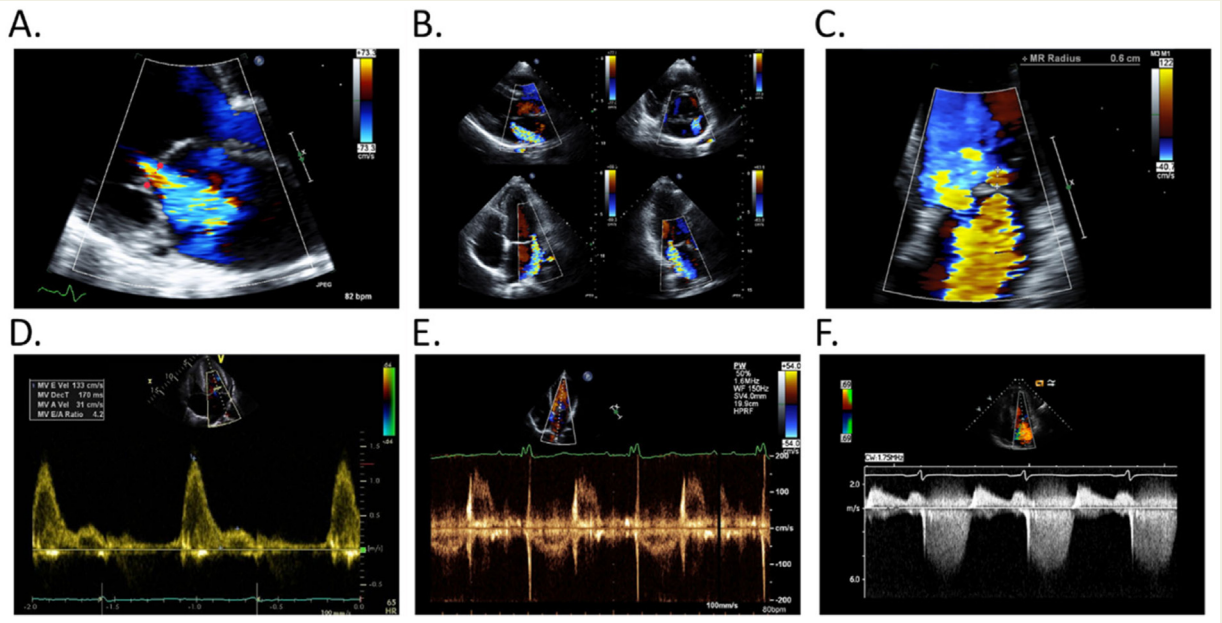

Figure F Semiquantitative methods for assessing MR severity. A. Vena contracta width. B. Colour jet area from multiple views. C. Flow convergence radius D. Pulsed-wave Doppler transmitral inflow signal. E. Pulsed-wave Doppler pulmonary venous signal. F. Continuous-wave Doppler of the MR jet.
Abbreviation: MR, mitral regurgitation.

Semiquantitative CDI parameters for MR include the VCW, FCR, and colour jet area (Figure F). Spectral Doppler parameters include the PWD transmitral inflow and pulmonary venous signals, and the CWD of the MR jet. Mild MR is supported by a VCW≤0.3cm, absent FCR or a FCR≤0.3cm at a colour Nyquist limit of 30-40cm/s, small and narrow colour jet area, an A-dominant transmitral inflow signal, a systolic dominant pulmonary venous flow, PWD MV VTI : LVOT VTI ratio <1, and faint CWD MR jet [68,70]. Criteria consistent with severe MR include a VCW ≥0.7cm, FCR ≥1.0cm at a colour Nyquist limit of 30–40cm/s, large colour jet area (>50% of the LA area), an E-dominant transmitral inflow signal (>1.2m/s), pulmonary venous systolic flow reversal, PWD MV VTI : LVOT VTI ratio >1.4, and a pansystolic dense CWD MR jet [68,70]. Adjunctive findings that suggest mild MR include a normal PASP or MR duration <30% of systole, while severe MR is supported by a dense triangular CWD profile, MR jet velocity <4.5m/s indicating high LA pressure, dilated LV/LA with no other cause, and PASP >50mmHg without an alternative explanation [103]. When multiple parameters are concordant, MR severity can be determined with high probability, especially when mild or severe.

MR quantification is strongly recommended. As with AR, quantitative measurements include the calculation of the EROA, RVol, and RF as measured by the PISA and volumetric methods (Figure G). 3D imaging may also be used to directly measure the VCA which corresponds to the EROA. When MR is pansystolic and measurements are accurately performed, an EROA ≥0.4cm^2, RVol ≥60mL, or RF ≥50% are highly specific for severe MR, while an EROA <0.2cm^2, RVol <30mL, or RF <30% are consistent with mild MR [68,70,103]. In secondary MR with an elliptical or crescentic regurgitant orifice, an EROA ≥0.3cm^2 is indicative of severe MR. Lower thresholds have been previously proposed to define severe MR (EROA ≥0.2cm^2, RVol ≥30mL) owing to their association with poorer prognosis [109]. However, the updated 2020 ACC/AHA and 2021 ESC/EACTS valvular heart disease guidelines have now recommended the same cut-off values for secondary severe MR as for primary MR (EROA ≥0.4cm^2, RVol ≥60mL) [46,47]. For patients with co-existent MS (rheumatic or degenerative) or being considered for an edge-to-edge clip, additional measurements including the MVA and transmitral mPG will be required [103].

The haemodynamic burden of chronic MR may result in LV/LA remodelling and dysfunction, post-capillary PHTN with secondary RV dilatation/dysfunction, and worsening of functional TR. Therefore, the TTE assessment of MR must also include comprehensive evaluation of left and right heart chamber sizes and function, and estimation of pulmonary pressures as detailed in the previous sections and societal guidelines [9]. More advanced TTE imaging techniques such as GLS may be useful to detect early impairment of LV systolic function, and incrementally improve risk stratification in asymptomatic patients with significant (≥3+) primary MR and preserved LVEF (≥60%) [110,111]. LV GLS >−19.9% has been previously demonstrated to predict long-term LV dysfunction post-repair and more recently a cut-off GLS >−20.6% was associated with increased mortality post-mitral valve surgery [112,113]. Notably, as LV GLS

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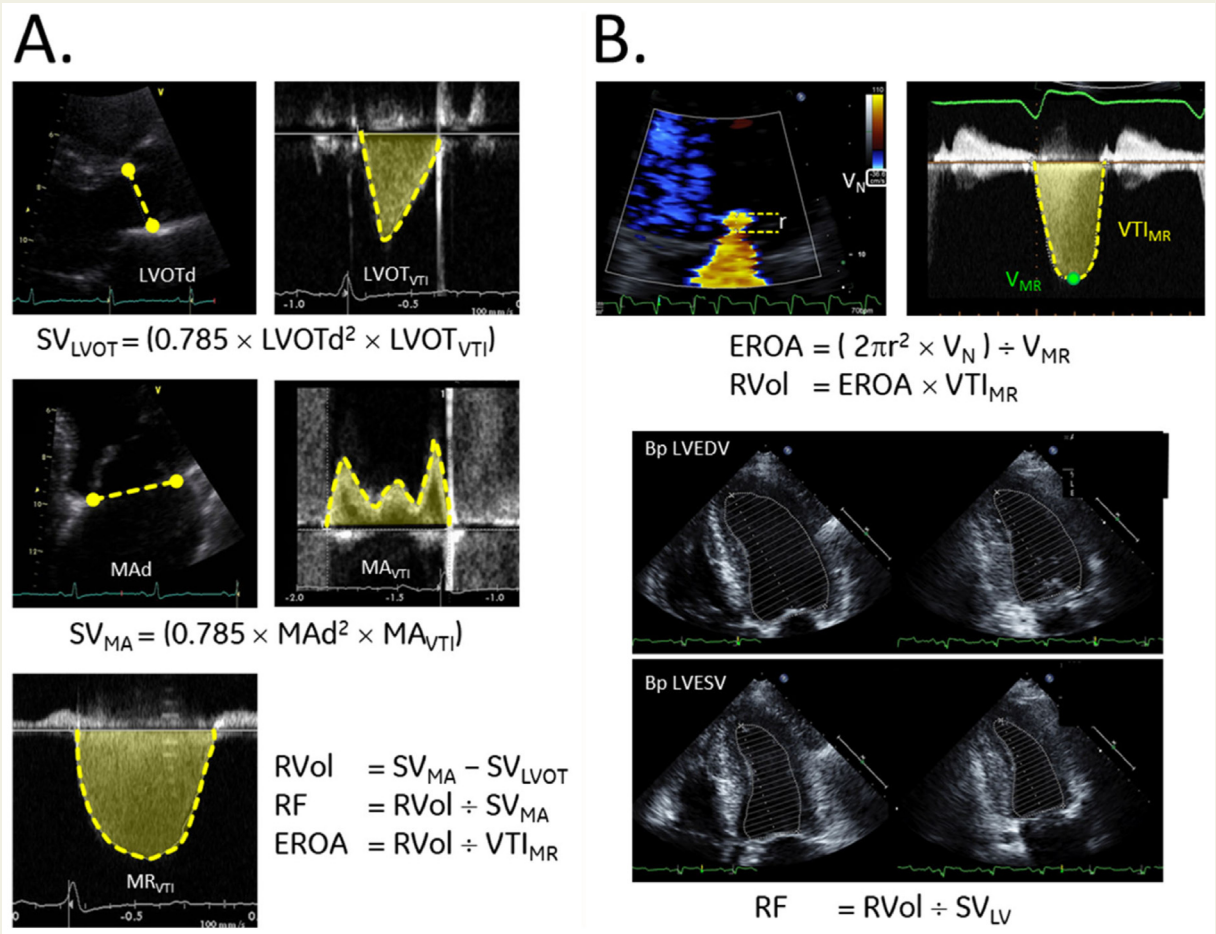

Figure G Quantitative methods for assessing MR severity. A. Volumetric method: the regurgitant volume (RVol), regurgitant fraction (RF), and the effective regurgitant orifice area (EROA) are derived from the LVOT diameter (LVOTd), LVOT velocity time integral ($LVOT_{VTI}$), the mitral annular diameter (MAd), the mitral annular VTI (MA_{VTI}), and the VTI of the MR CWD signal (VTI_{MR}). B. PISA method: the EROA and RVol are derived from the PISA radius (r), the colour Nyquist limit (V_N), the peak MR velocity (V_{MR}), and the MR VTI (VTI_{MR}). The RF can be derived from the RVol and the total LV stroke volume (SV_{LV}) as either: (1) the difference between the biplane left ventricular end diastolic (Bp LVEDV) and end systolic volumes (Bp LVESV) or (2) the LVOT stroke volume (SV_{LVOT}) + RVol (not shown).
Abbreviations: MR, mitral regurgitation; LVOT, left ventricular outflow tract; VTI, velocity time integral; CWD, continuous-wave Doppler; PISA, proximal isovelocity surface area.

measurements are load-dependent, higher-than-normal strain values have been reported in patients with MR, and therefore the reference ranges obtained in a healthy population do not apply for these patients [114]. Currently, there is no consensus on a "normal" LV GLS value in the setting of MR.

4.3. Mitral Valve Replacement (MVR) and Repair (MVr)

In the mitral position, prosthetic valves may also be surgically or percutaneously placed. Surgical mitral valve replacements (SMVR) include mechanical valves such as the bileaflet tilting disk, single tilting disk and ball-cage valves, and bioprosthetic valves which include stented and stentless prosthesis [85]. There are numerous transcatheter MVRs (TMVR) now available [115]. Valve-in-valve (ViV) and valve-in-ring (ViR) procedures are being performed where a transcatheter valve replacement is implanted within a degenerated bioprosthetic MVR or failed annuloplasty ring [116]. Both surgical (SMVr) and transcatheter mitral valve repair (TMVr) options are also available for patients with MR. Surgical approaches include the Alfieri stitch, annuloplasty rings, leaflet resection, artificial chords, and papillary muscle shortening. TMVr interventions include the edge-to-edge leaflet repair, direct annuloplasty systems, and artificial chords [117]. In Australia, the most common SMVr techniques include annuloplasty rings, leaflet resection, and artificial chords, while the most common TMVr approach is the edge-to-edge leaflet repair using the MitraClip™ device.

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Table 8 Key echocardiographic parameters for assessment of a SMVR, TMVR, and ViV.

Clinical	Date of valve replacement Type and size of the prosthetic valve Normal Doppler echocardiographic values for the prosthetic valve (where available) Height, weight, body surface area (BSA) Symptoms and related clinical findings Heart rate and rhythm Blood pressure
2D	Opening and closing motion of the prosthesis (leaflets for bioprosthesis/TMVR/ViV and occluders for mechanical prostheses) Presence of prosthetic leaflet calcification or abnormal echodensity attached to various components of the prosthesis Sewing ring seating, stability, and integrity
Spectral Doppler	MVR peak E velocity MVR mean gradient (Heart rate noted) Pressure half-time (PHT)—not for EOA (MVA) calculation Doppler velocity index (DVI) Effective orifice area (EOA) by continuity EOA indexed for BSA (IEOA)
Colour Doppler	If MR present, determine: • Normal or pathological • Transvalvular or paravalvular • Severity (assess as for native MR)
Other imaging data	LV and RV size and systolic function LA size Co-existent valvular disease PASP (RVSP)
Previous post-operative studies, when available	Comparison of above parameters in suspected prosthetic valvular dysfunction

Abbreviations: SMVR, surgical mitral valve replacement; TMVR, transcatheter mitral valve replacement; ViV, valve-in-valve; MVR, mitral valve replacement; MVA, mitral valve area; MR, mitral regurgitation; LV, left ventricular; RV, right ventricular; LA, left atrium; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure; 2D, two-dimensional.

All these procedures are expected to inherently create at least a mild degree of functional MS.

4.3.1. Recommendations for the performance of TTE post-MVR/MVr [3,22,46,47,81,85,86]

1. For baseline haemodynamic assessment ("fingerprint"):
   a. Surgical mitral valve replacement (SMVR)—4–6 weeks post-surgery.
   b. Transcatheter mitral valve replacement (TMVR)—pre-discharge.
   c. Surgical mitral valve repair (SMVr)—6 weeks–3 months post-surgery.
   d. Tricuspid mitral valve repair (TMVr)—pre-discharge.
2. Repeat TTE for routine surveillance in asymptomatic patients:
   a. Annual assessment beginning 5 years after implantation for bioprosthetic SMVR.
   b. Serial TTE assessment at 30 days and 1-year post-procedure, and then annually following TMVR.
   c. 3-yearly routine surveillance following SMVr.
   d. Serial TTE assessment initially at 1, 6, and 12 months, and then annually to 5 years following TMVr.
3. Repeat TTE is recommended with any new or changed clinical symptoms or suspected MVR/MVr dysfunction or complication.

Table 9 Key echocardiographic parameters for assessment of a MVr.

Clinical	Date of valve repair Type of repair+/-ring size Height, weight, body surface area (BSA) Symptoms and related clinical findings Heart rate and rhythm Blood pressure
2D	Thickness and mobility of mitral valve (MV) leaflets Presence of leaflet calcification or abnormal echodensity attached to valve ring (if relevant) Annular ring stability (if relevant) Presence or absence of systolic anterior motion of the anterior mitral valve leaflet
Spectral Doppler	MV peak E velocity MV mean gradient (Heart rate noted) PHT (not for EOA (MVA) calculation) Doppler velocity index (DVI) Effective orifice area (EOA) by continuity EOA indexed for BSA (IEOA)
Colour Doppler	Degree of MR (assess as for native MR)
Other imaging data	LV and RV size and systolic function LA size Co-existent valvular disease CWD of MR jet (if MR present) Pulmonary venous flow (if MR present) PASP (RVSP)

Abbreviations: MVr, mitral valve repair; PHT, pressure half-time; MVA, mitral valve area; LV, left ventricular; RV, right ventricular; LA, left atrial; CWD, continuous-wave Doppler; MR, mitral regurgitation; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure; 2D, two-dimensional.

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Table 10 Key echocardiographic parameters for assessment of transcatheter mitral edge-to-edge repair.

Clinical	Date of valve procedure
	Number of clips
	Height, weight, body surface area (BSA)
	Symptoms and related clinical findings
	Heart rate and rhythm
	Blood pressure
2D	Stability of clip(s)
	2D planimetry of mitral valve (MV) orifices
Spectral Doppler	MV peak E velocity
	MV mean gradient (Heart rate noted)
	PHT (not for MVA calculation)
Colour Doppler	Degree of MR by:
	• Jet area (with caution, multiple jets lead to overestimation of severity)
	• Vena contracta width (VCW)
	• FCR and corresponding Nyquist velocity
Other imaging data	LV and RV size and systolic function
	LA size
	Co-existent valvular disease
	CWD of MR jet
	Pulmonary venous flow
	Degree of shunting across the iatrogenic ASD
	PASP (RVSP)

Abbreviations: 2D, two-dimensional; PHT, pressure half-time; MVA, mitral valve area; MR, mitral regurgitation; FCR, flow convergence radius; LV, left ventricular; RV, right ventricular; LA, left atrial; CWD, continuous-wave Doppler; ASD, atrial septal defect; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure.

4. Repeat TTE is recommended for known MVR/MVr dysfunction when it would change management or guide therapy.

As previously described for AVRs, an initial TTE is also indicated within 4—6 weeks following SMVR for a baseline haemodynamic evaluation [46,47,85,86]. The frequency of serial TTE examinations is dependent upon the prosthesis type and the clinical status of the patient, with some variation between current AHA/ACC and ESC guidelines. Annual routine TTE is reasonable in patients with a bioprosthetic SMVR after the 5 year mark, even in the absence of a change in clinical status, due to the risk of SVD [3,22,46,47]. Earlier evaluation is also similarly indicated in selected patients at increased risk of early bioprosthetic MVR degeneration, including those with renal impairment, diabetes mellitus, abnormal calcium metabolism, systemic inflammatory disease, and in patients <60 years of age [22]. Routine annual TTE is not recommended for mechanical MVRs in the absence of symptoms or signs of valve dysfunction, if the initial post-operative baseline study was normal [22,44]. However, many of these patients may require ongoing clinical and TTE follow-up for other indications, such as residual LV systolic dysfunction, PHTN, aortic pathology, or concurrent valvular disease. Asymptomatic patients with prosthetic valve regurgitation or stenosis will also require more frequent follow-up to monitor for evidence of progressive LV dilatation and/or systolic dysfunction [22]. For patients with TMVRs, baseline TTE is recommended pre-discharge, at 30 days and 1-year post-implantation, and then annually thereafter [47].

For SMVr, an initial baseline TTE assessment should be performed 6 weeks to 3 months postoperatively [1]. Repeat TTE is appropriate for suspected MVr dysfunction at any stage but routine re-evaluation in patients without concerns should only be undertaken at an interval of ≥3 years. For TMVr, TTE for the re-assessment of MR severity and LV function is recommended pre-discharge, at 1, 6, and 12 months, and then annually to 5 years [2,81].

4.3.2. Requirements for comprehensive TTE assessment of MVRs and MVrs

A comprehensive overview of the echocardiographic assessment of MVR and MVr is beyond the scope of this document. Guideline recommendations have been provided by the ASE and EACVI detailing requirements for imaging, evaluation for complications, and grading of regurgitation and stenosis [81,85,86]. The key TTE parameters are briefly described and summarised in Tables 8, 9, and 10.

Mitral valve replacements (MVR). Comprehensive TTE assessment of the MVR also involves the use of multiple on- and off-axis views to evaluate the structural appearance of the prosthesis (leaflet or occluder morphology/mobility), seating/stability/integrity of the sewing ring, to identify the presence of calcification or abnormal structures on any of the various components of the MVR, and to determine the presence/type/ location/severity of MR.

The haemodynamic assessment of an MVR includes measurements of the peak E velocity, mPG, PHT, calculation of the EOA/IEOA, and DVI (Figure H). As with MS, heart rate reporting is essential as the mPG is dependent on the diastolic filling period. While the PHT is measured for MVRs, it cannot be used to estimate the EOA. Instead, the EOA is calculated via the continuity equation which assumes that the SV across the MVR and through the LVOT is the same (EOA can only be estimated when the degree of MR and/or AR are ≤1/4). Normal values for a MVR include a peak E velocity <1.9m/s, mPG <5mmHg, PHT <130ms, EOA ≥2.0cm^2, and DVI <2.2. The comparison of haemodynamic parameters with the baseline TTE or serial post-operative studies is important, especially if there is a suspicion of MVR dysfunction (comparison of the flow-

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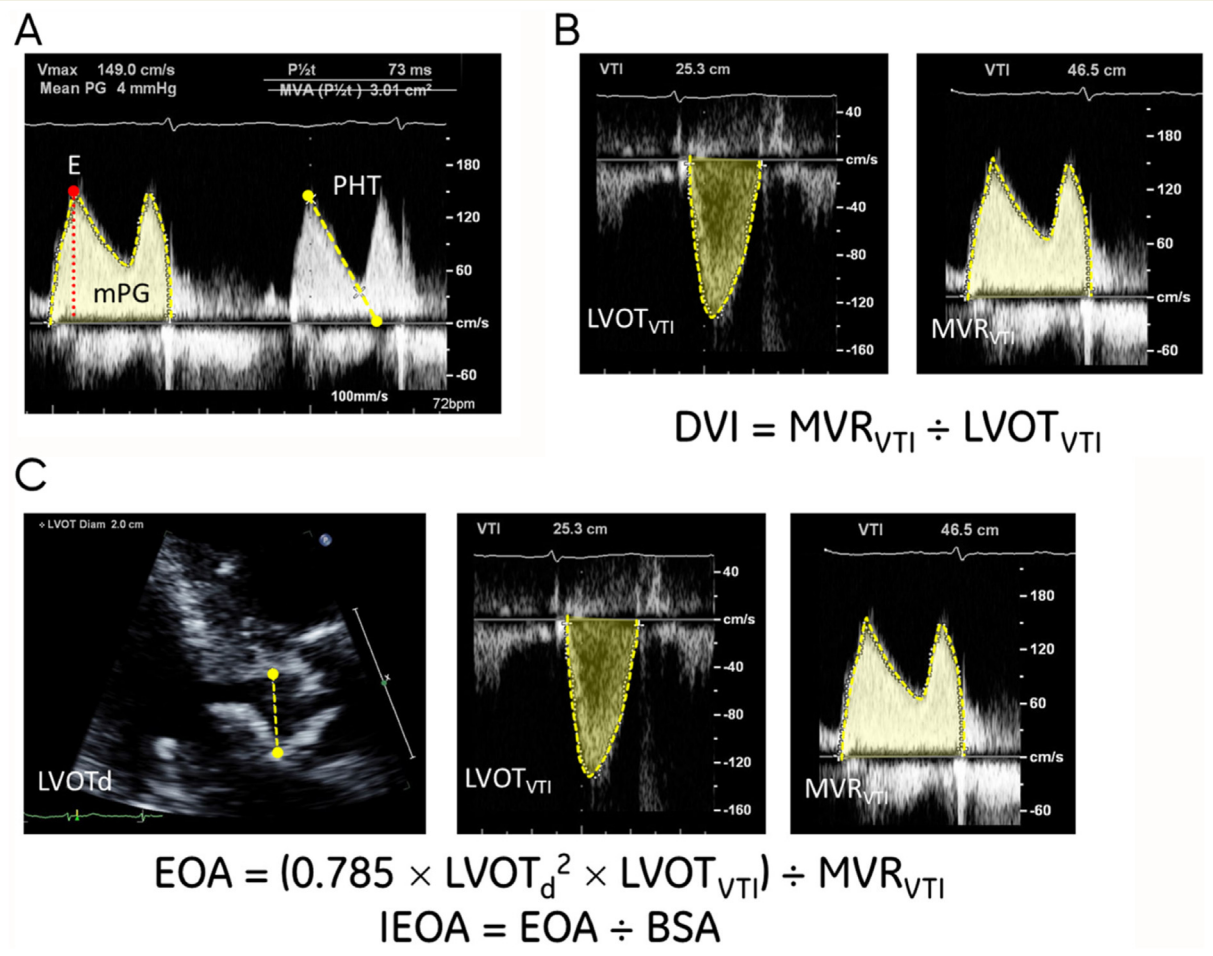

Figure H Haemodynamic assessment of a MVR. A. From the continuous-wave Doppler profile across the MVR, the peak E velocity, mean pressure gradient (mPG) and pressure half-time (PHT) are measured. Note that the PHT cannot be used to estimate the EOA (MVA) in MVRs. B. The Doppler velocity index (DVI) is derived from the MVR velocity time integral (MVR_{VTI}) and the LVOT velocity time integral ($LVOT_{VTI}$). C. The effective orifice area (EOA) is derived from the LVOT diameter (LVOTd), $LVOT_{VTI}$, and the MVR_{VTI}, and the indexed EOA (IEOA) is derived from the EOA and the body surface area (BSA). Abbreviations: MVR, mitral valve replacement; LVOT, left ventricular outflow tract; MVA, mitral valve area.

independent parameters such as the EOA, IEOA, or DVI would be most useful). The degree of pathologic MVR regurgitation should be assessed in a similar manner as for native valves, using the previously described integrated approach.

Mitral valve repair (MVr). A MVr should be assessed in a similar manner as for MVRs. Haemodynamic parameters include the peak transmitral velocities, mPG, PHT, and the EOA. As for MVRs, the PHT cannot be used to estimate the EOA, and the EOA calculation via the continuity equation is only valid when MR and/or AR are graded ≤1/4. Residual MR should be assessed in a similar manner as for native valves using the previously described integrated approach.

TMVr should be assessed in a similar manner as for SMVr but with a special focus on the evaluation of residual or recurrent MR. An integrated approach as for native MR is recommended [81]. Quantification of MR is difficult, as there are now two (or more) orifices, such that mitral annular stroke volume cannot be accurately obtained. Additionally, the PISA approach has not been validated for this double orifice geometry or for multiple MR jets which often exist. In addition to the standard haemodynamic assessment, 2D and/or 3D planimetry of each MV orifice may be performed to determine the degree of post-procedure stenosis. Furthermore, as the transcatheter edge-to-edge repair procedure usually includes a trans-septal puncture of the interatrial septum, an iatrogenic atrial septal defect (ASD) is created. Therefore, the degree of left-to-right shunting across this ASD should be evaluated by CDI. A significant shunt across the ASD may lead to PHTN. Consequently, right-sided chamber size/systolic function and pulmonary pressures should also be assessed.

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5. Right-Sided Valvular Heart Diseases

5.1. Tricuspid and Pulmonary Valve Disease

Unlike their left heart counterparts, the right-sided tricuspid and pulmonary valves are often overlooked. TR is an important prognostic factor, independent of LVEF and PHTN, in many disease processes. Tricuspid stenosis (TS) is also a significant cause of morbidity. The pulmonary valve, previously regarded as the least important intracardiac valve, has long been ignored in the adult population, as pulmonary valvular pathology was believed to have little impact on patient morbidity and mortality. However, this is clearly not the case, particularly in the subset of patients with congenital heart disease.

Right-sided valvular heart disease often has subtle clinical findings. As such, imaging is generally required for diagnosis and should be considered whenever a clinical suspicion arises. In particular, any clinical features of RHF should prompt TTE examination for possible valvular heart disease, if not previously performed.

5.1.1. Tricuspid valve disease

Tricuspid stenosis (TS) is rare in developed countries as rheumatic heart disease accounts for ~90% of all cases. In patients with rheumatic mitral valve disease, approximately 5% will develop concomitant tricuspid valve involvement. Other aetiologies of TS include carcinoid heart disease, congenital anomalies, endomyocardial fibrosis, and device lead-induced adhesions. RA tumours may also result in functional TS.

Tricuspid regurgitation (TR) is very common and may affect 65%–85% of the population [118]. Whilst mild TR is frequent and usually benign, moderate or severe TR can result in irreversible myocardial damage and adverse outcomes [119]. The tricuspid valve is a complex structure with an elliptical non-planar annulus and chordal insertions into both the papillary muscles as well as the RV walls. It has three leaflets—anterior (largest), posterior (multiple scallops), and septal (smallest)— within a partial fibrous annulus [120,121]. Primary (organic) TR implies pathology of the leaflets and accounts for 15% of TR cases.

Causes of primary TR may be degenerative (prolapse or flail), congenital (e.g., Ebstein's anomaly, tricuspid dysplasia or hypoplasia, clefts, or double orifice valves), or acquired (e.g., endocarditis, carcinoid or rheumatic heart disease, leaflet prolapse, tumour, trauma, or iatrogenic [radiation, drugs, or RV biopsy]) [68,70,122,123].

Secondary (functional) TR is more common (80% of TR cases), as changes affecting RV morphology and/or the tricuspid annular geometry will result in malcoaptation of the otherwise normal leaflets. The ellipsoid shape of the TV becomes flatter and more circular as it dilates. Causes of secondary TR include RV pressure and/or volume overload states resulting in RV dilatation (left-sided valvular heart disease, primary/secondary PHTN, pulmonary regurgitation [PR], or an ASD), myocardial disease (RV infarction, cardiomyopathies, AC), endomyocardial fibrosis, or tricuspid annular dilatation due to RA enlargement and dysfunction from chronic AF (idiopathic or atrial functional TR). Pacemaker or defibrillator lead-related TR (5% of TR cases) may also be primary (leaflet fibrosis/laceration/perforation/impingement/avulsion or chordal entanglement/rupture) or secondary (RV dyssynchrony/ remodelling due to pacing) [70,123].

Primary TR imparts pure volume overload on an initially normal right heart [119]. Conversely, RV enlargement is the major cause of ventricular secondary TR through leaflet tenting and annular dilatation [124]. Notably, as the RV is more load sensitive than the LV, initial pressure loading can rapidly translate into progressive RV dilatation with secondary TR, then resulting in a mixed pressure and volume overload state. Ultimately, TR begets more TR as a consequence of progressive RV dilatation, further increasing leaflet

Table 11 Key echocardiographic parameters for assessment of TS [44,47,94].

Clinical	Height, weight, body surface area (BSA)
	Heart rate and rhythm
	Blood pressure
2D/M-mode	Tricuspid valve (TV) morphology (leaflets and supporting apparatus)
	RA size
	IVC size and respiratory variation
	RV dimensions
	RV systolic function parameters (RV TAPSE, RV FAC)
Spectral Doppler	TV area (TVA; pressure half-time or continuity)
	TV mean gradient (Heart rate noted)
	TV inflow velocity and VTI
	RV systolic function parameters (RV S′)
Colour Doppler	TR severity
Other imaging data	Co-existent valvular disease (e.g. suggesting possible rheumatic involvement or carcinoid disease)
Advanced (if available and technically feasible)	3D TVA

Abbreviations: RA, right atrial; RV, right ventricular; IVC, inferior vena cava; TAPSE, tricuspid annular plane systolic excursion; FAC, fractional area changes; VTI, velocity time integral; RV S′, tissue Doppler-derived tricuspid lateral annular systolic velocity; TR, tricuspid regurgitation; 3D, three-dimensional.

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Table 12 Key echocardiographic parameters for assessment of chronic TR [46,47,68].

2D/M-mode	Tricuspid valve morphology (leaflets, annulus, and supporting apparatus)
	RV dimensions
	RV systolic function parameters (TAPSE, FAC)
	RA size
	IVC size and collapsibility
Spectral Doppler	Transtricuspid inflow
	Hepatic vein flow
	TR signal density and shape
	RV systolic function parameters (RV S′)
	PASP (RVSP)
Colour Doppler	TR jet area
	Vena contracta width (VCW)
	FCR and corresponding Nyquist velocity
Quantification (as required/feasible)	Regurgitant volume (RVol)
	Regurgitant fraction (RF)
	Effective regurgitant orifice area (EROA)
Advanced (if available and technically feasible)	3D vena contracta area (VCA)
	RV free wall longitudinal strain
	3D RVEF
Co-existent left heart disease or causes of RV volume overload	LV size and function
	Left-sided valvular function
	Pulmonary regurgitation (PR)
	Atrial septal defect (ASD)

Abbreviations: TR, tricuspid regurgitation; 3D, three-dimensional; RA, right atrial; RV, right ventricular; RVEF, right ventricular ejection fraction; LV, left ventricular; TAPSE, tricuspid annular plane systolic excursion; RV S′, tissue Doppler-derived tricuspid lateral annular systolic velocity; IVC, inferior vena cava; FCR, flow convergence radius; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure.

tenting and degree of regurgitation [125]. Increasing severity of TR is associated with worsening heart failure and reduced survival.

5.1.2. Pulmonary valve disease

Pulmonary stenosis (PS) can occur at many anatomic levels including sub-valvular, valvular (commonest), and supra-valvular locations. At least 95% of PS cases are related to congenital heart disease or genetic disorders (e.g., Tetralogy of Fallot [ToF], double outlet RV, Noonan's and Williams syndromes). Acquired causes of PS such as carcinoid and rheumatic heart disease are uncommon and generally have concomitant involvement of other valves. PS is generally diagnosed in a younger population and progression is usually slow with an overall survival of 96% at 25 years [126].

Table 13 Key echocardiographic parameters for assessment of PS [94,130].

Clinical	Height, weight, body surface area (BSA)
	Heart rate and rhythm
	Blood pressure
2D/M-mode	Pulmonic valve (PV) morphology
	RVOT and main and branch pulmonary arteries (assessment for sub/supravalvular stenosis)
	RV dimensions (including RVH)
	RV systolic function parameters (TAPSE, FAC)
	RA size
Spectral Doppler	PV peak velocity (PV V_{max})
	PV peak and mean gradients
	RVOT velocity
	PASP
	RV systolic function parameters (RV S′)
Colour Doppler	Pulmonary regurgitation (PR) severity
	Tricuspid regurgitation (TR) severity
Other imaging data	Co-existent valvular disease (e.g., suggesting possible carcinoid or rheumatic disease)
	Other features of underlying congenital heart disease (e.g., ToF, ventricular septal defect)

Abbreviations: PS, pulmonary stenosis; RVOT, right ventricular outflow tract; RV, right ventricular; RVH, right ventricular hypertrophy; TAPSE, tricuspid annular plane systolic excursion; FAC, fractional area change; RA, right atrial; PASP, pulmonary artery systolic pressure; RV S′, tissue Doppler-derived tricuspid lateral annular systolic velocity; ToF, tetralogy of Fallot.

Pulmonary regurgitation (PR) is the most important residual lesion after initial surgical correction of ToF or surgical valvotomy for PS. Other causes of PR include rheumatic or carcinoid heart disease, endocarditis, PHTN, pulmonary artery/annular dilatation or trauma [127]. Significant PR over time results in RV dilatation, volume overload, systolic dysfunction, arrhythmias, and sudden death.

5.2. Recommendations for the Performance of TTE for Assessment of Right-Sided Valvular Heart Disease [46]

1. To establish the diagnosis in patients with symptoms or signs of right-sided valvular heart disease or heart failure (to determine aetiology, severity, haemodynamic consequences [including RV size and systolic function, RA and IVC size, right-sided filling and pulmonary pressures] and assessment of any associated left heart disease).
2. Re-evaluation of known TR or TS in patients with a significant change in clinical status.

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Table 14 Key echocardiographic parameters for assessment of chronic PR [68].

Clinical	Height, weight, body surface area (BSA)
	Heart rate and rhythm
	Blood pressure
2D/M-mode	Pulmonic valve morphology (leaflets)
	Pulmonic annulus and main and branch pulmonary arteries
	RVOT
	RV and RA size
	RV systolic function parameters (TAPSE, FAC)
Spectral Doppler	PR signal density
	PR deceleration and/or pressure half-time
	Diastolic flow reversal (main and branch pulmonary arteries)
	PASP (RVSP)
	RV systolic function parameters (RV S′)
Colour Doppler	PR jet length and width in the RVOT
Advanced (if available and technically feasible)	RV free wall longitudinal strain
	3D RVEF
Other imaging data	Co-existent valvular disease (e.g., suggesting possible carcinoid disease)
	Other features of underlying congenital heart disease (e.g., repaired ToF)

Abbreviations: PR, pulmonary regurgitation; RVOT, right ventricular outflow tract; RV, right ventricular; RA, right atrial; TAPSE, tricuspid annular plane systolic excursion; FAC, fractional area change; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure; RV S′, tissue Doppler-derived tricuspid lateral annular systolic velocity; RVEF, right ventricular ejection fraction; 3D, three-dimensional; ToF, tetralogy of Fallot.

3. Re-evaluation of known PR or PS in patients with a significant change in clinical status.

5.3. Recommendations for Serial TTE Assessment of Right-Sided Valvular Heart Disease [2]

1. In patients with risk factors for developing right-sided valvular heart disease (e.g., previous corrective surgery for congenital heart disease or rheumatic heart disease), routine surveillance TTE every 3–5 years is recommended.
2. In patients with known right-sided valvular heart disease, repeat TTE should be performed when there are new or changed symptoms or physical examination findings.
3. In patients who are asymptomatic or have stable symptoms, the recommended TTE surveillance intervals for mild, moderate, and severe valvular heart disease are every 3–5 years, 1–2 years, and 6–12 months, respectively.

5.4. Requirements for Comprehensive TTE Assessment of Right-Sided Valvular Heart Disease

TTE plays a pivotal role in the initial diagnosis and serial monitoring of right-sided valvular heart disease, providing insights into both aetiology and potential interventional options. Evaluation of lesion severity mirrors the same approaches as for left-sided valvular stenosis or regurgitation as previously discussed. Key TTE parameters for the assessment of chronic TS and TR are summarised in Tables 11 and 12 respectively.

TTE parameters for grading severity of TS are less well-validated than for MS. In addition to the 2D anatomical appearances of valve and/or chordal thickening, spectral Doppler features specific for haemodynamically significant TS include a tricuspid mPG ≥5mmHg, tricuspid inflow VTI >60cm, PHT ≥190msec, and tricuspid valve area (TVA; continuity equation) ≤1.0cm^2. RA and IVC dilatation would also support severe TS [44,47,94].

The haemodynamic severity of TR is determined via an integrated approach, including RV and RA chamber sizes, and qualitative, semiquantitative, and quantitative Doppler assessments [68,70,128]. Specific criteria for severe TR include significant leaflet abnormalities (e.g., flail), annular dilatation with resultant leaflet malcoaptation, large TR jet area occupying >50% of the RA, VCW ≥0.7cm, FCR >0.9cm at a colour Nyquist limit of 30–40cm/s, dense triangular CWD profile, hepatic venous systolic flow reversal, and RV dilatation [68]. A transtricuspid inflow PWD E-velocity >1m/s in the absence of TS is also suggestive of severe TR [70]. Similar to MR, quantification (by the PISA method) is recommended where possible with cut-offs of EROA >0.4cm^2 and RVol ≥45mL defining the conventional threshold of severe TR. However, with evolution of transcatheter tricuspid therapies, there has been a proposed extension of the grading of "severe" to account for relative reductions in the degree of TR and allowing for comparison of clinical outcomes, despite quantitative parameters of the residual TR post-intervention still remaining above the traditional severe cut-offs in some patients [47,122,128,129]. With no change to the mild and moderate TR criteria, the new suggested grades of severe/massive/torrential are characterised by VCWs of 0.7–1.3/1.4–2.0/≥2.1cm, EROAs of 0.40–0.59/0.60–0.79/≥0.80cm^2, RVols of 45–59/60–74/≥75mL, and 3D VCAs of 0.75–0.94/0.95–1.14/≥1.15cm^2, respectively. TTE assessment of TR must also include a

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Table 15 Key echocardiographic parameters for assessment of a TVR [86].

Clinical	Date of valve replacement Type and size of the prosthetic valve Height, weight, body surface area (BSA) Symptoms and related clinical findings Heart rate and rhythm Blood pressure
2D	Opening and closing motion of the prosthesis (leaflets for bioprosthesis and occluders for mechanical prostheses) Presence of leaflet calcification or abnormal echodensity attached to various components of the prosthesis Sewing ring seating, stability, and integrity
Spectral Doppler	TVR peak E velocity TVR mean gradient (Heart rate noted) PHT (not for TVA calculation) TVR velocity time integral (VTI) Doppler velocity index (DVI) Effective orifice area (EOA) (no validated cut-off values)
Colour Doppler	If TR present, determine: • Normal or pathological • Transvalvular or paravalvular • Severity (assess as for native TR)
Other imaging data	RV size and systolic function RA size IVC size and inspiratory variation Hepatic venous flow pattern Co-existent valvular disease PASP (RVSP)
Previous post-operative studies, when available	Comparison of above parameters in suspected prosthetic valvular dysfunction

Abbreviations: TVR, tricuspid valve replacement; PHT, pressure half-time; TVA, tricuspid valve area; TR, tricuspid regurgitation; RV, right ventricular; RA, right atrial; IVC, inferior vena cava; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure.

Table 16 Key echocardiographic parameters for assessment of a PVR [86].

Clinical	Date of valve replacement Type and size of the prosthetic valve Height, weight, body surface area (BSA) Symptoms and related clinical findings Heart rate and rhythm Blood pressure
2D	Opening and closing motion of the prosthesis (leaflets for bioprosthesis and occluders for mechanical prostheses) Presence of leaflet calcification or abnormal echodensity attached to various components of the prosthesis Sewing ring seating, stability, and integrity Pulmonary conduit appearance
Spectral Doppler	PVR peak velocity PVR peak and mean gradients RVOT velocity and VTI
Colour Doppler	If PR present, determine: • Normal or pathological • Transvalvular or paravalvular • Severity (assess as for native PR)
Other imaging data	RV size, systolic function, and hypertrophy Pulmonary artery dimensions Co-existent valvular disease RVSP
Previous post-operative studies, when available	Comparison of above parameters in suspected prosthetic valvular dysfunction

Abbreviations: PVR, pulmonary valve replacement; 2D, two-dimensional; RVOT, right ventricular outflow tract; VTI, velocity time integral; PR, pulmonary regurgitation; RV, right ventricular; RVSP, right ventricular systolic pressure.

comprehensive evaluation of RV systolic function (TAPSE, FAC, RV S′, and RV free wall longitudinal strain and 3D RVEF, where possible).

Key TTE parameters for the assessment of chronic PS and PR are summarised in Tables 13 and 14, respectively.

TTE plays an important role to detect the anatomic level of PS, establishing the aetiology, quantifying stenosis severity, and guiding management. Features of severe PS on TTE may include thickened distorted pulmonic valve (PV) leaflets and/or sub/supra-valvular abnormalities, in association with a peak velocity (PV V_{max}) >4m/s, corresponding peak gradient >64mmHg, and mean gradient >35mmHg [94,130]. There may be evidence of RV hypertrophy, RV/RA enlargement, and potentially post-stenotic dilatation of the main pulmonary artery [44].

Pulmonary regurgitation (PR) is commonly a latent insidious consequence of previous intervention for congenital heart disease including ToF repairs. Semiquantitative and qualitative markers of severe PR include a broad diastolic colour jet in the RVOT, ratio of the jet width : PV annulus

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diameter ≥0.7, dense CWD spectral intensity with a steep deceleration slope and early termination in diastole (correlating with a short deceleration time of <260ms or PHT <100ms), flow reversal in the branch pulmonary arteries on PWD interrogation, and associated RV dilatation with features of volume overload [68,70]. A PR index (ratio of CWD PR duration : total diastolic time) of <0.77 is also supportive of, but not specific for, severe PR. Of note, the regurgitant jet of severe PR, particularly in the absence of elevated pulmonary pressures, may not be well-visualised on CDI as it may be of low velocity, laminar rather than turbulent flow, and brief as a result of rapid pressure equalisation. As for chronic TR, TTE assessment of PR must also include a comprehensive evaluation of RV size and systolic function as previously described.

5.5. Right-Sided Heart Valve Replacement and/or Repair

5.5.1. Tricuspid valve replacement (TVR) and repair (TVr)

In parallel with the mitral valve, tricuspid valve interventions (TVI) also include surgical TVR (STVR) or TVr (STVr), and a myriad of evolving transcatheter approaches including transcatheter TVR (TTVR) and TVr (TTVr) techniques (e.g., edge-to-edge clips, spacer devices, annular reduction, and caval valve implantation).

As with all other valve replacements or repairs, an initial TTE would be indicated for a baseline "fingerprint" haemodynamic assessment post-STVR or STVr. The British Heart Valve Society and British Society of Echocardiography (BSE) guidelines recommend routine serial TTE assessments on an annual basis, beginning 5 years after implantation of a bioprosthetic STVR [77]. No routine TTE follow-up is required for mechanical TVRs if the initial baseline study was normal and in the absence of an alternate indication for ongoing imaging surveillance (e.g. new or persistent RV systolic dysfunction, PHTN, or other concurrent valvular disease). No specific guidelines exist for routine TTE surveillance post-STVr although adopting a similar approach to the post-SMVr patient would not be unreasonable (i.e., ≥3 years). Following transcatheter TVIs, the timing of the baseline and follow-up TTEs would be guided by the clinical trial or registry requirements. A repeat TTE would be required for re-assessment at any time if there is any change in the patient's clinical status or suspected TVR/TVr dysfunction or complications.

A comprehensive review of echocardiographic prosthetic TVR evaluation is beyond the scope of this document but has been detailed in the relevant societal guidelines [85,86]. Key TTE parameters for assessment of TVRs are summarised in Table 15.

5.5.2. Pulmonary valve replacement (PVR)

Surgical or transcatheter PVRs are most commonly performed in patients with congenital heart disease for the treatment of PR (or PS not suitable for percutaneous valvuloplasty or surgical repair). As with all other valvular interventions, an initial TTE would be indicated for a baseline haemodynamic assessment, but follow-up studies would be based on clinician/institutional practices and guided by prosthesis and/or patient factors (e.g., underlying congenital heart disease necessitating ongoing TTE follow-up).

A comprehensive review of echocardiographic prosthetic PVR evaluation is beyond the scope of this document but has been detailed in the relevant societal guidelines [85,86]. Key TTE parameters for assessment of PVRs are summarised in Table 16.

6. Pericardial Diseases

Pericardial diseases are part of the differential diagnosis in patients with a variety of symptoms ranging from non-specific dyspnoea or chest pain to more sinister presentations with heart failure or hypotension. Pericardial diseases are increasingly more prevalent, not only related to infective pathogens, but also attributable to rising numbers of surgical and medical therapeutic interventions [131]. They are commonly encountered in various clinical settings and may result in significant morbidity as well as increased mortality [132].

6.1. Recommendations for Performance of TTE for Assessment of Pericardial Diseases [133]

1. Assessment of non-specific dyspnoea, chest pain, heart failure, or unexplained hypotension where pericardial disease is the suspected culprit.
2. Acute or recurrent pericarditis (to assess for a pericardial effusion, tamponade physiology, and myocardial involvement as in myopericarditis).
3. Incidental moderate–large pericardial effusion identified on other imaging (to assess possible aetiology, extent, and haemodynamic significance of the pericardial effusion).
4. Clinically suspected cardiac tamponade or post-pericardiotomy syndrome.
5. Clinically suspected constrictive pericarditis based on symptoms and patient's background history.
6. Suspected pericardial tumour based on clinical symptoms or other imaging.
7. Evaluation (and surveillance) of pericardial cyst identified on previous imaging.
8. Evaluation of congenital absence of the pericardium suspected clinically or identified on other imaging.
9. Procedural guidance for diagnostic or therapeutic pericardiocentesis.

Although multimodality imaging of the pericardium is often required in complex cases, TTE remains the initial investigation of choice due to its ease of use, widespread availability, diagnostic accuracy, and cost-effectiveness [134]. Ideally imaging should be preceded by a comprehensive history, physical examination, and basic

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Table 17 Key echocardiographic parameters for assessment of pericardial diseases.

2D assessment of pericardial effusion, thickness, and features of raised intrapericardial pressures	Pericardial effusion size, location, character (free flowing vs organising), and suitability for pericardiocentesis if indicated Collapse of right-sided chambers, IVC plethora, pleural effusion(s), ascites
2D assessment of enhanced ventricular interdependence	Early diastolic septal bounce, respiratory shift of the interventricular septum (requires acquisition over multiple respiratory cycles) Peri-myocardial tethering
M-mode assessment of enhanced ventricular interdependence	Early diastolic septal bounce, respiratory shift of the interventricular septum (requires acquisition over multiple respiratory cycles) Respiratory variation of ventricular size (requires acquisition over multiple respiratory cycles) Flattening of the posterior wall during diastole
Spectral Doppler assessment of exaggerated respiratory variation in diastolic filling	Reciprocal respiratory changes of mitral and tricuspid inflow velocities Reciprocal respiratory changes of diastolic forward flow velocities and end-diastolic flow reversal in the hepatic veins
Tissue Doppler and 2D speckle tracking	Tissue Doppler derived early diastolic velocities at the medial and lateral mitral annulus (e.g., evidence of annulus reversus and paradoxus) Strain imaging by 2D STE where possible

Abbreviations: M-mode, motion-mode; 2D, two-dimensional; IVC, inferior vena cava; STE, speckle-tracking echocardiography.

investigations such as an ECG and a chest x-ray. This systematic approach will avoid unnecessary testing and associated excessive costs.

6.2. Recommendations for Serial TTE Assessment of Pericardial Disease

Patients with pericardial diseases often require serial TTE assessments to guide their ongoing management. The need for a repeat study is predominantly driven by the patient's symptoms or treating physician's clinical discretion.

Indications for repeat TTE assessment of pericardial diseases [135,136]

1. In patients with a "concerning" non-physiological pericardial effusion, repeat TTE may be performed in 1–2 weeks, after 1 month, and at 6 months, to monitor for worsening of the effusion or development of tamponade physiology or constrictive pericarditis.
2. In patients with stable idiopathic "moderate" pericardial effusions, repeat TTEs may be performed every 6 months.
3. In patients with stable severe or large pericardial effusions, repeat TTEs every 3–6 months is reasonable.
4. In patients with a worsening pericardial effusion or symptoms, repeat TTE may be performed at a shorter time interval at the discretion of the treating physician.
5. In patients with transient constriction or effusive–constrictive pericarditis under medical therapy, a repeat TTE may be performed within 3 months to assess the efficacy of treatment.
6. In patients post-pericardiocentesis, repeat TTE is recommended immediately if there are ongoing symptoms or a large amount of fluid was drained, or within 24–48 hours for re-assessment of residual effusion and its haemodynamic effect. Timing and frequency of subsequent TTE assessments will depend on the patient's clinical status.
7. In patients post-pericardiectomy, repeat TTE is recommended within 24–48 hours for haemodynamic assessment and to identify any immediate post-operative complications. Timing and frequency of subsequent TTE assessments to evaluate the efficacy of surgery (pericardial thickening or recurrence of constrictive physiology) and monitor for changes in cardiac function will depend on the patient's clinical status as determined by the treating physician.

Ongoing follow-up imaging of a pericardial effusion will depend on the underlying aetiology and size. Moderate–large effusions and effusions related to bacterial infection, post-radiation pericardial diseases, and pericardial injury syndromes have an increased risk of developing complications (e.g., early risk of cardiac tamponade or recurrence and later complications of constrictive pericarditis) [135]. Uncomplicated acute pericarditis and idiopathic mild–moderate pericardial effusions have a very low likelihood of complications. Risk of constrictive pericarditis resulting from idiopathic pericarditis is also very low [137].

6.3. Requirements for Comprehensive TTE Assessment of Pericardial Diseases

Assessment of pericardial diseases (acute pericarditis, constrictive pericarditis, and pericardial effusion/tamponade) requires comprehensive evaluation of both anatomy and physiology, as outlined in the 2013 ASE Clinical Recommendations for Multimodality Cardiovascular Imaging of Patients with Pericardial Disease [134]. Key TTE parameters

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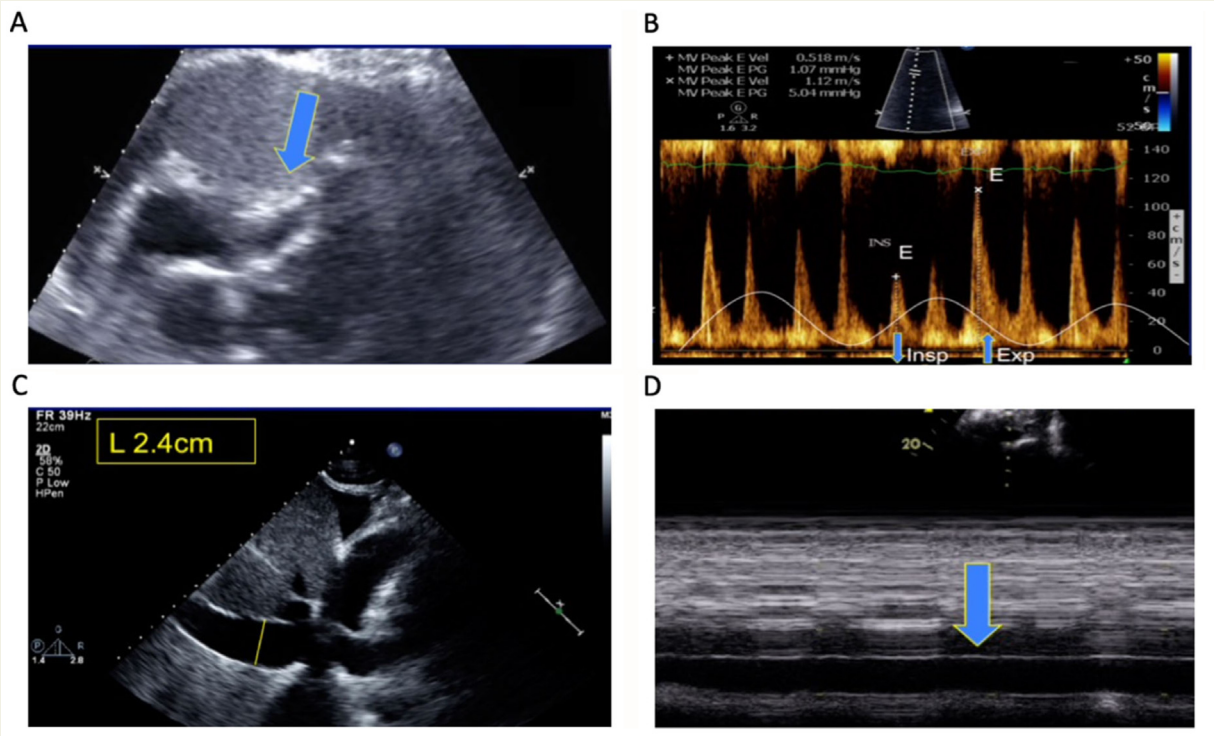

Figure I Key echocardiographic findings in cardiac tamponade. A. Subcostal TTE image of a large pericardial effusion with diastolic collapse of the RV (arrow). B. Pulsed-wave Doppler assessment of the mitral inflow with a respirometer demonstrating significant (>25%) respirophasic velocity variation (Mitral inflow velocity decreases during inspiration [Insp] and increases with expiration [Exp]). C: Subcostal TTE image depicting a dilated IVC. D: M-mode echocardiography demonstrating a dilated plethoric IVC (arrow) without respiratory size variation consistent with raised intrapericardial pressures. Abbreviations: TTE, transthoracic echocardiography; RV, right ventricle; IVC, inferior vena cava; M-mode, motion-mode.

for the assessment of pericardial disease are summarised in Table 17.

In addition to the general assessment of ventricular and valvular structure and function, the TTE examination in pericardial diseases must be targeted towards detailed assessment of the pericardium (effusion, thickening/stranding/tethering/calcification) and any associated features of raised intrapericardial pressures from the effusion (chamber compression) or enhanced ventricular interdependence that can be seen in both tamponade and constrictive pericarditis, resulting in abnormal respirophasic variation in ventricular filling (Figure I) [134]. All available echocardiographic tools including 2D imaging, M-mode, tissue Doppler, and 2D STE should be used to complete the evaluation [138]. Simultaneous recording of ventricular filling and central venous flows with a respirometer (slow sweep speed between 25-50mm/s) should be used for precise assessment of respirophasic variation [134]. 2D and/or M-mode assessment of the IVC (size and collapsibility) and PWD of the hepatic vein flows are also important [134,139].

2D STE strain imaging has been proposed as an additional tool to differentiate constrictive pericarditis from a restrictive cardiomyopathy. Contemporary work has shown regional differences in longitudinal strain in constriction (lower LV lateral wall strain values in comparison to the septum) with improvement in the LV/RV free wall longitudinal strain post-pericardiectomy [140]. Regional longitudinal and global circumferential strain is impaired in constrictive pericarditis whereas global longitudinal strain is reduced in restrictive cardiomyopathy [140,141].

6.4. Multimodality Imaging in Assessment of Pericardial Diseases

CMRI or CCT may provide complimentary information in the work-up of complex pericardial syndromes [142]. These modalities are able to assess the pericardial effusion, degree and extent of pericardial thickening, haemodynamic effects of the pericardial pathology, and offer additional incremental value for detection of pericardial inflammation and calcification (best appreciated by CCT). Positron emission tomography (PET) with ^{18}F-fluorodeoxyglucose (^{18}F-FDG) may also be useful for further characterisation of a pericardial mass when malignancy is suspected to be the underlying aetiology [143,144]. If complementary information is available from other imaging modalities such as CMRI, and confirms that the pericardial effusion is benign and physiological, frequent echocardiograms may be avoided in the absence of new symptoms.

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7. Aortic Diseases

Aortic assessment is a routine part of TTE (and TOE) examinations. TTE allows imaging of the aortic root, sinotubular junction, and proximal ascending aorta, with variable views of the more distal ascending aorta, arch, descending thoracic, and abdominal aorta. TOE provides excellent (and complementary) visualisation of the aortic root, proximal–mid ascending aorta, distal aortic arch, and proximal–distal descending thoracic aorta. However, the distal ascending aorta and proximal arch may not be well-visualised due to the TOE "blind spot", and the evaluation of the origins of the cerebral vessels may also be limited. TOE requires sedation, is invasive, and not as readily accessible as TTE.

7.1. Recommendations for Performance of TTE for Assessment of Aortic Diseases

A comprehensive TTE performed for any indication should include an evaluation of the thoracic and abdominal aorta, where possible. Primary indications focussing on the aorta may range from emergent assessments in suspected acute aortic syndromes (AAS) to routine screening studies (TTE is frequently superseded by CT or MR angiography in the former scenario). TTEs undertaken for other clinical reasons may also identify unsuspected or incidental aortic pathologies the most common of which are aortic aneurysms.

7.1.1. Acute aortic syndromes (AAS) and other aortic disorders

AAS consist of three inter-related conditions—aortic dissection, intramural haematoma, and penetrating aortic ulceration. Aortic dissection results from disruption of the media, with bleeding within and along the aortic wall. The separation of the aortic layers with intimal disruption results in the classic appearance of a thin linear filling defect or a flap between the true and false lumens. The true lumen is usually in continuity with the undissected portion of the aorta while the false lumen is often partially thrombosed. Dissections can be classified according to the DeBakey or Stanford systems used to guide subsequent management strategies [145]. Stanford type A dissection involves the ascending aorta, with (DeBakey type I) or without (Debakey type II) aortic arch and descending aorta propagation. Conversely, Stanford type B dissections do not involve the ascending aorta, while DeBakey type III is defined by its origin distal to the left subclavian artery in the descending aorta and limited to either above (DeBakey type IIIa) or extending below (DeBakey type IIIb) the diaphragm. An intramural haematoma can originate from a spontaneous haemorrhage of the medial vasa vasorum or microscopic aortic intimal tears and may propagate antegrade and/or retrogradely. The presentation mimics the classic dissection but without a false lumen or an intimal lesion on imaging. Penetrating atherosclerotic ulcers are most commonly located in the descending thoracic aorta. The ulcerations penetrate the internal elastic lamina, permitting haematoma formation within the media, leading to an intramural haematoma, aortic dissection, or aortic rupture. These are challenging to identify on TTE or even TOE. Traumatic aortic injury may result from penetrating or blunt force to the artery. While locations of injury to the aorta can vary, blunt deceleration trauma often affects the aortic isthmus, the transition zone from the unfixed aortic arch to the fixed descending aorta.

TTE is often used in the initial assessment of suspected AAS or traumatic injury to the thoracic aorta, particularly when CT is unavailable or likely to be delayed. While TTE sensitivity may be improved with UEAs, allowing identification of approximately 85% of type A dissections, definitive aortic assessment in these scenarios should still be undertaken with CT aortography as soon as possible [146–148]. Importantly, TTE is able to rapidly provide initial assessment of associated complications such as myocardial ischaemia, acute AR, and/or presence of a pericardial effusion/tamponade that may influence management decisions [149]. TOE may have a role for acute diagnosis, intraoperatively during surgery, or in re-assessment in the post-operative phase. While TOE is equally reliable for exclusion of acute thoracic aortic dissections, CT or MR angiography are able to better assess the dissection location/extent, affected/unaffected native aorta dimensions, and identify other potential complications (including dissection progression, aneurysmal degeneration, branch vessel involvement with resultant end-organ perfusion deficits, or aortic rupture) [150].

Other less common aortic pathologies include infectious or inflammatory aortopathies, such as mycotic aortic aneurysms and Takayasu or giant cell arteritis. Due to the anatomical details required for initial diagnosis and follow-up of these conditions, CT or MRI are often the imaging modalities of choice, with TTE playing a complementary role in selected cases.

7.1.2. Congenital anomalies

Aortic coarctation is characterised by a focal narrowing of the thoracic aorta most commonly affecting the isthmus. TTE is often the first imaging modality to raise the suspicion of a possible coarctation and is able to provide both basic anatomic and haemodynamic information. The suprasternal view on a TTE examination allows visualisation of the aortic arch, proximal descending thoracic aorta, and the origin of the left subclavian artery. Flow acceleration on CDI can be quantified with spectral Doppler measurements of the peak velocity if the angle of interrogation is well-aligned. Further detailed assessment of the coarctation including the location/severity/length of narrowing, associated arch hypoplasia or interruption, aneurysmal degeneration proximal or distal to the narrowing, and delineation of the extent of any collateral circulation is best done by CT and/or MRI. Concomitant aortic pathology may also be discovered during an initial TTE study performed for assessment of other complex congenital heart diseases.

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Table 18 Key echocardiographic parameters for assessment of aortic diseases.

Imaging window	Focus	Measurements
Parasternal window	Aortic root and ascending aorta long-axis view	LVOT diameter
		Sinuses of Valsalva diameter
	Aortic root short-axis view	Sinotubular junction diameter
	Aortic valve (AV) long and short-axis views	Ascending aorta diameter
		3D volume dataset of aortic annulus where possible (for TAVR)
		AV morphology and function (co-existent AS or AR [CDI])
Apical window	Aortic root long-axis view	AV function (co-existent AS or AR [CDI and spectral Doppler])
	AV long-axis view	
Suprasternal window	Aortic arch view	Aortic arch dimension
	Proximal descending thoracic aorta view	Proximal descending thoracic aorta dimension
		Doppler flow pattern in descending thoracic aorta (CDI and spectral Doppler)
Subcostal window	Abdominal aorta view	Abdominal aorta dimension (visualised portion)
		Doppler flow pattern in abdominal aorta (CDI and spectral Doppler)

Abbreviations: CDI, colour Doppler imaging; LVOT, left ventricular outflow tract; AS, aortic stenosis; AR, aortic regurgitation; TAVR, transcatheter aortic valve replacement; 3D, three-dimensional.

7.1.3. Screening for aortic aneurysms

TTE is often the first imaging modality utilised for the screening of the proximal aorta in patients with known aortic dilation, other conditions with associated aortopathies such as BAVs or genetic syndromes (e.g., Marfan, Loeys–Dietz, Ehlers–Danlos, Turners), and non-syndromic familial thoracic aortic diseases [149].

True aortic aneurysms involve the intima, media, and adventitia. The incidence has been increasing with the ageing population. Aortic sinus aneurysms are common in patients

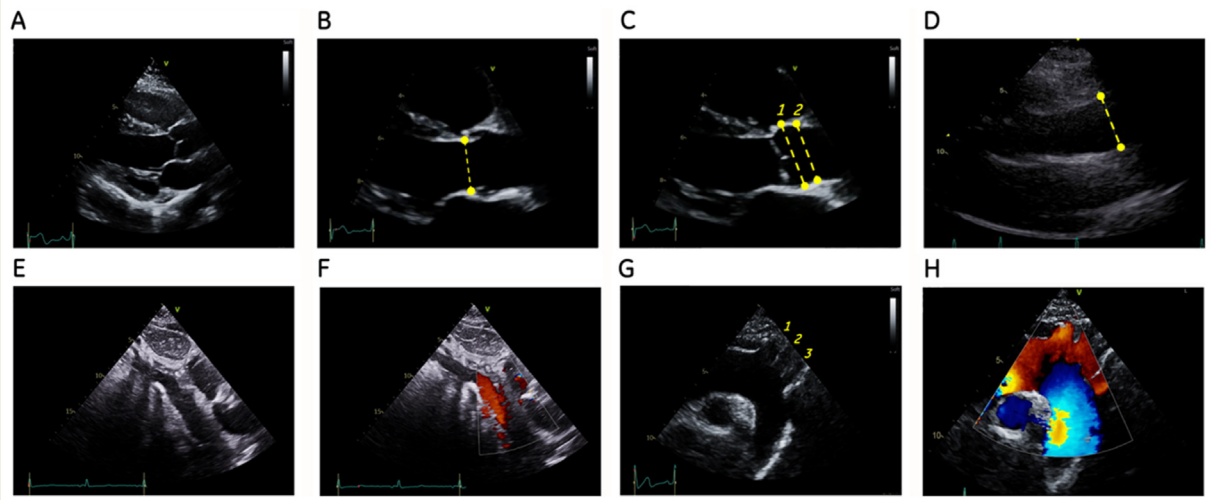

Figure J Echocardiographic assessment of the aorta. A. TTE parasternal long-axis view. B. Zoomed TTE parasternal long-axis view for measurement of the aortic annulus (inner edge-to-inner in mid-systole). C. Zoomed TTE parasternal long-axis view for (1) sinuses of Valsalva and (2) sinotubular junction diameter measurements (leading-edge-to-leading edge at end-diastole). D. Modified TTE parasternal long-axis view for measurement of the proximal ascending aorta (leading-edge-to-leading edge at end-diastole). E. TTE subcostal view of the abdominal aorta. F. TTE subcostal view of the abdominal aorta with colour Doppler imaging. G. TTE suprasternal view of the aortic arch, arch branches (innominate [1], left common carotid [2], and left subclavian [3]), and proximal descending thoracic aorta. H. TTE suprasternal view of the aortic arch, arch branches, and proximal descending thoracic aorta with colour Doppler imaging.
Abbreviations: TTE, transthoracic echocardiography; LVOT, left ventricular outflow tract.

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with BAVs (up to 20%) and familial thoracic aortic abnormalities. Aneurysms of the tubular portion of the ascending aorta are also seen in conjunction with BAVs but may also be associated with idiopathic, atherosclerotic, or inflammatory processes. Pseudoaneurysms occur when there is a contained rupture resulting in the destruction of the intima and media such that they do not contain all three aortic wall layers.

Various terms are used to describe specific imaging features of aortic aneurysms:

1. Fusiform aneurysms—symmetrical dilation of the entire aortic wall circumference.
2. Saccular aneurysms—localised outpouching of a portion of the aortic wall.
3. Annulo-aortic ectasia—sinotubular junction effacement resulting in the classic "tulip bulb" appearance and is commonly seen in Marfan, Loeys–Dietz, Ehlers–Danlos, and other familial aortic syndromes.
4. Sinus of Valsalva aneurysm—sinus of Valsalva dilation resulting from failure of the development of the aortic media elastic component, most commonly affecting the right and non-coronary sinuses.

7.1.4. Aortic atherosclerotic disease

Aortic atherosclerosis may be assessed by TTE but is more accurately evaluated by TOE. Extent of aortic atherosclerosis is particularly relevant to the preoperative assessment for cardiac procedures involving the aorta as patients may be at high risk of aortic cross clamp cerebrovascular accidents. Furthermore, the demonstration of aortic atherosclerosis may alter management decisions in patients following a stroke or peripheral embolism.

7.2. Recommendations for Serial TTE Assessment of Aortic Diseases

While tomographic imaging such as CT and MRI offer measurements of aortic dimensions that are independent of image orientation, TTE has a significant role in ongoing surveillance due to its advantages in cost and lack of radiation and contrast exposure. This is particularly relevant when TTE measurements correlate well with the other imaging modalities. Follow-up surveillance studies should only be undertaken if it will influence management decisions (e.g., patient is eligible for aortic repair).

7.2.1. Follow-up assessment for aortic aneurysms

The purpose of a follow-up TTE for this indication is to establish if a patient is at or near the treatment threshold, the rate of progression, or to investigate a change in clinical symptoms or examination findings. Surveillance intervals have not been studied extensively and any such recommendations are based on expert opinion and should be tailored to the individual patient's underlying condition. For example, rates of aortic expansion are greater in those with larger calibre aortas, aortopathies, BAVs, and females. The 5-year aortic dissection or rupture risk has been estimated at 0.4%, 1.1% and 2.9% for baseline aortic diameters of 45mm, 50mm, and 55mm, respectively [151]. Shorter surveillance intervals should be considered in patients with aortic dimensions close to surgical thresholds, documented increasing rates of dilatation, familial aortopathies, or a family history of aortic dissection.

After the initial baseline study, a repeat TTE usually at one year is indicated to establish interval change in those with aortic diameters over 45mm. Annual imaging surveillance is recommended for cases close to surgical thresholds (50–55mm), with shorter surveillance intervals being rarely appropriate, unless surgery was unduly delayed for other reasons. In the absence of a rapid rate of expansion, imaging surveillance should be performed every 2 years for aortic dimensions 45–49mm, and every 5 years for aneurysms measuring 40–44mm. It is also important to consider the inherent cross-modality differences in both the measurement technique (leading edge-to-leading edge vs inner edge-to-inner edge) and planes [149]. In general, comparative measurements are best made within the same modality and methodology.

7.2.2. Follow-up after thoracic aortic intervention

Follow-up post-intervention of aortic diseases will vary depending on the specific pathology, type of repair, and presence of concomitant valvular lesions. Serial TTE assessment is essential as tomographic imaging alone with CT may not provide adequate information about valvular function. MRI can be safely used for nitinol-based stent-grafts. TOE may rarely be indicated if a patient is unable to undergo a CT or MRI. The 2015 ASE Multimodality Imaging of Diseases of the Thoracic Aorta in Adults guideline suggests initial follow-up imaging at 1, 3, 6, and 12 months post-acute aortic dissection diagnosis and management, with annual examinations thereafter [152]. The writing committee recommends an initial study be performed at 4–6 weeks post-intervention, followed by further surveillance imaging at 6 months in some patients, or 12 months in most others. Yearly assessments are recommended but the interval may be extended once stability has been demonstrated.

7.2.3. Follow-up of congenital anomalies

Surveillance intervals need to be individualised depending on the primary lesion severity and associated abnormalities. Detailed discussion of this topic is beyond the scope of this document and readers should refer to the previously published 2018 AHA/ACC Guideline for the Management of Adults with Congenital Heart Disease [130].

7.3. Requirements for Comprehensive TTE Assessment of Aortic Diseases

Echocardiographic assessment of the aorta differs from other structures in that it is often descriptive, with measurements of relevant landmarks, and emphasis placed on the nature, extent, and severity of the specific pathology (e.g., aneurysmal dilation). Key TTE parameters for the assessment of aortic diseases are summarised in Table 18 and Figure J.

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The aortic root originates from the semilunar-shaped AV leaflets that are attached along the length of the aortic sinuses and in direct continuity with the LVOT. The aortic root therefore extends from the basal attachments of the AV leaflets (on LV myocardium) that form a virtual ring to the sinotubular junction [153]. Understanding and differentiating this virtual ring from the true anatomic ventriculo-arterial junction is crucial in the imaging work-up for TAVRs. The former is where the aortic annulus diameter is measured. The ascending aorta starts distal to the sinotubular junction, courses superiorly in the chest (slightly anterior and to the right of the main pulmonary trunk), continuing as the aortic arch, before coursing inferiorly towards the abdomen in a "candy cane" configuration.

Adult aortic dimensions are correlated with age, gender, and body size [10,154,155]. Normal ranges are important for the accurate and reproducible interpretation of imaging studies. Measurements may be reported as absolute values or indexed to the patient's BSA. The recommended normal ranges by the EACVI for standardised reporting are as follows [10]:

1. Annulus— $\leq$14mm^2 (Male [M] and female [F]).
2. Sinus of Valsalva—$\leq$19mm/m^2 (M), $\leq$20mm/m^2 (F).
3. Sinotubular junction—$\leq$17mm/m^2 (M and F).
4. Proximal ascending aorta—$\leq$17mm/m^2 (M), $\leq$19mm/m^2 (F).

There is currently insufficient normative data by ethnicity in the Māori, Australian Indigenous, and Pasifika patients in Australasia although this may become available with newer population studies [156]. The relevance of ethnicity was demonstrated in a large New Zealand cohort study where Pasifika patients accounted for 28% of acute aortic dissections, who in turn only represent 7% the total population [157].

With the exception of the aortic annulus, all other linear diameter measurements of the aortic root and ascending aorta by echocardiography are performed using the leading edge-to-leading edge technique at end-diastole. This is in contrast to the inner edge-to-inner edge convention in CT aortography. Echocardiographic measurements may also be inaccurate due to the complex 3D geometry of the aortic valve and root. The aortic trans-sinus dimensions acquired from echocardiography are often discrepant with those measured by multiplanar reconstruction as performed by CT or MRI. Because the 2D imaging plane is variably orientated, and almost always off-axis to the standard sinus-commissure tomographic measurements, echocardiographic values are more often closer to the larger sinus–sinus diameters. Furthermore, it is well-recognised that the aortic annulus and LVOT are commonly oval in shape, such that the standard 2D TTE parasternal long-axis view results in the measurement of the smaller anterior–posterior diameter. This limitation can be addressed by 3D echocardiography which may serve as a reliable alternative to CT if the use of contrast is contraindicated.

8. Infective Endocarditis (IE)

Infective endocarditis (IE) remains a deadly condition in Australia and around the world [158]. In more recent times, healthcare-associated IE represents up to 30% of cases. This is a consequence of newer predisposing factors, including increased intracardiac device implantations or deployment and use of invasive procedures with inherent risks for bacteraemia [159]. Echocardiography remains a central imaging tool for the diagnosis, management, prognostication, and surveillance of IE.

8.1. Recommendations for Performance of TTE for Assessment of IE

8.1.1. Diagnosis of IE

The modified Duke criteria remain the cornerstone for diagnosis of IE [160]. The two major criteria for clinical diagnosis include positive blood cultures with a typical organism and imaging evidence of pathological lesions (vegetation, abscess, pseudoaneurysm, or new dehiscence of a prosthetic valve) for which echocardiography (TTE and/or TOE) is the primary imaging modality. In any clinical syndrome where IE is suspected, a TTE should be performed as the initial investigation usually within 12–24 hours, or more urgently in cases complicated by pulmonary congestion or haemodynamic instability. As the sensitivity of TTE for detection of vegetations is only 50%–60%, and will not exclude the diagnosis, the majority of patients with suspected IE will ultimately require TOE assessment [161]. Nonetheless, an initial TTE still has distinct advantages as it can provide complementary diagnostic information regarding potential endocarditic involvement of anterior cardiac structures in the right heart, the anterior aspect of an aortic valve prosthesis and aortic root, as well as better interrogation of lesions on the ventricular side of a mitral or tricuspid prosthesis. TTE allows accurate assessment of chamber size and function, presence of a pericardial effusion, estimation of intracardiac pressures, and atrio–ventricular valvular regurgitation without the need for sedation. Additionally, the negative predictive value (NPV) of a 'near normal' TTE is not insignificant. In an Australian study of 622 consecutive patients with high-risk/suspected IE (77% positive blood cultures) referred for both TTE and TOE assessments, 44% of the patients had an initial 'negative' TTE (i.e., satisfactory images with no intracardiac prosthesis, no pathological lesions, $\leq$mild valvular thickening and/or regurgitation), which conferred a NPV of 97% for vegetations on subsequent TOE (99% for those with non-staphylococcal and 95% for *Staphylococcus aureus* bacteraemia) [161]. This suggests that a proportion of patients with suspected IE may not require an immediate TOE, and those with non-staphylococcal bacteraemia may not require further imaging, unless there is an ongoing high index of suspicion or concern (e.g., intractable fevers, embolism, new cardiac symptoms/signs). In the context of a negative TTE, patients with a staphylococcal bacteraemia will still likely require a

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TOE, but the timing of which could be delayed for 5–7 days or even a repeat TTE considered, as guided by clinical suspicion. TOE should be performed in patients with a positive TTE (if clinically appropriate) to confirm the findings, exclude local complications, or to assist with the diagnosis in the setting of intracardiac prosthesis/devices or an equivocal TTE (poor images, more than mild valvular regurgitation or thickening) [46,159]. If TTE and TOE images are concordant in patients with documented IE, this may allow ongoing serial TTE surveillance during treatment.

8.1.2. Prognosis of IE

Prognosis of IE is influenced by four main factors - patient characteristics, presence or absence of cardiac and non-cardiac complications, the infecting organism, and the echocardiographic assessment [159]. Imaging findings portending a poor prognosis include reduced LVEF, severe left-sided valve regurgitation, severe prosthetic valve dysfunction, elevated LA and pulmonary pressures, large vegetations (>10mm), and peri-annular complications. Patients with left-sided IE and heart failure, peri-annular complications, and/or Staphylococcus aureus infections are at highest risk of death and require urgent surgery during the active phase of the disease [162,163].

8.2. Recommendations for Serial TTE Assessment of IE

The clinical course of IE is highly variable, and depends on the causative microorganism, presence or absence of pre-existing cardiac disease, prosthetic valves or intracardiac devices, and the mode of presentation. Successful treatment of IE relies on microbial eradication by antibiotics which commonly requires up to 6 weeks of intravenous treatment. Surgical intervention involves complete removal of all infected material, draining abscesses, and repair or replacement of the damaged valve(s) [159]. There is little evidence to guide specific recommendations for serial imaging assessment in patients with suspected or definite IE.

8.2.1. Serial assessment for diagnosis

Consistent with the ACC/AHA and ESC guidelines, in patients with suspected IE and a negative initial TTE, further re-evaluation in 5–7 days is appropriate if the clinical suspicion of endocarditis remains high [46,159]. However, the incremental yield from a third evaluation is low, and no additional information is obtained from more than three imaging assessments [164].

8.2.2. Treatment surveillance

In patients with uncomplicated IE undergoing antibiotic treatment, repeat TTE and/or TOE may be considered during follow-up in order to detect new silent complications and monitor vegetation size. The timing and mode (TTE or TOE) of the repeat examination will depend on the initial imaging findings, type of microorganism, and initial response to therapy. Repeat TTE and/or TOE is recommended if a new complication of IE is suspected (e.g., new murmur, embolism, persisting fever, heart failure, abscess, or atrioventricular block). At completion of antibiotic therapy, repeat TTE can also be performed to document a "new" baseline [159].

8.3. Requirements for Comprehensive TTE Assessment of IE

TTE imaging should be performed in the standard imaging planes (parasternal, apical, subcostal, and suprasternal windows) with the use of off-axis imaging windows as required. Side-by-side comparison with previous study images (where available) is recommended for all cases, to determine if interval changes are present. Specific focus should be placed on:

1. Identification and assessment of vegetation(s) (e.g., size, location, mobility).
2. Evidence of aortic root thickening.
3. Evaluation of native valve anatomy and valvular regurgitation if present.
4. Prosthetic valvular function (assessment of antegrade flow and degree of valvular or para-prosthetic regurgitation).
5. Diastolic MR that may indicate atrio-ventricular block or severe AR.
6. LV/RV size and systolic function and estimation of intracardiac filling and pulmonary pressures.
7. Pericardial effusion and associated haemodynamic significance if present.

8.4. Multimodality Imaging in Assessment of IE

The modified Duke criteria have demonstrated lower diagnostic sensitivity in cases of prosthetic valve or cardiac device-related IE. Advances in alternative imaging modalities have resulted in improved identification of endocardial involvement and complications of IE (i.e., abscess, pseudoaneurysms, fistula) in these settings. Cardiac/whole-body CT or cerebral MRI are able to detect clinically silent embolic events or infectious aneurysms. ^{18}F-fluorodeoxyglucose PET/CT may improve the initial diagnosis of prosthetic valve endocarditis and can be performed serially to monitor treatment response [159].

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9. Cardiac Masses

Common aetiologies of cardiac masses can be considered under five broad headings [165]:

1. Intracardiac thrombus—usually attached to the atrial or ventricular endocardium and are often associated with arrhythmias (e.g., AF), cardiac structural abnormalities (e.g., aneurysmal dilatation), or an underlying thrombogenic disorder.
2. Cardiac tumours—the most common primary benign tumours in the adult population are myxomas, lipomas, or valvular papillary fibroelastomas. Primary malignant tumours are extremely rare and include sarcomas, lymphomas, and mesotheliomas. Secondary (metastatic) tumours are usually detected in the context of a known primary malignancy (e.g., lung, breast, gastrointestinal, lymphoma, or leukaemia) and rarely present as an isolated cardiac mass.
3. Infective vegetation or inflammatory mass.
4. Unusual anatomic variants of a normal cardiac structure (e.g., prominent crista terminalis in the RA or moderator band in the RV).
5. An apparent mass caused by an ultrasound imaging artefact.

9.1. Recommendations for Performance of TTE for Assessment of Cardiac Masses

TTE is considered the most appropriate first-line investigation for assessment of a known or suspected cardiac mass. TTE is indicated when:

1. A cardiac mass has been detected with an alternate imaging modality such as a chest CT or PET imaging.
2. When a cardiac mass is suspected on clinical grounds (e.g., young patient with systemic embolic events in multiple vascular territories).

A cardiac mass may also be an incidental finding when a TTE is performed for another indication.

9.2. Recommendations for Serial TTE Assessment of Cardiac Masses

In the majority of clinical situations, once a cardiac mass is detected on TTE, the typical approach would be to utilise further imaging with TOE, CCT, or CMR to confirm the diagnosis and establish the aetiology, followed by a definitive management strategy such as surgical resection of a primary tumour or anticoagulation for intracardiac thrombi. An early follow-up TTE post-intervention is recommended to assess for any residual impact on cardiac structure or function, and to serve as a baseline for future comparison should there be a recurrence of symptoms or any other relevant change in the patient's clinical status. Repeat imaging may also be reasonable following surgical resection of a primary tumour to monitor for recurrent disease. Resected benign tumours may recur in the absence of tumour-free margins [166]. Surveillance TTEs may be reasonably repeated at 3–5 yearly intervals.

While benign cardiac tumours are typically slow-growing, evidence regarding the natural history is largely limited to case reports and relatively small observational studies. Comparison of progression rates and outcomes between studies performed at different centres is confounded by variations in imaging protocols and management approaches. Serial TTE imaging may be indicated in selected cases where patients are managed with an initial watch-and-wait approach. For example, a small valvular mass (presumed to be a papillary fibroelastoma) that is not associated with significant valvular dysfunction may be managed conservatively in the first instance, particularly if the patient is elderly or has significant comorbidities associated with increased surgical risk. In such cases, it would be reasonable to repeat the TTE study when there is an alteration in the patient's clinical status (e.g., new or progressive murmur), to assess for significant interval change that would influence management decisions (e.g., monitoring the size of a presumed benign tumour where progression to a high risk of valvular obstruction would lead to intervention), or at the discretion of the treating clinician.

9.3. Requirements for Comprehensive TTE Assessment of Cardiac Masses

Echocardiographic imaging should be performed from standard parasternal, apical, subcostal, and suprasternal windows, as well as additional off-axis views (e.g., paraapical imaging) to fully assess the anatomical relationships of the cardiac mass and exclude the presence of additional lesions. Essential characteristics that must be clearly defined include the size (measured in multiple dimensions), base of attachment, involvement of adjacent cardiac structures, mobility, and potential haemodynamic consequences (e.g., functional valvular obstruction with myxomas or regurgitation in the case of fibroelastomas) [167]. Benign masses are usually confined to the endocardium in comparison with malignant neoplasms that may invade across tissue planes or anatomic boundaries and are more often associated with pericardial effusions. The presence of independently mobile elements may increase the risk of embolic complications and should be highlighted in the report.

The use of UEAs may be beneficial in patients with suboptimal image quality to aid in delineating the attachment of the mass to the underlying endocardium or valvular structures. Myocardial contrast echocardiography may provide additional diagnostic insight into the pathology of a mass by assessing for the presence of vascularity [8,165,167]. Absence of contrast enhancement or vascularity would be consistent with a thrombus or vegetation, depending on the clinical setting. The degree of enhancement may help distinguish between benign lesions, such as myxomas, which typically partially opacify or enhance less than the surrounding myocardium, while malignant tumours fully opacify or have exaggerated perfusion. 3D TTE may allow better

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appreciation of the shape and attachment of cardiac masses, but its utility is limited by lower spatial and temporal resolutions.

Due to its high spatial resolution and the close proximity of the probe to cardiac structures, TOE imaging can provide exceptional detail in the assessment of cardiac masses, particularly those located in the atria or attached to the interatrial septum. The high temporal resolution also makes this modality the gold standard technique for assessment of mobile masses attached to the valves. 3D TOE can be especially useful for pre-surgical planning by providing more accurate evaluation of the anatomical relationships, size, and shape of the mass.

9.4. Multimodality Imaging in Assessment of Cardiac Masses

CCT and CMRI are strongly recommended for the investigation of cardiac masses, when echocardiography indicates myocardial or pericardial invasion. These modalities can provide incremental information regarding tissue characterisation, and also assess for extra-cardiac involvement or metastases within the larger field of view.

10. Pulmonary Hypertension (PHTN)

Routine TTE examinations should include rigorous assessment of pulmonary pressures along with right and left heart function. PHTN is simply the identification of elevated pulmonary artery pressures, whereas pulmonary arterial hypertension (PAH) is the term reserved for PHTN in the setting of normal left heart filling pressures and elevated pulmonary vascular resistance. New diagnostic criteria have defined PHTN as a mean pulmonary artery pressure (mPAP) $\geq$20mmHg at right heart catheterisation (RHC), recognised echocardiographically by an RVSP above approximately 30mmHg [168]. TTE plays a pivotal role in the initial detection and assessment of possible PHTN for the purposes of aetiological diagnosis, treatment, and prognosis [169]. The following guiding principles should be applied routinely to any comprehensive TTE study [170]:

1. Calculation of the PASP (RVSP) as a screening tool for PHTN.
2. Identification of any left heart disease that may be a contributor (Group 2 PHTN, see below).
3. Determination of the effects of PHTN on the right heart.

10.1. Recommendations for Performance of TTE for Assessment of PHTN

1. Evaluation of patients with symptoms or signs that may suggest PHTN (e.g., breathlessness).
2. Evaluation of patients with conditions that may increase the likelihood of PHTN (e.g., connective tissue diseases, left heart diseases, lung diseases, pulmonary artery obstruction, systemic diseases).
3. Follow-up of patients with known PHTN (see below for recommendations).

10.2. Recommendations for Serial TTE Assessment of PHTN

Regular TTE follow-up is essential to monitor therapeutic response (improvement or deterioration) to disease-specific treatments. The recommended intervals for repeat studies are dependent on the World Health Organization clinical class or group of PHTN: [30,168]

1. Group 1: PAH
   a. Stable disease receiving Pharmaceutical Benefits Scheme (PBS)-subsidised therapy—3 months after initiating therapy, and 6-monthly thereafter (unless a separate clinical indication exists).
   b. Stable disease not receiving PBS-subsidised therapy—annually.
   c. Any unstable or worsening disease—as clinically indicated.
2. Group 2: PHTN due to left heart disease

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Table 19 Key echocardiographic parameters for assessment of PHTN.

Parameter	Measurement
Evaluation of the left heart (comparison to initial or previous TTE on follow-up studies)	Measurement of LV and LA volumes Evaluation of LV systolic and diastolic function Assessment of mitral and aortic valves (re-evaluation on serial studies if previous abnormality identified)
Evaluation of RV size and shape	RV dimensions RV area and/or volume RV shape (sphericity, septal flattening in systole and/or diastole) RV wall thickness
Evaluation of RV function	RV systolic function (FAC, TAPSE, RV S') Consider RV free wall longitudinal strain, where possible
Measurement of RA parameters	RA area and/or volume RA pressure (IVC size and respiratory variation)
Measurement of pulmonary artery pressures	PASP (RVSP using the peak TR V_{max}; If insufficient TR is present consider use of agitated saline contrast or UEAs and/or pulmonary artery acceleration time) Estimation of pulmonary artery mean and diastolic pressures using the early- and end-diastolic PR velocities
Estimation of pulmonary vascular resistance	Consider use of echocardiographic estimates of pulmonary vascular resistance
Pericardium	Assessment of pericardial effusion Assessment for pericardial disease including constrictive physiology

Abbreviations: PHTN, pulmonary hypertension; TTE, transthoracic echocardiography; LA, left atrial; LV, left ventricular; RA, right atrial; RV, right ventricular; FAC, fractional area change; TAPSE, tricuspid annular plane systolic excursion; RV S', tissue Doppler-derived tricuspid lateral annular systolic velocity; IVC, inferior vena cava; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure; TR, tricuspid regurgitation; PR, pulmonary regurgitation; UEA, ultrasound enhancing agents.

   a. Determined by progression and follow-up recommendations for underlying left heart disease (see LV and valvular heart disease recommendations).
   b. Stable disease receiving PHTN treatment (either PBS-subsidised or otherwise)— annually.
   c. Any unstable or worsening disease—as clinically indicated.
3. Group 3: PHTN due to lung diseases or hypoxia
   a. 3 months after initiating PHTN therapy, and 6–monthly thereafter (unless a separate clinical indication exists) as determined by progression of the underlying lung disease.
   b. Stable disease with known PHTN—every 1–2 years.
   c. Any unstable or worsening disease—as clinically indicated.
4. Group 4: PHTN due to pulmonary artery obstructions
   a. 3 months after initiating PHTN therapy, and 6–monthly thereafter (unless a separate clinical indication exists).
   b. Stable disease not receiving PBS therapy— annually.
   c. Any unstable or worsening disease—as clinically indicated.
5. Group 5: PHTN with unclear and/or multifactorial mechanisms (miscellaneous and systemic diseases)
   a. 3 months after initiating PHTN therapy, and 6–monthly thereafter (unless a separate clinical indication exists) as determined by progression of the underlying disease.
   b. Stable disease with known PHTN—every 1–2 years.
   c. Any unstable or worsening disease—as clinically indicated.

10.3. Requirements for Comprehensive TTE Assessment of PHTN

Although the formal diagnosis of PHTN requires the gold standard invasive RHC, TTE remains the most accessible primary non-invasive tool for the baseline assessment of pulmonary haemodynamics and probability of PHTN [38,41]. TTE is also able to provide diagnostic insights into the potential underlying aetiology of the PHTN, surrogate features that may prompt further investigation for PHTN despite apparent "normal" RVSP values (e.g., unexplained RV dilatation or dysfunction, functional TR), and offer incremental prognostic information from advanced echocardiographic techniques (e.g., RV strain, 3D TTE RV volumes, and RVEF). Serial TTE assessments are also required for ongoing prescribing of disease-specific therapies as mandated by the PBS. Key echocardiographic parameters for the initial and serial assessment of PHTN are summarised in Table 19.

A complete discussion regarding the technical aspects of echocardiographic assessment of PHTN is beyond the scope of this document but the details are comprehensively reviewed in a state-of-the-art review by Cordina et al. [38].

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11. Cardio-Oncology

Serial evaluation of LV function is emerging as an important area in the longitudinal assessment of patients during and after cancer treatment. With improved long-term cancer survival, the potential short, intermediate, and long-term cardiovascular toxicity of anti-cancer agents have become relevant [171]. Cardiotoxicity from chemotherapeutic agents and radiotherapy includes cardiac dysfunction and heart failure, arrhythmias, hypertension, coronary artery disease, valvular damage, pericardial diseases, thromboembolism, and PHTN. This section will focus on the detection and monitoring of LV dysfunction.

Potential cardiotoxic effects from chemotherapy used in the treatment of patients with cancer are well-established [172–174]. The frequency of cardiotoxicity resulting in LV dysfunction is <10% with heart failure occurring in <5% [175]. Radiation therapy to the mediastinum or left side of the thorax can also increase the risk of developing cardiac dysfunction in particular coronary artery disease [176]. The timing at which cardiotoxicity develops depends on the chemotherapeutic agent, with damage caused by anthracyclines occurring immediately or in the short term after exposure, whereas damage from radiation therapy may take many years to manifest. As there is usually significant underlying cardiac reserve, overt LV dysfunction may not appear for a considerable time after treatment administration [177].

Chemotherapy cardiotoxicities or cancer therapeutics-related cardiac dysfunction (CTRCD) are classified into two pathophysiologic subtypes—type 1 CTRCD (cardiotoxic) is dose-dependent and results in irreversible myocyte damage while type 2 CTRCD (cardioinhibitory) is not related to cumulative dosing and causes myofibrillar damage which is mostly reversible [177]. Typically type 1 CTRCD is due to anthracycline therapy (e.g., doxorubicin, epirubicin) and type 2 CTRCD results from monoclonal antibodies, such as anti-human epidermal growth factor receptor-2 (HER2) agents (e.g., trastuzumab, pertuzumab, trastuzumab emtansine [T-DM1]), and tyrosine kinase inhibitors (e.g., neratinib, lapatinib, imatinib).

11.1. Recommendations for Performance of TTE for Assessment of CTRCD

Echocardiography has emerged as the dominant imaging modality for the diagnosis of both asymptomatic and symptomatic CTRCD [177]. Recommendations for TTE evaluation of cardiac function in an oncology patient include:

Table 20 Recommendations for performance of TTE for assessment of CTRCD.

Indications	Key echocardiographic parameters
Baseline evaluation prior to chemotherapy Serial monitoring of LV function • Evaluation after appropriate time interval during chemotherapy (e.g., 3 monthly with trastuzumab therapy) • In high-risk patients more frequent monitoring may be required (i.e., prior to each dose of chemotherapy [e.g., 3-weekly prior to trastuzumab]) Significant clinical deterioration Significant change in therapy 3–6 months following completion of chemotherapy Routine monitoring of LV function in stable patients (following anthracycline therapy - annually for 2–3 years then 3–5 yearly as for low/medium risk patients)	Quantitative measurement of LV size and function • Minimum dataset should include LV volumes and LVEF using 2D biplane Simpson's method • Where possible 3D LV volumes and LVEF and GLS should be performed • Use of UEAs where LV endocardial definition is suboptimal Quantitative measurement of RV size and systolic function • Minimum dataset should include RV basal dimension, RVOT proximal dimension, TAPSE, RV S ', and FAC • Where possible 3D RV volumes and RVEF and free wall strain should be performed Measures of diastolic function • Minimum dataset should include mitral valve inflow Dopplers, mitral annular e' velocities, E/e' ratio, TR V_{max}, and LAVI IVC size and respiratory variation PASP (RVSP)

Abbreviations: TTE, transthoracic echocardiography; CTRCD, cancer therapeutics-related cardiac dysfunction; IVC, inferior vena cava; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure; LV, left ventricular; LVEF, left ventricular ejection fraction; 2D, two-dimensional; 3D, three-dimensional; UEAs, ultrasound enhancing agents; RV, right ventricular; RV S', tissue Doppler-derived tricuspid lateral annular systolic velocity; RVOT, right ventricular outflow tract; TAPSE, tricuspid annular plane systolic excursion; FAC, fractional area change; TR, tricuspid regurgitation; LAVI, left atrial volume index.

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Table 21 Recommended protocol for initial cardio-oncology TTE (40 minutes).

Comprehensive TTE study (including heart rate and blood pressure)
2D biplane Simpson's LVEF and 3D LVEF and GLS where possible
Full report with LVEF and GLS values included
Follow-up recommendations (guided by baseline risk assessment)

1. Prior to initiation of systemic chemotherapy or radiotherapy (left chest or mediastinal) as a baseline reference or comparison for serial studies and risk stratification of patients at higher risk of cardiovascular complications (e.g., screening for pre-existing cardiac dysfunction).
2. At regular intervals during chemotherapy.
3. After conclusion of chemotherapy or radiation treatment.
4. If there is a change in the patient's symptoms or physical examination findings or clinical deterioration (e.g., heart failure).

Early detection of CTRCD may prompt initiation of cardioprotective therapy to potentially reverse the cardiotoxicity and prevent the development of heart failure.

Table 22 Recommended protocol for cardio-oncology TTE during treatment (20 minutes).

Imaging Window	Focus	Measurements
Parasternal long axis	Deep PLAX image (check for pericardial effusion)	2D LV dimensions
(PLAX; 5-6 images)	Standard PLAX image and measurements	
	CDI assessment of the mitral and aortic valves	
Parasternal short axis	Basal LV	
(PSAX; 3 images)	Mid LV	
	Distal LV/apex	
Apical 4-chamber	Full depth A4C view	LVEF, GLS
(A4C; 10-11 images)	Reduced depth focused view of the LV	LA biplane volume
	Focused view of the LA	MV E and A velocities and deceleration time
	CDI assessment of the MV	Medial/lateral/average e' velocities and E/e' ratio
	PWD at mitral leaflet tips	TR V_{max}; PASP (RVSP)
	TDI for mitral annular medial and lateral e' velocities	
	CDI assessment of the TV	
Apical 5-chamber	CDI assessment of the AV	AV peak velocity
(A5C; 2 images)	CWD across the AV	
Apical 2-chamber	Full depth A2C view	LVEF, GLS
(A2C; 5 images)	Reduced depth focused view of the LV	LA biplane volume
	Focused view of the LA	
	CDI assessment of the MV	
Apical long axis	Full depth ALAX view	GLS
(ALAX; 3 images)	Reduced depth focused view of the LV	
	CDI assessment of the MV and AV	
Report		
Minimum requirements	Blood pressure and heart rate; Timing of last chemotherapy or monoclonal antibody infusion	
	LV size and systolic function (2D biplane Simpson's LVEF; 3D LVEF and GLS where possible)	
	MV and AV summary	
	PASP (RVSP)	
	Any other significant changes	
	Follow-up recommendations (guided by interval stability/change and specific chemotherapeutic agent[s])	

Abbreviations: TTE, transthoracic echocardiography; MV, mitral valve; AV, aortic valve; TV, tricuspid valve; PASP, pulmonary artery systolic pressure; RVSP, right ventricular systolic pressure; LV, left ventricle/ventricular; LVEF, left ventricular ejection fraction; LA, left atrium/atrial; GLS, global longitudinal strain; PWD, pulsed-wave Doppler; CDI, colour Doppler imaging; 2D, two-dimensional; 3D, three-dimensional; TR, tricuspid regurgitation.

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Table 23 Recommended protocol for cardio-oncology TTE following completion of treatment (40 minutes).

- Comprehensive TTE study (including heart rate and blood pressure)
- 2D biplane Simpson's LVEF; 3D LVEF and GLS where possible
- Full report with LVEF and GLS values included
- All progressive LVEF and GLS measurements

Abbreviations: TTE, transthoracic echocardiography; LVEF, left ventricular ejection fraction; GLS, global longitudinal strain; 2D, two-dimensional; 3D, three-dimensional.

11.2. Recommendations for Serial TTE Assessment of CTRCD

Current ASE/EACVI, BSE, British Cardio-Oncology Society (BCOS), and European Society of Medical Oncology (ESMO) expert consensus statements and guidelines all recommend serial monitoring of LV function by TTE to allow early detection of CTRCD [174,177–179]. CTRCD is defined as an absolute decrease in LVEF of >10% to a value of <50%. Furthermore, a >15% relative decrease in LV GLS compared to baseline is regarded as clinically significant or abnormal, while a <8% reduction is not considered indicative of CTRCD. Any decline in LVEF or GLS requires confirmation with a repeat study in 2–3 weeks.

TTE surveillance for anthracycline chemotherapy should be guided by the baseline risk assessment for cardiotoxicity based on patient (age, pre-existing cardiovascular disease or risk factors, impaired LV function) and treatment-related (type and dose) factors [179]. It is recommended that surveillance TTE studies during treatment be undertaken according to the number of cycles completed or cumulative dose received as determined by the risk category (e.g., after completion of 4 or 6 cycles of anthracycline-based chemotherapy regimes in breast cancer patients, and/or a cumulative dose of 250mg/m^2 for low-risk patients). Further monitoring for delayed or late anthracycline toxicity within the first year should also be considered at 6 and/or 12 months following completion of therapy.

Trastuzumab is usually administered for a total of 12-18 months. Currently, evaluation of LV function is recommended at 3-monthly intervals during treatment. In patients with pre-existing cardiac dysfunction (e.g., co-existent cardiomyopathy, previous ischaemic heart disease), more frequent evaluation may be necessary (i.e., every 3 weeks prior to administration of the next dose of chemotherapy). Following completion of trastuzumab therapy, further surveillance TTEs can be considered at 3–6 months, and at 12 months, in patients deemed to be at medium or high risk for cardiotoxicity during the baseline assessment. If neoadjuvant or adjuvant anthracycline therapy is administered, further follow-up as described above can be considered.

A comprehensive review of echocardiographic monitoring with other anti-cancer agents, including vascular endothelial growth factors and tyrosine kinase, proteosome or immune checkpoint inhibitors, is beyond the scope of this document but have been detailed in the relevant societal guidelines, consensus statements and expert reviews [174,177–179].

Additionally, if there is a deterioration in the clinical condition of an oncology patient suggestive of cardiotoxicity, a repeat TTE should be performed as dictated by the treating clinician.

11.3. Requirements for Comprehensive TTE Assessment of CTRCD

The recommended indications and protocols for TTE examinations at baseline, during, and after completion of anti-cancer therapies are summarised in Tables 20–23. The patient's height, weight, and blood pressure should be recorded as part of the examination. Comprehensive assessment of LV size and systolic/diastolic function is central to the evaluation for CTRCD. 2D biplane Simpson's LVEDV and LVESV should be measured, and the quantitative numerical LVEF reported. 3D LV volumes and LVEF are preferable where possible, due to the superior reproducibility and lesser inter/intra-observer variability [180]. Where available, it is recommended that LV GLS be performed during the baseline pre-treatment TTE and on all serial scans. GLS can detect changes in LV function prior to any overt alteration in LVEF, may be a more sensitive marker of preclinical/subclinical cardiotoxicity, and a better reflection of response to cardioprotective therapy [181]. Repeat TTE studies should also be optimised to improve reproducibility and consistency, with use of the same methodology for LVEF calculations, the same offline vendor platform for GLS measurements, and ideally performed in the same laboratory.

Conclusion

The optimal use and high-quality performance and reporting of TTEs will prevent over or under-utilisation of this resource, such as the inappropriate frequent surveillance of patients with stable chronic cardiac disease in the absence of clinical change, or unnecessary downstream testing due to technically incomplete or suboptimal studies. While this document serves as a broad guideline for the performance of TTE in adults, the indication(s) for an initial or repeat study and timing of serial examinations must still be stewarded by the treating clinician's discretion, tailored to the individual patient's condition and clinical state.

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