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Guidelines for the management of valvular heart disease (VHD) and SHSCT echocardiography service

Table of contents:

These guidelines are a summary of the current guidelines and consensus documents of the ESC / EACVI and are intended for internal use within the SHSCT only.

These guidelines are based predominantly on:

ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC). European Heart Journal (2016) 37: 2893–2962.

Recommendations for the echocardiographic assessment of native valvular regurgitation: an executive summary from the European Association of Cardiovascular Imaging. European Heart Journal – Cardiovascular Imaging (2013): 14:611-644.

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1.1 Abbreviations:

ACE	Angiotensin converting enzyme
AF	Atrial fibrillation
ARB	Angiotensin receptor blocker
AVA	Aortic valve area
AVS	Aortic valve surveillance
BAV	Balloon aortic valvuloplasty
BHSCT	Belfast health and social care trust
BNP	B type natriuretic peptide
BSA	Body surface area
CABG	Coronary artery bypass grafting
CAD	Coronary artery disease
CKD	Chronic kidney disease
CMR	Cardiac magnetic resonance
CRT	Chronic resynchronisation therapy
CT	Computerised tomography
CVA	Cerebrovascular accident
CVRU	Cardiovascular research unit
CW	Continuous wave
DCC	Direct current cardioversion
ECG	Electrocardiogram
EROA	Effective regurgitant orifice area
ESC	European society of cardiology
FBP	Full blood picture
HTM	Heart team meeting
LA	Left atrium
LFT	Liver function tests
LVEDD	Left ventricular end diastolic diameter

LVESD	Left ventricular end systolic diameter
LVEF	Left ventricular ejection fraction
LVSVI	Left ventricular stroke volume indexed
NOAC	Non-vitamin k antagonist oral anticoagulant
NYHA	New York heart association
OAC	Oral anticoagulant
PAF	Paroxysmal atrial fibrillation
PMC	Percutaneous mitral commissurotomy
PISA	Proximal isovelocity surface area
PVI	Pulmonary vein isolation
TAVI	Transcatheter aortic valve implantation
TFT	Thyroid function tests
TOE	Transoesphageal echocardiography
TTE	Transthoracic echocardiogram
TTR	Time in therapeutic range
VC	Vena contracta
VHD	Valvular heart disease
VKA	Vitamin K antagonist
SHSCT	Southern health and social care trust
U+E	Urea and Electrolytes

1.2 Key messages:

General messages:

- Precise evaluation of the patient's history, symptomatic status and physical examination are crucial for the diagnosis and management of VHD.
- Echocardiography is the key technique to diagnose VHD and assess its severity and prognosis. Other non-invasive investigations such as stress testing, CMR, CT, fluoroscopy and biomarkers are complementary, and invasive investigation beyond preoperative coronary angiography is restricted to situations where non-invasive evaluation is inconclusive.
- Risk stratification is essential for decision making to weigh the risk of intervention against the expected natural history of VHD.
- Decision making in elderly patients requires special considerations, including life expectancy and expected quality of life, with regards to comorbidities and general condition (frailty).
- NOACs may be used in patients with atrial fibrillation and aortic stenosis, aortic regurgitation, mitral regurgitation or aortic bioprostheses >3months after implantation but are contraindicated in mitral stenosis and mechanical valves.

Aortic stenosis:

- The diagnosis of severe aortic stenosis requires consideration of AVA together with flow rate, pressure gradients (the most robust measurement), ventricular function, size and wall thickness, degree of valve calcification and blood pressure, as well as functional status.
- The assessment of the severity of aortic stenosis in patients with low gradient and preserved ejection fraction remains particularly challenging.
- The strongest indication for intervention remains symptoms of aortic stenosis (spontaneous or on exercise testing).
- The presence of predictors of rapid symptom development can justify early surgery in asymptomatic patients, particularly when surgical risk is low.
- Although current data favour TAVI in elderly patients who are at increased risk for surgery, particularly when a transfemoral access is possible, the decision between TAVI and SAVR should be made by the Heart Team after careful, comprehensive evaluation of the patient, weighing individually the risks and benefits. Evidence is emerging on intermediate and low surgical risk patients.

Aortic regurgitation:

- The evaluation of aortic regurgitation requires consideration of valve morphology and the mechanism and severity of regurgitation, including careful assessment of aortic dilatation.
- In asymptomatic patients with severe aortic regurgitation, careful follow-up of symptomatic status and LV size and function is mandatory.

- The strongest indication for valve surgery is the presence of symptoms (spontaneous or on exercise testing) and/or the documentation of LVEF<50% and/or end-systolic diameter >50mm.
- In patients with a dilated aorta, definition of the aortic pathology and accurate measurements of aortic diameters are crucial to guide the timing and type of surgery.
- Aortic valve repair and valve-sparing aortic surgery instead of aortic valve replacement should be considered in selected cases in experienced centres.

Mitral stenosis:

- Most patients with severe mitral stenosis and favourable valve anatomy currently undergo percutaneous mitral commissurotomy PMC.
- Decision making as to the type of intervention in patients with unfavourable anatomy is still a matter of debate and must take into account the multifactorial nature of predicting the results of PMC

Mitral regurgitation:

- Echocardiography is essential to assess the aetiology of mitral regurgitation, as well as valve anatomy and function. An integrative approach is needed to assess the severity of mitral regurgitation.
- Indication for intervention in primary mitral regurgitation is guided by symptoms and risk stratification that includes the assessment of ventricular function and size, atrial fibrillation, systolic pulmonary pressure and LA size.
- In secondary mitral regurgitation, there is no conclusive evidence for a survival benefit after mitral valve intervention. Mitral surgery is recommended concomitantly in patients with an indication for CABG and may be considered in patients who are symptomatic despite optimal medical therapy including CRT if indicated or who have a low surgical risk when revascularization is not indicated.
- Mitral valve repair is the preferred method, but mitral valve replacement should be considered in patients with unfavourable morphological characteristics.
- Outcomes of mitral valve repair depend on surgeon experience and centre-related volume.
- Percutaneous edge-to-edge repair may be considered in patients at high surgical risk.

Tricuspid regurgitation

- For appropriate management, secondary tricuspid regurgitation has to be clearly distinguished from primary tricuspid regurgitation.
- Similar to mitral regurgitation, primary tricuspid regurgitation requires intervention sufficiently early to avoid secondary damage of the RV, which is associated with poor outcome.

- Secondary tricuspid regurgitation should be liberally treated at the time of left-sided valve surgery.
- Consideration of isolated surgery of secondary tricuspid regurgitation after previous left-sided valve surgery requires comprehensive assessment of the underlying disease, pulmonary haemodynamics and RV function.

Prosthetic heart valves

- The choice between a mechanical prosthesis and a bioprosthesis should not overstress the role of age and should take into account the wishes of the informed patient.
- Patients with a mechanical prosthesis require lifelong treatment using a VKA with a target INR adapted to the prosthesis and patient characteristics.
- Low-dose aspirin should be added to VKA only in selected patients with a mechanical prosthesis who have atherosclerosis or recurrent embolism.
- The risk of thromboembolism and bleeding is higher during the postoperative period and requires increased awareness of the monitoring of anticoagulant therapy.
- The management of anticoagulant therapy during non-cardiac surgery should be adapted to the type of surgery. Minor surgical procedures generally do not require interruption of anticoagulation.

2. Introduction

Decision making in valvular heart disease (VHD) involves accurate diagnosis, timing of intervention, risk assessment and, based on these, selection of the most suitable type of intervention.

Many factors ultimately determine the most appropriate treatment in individual patients within a given community. These factors include the availability of diagnostic equipment, the expertise of cardiologists and surgeons, especially in the field of valve repair and percutaneous intervention and, notably, the wishes of well-informed patients. Furthermore, owing to the lack of evidence based data in the field of VHD, most recommendations are largely the result of expert consensus opinion.

The aims of the evaluation of patients with VHD are to diagnose, quantify and assess the mechanism of VHD as well as its consequences. Decision making for intervention should be made by a heart team meeting (HTM) with a particular expertise in VHD, comprising cardiologists, cardiac surgeons, imaging specialists and anaesthetists. The HTM approach is particularly advisable in the management of high-risk patients.

The essential questions in the evaluation of a patient for valvular intervention are summarised in table 1.

Table 1: Essential questions in the evaluation of patients for valvular intervention

Questions
• How severe is VHD?
• What is the aetiology of VHD?
• Does the patient have symptoms?
• Are symptoms related to valvular disease?
• Are any signs present in asymptomatic patients that indicate a worse outcome if the intervention is delayed?
• What are the patient's life expectancy[a] and expected quality of life?
• Do the expected benefits of intervention (versus spontaneous outcome) outweigh its risks?
• What is the optimal treatment modality? Surgical valve replacement (mechanical or biological), surgical valve repair, or catheter intervention?
• Are local resources (local experience and outcome data for a given intervention) optimal for the planned intervention?
• What are the patient's wishes?

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3. Purpose of this policy

VHD and its associated symptoms of dyspnoea, palpitations, dizziness and chest discomfort are very common presenting complaints to the emergency department and/or acute medical unit. Assessment of these patients with VHD should include clinical evaluation, 12 lead electrocardiogram (ECG) and transthoracic echocardiogram (TTE) evaluation. Prompt therapy should be planned and instigated to minimise associated mortality and morbidity.

This policy aims to assist the attending health care professionals in treating patients with VHD in both the acute and chronic setting.

4. Scope

This document provides guidance for any professional involved in the clinical management of patients presenting to either primary or secondary care with AF. This will include:

- Consultants
- SAS doctors
- SpRs
- Junior Doctors
- Specialist Nurses
- Nursing Staff
- General Practitioners

5. Patient evaluation

Precise evaluation of the patient's history and symptomatic status as well as proper physical examination, in particular auscultation and search for heart failure signs, are crucial for the diagnosis and management of VHD. In addition, assessment of the extra cardiac conditions and comorbidities require particular attention.

5.1 Echocardiography

Following adequate clinical evaluation, TTE is the key technique used to confirm the diagnosis of VHD as well as to assess its severity and prognosis. An integrated approach including various criteria is strongly recommended instead of referring to single measurements. Echocardiography is also key to assess valve morphology and function as well as to evaluate the feasibility and indications of a specific intervention. Indices of left ventricular enlargement and function are strong prognostic factors. Pulmonary artery pressure should be estimated as well as right ventricular function. Transoesophageal echocardiography (TOE) should be considered when TTE is of suboptimal quality or when thrombosis, prosthetic valve dysfunction or endocarditis is suspected. The following are deemed essential for evaluation of VHD:

- Assessment of valve morphology: tricuspid, bicuspid, unicuspid or quadricuspid valve.
- Determination of the direction of the regurgitation (central or eccentric).
- Identification of the mechanism.
- Quantification of stenosis or regurgitation should follow an integrated approach considering all qualitative, semi quantitative and quantitative parameters.
- Measurement of LV function and dimensions. Indexing LV diameters for body surface area (BSA) is recommended.
- New parameters obtained by three-dimensional (3D) echocardiography, tissue Doppler and strain rate imaging may be useful, particularly in patients with borderline left ventricular ejection fraction (LVEF), where they may help in the decision for surgery.
- Measurement of the aortic root and ascending aorta (leading edge to leading edge at end diastole) at four levels: annulus, sinuses of Valsalva, sinotubular junction and tubular ascending aorta.
- The calculation of indexed values has been recommended to account for body size.

5.2 Stress testing

The primary purpose of exercise testing is to unmask the objective occurrence of symptoms in patients who claim to be asymptomatic or have non-specific symptoms, and is especially useful for risk stratification in aortic stenosis. Exercise testing will also determine the level of recommended physical activity, including participation in sports. Exercise echocardiography may identify the cardiac origin of dyspnoea. The prognostic impact has been shown mainly for aortic stenosis and mitral regurgitation. The search for flow reserve (also called 'contractile reserve') using low-dose dobutamine stress echocardiography is useful for assessing aortic stenosis severity and for operative risk stratification in low-gradient aortic stenosis with impaired LV function as well as to assess the potential of reverse remodelling in patients with heart failure and functional mitral regurgitation after a mitral valve procedure.

5.3 Cardiac MRI (CMR)

In patients with inadequate echocardiographic quality or discrepant results, cardiac magnetic resonance (CMR) should be used to assess the severity of valvular lesions, particularly regurgitant lesions, and to assess ventricular volumes, systolic function, abnormalities of the ascending aorta and myocardial fibrosis. CMR is the reference method for the evaluation of RV volumes and function and is therefore particularly useful to evaluate the consequences of tricuspid regurgitation.

5.4 Cardiac CT

Cardiac computerised tomography (CT) may contribute to evaluation of the severity of valve disease, particularly in aortic stenosis, and of the thoracic aorta. Cardiac CT plays an important role in the workup of patients with VHD considered for transcatheter intervention, in particular transcatheter aortic valve implantation (TAVI), and provides valuable information for pre-procedural planning. Owing to its high negative predictive value, CT may be useful to rule out coronary artery disease (CAD) in patients who are at low risk of atherosclerosis.

5.5 Cinefluroscopy

Cinefluoroscopy is particularly useful for assessing the kinetics of the occluders of a mechanical prosthesis.

5.6 Biomarkers

B-type natriuretic peptide (BNP) serum levels are related to New York Heart Association (NYHA) functional class and prognosis, particularly in aortic stenosis and mitral regurgitation. Natriuretic peptides may be of value for risk stratification and timing of intervention, particularly in asymptomatic patients.

5.7 Coronary angiography

Coronary angiography is indicated for the assessment of CAD when surgery or an intervention is planned, to determine if concomitant coronary revascularization is indicated (see following table of recommendations). Alternatively, CT can be used to rule out CAD in patients at low risk for the condition.

5.8 Risk stratification

Risk stratification applies to any sort of intervention and is required for weighing the risk of intervention against the expected natural history of VHD as a basis for decision making. Most experience relates to surgery and TAVI. The EuroSCORE I (http://www.euroscore.org/) overestimates operative mortality and its calibration of risk is poor. Consequently, it should no longer be used to guide decision making. The EuroSCORE II and the Society of Thoracic Surgeons (STS) score (http://riskcalc.sts.org/stswebriskcalc/#/) more accurately discriminate high and low risk surgical patients and show better calibration to predict postoperative outcome after valvular surgery.

6. Aortic stenosis

Aortic stenosis is the most common primary valve disease leading to surgery or catheter intervention in Europe and North America, with a growing prevalence due to the ageing population. Echocardiography is the key diagnostic tool. It confirms the presence of aortic stenosis; assesses the degree of valve calcification, LV function and wall thickness; detects the presence of other associated valve disease or aortic pathology and provides prognostic information. Doppler echocardiography is the preferred technique for assessing the severity of aortic stenosis.

6.1 Types of aortic stenosis

- High-gradient aortic stenosis (valve area <1.0 cm2, peak gradient >64mmHg, mean gradient >40mmHg). Severe aortic stenosis can be assumed irrespective of whether LVEF and flow are normal or reduced.

- Low-flow, low-gradient aortic stenosis with reduced ejection fraction (valve area <1.0 cm2, peak gradient < 64mmHg, mean gradient <40mmHg, ejection fraction <50%, LV stroke volume index (LVSVi) <35mL/m2). Low-dose dobutamine echocardiography is recommended in this setting to distinguish truly severe aortic stenosis from pseudosevere aortic stenosis, which is defined by an increase to an AVA of> 1.0 cm2 with flow normalization. In addition, the presence of flow reserve (also termed contractile reserve; increase of stroke volume >20%) has prognostic implications because it is associated with better outcome.

- Low-flow, low-gradient aortic stenosis with preserved ejection fraction (valve area <1.0 cm2, mean gradient <40mmHg, ejection fraction >50%, LVSVi <35mL/m2). This is typically encountered in the elderly and is associated with small ventricular size, marked LV hypertrophy and frequently a history of hypertension. The diagnosis of severe aortic stenosis in this setting remains challenging and requires careful exclusion of measurement errors and other reasons for such echocardiographic findings. The degree of valve calcification by MSCT is related to aortic stenosis severity and outcome. Its assessment has therefore gained increasing importance in this setting.

- Normal-flow, low-gradient aortic stenosis with preserved ejection fraction (valve area <1.0 cm2, mean gradient <40mmHg, ejection fraction >50%, LVSVi >35mL/m2). These patients will in general have only moderate aortic stenosis.

Figure 1 and table 2 highlights a practical approach to assessment of aortic stenosis severity.

Figure 1: A practical stepwise approach for the assessment of aortic stenosis severity

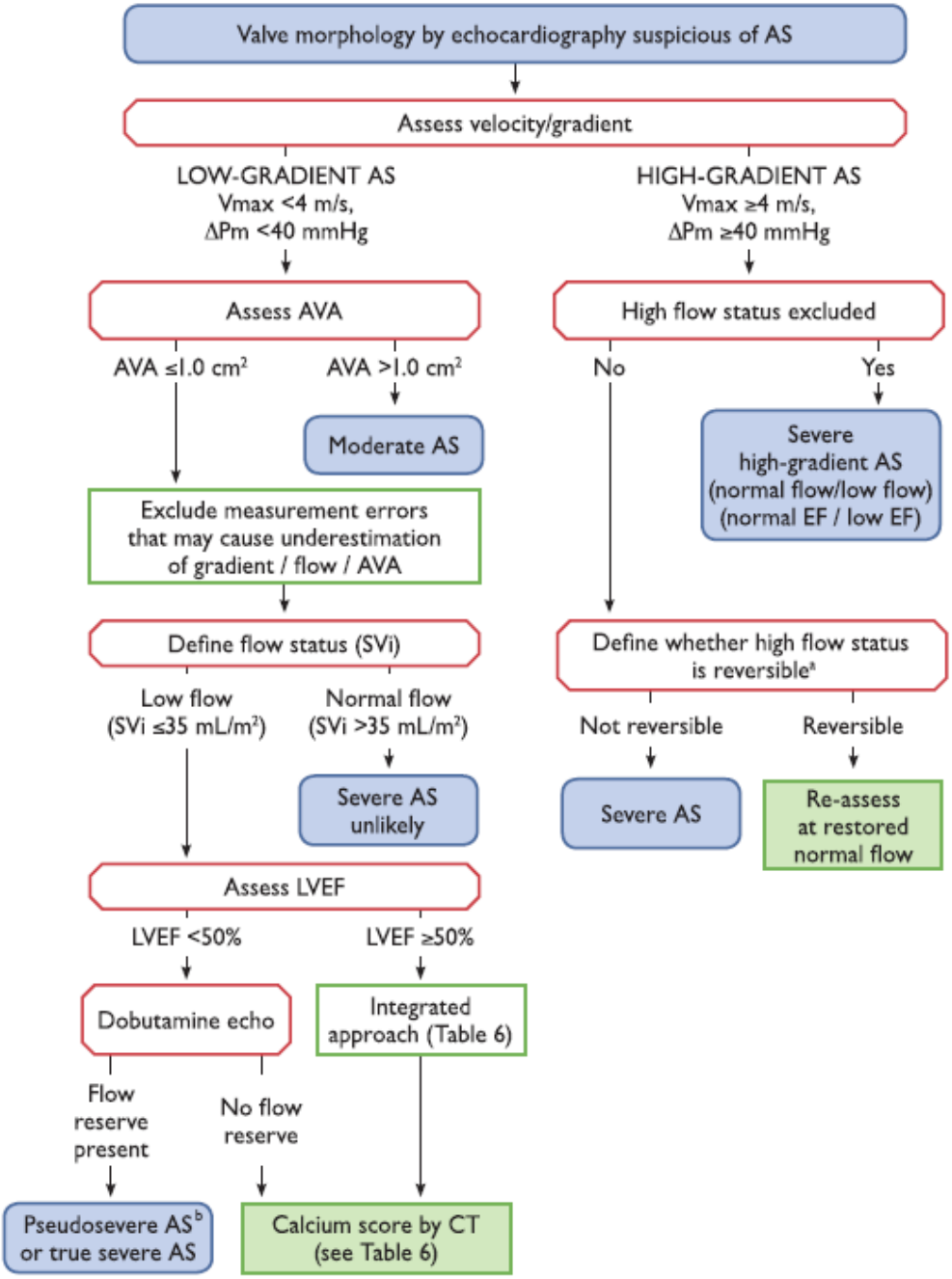

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Table 2: Parameters of aortic stenosis severity

Aortic stenosis	Mild	Moderate	Severe
Peak velocity (m/s)	<2.9	3.0-3.9	>4.0
Mean pressure drop (mmHg)	<25	25-40	>40
Valve area (cm^2)	1.5-2.0	1.0-1.4	<1.0
Velocity or VTI ratio	≥0.5	0.25-0.5	≤0.25

6.2 Indications for intervention

The diagnosis of severe aortic stenosis requires consideration of AVA together with flow rate, peak and mean pressure gradients (the most robust measurement), ventricular function, size and wall thickness, degree of valve calcification and blood pressure, as well as functional status. The assessment of the severity of aortic stenosis in patients with low gradient and preserved ejection fraction remains particularly challenging.

The strongest indication for intervention remains symptoms of aortic stenosis (spontaneous or on exercise testing). The presence of predictors of rapid symptom development can justify early surgery in asymptomatic patients, particularly when surgical risk is low.

Although current data favour TAVI in elderly patients who are at increased risk for surgery, particularly when a transfemoral access is possible, the decision between TAVI and SAVR should be made by the HTM after careful, comprehensive evaluation of the patient, weighing individually the risks and benefits.

Asymptomatic patients

In the asymptomatic patient, the wide variability in the rate of progression of aortic stenosis stresses the need for patients to be carefully educated about the importance of follow-up and reporting symptoms as soon as they develop.

Exercise testing is recommended in physically active patients for unmasking symptoms and for risk stratification of asymptomatic patients with severe aortic stenosis. Stress tests should determine the recommended level of physical activity. Follow-up evaluation should focus on haemodynamic progression, LV function and hypertrophy and dimensions of the ascending aorta.

Asymptomatic severe aortic stenosis should be re-evaluated at least every 6months for the occurrence of symptoms (change in exercise tolerance, ideally using exercise testing if symptoms are doubtful) and change in echocardiographic parameters. Measurement of natriuretic peptides should be considered. In the presence of significant calcification, mild and moderate aortic stenosis should be re-evaluated yearly and have interval TTE. In younger patients with mild aortic stenosis and no significant calcification, intervals may be extended to 2–3 years. In the SHSCT this is usually facilitated though out-patient clinics or more recently the aortic valve surveillance clinic (AVS) clinic (see SHSCT aortic valve surveillance section 18 and appendix 3). Patients are then referred to discussion at the HTM if symptoms are progressing and TTE shows advancing disease.

- ✓ **General considerations:**

- Surgical aortic valve replacement (SAVR) is indicated in asymptomatic patients with severe aortic stenosis and systolic LV dysfunction (LVEF <50%) not due to another cause.
- SAVR is indicated in asymptomatic patients with severe aortic stenosis and an abnormal exercise test showing symptoms on exercise clearly related to aortic stenosis.
- SAVR is indicated in patients with moderate or severe aortic stenosis undergoing CABG or surgery of the ascending aorta or of another valve.
- SAVR should be considered in asymptomatic patients with normal ejection fraction and none of the above-mentioned exercise test abnormalities if the surgical risk is low and one of the following findings is present:

    - ✓ Very severe aortic stenosis defined by a Vmax >5.5 m/s
    - ✓ Severe valve calcification and a rate of Vmax progression ≥0.3 m/s/year
    - ✓ Markedly elevated BNP levels (>threefold age and sex corrected normal range) confirmed by repeated measurements without other explanations
    - ✓ Severe pulmonary hypertension (systolic pulmonary artery pressure at rest >60mmHg confirmed by invasive measurement) without other explanation.

Symptomatic patients

Early therapy should be strongly recommended in all symptomatic patients with severe aortic stenosis because of their poor prognosis. The only exceptions are patients with severe comorbidities indicating a survival of< 1 year and patients in whom severe comorbidities or their general condition at an advanced age make it unlikely that the intervention will improve quality of life or survival. As long as the mean gradient remains >40mmHg, there is virtually no lower ejection fraction limit for intervention, whether surgery or TAVI.

- ✓ **General considerations:**

- Intervention is indicated in symptomatic patients with severe, high-gradient aortic stenosis (peak gradient > 64mmHg, mean gradient >40mmHg or peak velocity >4.0 m/s).
- Intervention is indicated in symptomatic patients with severe low-flow, low-gradient (<40 mmHg) aortic stenosis with reduced ejection fraction and evidence of flow (contractile) reserve excluding pseudosevere aortic stenosis. LV function usually improves after intervention.
- Intervention should be considered in symptomatic patients with low-flow, low-gradient (<40 mmHg) aortic stenosis with normal ejection fraction after careful confirmation of severe aortic stenosis. Data on their natural history and outcome after surgical or catheter intervention remain controversial. In such cases, intervention should only be performed when symptoms are present and if comprehensive evaluation suggests significant valve obstruction.
- Intervention should be considered in symptomatic patients with low-flow, low-gradient aortic stenosis and reduced ejection fraction without flow (contractile) reserve, particularly when CT calcium scoring confirms severe aortic stenosis.
- Intervention should not be performed in patients with severe comorbidities when the intervention is unlikely to improve quality of life or survival.

The management of patients with low-gradient aortic stenosis is more challenging.

- In patients with low-flow, low-gradient aortic stenosis and reduced ejection fraction in whom the depressed ejection fraction is predominantly caused by excessive afterload, LV function usually improves after intervention.
- Patients with low-flow, low-gradient aortic stenosis and preserved ejection fraction are the most challenging subgroup. Data on their natural history and outcome after surgical or catheter intervention remain controversial. In such cases, intervention should only be performed when symptoms are present and if comprehensive evaluation suggests significant valve obstruction.

6.3 Choice of intervention

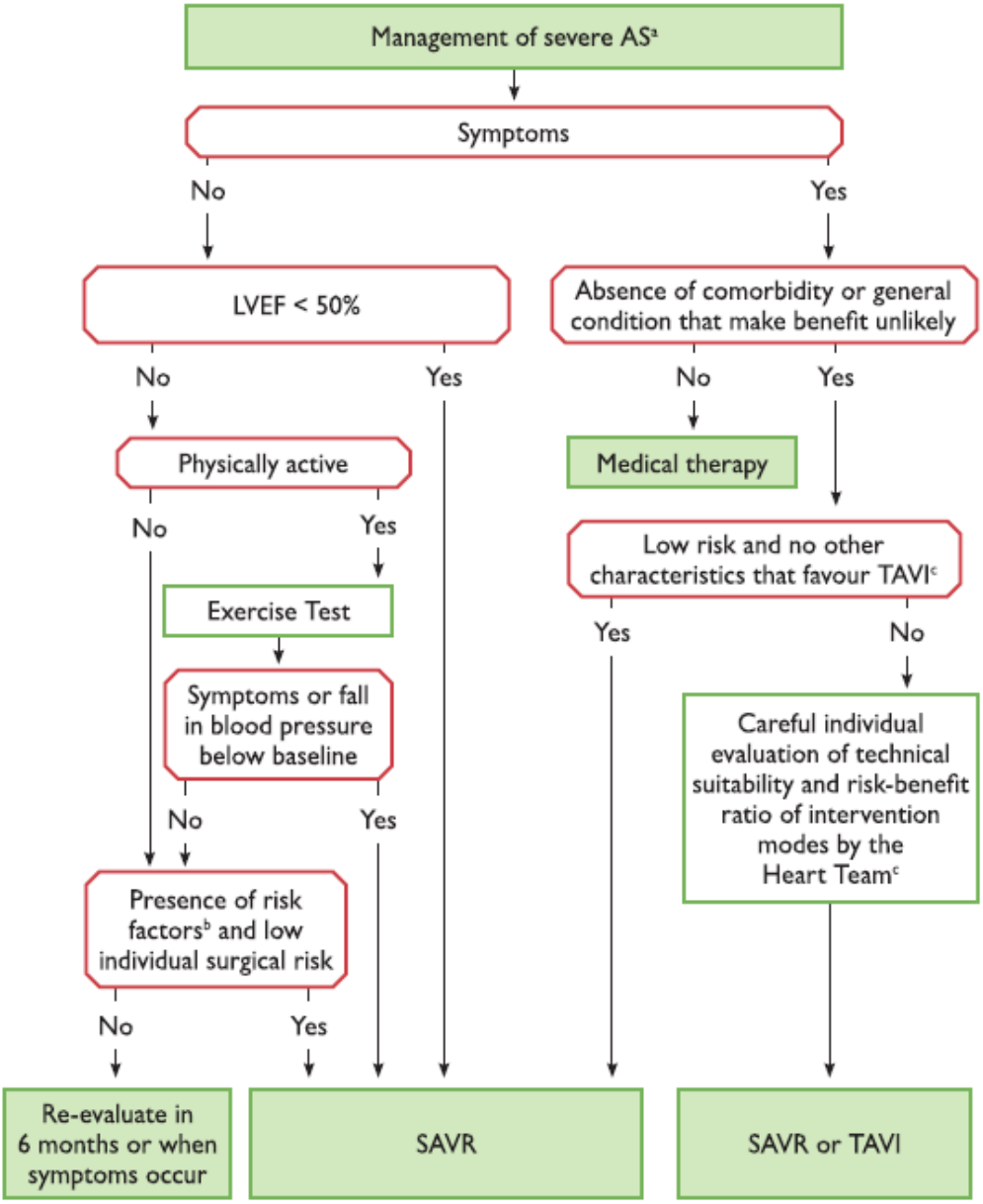
The choice of the intervention mode should take into account the cardiac and extracardiac characteristics of the patient, the individual risk of surgery, which is assessed by the judgement of the HTM in addition to scores, the feasibility of TAVI and the local experience and outcome data.

- The choice for intervention must be based on careful individual evaluation of technical suitability and weighing of risks and benefits of each modality.
- SAVR is recommended in patients at low surgical risk (STS or EuroSCORE II < 4% or logistic EuroSCORE I < 10% and no other risk factors not included in these scores, such as frailty, porcelain aorta, sequelae of chest radiation).
- TAVI is recommended in patients who are not suitable for SAVR as assessed by the HTM.

- In patients who are at increased surgical risk (STS or EuroSCORE II ≥4% or logistic EuroSCORE I ≥10% or other risk factors not included in these scores such as frailty, porcelain aorta, sequelae of chest radiation), the decision between SAVR and TAVI should be made by the HTM according to the individual patient, with TAVI being favoured in elderly patients suitable for transfemoral access.
- Evidence is evolving on the use of TAVI in intermediate and low risk patients.
- Balloon aortic valvotoplasty (BAV) may be considered as a bridge to SAVR or TAVI in haemodynamically unstable patients or in patients with symptomatic severe aortic stenosis who require urgent major non-cardiac surgery.

Figure 2 highlights the management of severe aortic stenosis.

Figure 2: Management of severe aortic stenosis



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6.4 Medical therapy

No medical therapy for aortic stenosis can improve outcome compared with the natural history. Randomised trials have consistently shown that statins do not affect the progression of aortic stenosis. Patients with symptoms of heart failure who are unsuitable candidates for surgery or TAVI or who are currently awaiting surgical or catheter intervention should be medically treated according to the heart failure guidelines. Coexisting hypertension should be treated. Medical treatment should be carefully titrated to avoid hypotension and patients should be re-evaluated frequently. Maintenance of sinus rhythm is important.

6.5TAVI workup

CT is the preferred imaging tool to assess the anatomy and dimensions of the aortic root, size and shape of the aortic valve annulus, its distance to the coronary ostia, the distribution of calcifications and the number of aortic valve cusps. It is essential to evaluate the feasibility of the various access routes, as this provides information on minimal luminal diameters, atherosclerotic plaque burden, the presence of aneurysms or thrombi, vessel tortuosity and thoracic and LV apex anatomy.

7. Aortic regurgitation

Aortic regurgitation (AR) can be caused by primary disease of the aortic valve cusps and/or abnormalities of the aortic root and ascending aortic geometry. Degenerative tricuspid and bicuspid aortic regurgitation are the most common aetiologies in Western countries, accounting for approximately two-thirds of the underlying aetiology of aortic regurgitation in the Euro Heart Survey on VHD. Other causes include infective and rheumatic endocarditis. Acute severe aortic regurgitation is mostly caused by infective endocarditis and less frequently by aortic dissection.

7.1 Anatomy and function of the aortic valve

The aortic valve consists of a complex of structures surrounding the aortic orifice along the outflow tract of the left ventricle. Typically, the valve has three cusps, which are semi-lunar in shape. Each cusp is attached along its curved edge, and the cusps meet at three commissures that are equally spaced along the circumference of the sleeve at the supra-aortic ridge. In normal aortic valve, the cusps are symmetrical mobile, and free at the commissures, with equal overlap on the closure. The cusps are named left, right, and non-coronary cusps based on the location of the coronary ostia.

7.2 Aetiology and mechanisms of aortic regurgitation

AR results from disease of either the aortic leaflets or the aortic root that distorts the leaflets and prevents their correct apposition. Common causes of leaflet abnormalities that result in AR include senile leaflet calcifications, bicuspid aortic valve, infective endocarditis, and rheumatic fever. Aortic causes of AR include annulo-aortic ectasia (idiopathic root dilatation), Marfan's syndrome, aortic dissection, collagen vascular disease, and syphilis.

7.3 Echocardiographic evaluation of aortic regurgitation

The parasternal long-axis view is classically used to measure the LV outflow tract, the aortic annulus, and the aortic sinuses dimensions. Leaflet thickness and morphology can be visualized from this window as well as from the parasternal short-axis view and the apical five-chamber view. However, not uncommonly, 2D TTE does not allow a complete assessment of the anatomy and causes of AR. In this situation, if the acoustic window is optimal, 3D echo could provide better delineation of the aortic valve morphology. In some cases, TOE is needed particularly when TTE is insufficient for assessing the mechanisms and causes of AR, as well as the aortic root dimensions and morphology

7.4 Assessment of severity of aortic regurgitation

Using colour flow Doppler, the regurgitant jet to LVOT (left ventricular outflow tract) ratio in diastole can be used for detection and initial visual assessment of AR. These measurements suffer from a high inter-observer variability. Central jets are suggestive of rheumatic disease, whereas eccentric jets are often associated with aortic valve prolapse or perforation. A jet/LVOT of ≥65% suggests severe AR whereas a jet/LVOT <25% suggests mild AR.

Imaging of the vena contracta (VC), the regurgitant jet as it traverses the aortic orifice or the effective regurgitant area, is obtained from the parasternal long-axis view. Practically, the VC represents the smallest flow diameter at the level of the aortic valve in the LV outflow tract, immediately below the flow convergence region. Using a Nyquist limit of 50–60 cm/s, a VC width of <3 mm correlates with mild AR, whereas a width >6 mm indicates severe AR. The measurement of the VC is affected by several factors as the presence of multiple jets. In this situation, the respective widths of the VC are not additive.

Aortic regurgitation can lead to diastolic flow reversal in the aorta. The flow reversal is best imaged in the upper descending aorta at the aortic isthmus level from a suprasternal view by using PW Doppler. Continuous wave (CW) Doppler of the AR jet is classically best obtained from the apical five-chamber view. A pressure half-time of <200 ms is consistent with severe AR, whereas a value >500 ms suggests mild AR.

The assessment of the flow convergence zone has been less extensively studied in AR than in mitral regurgitation. Imaging of the flow convergence zone is obtained from the apical three or five-chamber or parasternal long-axis or upper right parasternal views. The radius of the proximal isovelocity surface area (PISA) is measured at diastole using the first aliasing. The flow convergence or PISA method has several limitations. Firstly, it is not feasible in a

significant percentage of patients with AR due to interposition of valve tissue and difficulty in correctly identifying the flow convergence zone. Non-planar or confined flow convergence zones that invalidate the hemispheric assumption are potential causes of either under or over-estimation of AR severity by the PISA method. Accordingly, caution should be exercised when using the PISA method in patients with obtuse flow convergence angles, such as those with aneurysmal dilation of the ascending aorta or those with confined flow convergence zone such as could occur in patients with cusp perforation or commissural leaks. The PISA method is used to calculate the effective regurgitant orifice area (EROA), regurgitation volume and regurgitation fraction. Table 3 summaries the main parameters of AR severity.

Table 3: Parameters of AR severity

Aortic regurgitation	Mild	Moderate	Severe
Vena contracta width (cm)	<0.3		>0.6
Jet width/LVOT diam. (%)	<25		≥65
Regurgitant volume (mL)	≤30	31-59	≥60
Regurgitant fraction (%)	≤30	31-49	≥50
Regurgitant orifice area (cm^2)	≤0.10	0.11-0.29	≥0.30
Pressure half time (ms)	>500		<200
End Diastolic Velocity (upper DAo) (cm/s)			≥20

The presence of severe AR has significant haemodynamic effects, primarily on the LV. AR imposes additional volume load on the LV. In acute AR, the LV is classically not enlarged, while in the chronic situation, the LV progressively dilates and irreversible LV damage may occur. Hence, dilatation is sensitive for chronic significant AR while the normal size almost excludes severe chronic AR.

7.5 CT, CMR and dilated aorta

CMR should be used to quantify the regurgitant fraction when echocardiographic measurements are equivocal. In patients with aortic dilatation, gated cardiac CT is recommended to assess the maximum diameter. CMR can be used for follow-up, but indication for surgery should preferably be based on CT measurements. Different methods of aortic measurements have been reported and this may result in diameter discrepancies of 2–3mm that could influence therapeutic management. To improve reproducibility, it is recommended to measure diameters using the inner to inner edge technique at end diastole. Diameters at the annulus, sinus of Valsalva, sinotubular junction, tubular ascending aorta and aortic arch level should be reported.

7.6 Management of aortic regurgitation

Acute aortic regurgitation may require urgent surgery. It is primarily caused by infective endocarditis and aortic dissections. Specific ESC guidelines deal with these entities. The indications for intervention in chronic aortic regurgitation are related to symptoms, assessment of severity on TTE / TOE status of the LV or dilatation of the aorta. In symptomatic patients, surgery is recommended irrespective of the LVEF value, except for extreme cases, as long as aortic regurgitation is severe and the operative risk is not prohibitive.

All patients should be discussed at the HTM. Figure 3 summarises the management of severe aortic regurgitation. Although surgical aortic valve replacement (SAVR) is the standard procedure in the majority of patients with aortic regurgitation, valve repair or valve sparing surgery should be considered in patients with pliable noncalcified tricuspid or bicuspid valves who have a type I (enlargement of the aortic root with normal cusp motion) or type II (cusp prolapse) mechanism of aortic regurgitation. In experienced centres, valve-sparing root replacement and valve repair, when feasible, yield good long-term results with low rates of valve-related events as well as better quality of life.

In asymptomatic patients with severe aortic regurgitation, impairment of LV function (LVEF ≤50%) and LV enlargement with an LV end-diastolic diameter (LVEDD) >70mm or left ventricular end-systolic diameter (LVESD) >50mm are associated with worse outcome and surgery should therefore be pursued when these cut-offs are reached.

In patients with small body size, LVESD should be related to BSA and a cut-off of 25mm/m2 BSA appears to be more appropriate. In patients not reaching the thresholds for surgery, close follow-up is needed and exercise testing should be performed to identify borderline symptomatic patients. In truly asymptomatic patients, regular assessment of LV function and physical condition are crucial to identify the optimal time for surgery. A rapid progression of ventricular dimensions or decline in ventricular function on serial testing is a reason to consider surgery.

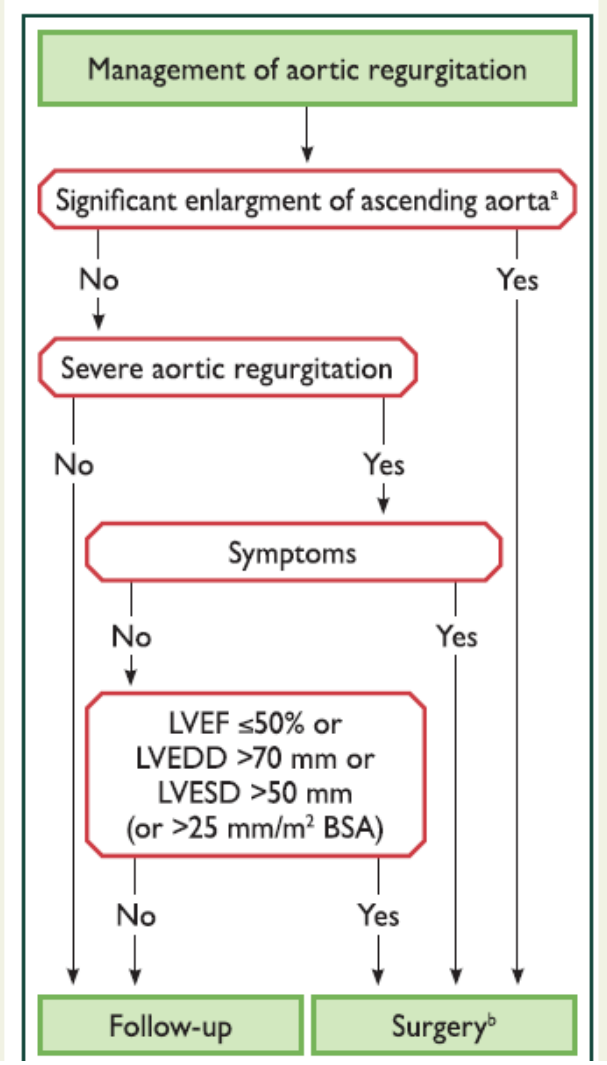
7.7 Management of concomitant aortic dilatation

In patients with a dilated aorta, the rationale for surgery has been best defined in patients with Marfan syndrome and root dilation. Root aneurysms need to have root replacement, with or without preservation of the native aortic valve, but definitely with coronary reimplantation. In contrast, tubular ascending aortic aneurysms require only a supracommissural tube graft replacement without coronary reimplantation.

In patients with aortic dilatation, the family history, age and anticipated risk of the procedure should be taken into consideration. In individuals with hypertensive aortopathy, prophylactic surgery should be considered with a diameter of ≥55mm. For a bicuspid aortic valve, prophylactic surgery should be considered with aortic diameters of ≥50mm especially when additional risk factors or coarctation are present. Surgery is indicated in all patients with Marfan syndrome and a maximal aortic diameter ≥50 mm. In patients with Marfan syndrome

and additional risk factors (e.g. family history of aortic dissection) and in patients with a TGFBR1 or TGFBR2 mutation (including Loeys–Dietz syndrome), surgery should be considered at a maximal aortic diameter ≥45mm.

Figure 3: Management of severe aortic regurgitation



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7.8 Medical therapy

Medical therapy can provide symptomatic improvement in individuals with chronic severe aortic regurgitation in whom surgery is not feasible. In patients who undergo surgery but continue to suffer from heart failure or hypertension, angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs) and beta-blockers are useful.

7.9 Serial testing

All asymptomatic patients with severe aortic regurgitation and normal LV function should be seen for follow-up at least every year. In patients with a first diagnosis, or if LV diameter and/or ejection fraction show significant changes or come close to thresholds for surgery, follow-up should be continued at 3–6-month intervals. In inconclusive cases, BNP may be helpful, as its elevation during follow up has been related to deterioration of LV function. Patients with mild to moderate aortic regurgitation can be reviewed on a yearly basis and echocardiography performed every 2 years. If the ascending aorta is dilated (>40mm) it is recommended to perform CT or CMR. Follow-up assessment of the aortic dimension should be performed using echocardiography and/or CMR. Any increase >3mm should be validated by CT angiography/CMR and compared to baseline data.

8 Mitral stenosis

The incidence of rheumatic mitral stenosis has greatly decreased in industrialized countries. Degenerative calcific mitral valve disease is now encountered mainly in elderly patients. Percutaneous mitral commissurotomy has had a significant impact on the management of rheumatic mitral stenosis.

8.1 Evaluation of mitral stenosis

Echocardiography is the preferred method for diagnosing mitral stenosis and for assessing its severity and haemodynamic consequences. However, several specific issues should be considered. Valve area using planimetry is the reference measurement of mitral stenosis severity, whereas mean transvalvular gradient and pulmonary pressures reflect its consequences and have a prognostic value. TTE / TOE usually provides sufficient information for assessment. Stress testing is indicated in patients with no symptoms or symptoms equivocal or discordant with the severity of mitral stenosis. Exercise echocardiography may provide additional objective information by assessing changes in mitral gradient and pulmonary artery pressure.

Table 4 highlights the TTE parameters of mitral stenosis severity. Severe mitral stenosis equates to a pressure half time of ≥220ms, a mean gradient of >10mmHg and a mitral valve area of <1.0cm2.

Table 4: TTE parameters of mitral stenosis severity

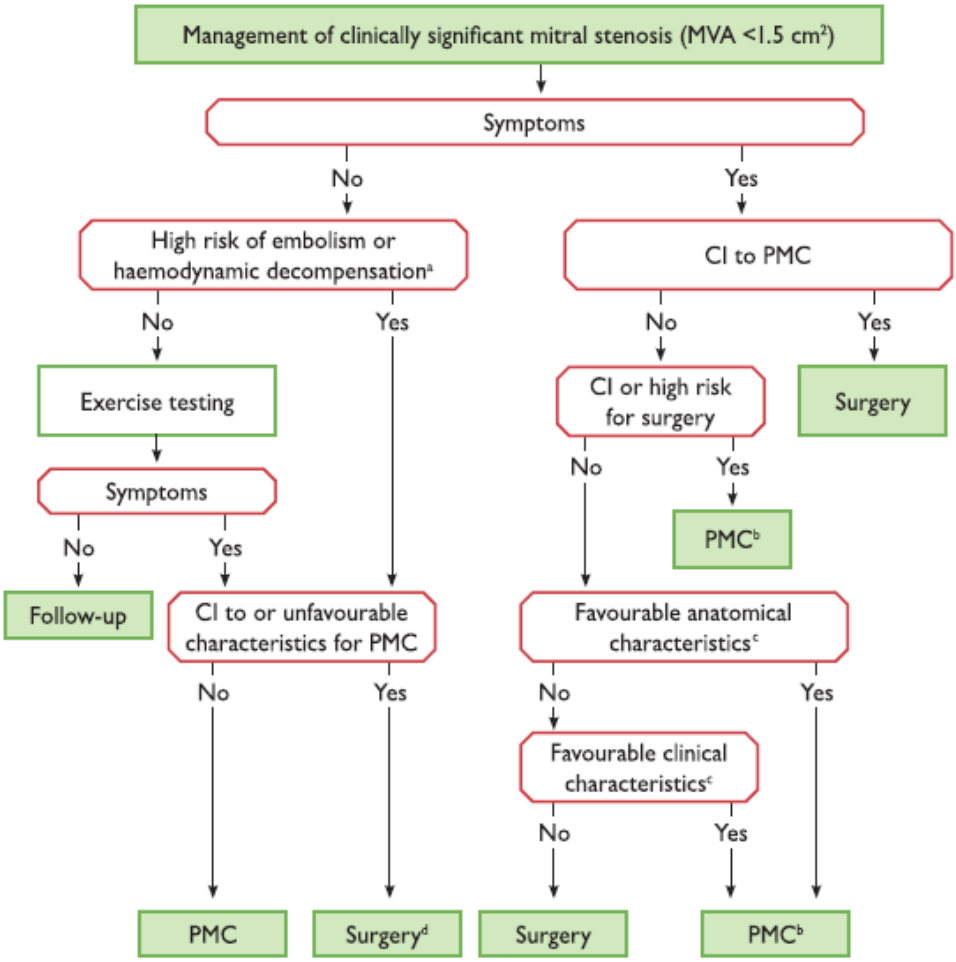
Mitral stenosis

	Mild	Moderate	Severe
Pressure half time (ms)	71-139	140-219	≥220
Mean pressure drop (mmHg)	<5	5-10	>10
Valve area (cm^2)	1.6-2.0	1.0-1.5	<1.0

8.2 Indications for intervention

The type of treatment, as well as its timing, should be decided on the basis of clinical characteristics, valve anatomy and local expertise. In general, indication for intervention should be limited to patients with clinically significant (moderate to severe) mitral stenosis (valve area <1.5 cm2). However, PMC may be considered in symptomatic patients with a valve area >1.5 cm2 if symptoms cannot be explained by another cause and if the anatomy is favourable. Intervention should be performed in symptomatic patients. Most patients with favourable valve anatomy currently undergo PMC, however, open commissurotomy may be preferred by experienced surgeons in young patients with mild to moderate mitral regurgitation. In patients with unfavourable anatomy, decision making as to the type of intervention is still a matter of debate and must take into account the multifactorial nature of predicting the results of PMC. Surgery, which is mostly valve replacement, is indicated in the other patients. Figure 4 highlights the management of mitral stenosis.

Figure 4: Management of mitral stenosis



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8.3 Medical therapy

Diuretics, beta-blockers, digoxin or heart rate–regulating calcium channel blockers can transiently improve symptoms. Anticoagulation with a target international normalized ratio (INR) between 2 and 3 is indicated in patients with either new-onset or paroxysmal atrial fibrillation. Patients with moderate to severe mitral stenosis and persistent atrial fibrillation should be kept on vitamin K antagonist (VKA) treatment and should not receive NOACs.

8.4 Serial testing

Asymptomatic patients with clinically significant mitral stenosis who have not undergone intervention should be followed up yearly by means of clinical and echocardiographic examinations and at longer intervals (2–3 years) in case of moderate stenosis. Management of patients after successful PMC is similar to that of asymptomatic patients. Follow-up should

be more frequent if asymptomatic restenosis occurs. When PMC is not successful, surgery should be considered early unless there are definite contraindications.

9. Mitral regurgitation

Mitral regurgitation is the second-most frequent indication for valve surgery in Europe. It is essential to distinguish primary from secondary mitral regurgitation, particularly regarding surgical and transcatheter interventional management.

9.1 Primary mitral regurgitation

In primary mitral regurgitation, one or several components of the mitral valve apparatus are directly affected. The most frequent aetiology is degenerative (prolapse, flail leaflet). Endocarditis as one of the causes of primary mitral regurgitation is discussed in specific ESC guidelines.

9.2 Evaluation in primary mitral regurgitation

Echocardiography is the principal investigation used to assess the severity and mechanism of mitral regurgitation, its consequences for the LV (function and remodelling), LA and pulmonary circulation, as well as the likelihood of repair. Quantification should be performed in an integrative way, including qualitative, semi-quantitative and quantitative parameters. TTE is diagnostic in most cases, but TOE is recommended, particularly in the presence of suboptimal image quality. Three dimensional echocardiography provides additional information for selecting the appropriate repair strategy. The consequences of mitral regurgitation on ventricular function are assessed by measuring LV size and ejection fraction. LA volume, systolic pulmonary artery pressure, tricuspid regurgitation and annular size and RV function are important additional parameters.

Severe primary mitral regurgitation is defined as a vena contracta >7mm, a PISA >1.0cm, a regurgitation volume of >60mls, a regurgitation fraction of >50% and an EROA >0.4cm2. In secondary mitral regurgitation, a regurgitation volume of >30mls and an EROA >0.2cm2 is considered severe. Table 5 summarises the parameters of mitral regurgitation severity.

Table 5: Parameters of mitral regurgitation severity.

Mitral regurgitation (Primary Organic)

	Mild	Moderate	Severe
Vena contracta (cm)	<0.3		≥0.7
PISA radius (Nyquist 40cm/s)	<0.4		>1.0
Regurgitant volume (mL)	≤30	31-59	≥60
Regurgitant fraction (%)	≤30	31-49	≥50
Regurgitant orifice area (cm^2)	<0.20	0.21-0.39	≥0.40
MV Inflow $^{(VTI)}$/LVOT$^{(VTI)}$			>1.4

In asymptomatic patients, the significant increase of pulmonary artery pressure with exercise (>60mmHg) has been reported to be of prognostic value. The use of global longitudinal strain could be of potential interest for the detection of subclinical LV dysfunction but is limited by inconsistent algorithms used by different echocardiographic systems. Neurohormonal activation is observed in mitral regurgitation, with a potential value of elevated BNP levels and a change in BNP as predictors of outcome (particularly of symptom onset). In particular, low plasma BNP has a high negative predictive value and may be helpful in the follow-up of asymptomatic patients.

9.3 Indications for intervention in primary mitral regurgitation

Urgent surgery is indicated in patients with acute severe mitral regurgitation. In the case of papillary muscle rupture as the underlying disease, valve replacement is in general required. Surgery is indicated in symptomatic patients with severe primary mitral regurgitation. An LVEF ≤60% or LVESD ≥45mm, atrial fibrillation and a systolic pulmonary pressure ≥50mmHg predict a worse postoperative outcome independent of the symptomatic status and have therefore become triggers for surgery in asymptomatic patients. Figure 5 highlights the management of primary mitral regurgitation.

Watchful waiting is a safe strategy in asymptomatic patients with severe primary mitral regurgitation and none of the above indications for surgery.

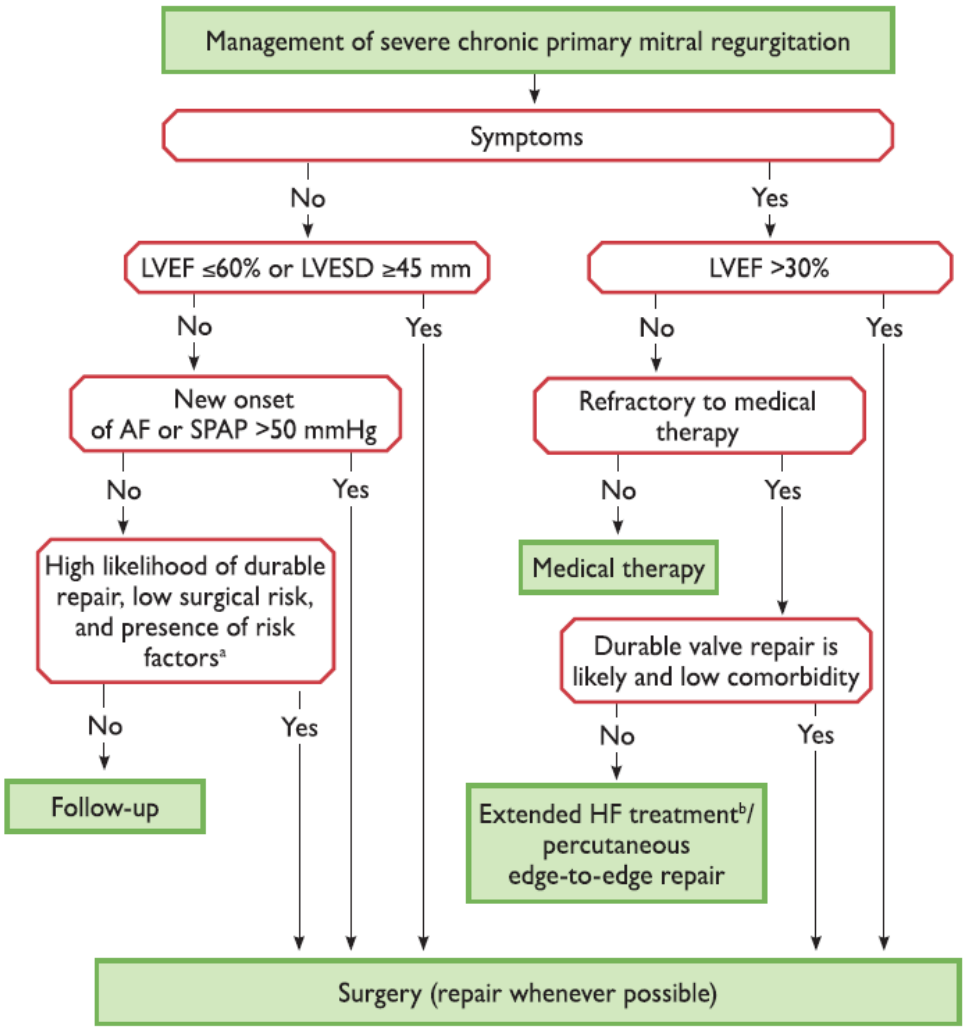
Despite the absence of a randomized comparison between the results of valve replacement and repair, it is widely accepted that, when feasible, valve repair is the preferred treatment. Achieving a durable valve repair is essential. Degenerative mitral regurgitation due to segmental valve prolapse can be repaired with a low risk of mitral regurgitation recurrence and reoperation. The reparability of rheumatic lesions, extensive valve prolapse and even more so mitral regurgitation with leaflet calcification or extensive annular calcification is more

challenging. Patients with a predictably complex repair should undergo surgery in experienced repair centres with high repair rates, low operative mortality and a record of durable results.

When repair is not feasible, mitral valve replacement with preservation of the subvalvular apparatus is favoured.

Transcatheter mitral valve interventions (mitraclip, Neochord) have been developed to correct primary mitral regurgitation either through a transseptal or a transapical approach. Among the transcatheter procedures, currently the edge-to-edge mitral repair is widely adopted. Experience with transcatheter annuloplasty, transapical chordal implantation or valve replacement is still limited and general recommendations cannot yet be made. Transcatheter mitral valve treatment should be discussed by the HTM in symptomatic patients who are at high surgical risk or are inoperable. Percutaneous edge-to-edge repair is generally safe and can improve symptoms and provide reverse LV remodelling. However, the rate of residual mitral regurgitation up to 5 years is higher than with surgical repair.

Figure 5: Management of primary mitral regurgitation



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9.4 Medical therapy

In acute mitral regurgitation, nitrates and diuretics are used to reduce filling pressures. Sodium nitroprusside reduces afterload and regurgitant fraction. Inotropic agents and an intra-aortic balloon pump are of use in hypotension and haemodynamic instability. In chronic mitral regurgitation with good ventricular function, there is no evidence to support the prophylactic use of vasodilators, including ACE inhibitors. However, ACE inhibitors should be considered when heart failure has developed in patients who are not suitable for surgery or when symptoms persist after surgery. Beta blockers and spironolactone (or eplerenone) should also be considered as appropriate.

9.5 Serial testing

Asymptomatic patients with severe mitral regurgitation and LVEF >60% should be followed clinically and echocardiographically every 6 months. When guideline indications for surgery are reached, early surgery is associated with better outcomes. Asymptomatic patients with moderate mitral regurgitation and preserved LV function can be followed on a yearly basis and echocardiography should be performed every 1–2 years.

9.6 Secondary mitral regurgitation

In secondary mitral regurgitation (previously also referred to as 'functional mitral regurgitation'), the valve leaflets and chordae are structurally normal and mitral regurgitation results from an imbalance between closing and tethering forces on the valve secondary to alterations in LV geometry. It is most commonly seen in dilated or ischaemic cardiomyopathies. Annular dilatation in patients with chronic atrial fibrillation and LA enlargement can also be an underlying mechanism.

9.7 Evaluation in secondary mitral regurgitation

Echocardiography is essential to establish the diagnosis of secondary mitral regurgitation. In secondary mitral regurgitation, lower thresholds have been proposed to define severe mitral regurgitation compared with primary mitral regurgitation (EROA >0.2cm2, regurgitation volume >30mls), owing to their association with prognosis. However, it is unclear if prognosis is independently affected by mitral regurgitation compared with LV dysfunction. So far, no survival benefit has been confirmed for reduction of secondary mitral regurgitation. For isolated mitral valve treatment (surgery or percutaneous edge-to-edge repair) in secondary mitral regurgitation, thresholds of severity of mitral regurgitation for intervention still need to be validated in clinical trials. The severity of secondary mitral regurgitation should be reassessed after optimized medical treatment. The severity of tricuspid regurgitation and RV size and function should also be evaluated.

9.8 Indications for intervention in secondary mitral regurgitation

The presence of chronic secondary mitral regurgitation is associated with impaired prognosis. However, in contrast to primary mitral regurgitation, there is currently no evidence that a reduction of secondary mitral regurgitation improves survival. The limited data regarding secondary mitral regurgitation result in a lower level of evidence for treatment recommendations and highlight the importance of decision making by the HTM. Heart failure specialists should be involved. In patients with CAD undergoing revascularization, the evaluation and decision to treat (or not to treat) ischaemic mitral regurgitation should be made before surgery, as general anaesthesia may significantly reduce the severity of regurgitation. When mitral regurgitation severity is assessed intraoperatively, the use of acute volume challenge and an increase in afterload may be helpful. The optimal surgical approach remains controversial. While mitral valve repair with an undersized complete ring to restore leaflet coaptation and valve competence is the preferred technique, valve

replacement should be considered in patients with echocardiographic risk factors for residual or recurrent mitral regurgitation.

9.9 Medical therapy in secondary mitral regurgitation

Optimal medical therapy in line with the guidelines for the management of heart failure should be the first step in the management of all patients with secondary mitral regurgitation. Indications for CRT should be evaluated in accordance with related guidelines. If symptoms persist after optimization of conventional heart failure therapy, options for mitral valve intervention should be evaluated.

10. Tricuspid stenosis

Tricuspid stenosis is often combined with tricuspid regurgitation, most frequently of rheumatic origin. It is therefore almost always associated with left-sided valve lesions, particularly mitral stenosis, that usually dominate the clinical presentation. Other causes are rare, including congenital, drug-induced valve diseases, Whipple's disease, endocarditis and large right atrial tumour.

10.1 Evaluation of tricuspid stenosis

Echocardiography provides the most useful information. Tricuspid stenosis is often overlooked and requires careful evaluation. Echocardiographic evaluation of the anatomy of the valve and its subvalvular apparatus is important to assess valve reparability. No generally accepted grading of tricuspid stenosis severity exists, but a mean gradient >_5mmHg at normal heart rate is considered indicative of clinically significant tricuspid stenosis. Catheterization is no longer used for evaluating the severity of tricuspid stenosis.
11.2 Indications for intervention. Table 6 summarises tricuspid stenosis severity.

Table 6: Parameters of tricuspid stenosis

Tricuspid stenosis	
	Severe
Mean pressure drop (mmHg)	≥5
Inflow velocity-time integral (cm)	>60
Valve area (cm^2)	<1.0

10.2 Intervention in tricuspid stenosis

The lack of pliable leaflet tissue is the main limitation for valve repair. Even though this is still a matter of debate, biological prostheses for valve replacement are usually preferred over mechanical ones because of the high risk of thrombosis carried by the latter and the satisfactory long-term durability of the former in the tricuspid position. Percutaneous balloon tricuspid dilatation has been performed in a limited number of cases, either alone or alongside PMC, but frequently induces significant regurgitation. There is a lack of data on long-term results. Intervention on the tricuspid valve is usually carried out at the time of intervention on the other valves in patients who are symptomatic despite medical therapy. The choice between repair or valve replacement depends on valve anatomy and surgical expertise.

10.3 Medical therapy

Diuretics are useful in the presence of heart failure but are of limited long-term efficacy.

11. Tricuspid regurgitation

Pathological tricuspid regurgitation is more often secondary, due to RV dysfunction following pressure and/or volume overload in the presence of structurally normal leaflets. Possible causes of primary tricuspid regurgitation are infective endocarditis (especially in intravenous drug addicts), rheumatic heart disease, carcinoid syndrome, myxomatous disease, endomyocardial fibrosis, Ebstein's anomaly and congenitally dysplastic valves, drug-induced valve diseases, thoracic trauma and iatrogenic valve damage.

11.1 Evaluation of tricuspid regurgitation

Echocardiography is the ideal technique to evaluate tricuspid regurgitation. In primary tricuspid regurgitation, the aetiology can usually be identified from specific abnormalities of the valve structure. In secondary tricuspid regurgitation, the degree of dilatation of the annulus, the RV dimension and function and the degree of tricuspid valve deformation should be measured.

Severe tricuspid regurgitation is defined as a vena contracta >7mm, a regurgitation volume of >45mls, an EROA >0.4cm2, a dilated right atrium and right ventricle and systolic flow reversal in the inferior vena cava. Table 7 summarises the parameters of mitral regurgitation severity.

Table 7: Parameters of tricuspid regurgitation

Tricuspid regurgitation	Mild	Moderate	Severe
VC width (cm)	Not defined	<0.7	>0.7
EROA (mm^2)			≥40
Regurgitant volume (mL)			≥45
CW jet density/contour	Soft/ parabolic	Dense/ variable	Dense/ triangular early peaking
RA/RV/IVC size	Normal	Normal/dilated	Usually dilated
Hepatic vein flow	Systolic dominance	Systolic blunting	Systolic reversal

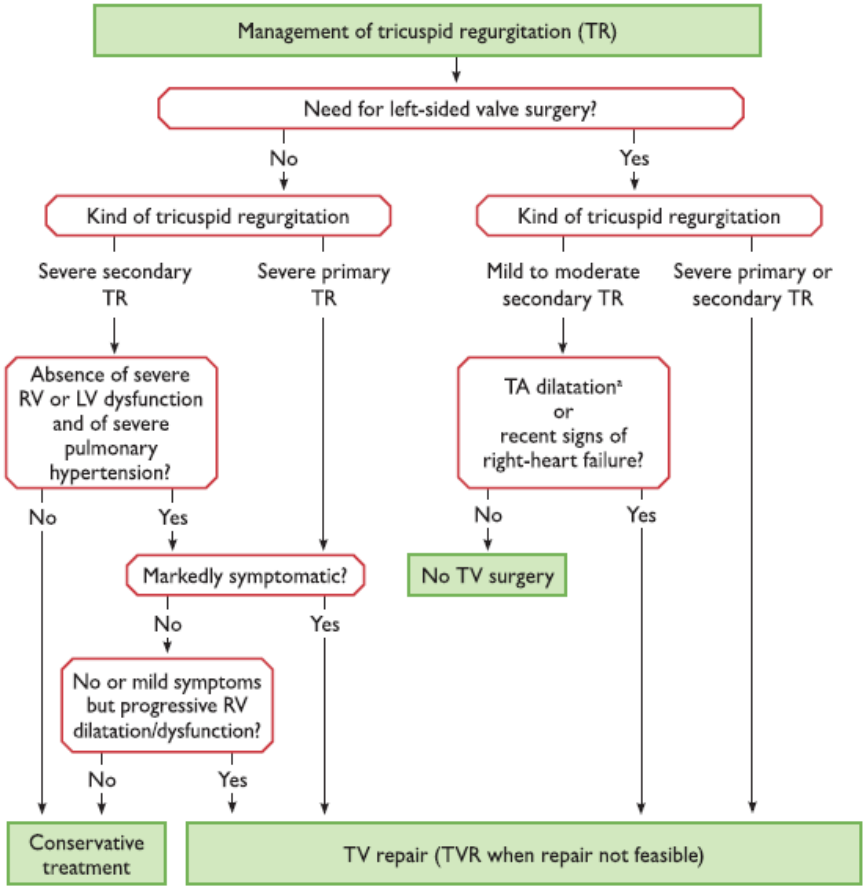
11.2 Indications for intervention

The timing of surgical intervention remains controversial, mostly due to the limited data available and their heterogeneous nature. Surgery should be carried out sufficiently early to avoid irreversible RV dysfunction. In severe primary tricuspid regurgitation, surgery is not only recommended in symptomatic patients but should also be considered in asymptomatic patients when progressive RV dilatation or decline of RV function is observed. Although these patients respond well to diuretic therapy, delaying surgery is likely to result in irreversible RV damage, organ failure and poor results of late surgical intervention.

In secondary tricuspid regurgitation, adding a tricuspid repair, if indicated, during left-sided surgery does not increase operative risk and has been demonstrated to provide reverse remodelling of the RV and improvement of functional status even in the absence of substantial tricuspid regurgitation when annulus dilatation is present. It should therefore be performed liberally.

If possible, valve repair is preferable to valve replacement. Ring annuloplasty, preferably with prosthetic rings, is key to surgery for secondary tricuspid regurgitation. Valve replacement should be considered when the tricuspid valve leaflets are significantly tethered and the annulus is severely dilated. Figure 6 highlights the management of tricuspid regurgitation.

Figure 6: Management of tricuspid regurgitation



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12. Prosthetic heart valves

Every valve prosthesis introduces a new disease process. In practice, the choice is between a mechanical and a biological prosthesis. Randomized trials comparing both prostheses consistently found similar survival, no significant difference in rates of valve thrombosis and thromboembolism, higher rates of bleeding with mechanical prostheses and higher rates of reintervention with bioprostheses.

12.1 Choice of prosthetic valve

The choice between a mechanical and a biological valve in adults is determined mainly by estimating the risk of anticoagulation-related bleeding and thromboembolism with a mechanical valve versus the risk of structural valve deterioration with a bioprosthesis and by considering the patient's lifestyle and preferences. Rather than setting arbitrary age limits, prosthesis choice should be discussed in detail with the informed patient, cardiologists and surgeons, taking into account the factors detailed below (see tables of recommendations in section 11.1). Bioprostheses should be considered in patients whose life expectancy is lower than the presumed durability of the bioprosthesis, particularly if comorbidities may necessitate further surgical procedures, and in those with increased bleeding risk. In women who wish to become pregnant, the high risk of thromboembolic complications with a mechanical prosthesis during pregnancy and the low risk of elective reoperation are incentives to consider a bioprosthesis, despite the rapid occurrence of structural valve deterioration in this age group.

12.2 Management after valve intervention

Thromboembolism and anticoagulant-related bleeding present the majority of complications experienced by prosthetic valve recipients.

All patients require lifelong follow-up by a cardiologist after valve surgery to detect early deterioration in prosthetic function or ventricular function or progressive disease of another heart valve. Clinical assessment should be performed yearly or as soon as possible if new cardiac symptoms occur. TTE should be performed if any new symptoms occur after valve replacement or if complications are suspected.

After transcatheter as well as surgical implantation of a bioprosthetic valve, echocardiography, including the measurement of transprosthetic gradients, should be performed within 30 days after valve implantation (i.e. baseline imaging), at 1 year / 3 years / 5 years after implantation and annually thereafter. TOE should be considered if TTE is of poor quality and in all cases of suspected prosthetic dysfunction or endocarditis. Cinefluoroscopy for mechanical valves provides useful additional information if valve thrombus or pannus are suspected to impair valve function.

Antithrombotic management should address effective control of modifiable risk factors for thromboembolism in addition to the prescription of antithrombotic drugs.

12.3 Management of valve thrombosis

Obstructive valve thrombosis should be suspected promptly in any patient with any type of prosthetic valve who presents with recent dyspnoea or an embolic event. The diagnosis should be confirmed by TTE and TOE, cinefluoroscopy or CT scan if promptly available. The management of mechanical prosthetic valve thrombosis is high risk, whatever the option taken. Surgery is high risk because it is most often performed under emergency conditions and is a reintervention. On the other hand, fibrinolysis carries risks of bleeding, systemic embolism and recurrent thrombosis that are higher than after surgery. Emergency valve

replacement is recommended for obstructive prosthetic valve thrombosis in critically ill patients without a contraindication to surgery (see figure 7).

Management of non-obstructive mechanical prosthetic valve thrombosis depends mainly on the occurrence of a thromboembolic event and the size of the thrombus (see figure 8). Surgery should be considered for a large (>10mm) non-obstructive prosthetic valve thrombus complicated by embolism or which persists despite optimal anticoagulation. Fibrinolysis may be considered if surgery is at high risk but carries a risk of bleeding and thromboembolism.

Valve thrombosis occurs mainly in mechanical prostheses. However, cases of thrombosis of bioprostheses have been reported after surgery or transcatheter valve implantation. Subclinical thrombosis of bioprostheses may be more frequent when assessed by cardiac CT and subclinical thrombosis of TAVI prostheses can be associated with a moderate increase in transprosthetic gradients, but the clinical consequences are unknown. Anticoagulation using a VKA and/or UFH is the first-line treatment of bioprosthetic valve thrombosis.

Figure 7: Management of left sided obstructive mechanical prosthetic thrombosis

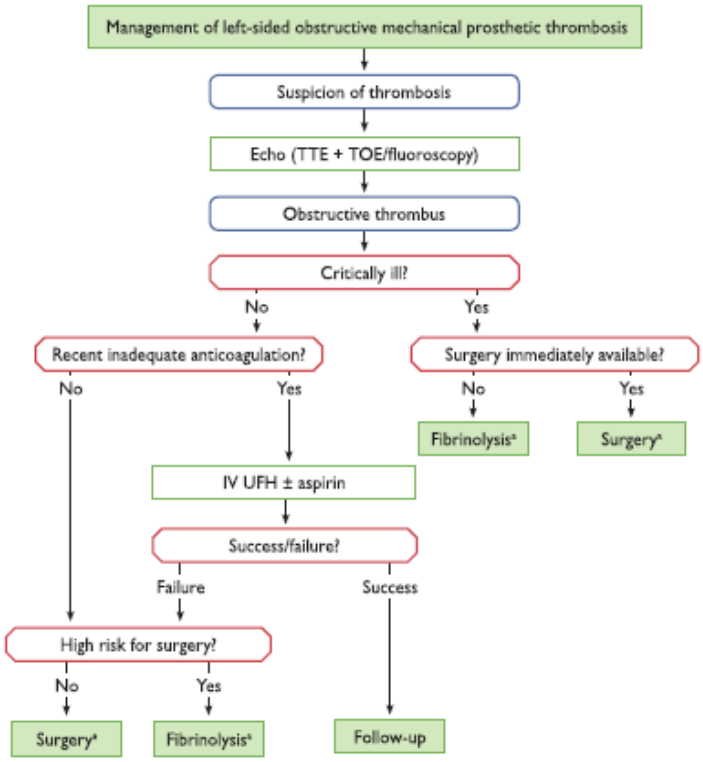

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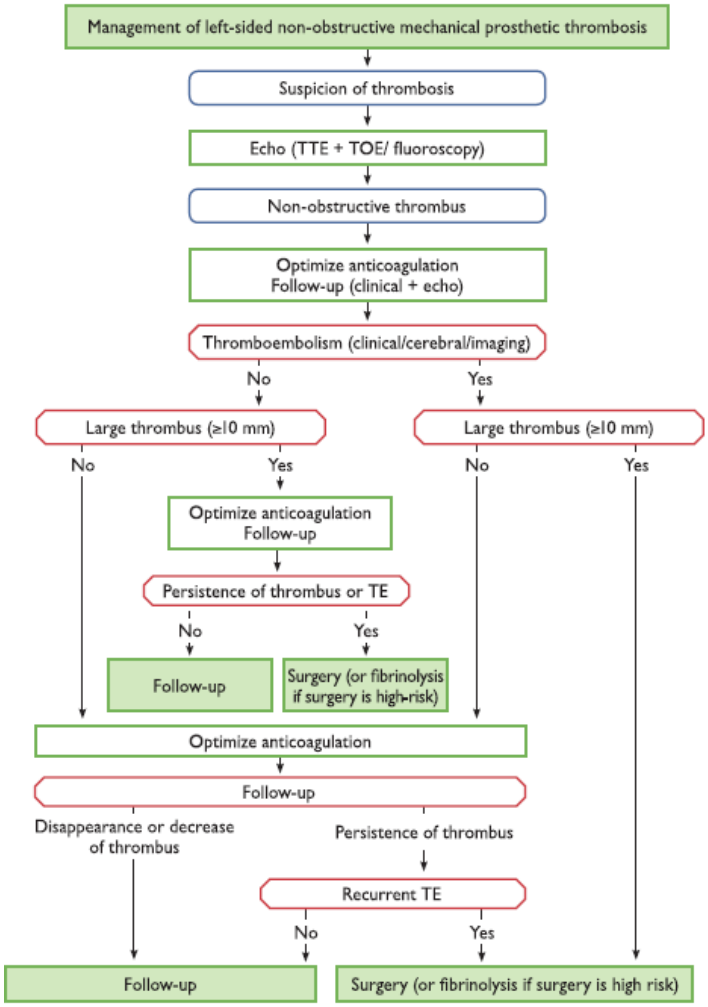
Figure 8: Management of left sided non-obstructive mechanical prosthetic thrombosis



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12.4 Management of haemolysis

Blood tests for haemolysis should be part of routine follow-up after valve replacement. Lactate dehydrogenase, although non-specific, is related to the severity of haemolysis. The diagnosis of haemolytic anaemia requires TOE to detect a paravalvular leak if TTE is not contributory. Reoperation is recommended if the paravalvular leak is related to endocarditis or causes haemolysis requiring repeated blood transfusions or leading to severe symptoms. Medical therapy, including iron supplementation, beta blockers and erythropoietin, is indicated in patients with severe haemolytic anaemia when contraindications to surgery are present. Transcatheter closure of a paravalvular leak is feasible, but experience is limited and there is presently no conclusive evidence to show a consistent efficiency.

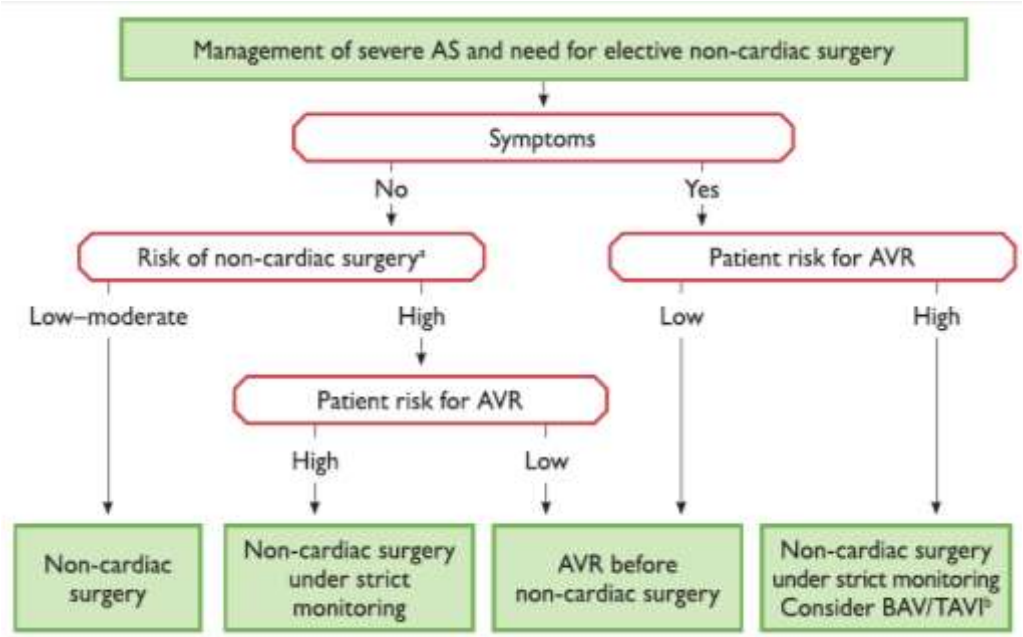
13. Perioperative assessment in non-cardiac surgery

Cardiovascular morbidity and mortality are increased in patients with VHD who undergo non-cardiac surgery. Symptomatic severe aortic stenosis or mitral stenosis may require valve replacement or percutaneous intervention before non-cardiac surgery. Echocardiography should be performed in any patient with VHD. Determination of functional capacity is a pivotal step in preoperative risk assessment, measured either by exercise test or ability to perform activities in daily life. The decision for management should be taken after multidisciplinary discussion involving cardiologists, surgeons and anaesthesiologists.

13.1 Aortic stenosis pre-assessment

In patients with severe aortic stenosis, urgent non-cardiac surgery should be performed under careful haemodynamic monitoring. The management related to elective non-cardiac surgery depends on the presence of symptoms and the type of surgery (see figure 9). In symptomatic patients, aortic valve replacement should be considered before non-cardiac surgery. In patients at increased surgical risk, TAVI is a therapeutic option. In asymptomatic patients, elective non-cardiac surgery can be performed safely, albeit with a risk of worsening heart failure.

Figure 9: Management of severe aortic surgery undergoing non cardiac surgery



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13.2 Mitral stenosis pre-assessment

Non-cardiac surgery can be performed safely in patients with nonsignificant mitral stenosis (valve area >1.5 cm2) and in asymptomatic patients with significant mitral stenosis and a systolic pulmonary artery pressure <50mmHg. In symptomatic patients or in patients with systolic pulmonary artery pressure >50mmHg, correction of mitral stenosis, by means of PMC whenever possible, should be attempted before non-cardiac surgery if it is high risk.

13.3 Aortic and mitral regurgitation pre-assessment

Non-cardiac surgery can be performed safely in asymptomatic patients with severe mitral regurgitation or aortic regurgitation and preserved LV function. The presence of symptoms or LV dysfunction should lead to consideration of valvular surgery, but this is seldom needed before non-cardiac surgery. If LV dysfunction is severe (ejection fraction <30%), non-cardiac surgery should be performed only if strictly necessary, after optimization of medical therapy for heart failure.

14. Endocarditis prophylaxis

Antibiotic prophylaxis should be considered for high-risk procedures in patients with prosthetic valves, including transcatheter valves, or with repairs using prosthetic material and those with previous episodes of infective endocarditis. Recommendations regarding dental and cutaneous hygiene and strict aseptic measures during any invasive procedures are advised in this population. Antibiotic prophylaxis should be considered in dental procedures involving manipulation of the gingival or periapical region of the teeth or manipulation of the oral mucosa. Antibiotic prophylaxis should be considered in patients with previous endocarditis or adult congenital heart disease.

15. Heart team meeting (HTM)

The main purpose of the HTM is to deliver better quality evidence based care. This is achieved through discussion of cases with a team of cardiologists and cardiac surgeons who share expertise in interventional and surgical management of vascular diseases and complications. The SHSCT HTM undertaken with the BHSCT cardiac surgeons occurs on Wednesdays in the cardiovascular research unit (CVRU) at 1pm. Ad hoc cases are discussed virtually with the BHSCT if needed.

16. SHSCT echocardiography service

The echocardiography service in the SHSCT is a busy unit of 8 dedicated cardiac physiologists who perform >10,000 TTEs per year. In addition, approximately 150-200 TOEs are performed per year.

The request form for TTE / TOE and dobutamine stress echocardiography (DSE) is located in appendix 2. Guidelines are found on the back of the form to assist referrers in making requests for imaging procedures. Appendix 3 is a useful flowchart to assist staff in determining if a TTE is indicted and proves useful requesting advice and advice on alternative diagnoses to consider.

17. Aortic valve surveillance (AVS) clinic

The aortic valve surveillance (AVS) clinic has been set up to review and arrange serial TTE studies for patients diagnosed with mild or moderate aortic stenosis. Appendix 4 is a useful flowchart to assist referrers in decision making in aortic stenosis cases. If patients become more symptomatic or progress to severe aortic stenosis then the AVS clinic will alert the responsible consultant who will then prepare to present the patient at the HTM for intervention.

18. Update and review

- This document will be updated every 3 years.
- Revisions will be made ahead of the review date if new, relevant national guidelines are published. Where the revisions are significant and the overall policy is changed, the authors will ensure the revised document is taken through the standard consultation, approval and dissemination processes.

19. References

ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC). European Heart Journal (2016) 37: 2893–2962.

Recommendations for the echocardiographic assessment of native valvular regurgitation: an executive summary from the European Association of Cardiovascular Imaging. European Heart Journal – Cardiovascular Imaging (2013): 14:611-644.

Appendix 1 – Governance information

Document title	Guidelines for the management of Valvular heart disease and the SHSCT echocardiography service
Date issued / approved:	**5th August 2020**
Date valid from:	**5th August 2020**
Date valid to:	**Initial: 5th August 2023** **Extended: 1st Jan 2027 (see below)**
Brief summary of contents	This document provides guidance for any professional involved in the clinical management of patients presenting to SHSCT with valvular heart disease.
Policy objectives	To provide clear speciality agreed guidelines and pathways for the diagnosis and clinical management of patients with valvular heart disease.
Keywords	Cardiology Valvular heart disease Aortic stenosis Mitral regurgitation Echocardiography
Authorship	Dr David Mc Eneaney (cardiology clinical lead, consultant cardiologist) Kay Carroll (cardiology head of service) Dr Mick Connolly (consultant cardiologist) Dr Ian Menown (consultant cardiologist)

Addendum Jan 2024:

This policy was reviewed by the consultant cardiologists and discussed at the cardiology governance meeting in Winter 2023. It was sent out for a 6 week consultation period. No amendments were suggested by the wider cardiology team. Following universal unanimous agreement by the consultant body at this governance meeting it has been approved for 3 further years until 1st Jan 2027.

Signed: Dr Mick Connolly Consultant cardiologist

Appendix 2: Echocardiogram request form

Southern Health and Social Care Trust

Echocardiogram Request

Name:
H+C:
Date of birth:
Address:
(Addressograph if available)

Transthoracic (TTE) ☐

Transoesphageal (TOE) ☐
NB: All TOE scope tests must be discussed with cardiology team

Dobutamine stress (DSE) ☐

OP ☐

IP ☐ Ward ☐

Mobility: Walk / Chair / Bed

Consultant:

DSE: Please record rate limiting drugs

PPM / ICD: Yes / No
Infection risk: Yes / No
Chaperone: Yes / No
Interpreter: Yes / No
Specify:

Indication

☐ LV / RV function ☐ Arrhythmia ☐ Reversible ischaemia (DSE)
☐ Valvular ☐ Endocarditis ☐ Low flow AS (DSE)
☐ Cardiomyopathy ☐ Pericardial ☐ Other

BNP result: (must be provided for ***De novo*** heart failure)
ECG / underlying rhythm:

Urgency: Urgent / Routine / Managed list
NB: Urgent requests must be agreed with cardiologist before submitting or else will be vetted as routine

Managed list date:

Previous TTE date (required):

EDD (required): (Requests received <24hours pre - EDD cannot be accommodated)

Specific clinical questions (required)

** ADMIN ONLY **
☐ Approved
☐ Declined (return to referrer)
Reason:

Signature	Print	Contact number	Date

Southern Health and Social Care Trust

Echocardiogram Request

Echocardiography is not indicated in the following circumstances (NI cardiac network guidelines)

Arrhythmias with TTE
- Infrequent APCs or infrequent VPCs without other evidence of heart disease
- Lightheadedness / presyncope when there are no other symptoms or signs of cardiovascular disease

Evaluation of Ventricular Function with TTE
- Routine surveillance of ventricular function with known CAD and no change in clinical status or cardiac exam

Perioperative Evaluation with TTE
- Routine perioperative evaluation of ventricular function with no symptoms or signs of cardiovascular disease

Native Valvular Stenosis with TTE
- Routine surveillance (<3 yr) of mild valvular stenosis without a change in clinical status or cardiac exam
- Routine surveillance (<1 yr) of moderate or severe valvular stenosis without a change in clinical status or cardiac exam

Native Valvular Regurgitation with TTE
- Routine surveillance of trace valvular regurgitation
- Routine surveillance (<3 yr) of mild valvular regurgitation without a change in clinical status or cardiac exam

Prosthetic Valves with TTE
- Routine surveillance (<3 yr after valve implantation) of prosthetic valve if no known or suspected valve dysfunction

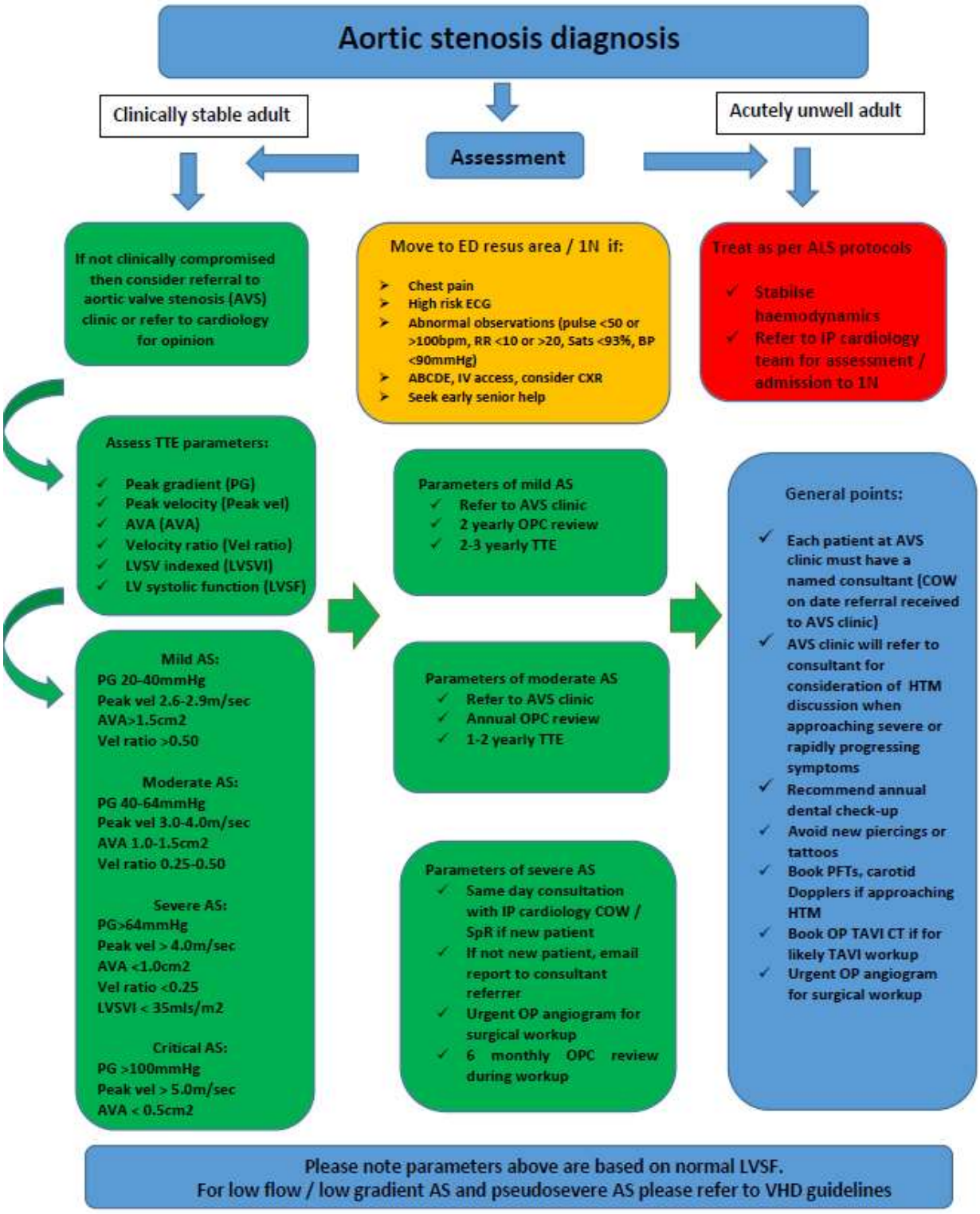
Infective Endocarditis (Native or Prosthetic Valves) with TTE
- Transient fever without evidence of bacteraemia or a new murmur

Cardiomyopathies with TTE
- Routine surveillance (<1 yr) of known cardiomyopathy without a change in clinical status or cardiac exam

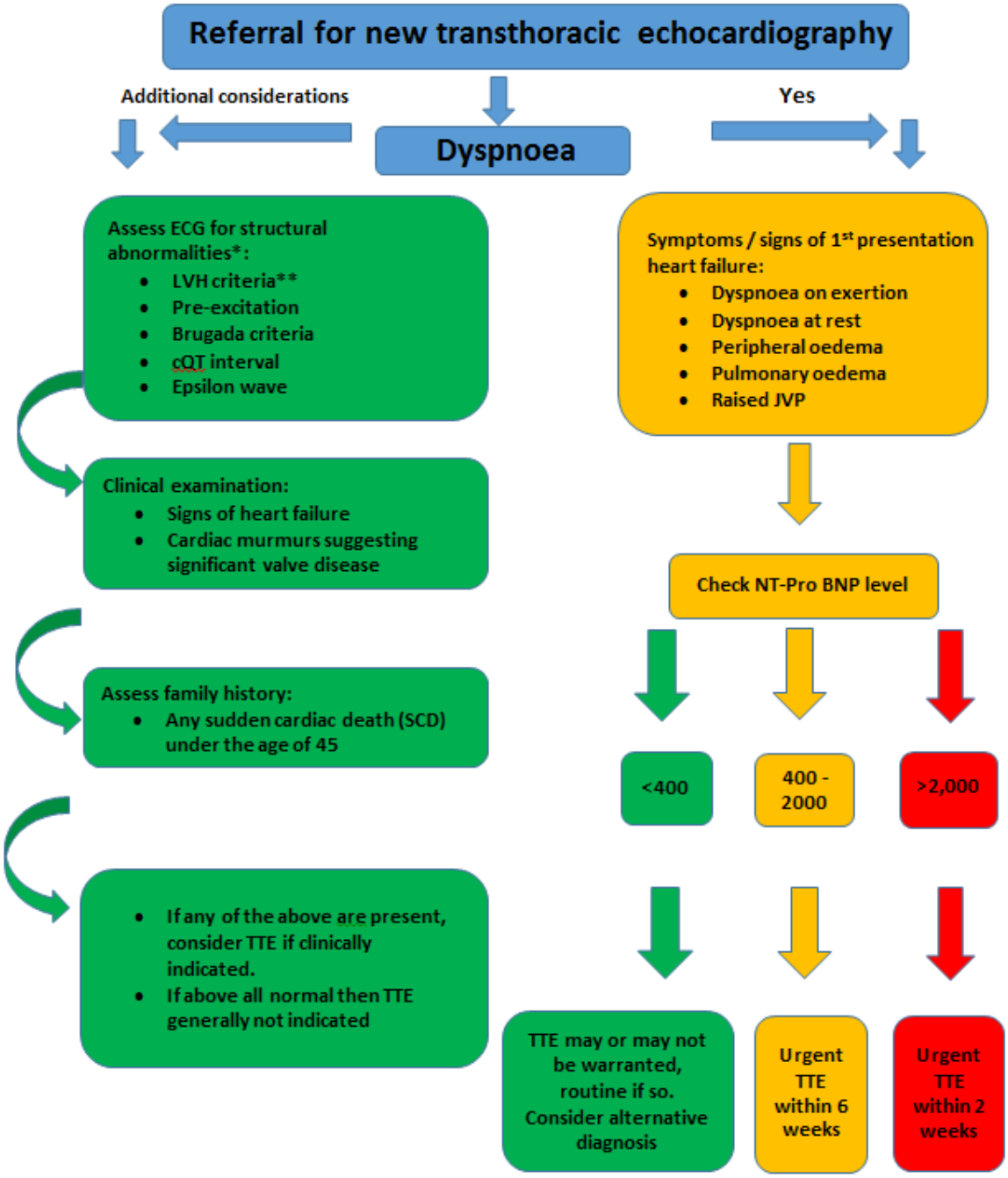
TTE for Evaluation of Hypertension, Heart Failure (HF) or Cardiomyopathy
- Routine evaluation of systemic hypertension without symptoms or signs of hypertensive heart disease
- Evaluation of LV function in suspected HF where ECG and BNP are within normal range
- Routine surveillance (<1 yr) of HF (systolic or diastolic) when there is no change in clinical status or cardiac exam

Note: a new diagnosis of HF should ideally have a TTE within 2 weeks if BNP >2000 or within 6 weeks if BNP >400

Appendix 3: Aortic valve surveillance (AVS) clinic referral



Appendix 4 : TTE requesting guidance



Please note this guideline does not cover all potential scenarios. If in doubt discuss with a member of the cardiology team

*See overleaf for examples

**LVH could suggest underlying HTN, aortic stenosis or a cardiomyopathy. This should be assessed using specific criteria e.g. deepest S wave in V1/V2 added to tallest R wave in V5/V6 >35mm (7 large squares), see overleaf

For palpitations / syncope / VHD please follow separate pathways