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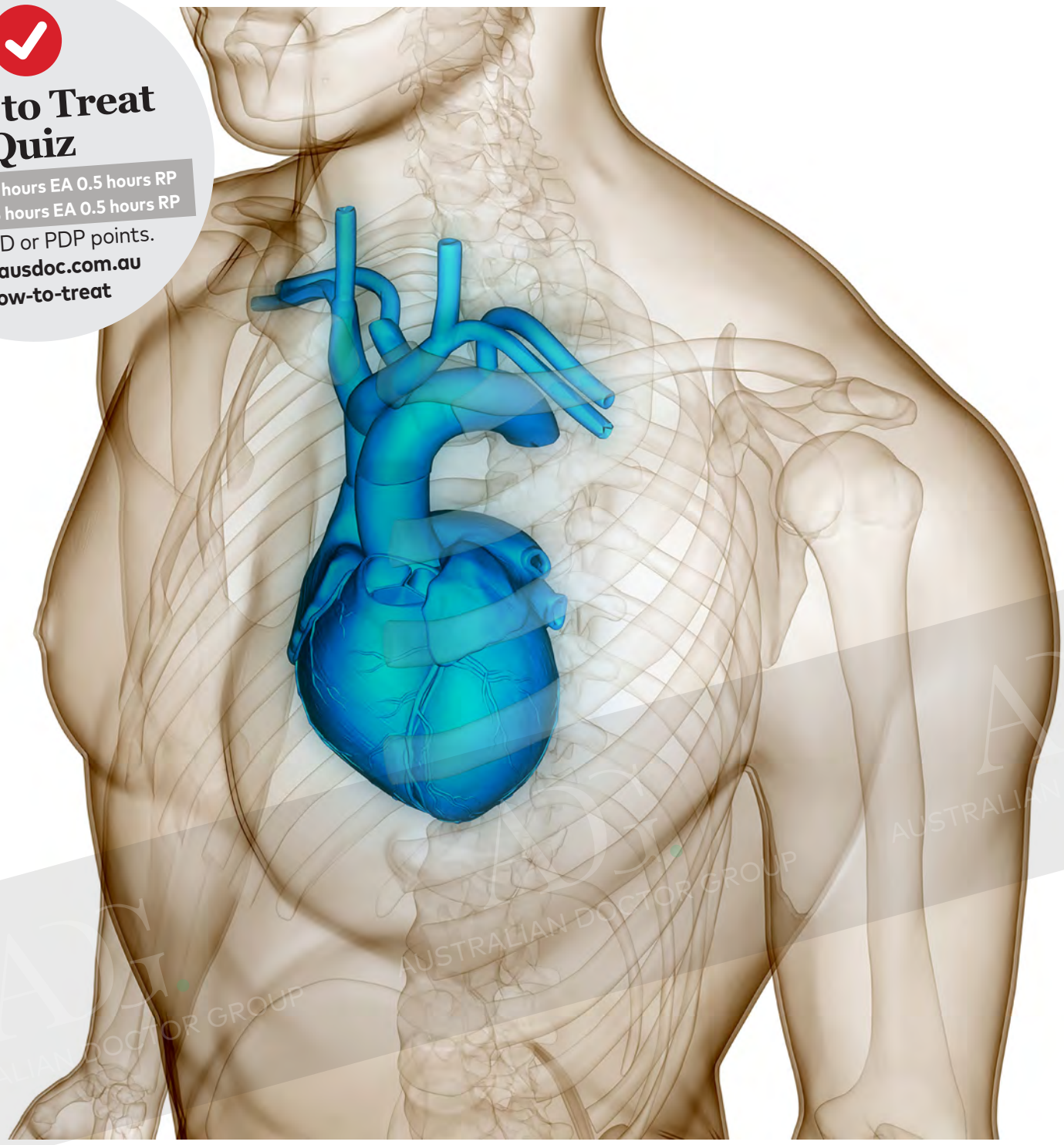

NEED TO KNOW

Mitral valve regurgitation is the most common valvular heart disease in Western counties, including Australia, particularly in ageing populations.

Left untreated, it can lead to heart failure and death.

Guidelines classify mitral valve regurgitation into either primary or secondary to determine therapeutic approaches.

Echocardiogram is paramount in investigation; it will diagnose the condition, determine severity, assess the aetiology and determine the choice of therapy.

A comprehensive assessment in a specialised heart valve centre is vital in providing an individualised treatment plan for each patient.

Surgery is the first choice for patients with primary and secondary mitral valve regurgitation, but there is a large proportion of patients in whom surgery carries a prohibitive risk.

Transcatheter mitral valve repair is an emerging technology in structural cardiology, and it is rapidly evolving as a safe and effective alternative in high surgical risk patients.

Mitral regurgitation

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BACKGROUND

MITRAL regurgitation (MR) is defined as backward flow of blood from the left ventricle (see figures 1 and 2) into the left atrium during systole.[1] This is the most common valvular heart disease in Western countries, including Australia, and occurs particularly in ageing populations.

In the US, the prevalence of mitral valve (MV) disease is greater than 10% in those older than 75 years, despite a reduction in the incidence of rheumatic heart disease, making it a significant public health issue.[2,3]

While those with MR may be asymptomatic early in the course of the disease, if left untreated, it can eventually lead to heart failure and death.[4]

Recent advances in cardiac multimodality imaging and surgical techniques, as well as the introduction of transcatheter interventions into practice, have transformed the care provided to patients with MR and significantly improved outcomes. It is essential that this condition is detected early in the primary care setting to ensure patients with MR receive appropriate treatment.

This How to Treat provides an overview of the epidemiology, diagnosis and management of MR. It aims to equip GPs with the knowledge to identify patients early in the course of the illness and initiate a timely treatment plan, including early referrals to specialised valve centres.

AETIOLOGY

THE MV apparatus is a dynamic and complex structure consisting of the mitral annulus, the anterior and posterior leaflets, chordae tendinea, and papillary muscles surrounded by left ventricular and left atrial walls (see figure 3).[5]

MV function is dependent on the appropriate interplay of these components. Disturbance in any part of this apparatus or surrounding structure can lead to MR.[6]

To determine appropriate therapeutic approaches, guidelines classify MR into either primary or secondary. Primary MR is defined as a structural abnormality in any component of the MV apparatus causing MV leak, while secondary MR is defined as disease of the left ventricle or left atrium that interferes with the integrity of the MV apparatus, thus causing a leak in the MV.[7]

Primary mitral regurgitation

While rheumatic heart disease (see figure 4) is a common cause of primary MR in low income and developing countries, the most common cause of primary MR in Western countries is degenerative disease affecting components of the MV apparatus, including myxomatous degeneration of the MV leaflets and/or redundant chordal apparatus.[8,9]

Redundant leaflets can prolapse back into the left atrium causing malcoaptation of the leaflets and consequent regurgitation. Degeneration and rupture of the chordae may lead to the MV becoming unsupported, which results in MR. Primary MR can also occur from leaflet perforation from pathologies like endocarditis. Other rare causes include drugs, radiation, and systemic disease-causing restricted leaflet motion from thickening of both leaflets and subvalvular apparatus. An additional recognised cause of MR in the elderly population is mitral annular calcification, which may start in the posterior annulus and extend into the subvalvular apparatus, affecting leaflet function.[10]

Secondary mitral regurgitation

The MV apparatus is normal in functional or secondary MR; however, ventricular or atrial dilatation and/or remodelling leads to an imbalance in coaptation of the MV, which causes MR.

Ventricular dilatation from either ischaemic or nonischaemic cardiomyopathy, as well as markedly depressed or even normal left ventricle (LV) function after an isolated infero-basal myocardial infarction leading to posterior leaflet tethering, can lead to gradual annular dilatation and/or leaflet malcoaptation.[11] This begins a cycle of increasing LV pressure and volume overload, leading to further LV dilatation and remodelling with progressive dysfunction and worsening MR.[1]

Secondary MR can also occur as a result of chronic atrial fibrillation that causes significant atrial ▸

◂ enlargement and annular dilatation with or without left ventricular dysfunction. This pathophysiological process has had more recognition in recent years.[12]

DIAGNOSIS

History and physical examination

ASSESSMENT of patients with chronic MR begins with a detailed medical history and physical examination. In those with chronic MR, symptoms may vary from asymptomatic to severely dyspnoeic. Absence of symptoms in the chronic phase may be explained by progressive left atrial enlargement; this allows for accommodation of the large regurgitant volume in the dilated left atrium without a significant increase in pressure, which does not then cause dyspnoea. Patients typically tend to reduce their activity to avoid symptoms. It is important to ask the patient and family members what the patient can currently undertake compared with their previous activities. Exercise testing may unmask symptoms in asymptomatic patients.[13]

As the compensatory mechanisms become overwhelmed, patients develop a gradual reduction in exercise tolerance and exertional shortness of breath. Fatigue and palpitations are also common symptoms of severe MR.

Regarding physical examination, complete assessment of MR always includes evaluation for fluid overload, signs of heart failure and suggestive heart sounds (see figure 5). Common signs of MR include a displaced apex beat, loud systolic murmur and cardiomegaly.[14] In patients with primary MR, the presence of S3 plus a short diastolic murmur is usually associated with a significant MR.[14] Radiation of the murmur of primary MR can provide a hint regarding the underlying leaflet pathology. Murmurs from the anterior leaflet are generally directed towards the axilla, while murmurs from a flail posterior leaflet radiate anteriorly. Secondary MR murmurs are generally best heard at the apex. Atrial fibrillation and other arrythmias are commonly associated with MR.[14]

Investigation

Transthoracic echocardiogram (TTE) and transoesophageal echocardiogram (TOE, the gold standard) are vital investigations to diagnose, evaluate severity, assess anatomy, aetiology, mechanism, associated findings, and guide choice of therapy in MR.[3] TTE (2D images and doppler) is the initial assessment, given its non-invasive nature and easy availability.

When assessing the severity of MR, guidelines recommend integration of qualitative (for example, MV morphology and colour flow jet area), semiquantitative (vena contracta [the narrowest part of the jet that is just distal to the regurgitant orifice] width and pulmonary vein flow), and quantitative (regurgitant volume and regurgitant orifice area) measures in the assessment.[15,16]

While in some cases TTE provides sufficient information for assessment of MR, TOE is mostly needed for a full assessment. TOE provides better quality images, and it is more accurate in the assessment of eccentric jets.[17] In addition, the 'en face' view of the MV obtained using 3D echo helps visualise the anatomic details of the MV apparatus and differentiates between primary and secondary MR with high level of certainty. 3D TOE is highly precise in locating prolapsing scallops (P1 [anterior or medial scallop], P2 [middle scallop], and P3 [posterior or lateral scallop]; see figures 6 and 7), and can be used to understand highly specific details required for both surgical and transcatheter repair.[18]

Other modalities (very rarely indicated) that can be useful in assessment of MR are non-invasive cardiac MRI (CMR) and invasive right and left heart catheterisation. CMR is generally more accurate and reproducible than catheterisation for quantitating MR volume, LV volume and LV ejection fraction (LVEF).[19] Left ventriculogram, invasive measurement of cardiac output and pulmonary pressures can provide additional information to inform decision making regarding treatment.

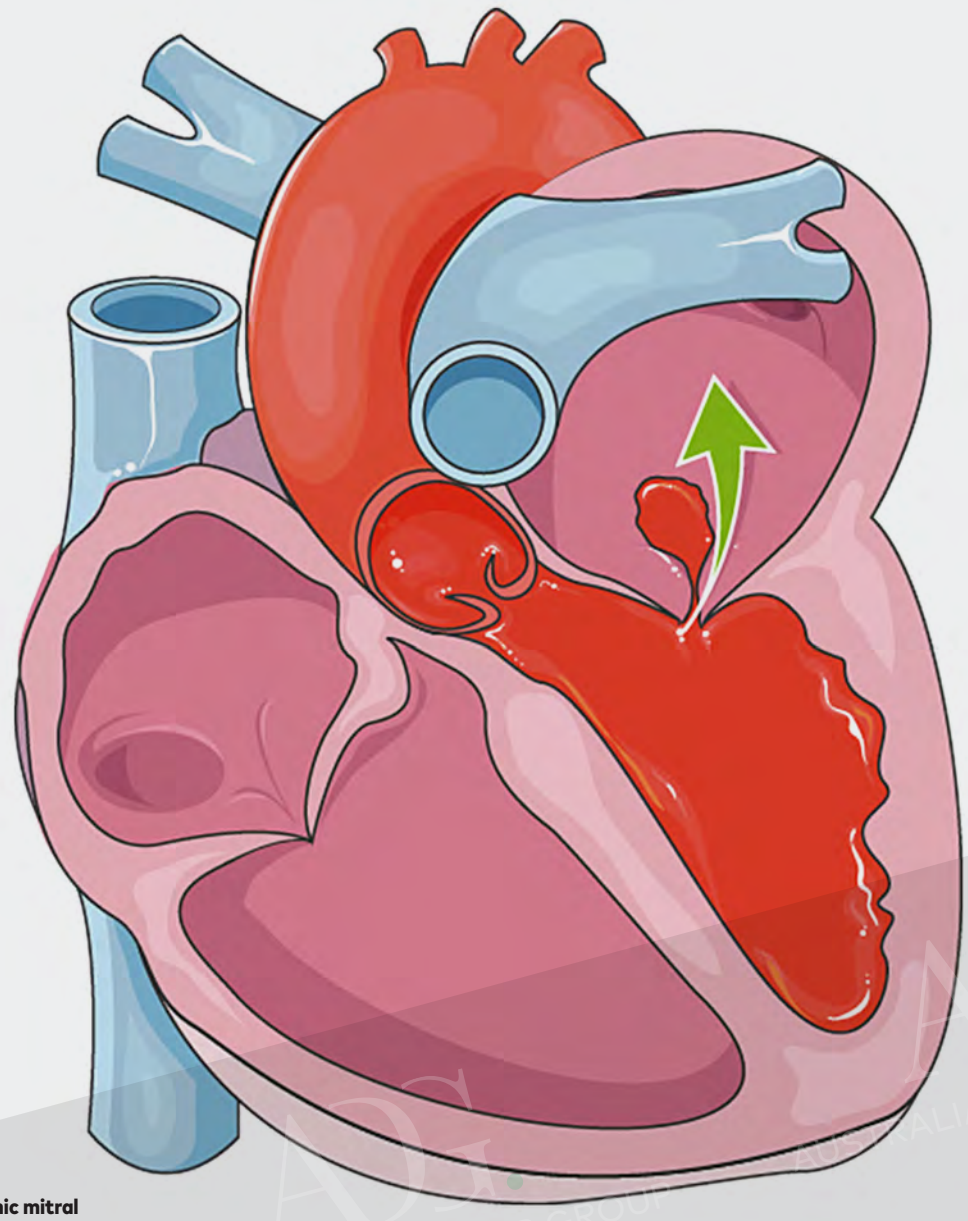

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Figure 1. Chronic mitral regurgitation.

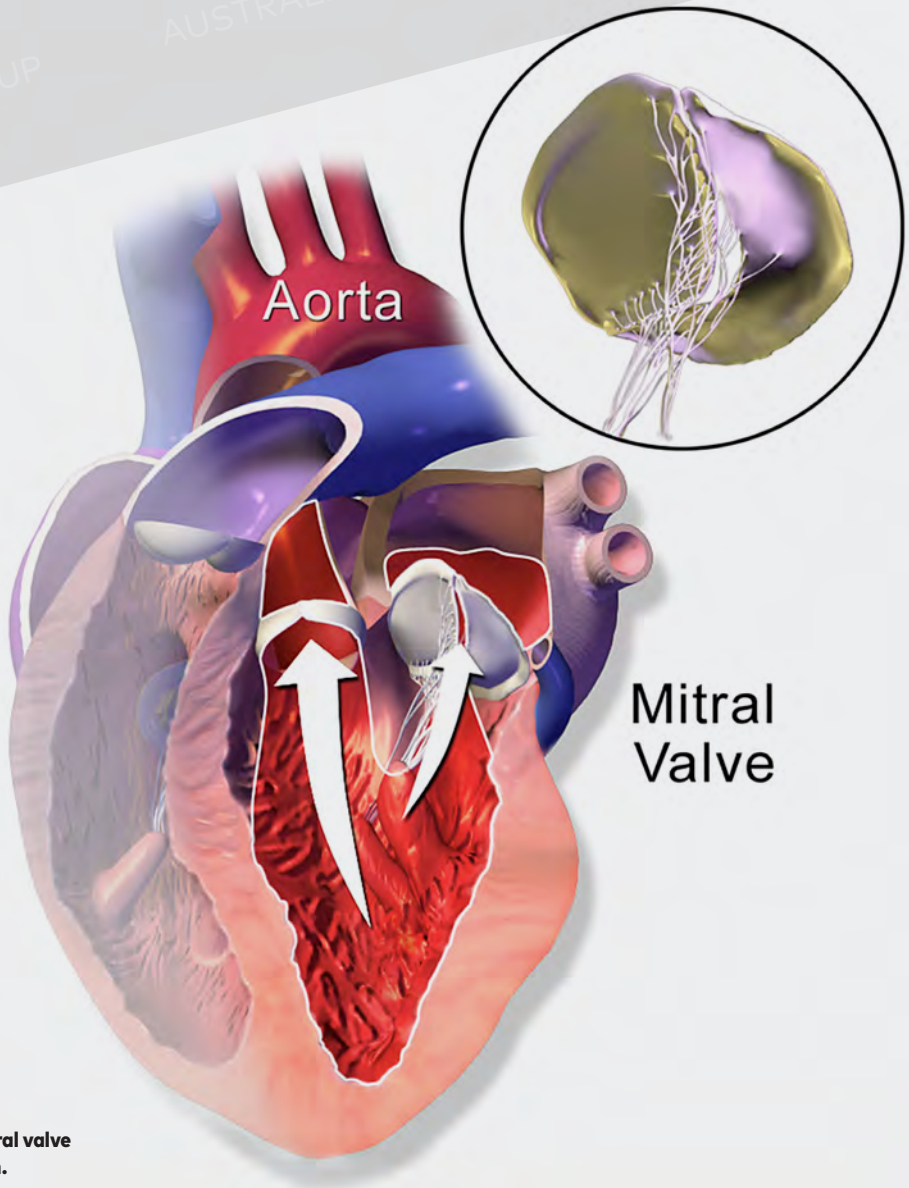

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Figure 2. Mitral valve regurgitation.

MANAGEMENT

General considerations

DECIDING on the best treatment for MR is based on multiple variables, including the type, severity, patient comorbidities, associated cardiac changes and experience of the treating centre. A comprehensive assessment of patient history, physical examination and diagnostic tests in a specialised heart valve centre is vital to provide individualised treatment for each patient.[10]

Primary mitral regurgitation

Medical therapy has a limited role in acute MR. Diuretics can be used to reduce filling pressures and inotropic agents to increase contractility. There is no evidence to support the use of medical therapy in chronic stable MR and preserved LVEF.[13] In patients with MR and reduced LVEF, guideline-directed heart failure therapy is needed, in conjunction with other treatments.[20]

INTERVENTION

The European Society of Cardiology guidelines for the treatment of MR appear in figure 8. In summary, intervention is indicated in all asymptomatic patients with severe MR. Intervention is also indicated in patients with severe MR and high-risk features like reduced LVEF, enlarged left atrium and increased pulmonary pressures.

If no symptoms or high-risk features are present, watchful waiting with close follow-up is a reasonable option.[15] When intervention is indicated surgery remains the first line of therapy, unless the heart team recommends against it, because of its greater higher efficacy in treating primary MR.[21]

If the heart team decide that the patient is suitable for surgery, then valve repair, not replacement, is the first surgical choice, because valve repair is associated with a greater life expectancy compared with MV replacement.[5] Successful repair of primary MR at the right time and performed in an experienced centre results in life expectancy similar to that of normal age-matched population.[22] When repair is not possible, MV replacement is a good alternative option.[22]

While surgery remains the first choice for patients with primary MR, surgical risk is prohibitive in a large percentage of patients because of old age and comorbidities. In these patients, transcatheter MV implantation is a safe option. Transcatheter MV repair is an emerging technology in cardiology, and it is rapidly evolving. Because of the complexity of MV apparatus, there are multiple devices in the experimental phase. Currently, transcatheter MV edge to edge repair (TEER) using mitral clip (Abbott Vascular) or PASCAL (Edwards Lifesciences) devices are the most mature, with the most

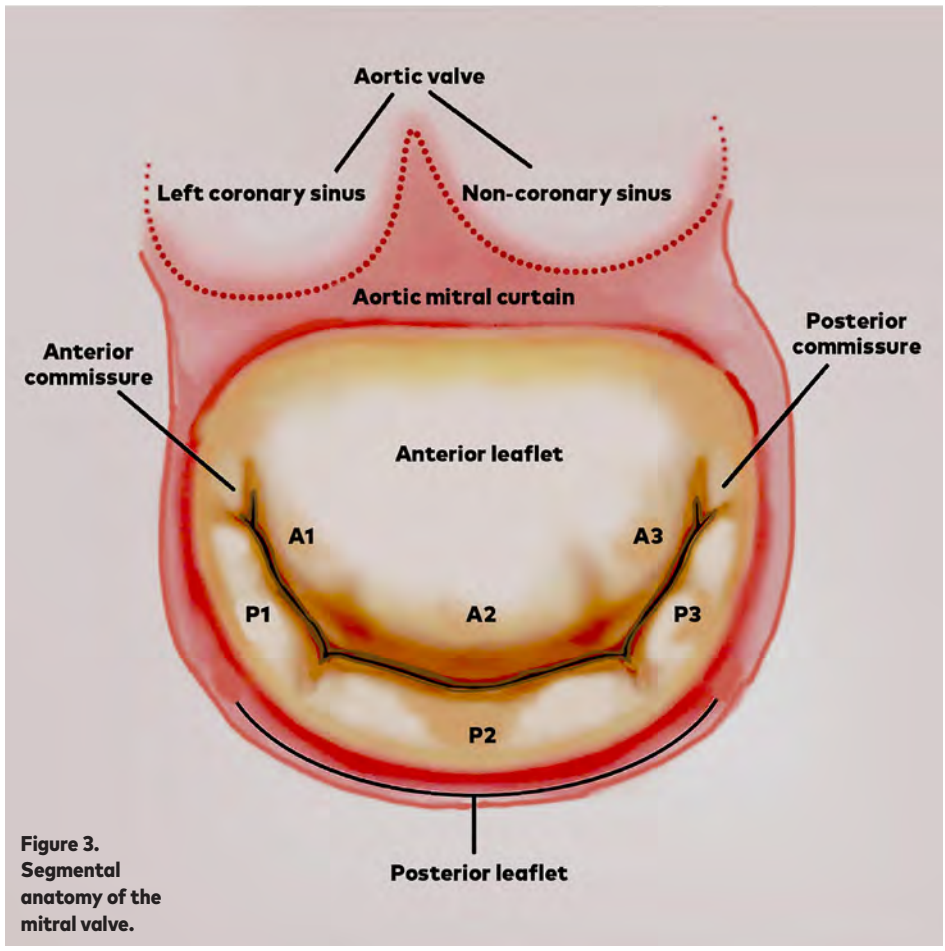
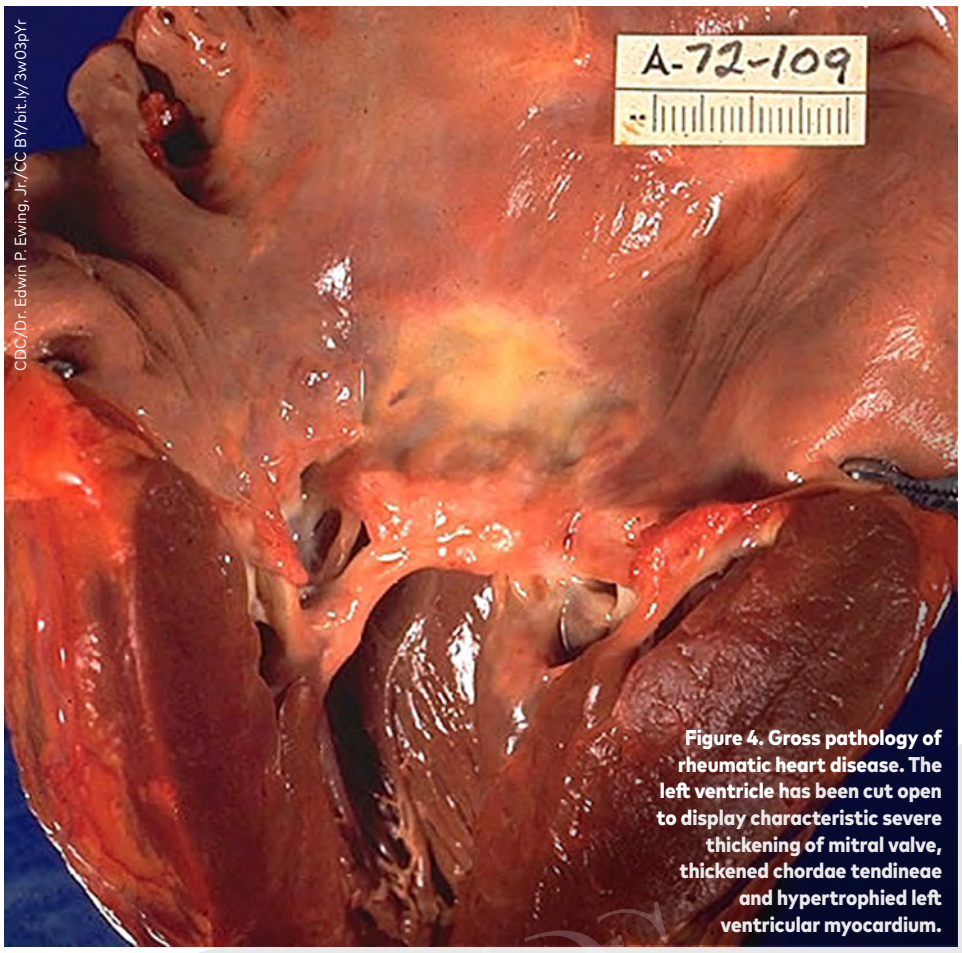


Figure 3. Segmental anatomy of the mitral valve.



CDC/Dr. Edwin P. Ewing, Jr./CC BY/bit.ly/3w03pYr

Figure 4. Gross pathology of rheumatic heart disease. The left ventricle has been cut open to display characteristic severe thickening of mitral valve, thickened chordae tendineae and hypertrophied left ventricular myocardium.

data.[13] This technique mimics the surgical Alfieri edge-to-edge leaflet repair and attaches the free edges of the anterior and posterior leaflets with a clip.[23]

The 2011 EVEREST II (Endovascular Valve Edge-to-Edge Repair Study) trial included 279 patients randomly assigned to either mitral clip or surgery. It showed that percutaneous repair, although less effective in reducing MR than surgery, was associated with improved left ventricular dimensions, improvement in New York Heart Association class and in quality of life.[21] Since this trial, real life registry data have confirmed the safety and efficacy of percutaneous MV edge to edge repair in patients with primary MR and prohibitive surgical risk.[24]

Secondary mitral regurgitation

Unlike primary MR, secondary MR is not a disease of the MV but a disease of the cardiac chambers that surround the MV apparatus. Secondary MR usually occurs because of LV or left atrial remodelling and enlargement, which causes papillary muscle displacement and/or annular dilatation. Another cause may be isolated posterior leaflet tethering in preserved LVEF following myocardial infarction.[1,12]

Given the aetiology is heterogenous and the MV structure is not the culprit, treatment focuses on the mechanism of the MR. In patients with a dilated ventricle, guideline-directed medical therapy, including device therapy (cardiac resynchronisation therapy), is first line and arguably the most important line of therapy.[20]

In ischaemic MR, in addition to medical therapy, consider the indications for coronary revascularisation.[13] Studies in patients with heart failure and secondary MR have shown improvements in both mortality and in quality of life scores with guideline-directed medical therapy.[11, 25]

INTERVENTION

Consider intervention if guideline-directed medical therapy fails. Decisions are made based on the recommendations of a multidisciplinary heart team.

In the case of functional MR associated with coronary artery disease, MV repair or replacement at the time of bypass surgery improves symptoms and survival, and should be considered.[26] In isolated secondary MR, the evidence to support surgical intervention remains limited because of the enormous procedural risks, high recurrence rates and lack of mortality benefits.[27]

In high-risk surgical patients, TEER is a viable option. The 2018 COAPT (Cardiovascular Outcomes Assessment of the MitraClip Percutaneous Therapy for Heart Failure Patients With Functional Mitral Regurgitation) trial randomly assigned more than 600 patients with symptomatic MR and reduced LVEF to either TEER plus guideline-directed medical therapy or guideline-directed medical therapy alone.[28] This trial showed that TEER was a safe procedure with a very low complication rate. Patients in the TEER arm had significantly fewer hospitalisations for heart failure and improved survival at two years.[28]

Interestingly, the MITRA-FR (Multicentre Study of Percutaneous Mitral Valve Repair MitraClip Device in Patients With Severe Secondary Mitral Regurgitation) trial, which randomised 300 patients to either medical therapy alone or TEER and medical therapy, showed no difference between the groups.[29]

A possible explanation for the different findings between these two studies is the timing of the intervention relative to the LV size and the severity of MR.

The patients enrolled in the COAPT trial had more severe MR and less advanced LV disease compared with those in the MITRA-FR trial, indicating that those with too severe LV dilation/dysfunction may not benefit from the MitraClip procedure.

The benefits of intervention are thought to be greater when the degree of MR is disproportionate to what is expected for the ventricular ▸

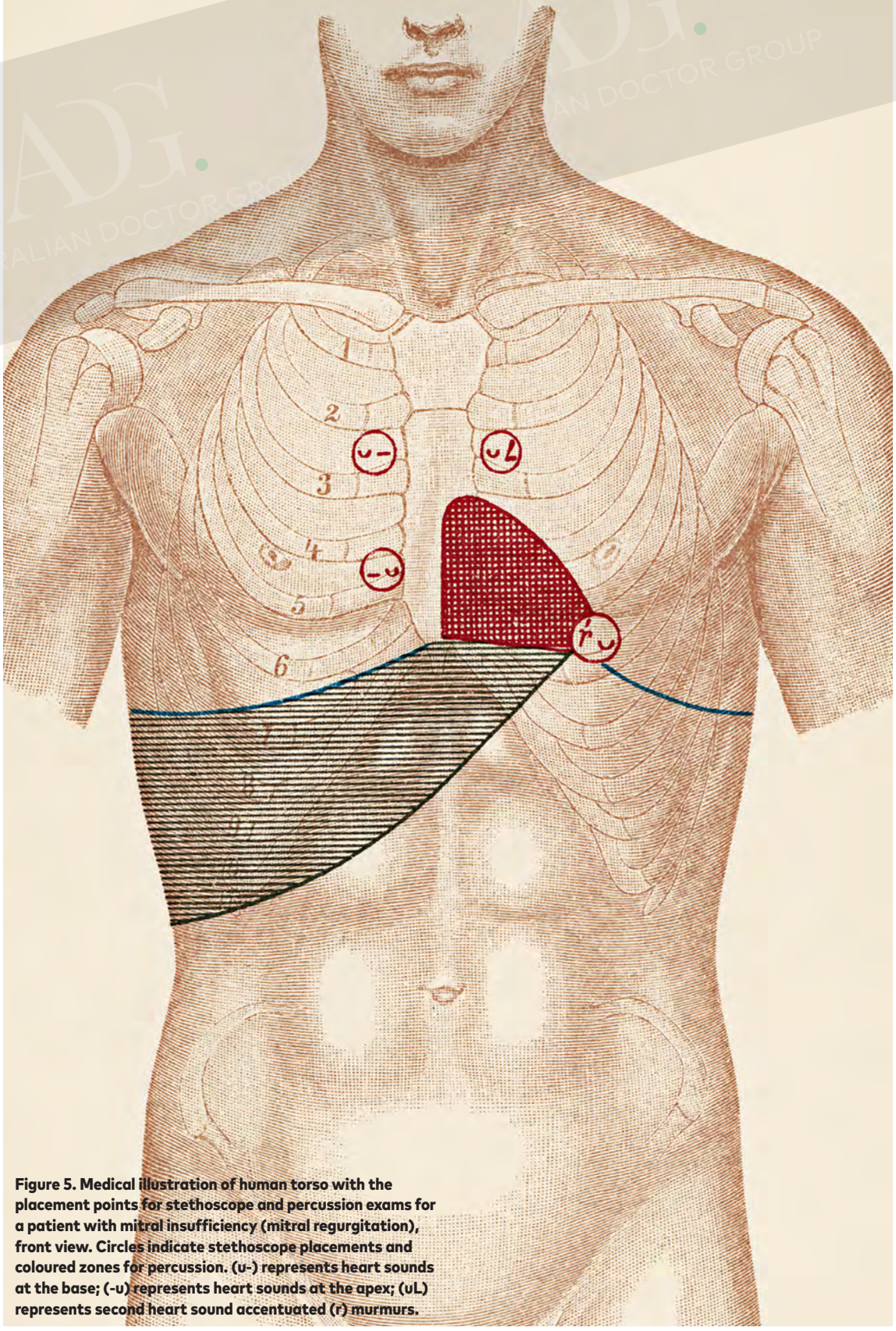


Figure 5. Medical illustration of human torso with the placement points for stethoscope and percussion exams for a patient with mitral insufficiency (mitral regurgitation), front view. Circles indicate stethoscope placements and coloured zones for percussion. (u-) represents heart sounds at the base; (-u) represents heart sounds at the apex; (uL) represents second heart sound accentuated (r) murmurs.

◂ size alone (that is, a bigger ventricle means more advanced cardiomyopathy; this implies dual pathology, which means correcting the MR may not result in improvement, or the damage is irreversible).[30]

The percentage of disproportionate MR was greater in the COAPT trail compared with the MITRA-FR trial. Thus, selecting the right patient for the right procedure at the right time is a complex and nuanced process that highlights the need for early referral to specialist centres.

CASE STUDIES

Case study one

FELIX, a 74-year-old male, has been feeling progressively short of breath over the past three months. This started when he was diagnosed with COVID-19 four months ago. Since then, he has not felt "normal". Six months ago, he was able to walk for more than 30 minutes. He can currently only walk 100 meters before he must stop to catch his breath. There is no shortness of breath at rest. Felix also gets occasional self-limiting palpitations that last a few minutes, but he does not have either chest pain or other cardiac symptoms.

He has a history of ischaemic heart disease (with a previous coronary artery angioplasty with a stent in 2018), hypertension, dyslipidaemia and right knee osteoarthritis. Felix lives with his wife at home, is independent with all activities of daily living and has no gait aids. He is a non-smoker and drinks minimal alcohol.

On examination, he has a soft pansystolic murmur to the left of the sternal border in the 5th intercostal space, which radiates to the axilla. There are no signs of heart failure, and he appears euvolemic.

Laboratory investigations show a normal haemoglobin, and his ECG is unremarkable. A chest X-ray does not show any significant changes that would explain his shortness of breath. A transthoracic echocardiogram shows severe MR. Felix's left atrium is mildly dilated, and his right atrial size is within normal limits. His left ventricular function is preserved. There is no other significant valvular disease.

Felix is referred to a specialised centre with experience in the treatment of MV pathology. He undergoes further workup with a TOE, which shows mildly thickened mitral leaflets (see figure 9). Additional findings include restricted P2 leaflet motion and anterior leaflet prolapse. MR proximal isovelocity surface area (PISA) = 1.1cm, MR effective regurgitant orifice is 0.43cm (severe = more 0.4cm), and MR regurgitant volume is 77mL.

His left and right ventricular functions are normal with no other significant abnormalities, and pulmonary pressures are normal. Right heart catheterisation shows mildly elevated pulmonary pressures with normal peripheral vascular resistance. Left heart catheterisation shows mild to moderate disease in all the main arteries with severe disease in a small calibre left anterior descending diagonal branch.

Felix's case is discussed in the heart team meeting, and given that he has no significant past medical history that precluded him from surgery, he is referred for a surgical MV repair.

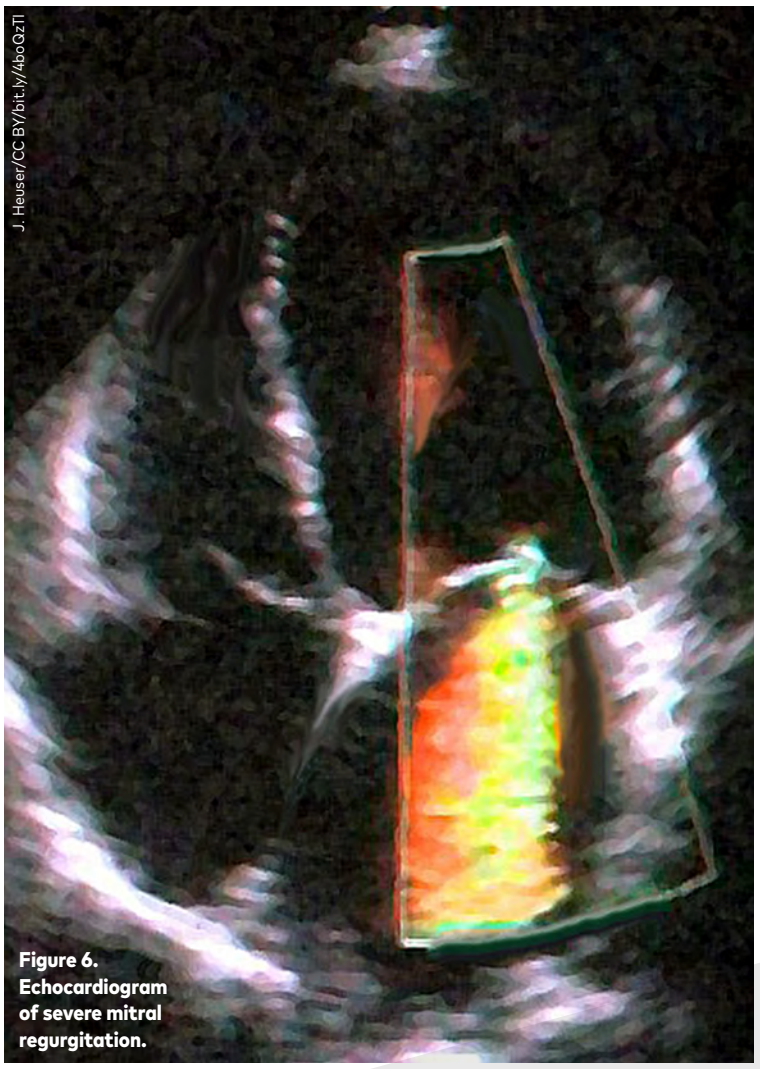


Figure 6. Echocardiogram of severe mitral regurgitation.

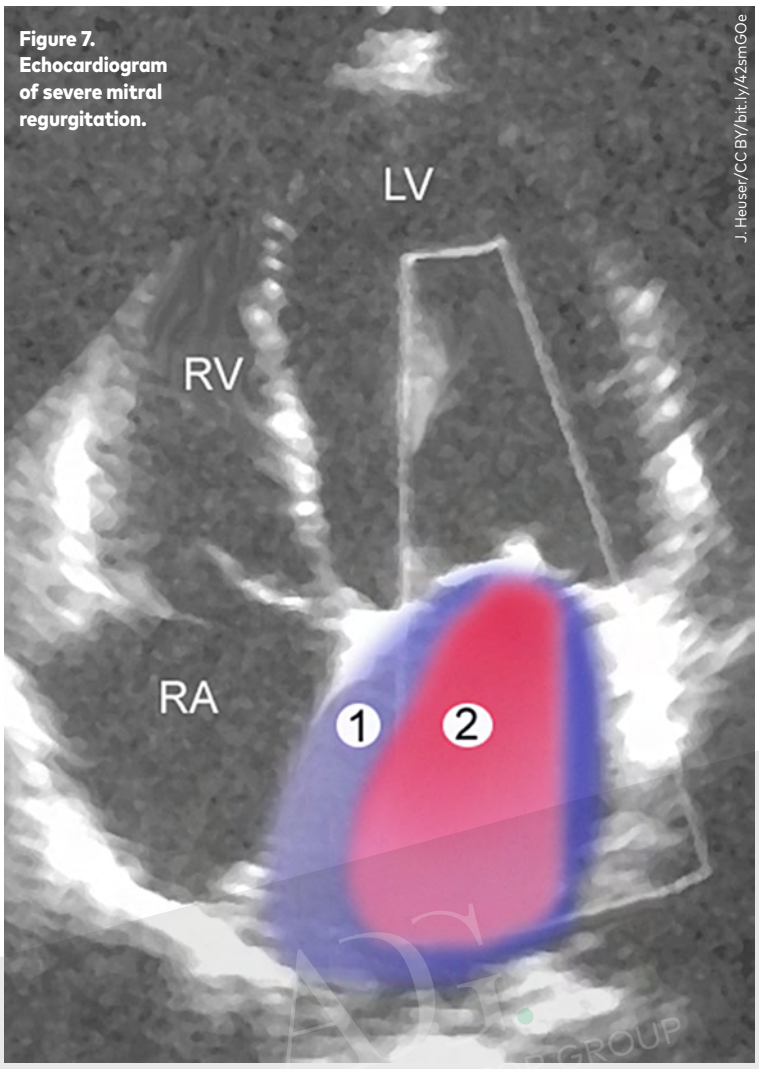
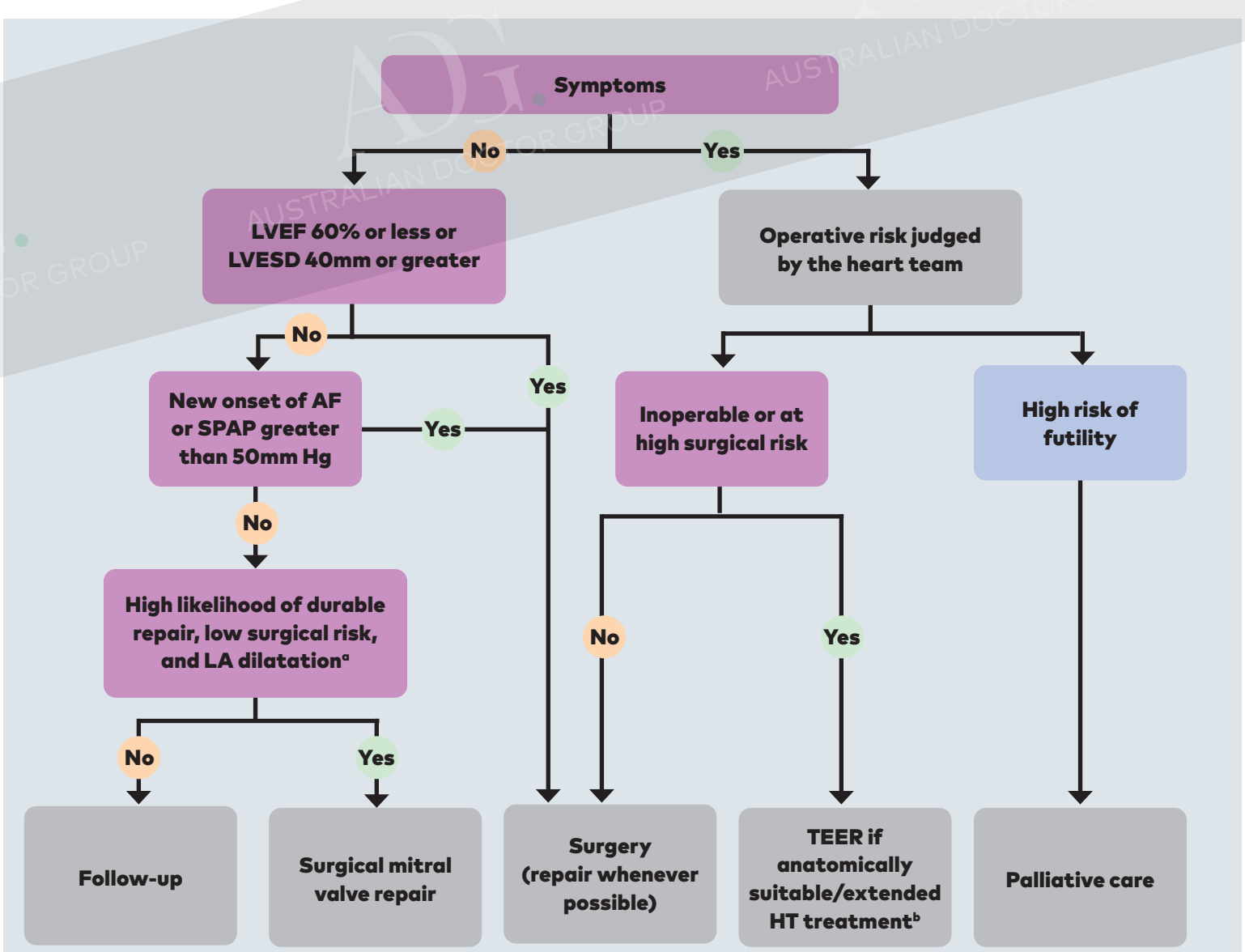


Figure 7. Echocardiogram of severe mitral regurgitation.



AF = atrial fibrillation; HF = heart failure; LA = left atrium/left atrial; LVEF = left ventricular ejection fraction; LVESD = left ventricular end-systolic diameter; SPAP = systolic pulmonary arterial pressure; TEER= transcatheter edge-to-edge repair.
[a] LA dilatation: volume index 60mL/m² or greater or diameter 55mm or greater at sinus rhythm.
[b] Extended heart failure treatment includes the following: cardiac resynchronisation therapy; ventricular assist devices; heart transplantation.
Based on Vahanian A et al 2021[15]

Figure 8. Management of patients with primary mitral regurgitation.

Case study two

Anne, an 86-year-old female, presents to ED with acute decompensated heart failure requiring admission.

Her symptoms have progressed over the preceding few weeks. She initially started to have shortness of breath only after walking long distances or doing a moderate amount of exercise. The symptoms progressed quickly to shortness of breath with shorter distances.

Before presenting to hospital, she was waking up from sleep frequently with a feeling of suffocation and needed to use three pillows to be able to sleep for only few hours.

Her past medical history includes type 2 diabetes mellitus, chronic kidney disease, mild cognitive impairment and known severe MR. Despite her mild cognitive impairment, Anne manages to live alone in a unit that is close to her children. She does not use any walking aids, is a distant ex-smoker and does not drink alcohol.

PAGE 20 ▸

◂PAGE 18 She has had two admissions for heart failure in the past six months. After the initial admission, it was decided to manage Anne medically.

On examination, she has an elevated jugular venous pressure, widespread crepitations in both lungs and bilateral pitting leg oedema suggestive of decompensated heart failure. Her heart auscultation demonstrates a pan-systolic murmur radiating to axilla. ECG shows atrial fibrillation with ventricular rate of 110.

Blood tests show only a slight deterioration of creatinine compared with her baseline. Chest X-ray shows findings consistent with pulmonary oedema in keeping with acute decompensated heart failure.

Repeat echo shows severe MR with mild global left ventricular dysfunction (see figure 10). There is no other significant valvular pathology.

Anne's left heart catheterisation reveals severe stenosis in the mid-section of the right coronary artery. Her right heart catheterisation reveals mildly elevated pulmonary pressures.

A TOE confirms the severity of the MR, with the aetiology thought to be posterior MV prolapse with flail P2 segments.

The case is discussed in the heart team meeting. Anne is deemed an unsuitable candidate for surgical repair given her age, mild cognitive impairment and chronic kidney disease. Her anatomy, as assessed during the TOE, is thought to be suitable for a mitral clip procedure.

The treatment recommendation is for initial stabilisation of her decompensated heart failure, followed by treatment of coronary artery disease and then the mitral clip procedure, given the favourable anatomy for transcatheter end to end repair.

Case study three

Trevor, an 87-year-old male, has regularly been seeing his local physician for known stable moderate MR. He has recently become more fatigued than usual. There is no chest pain and no palpitation.

He has history of chronic atrial fibrillation (more than 10 years), diabetes mellitus and hypertension. He also has history of lung cancer that was treated with radiation and lobectomy eight years earlier. He has since had yearly reviews for his lung cancer and has been in remission.

Trevor lives at home with his wife, is independent in all activities of daily living and still drives. He is an ex-smoker of 20 pack-years and rarely drinks alcohol.

On examination, he is in rate-controlled atrial fibrillation, and is euvolemic. His heart auscultation demonstrates a pan-systolic murmur with no radiation. His chest is clear and there are no signs of heart failure.

His previous six-monthly echocardiograms had been stable for two years. However, his most recent transthoracic echocardiogram that was performed before this presentation showed severe MR and severe bi-atrial enlargement. His left ventricle was normal in size and function and there was no other significant valvular pathology.

Transoesophageal echocardiogram showed a restricted posterior MV leaflet resulting in an overriding anterior leaflet, with a PAGE 22 ▸

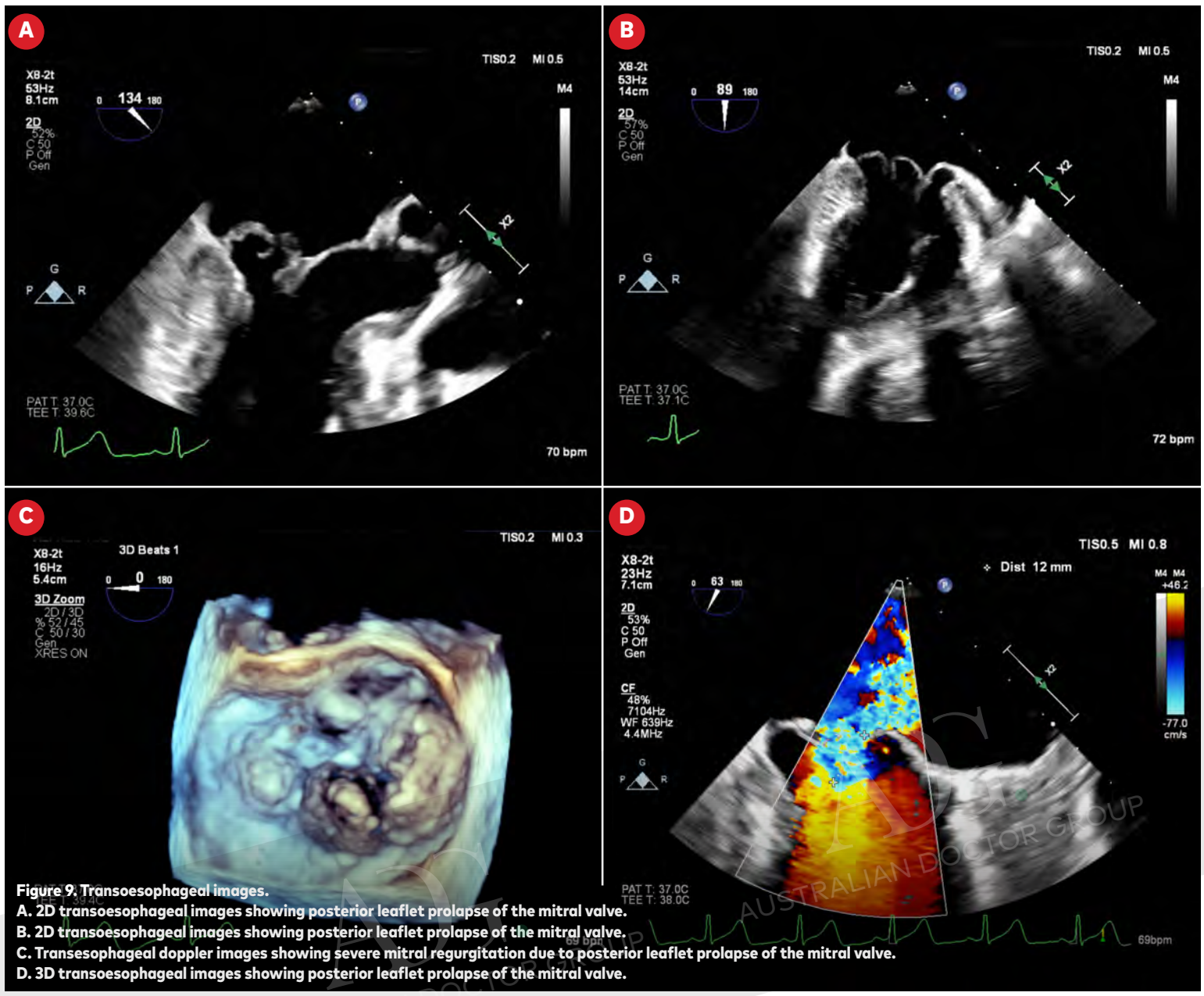


Figure 9. Transoesophageal images.
A. 2D transoesophageal images showing posterior leaflet prolapse of the mitral valve.
B. 2D transoesophageal images showing posterior leaflet prolapse of the mitral valve.
C. Transesophageal doppler images showing severe mitral regurgitation due to posterior leaflet prolapse of the mitral valve.
D. 3D transoesophageal images showing posterior leaflet prolapse of the mitral valve.

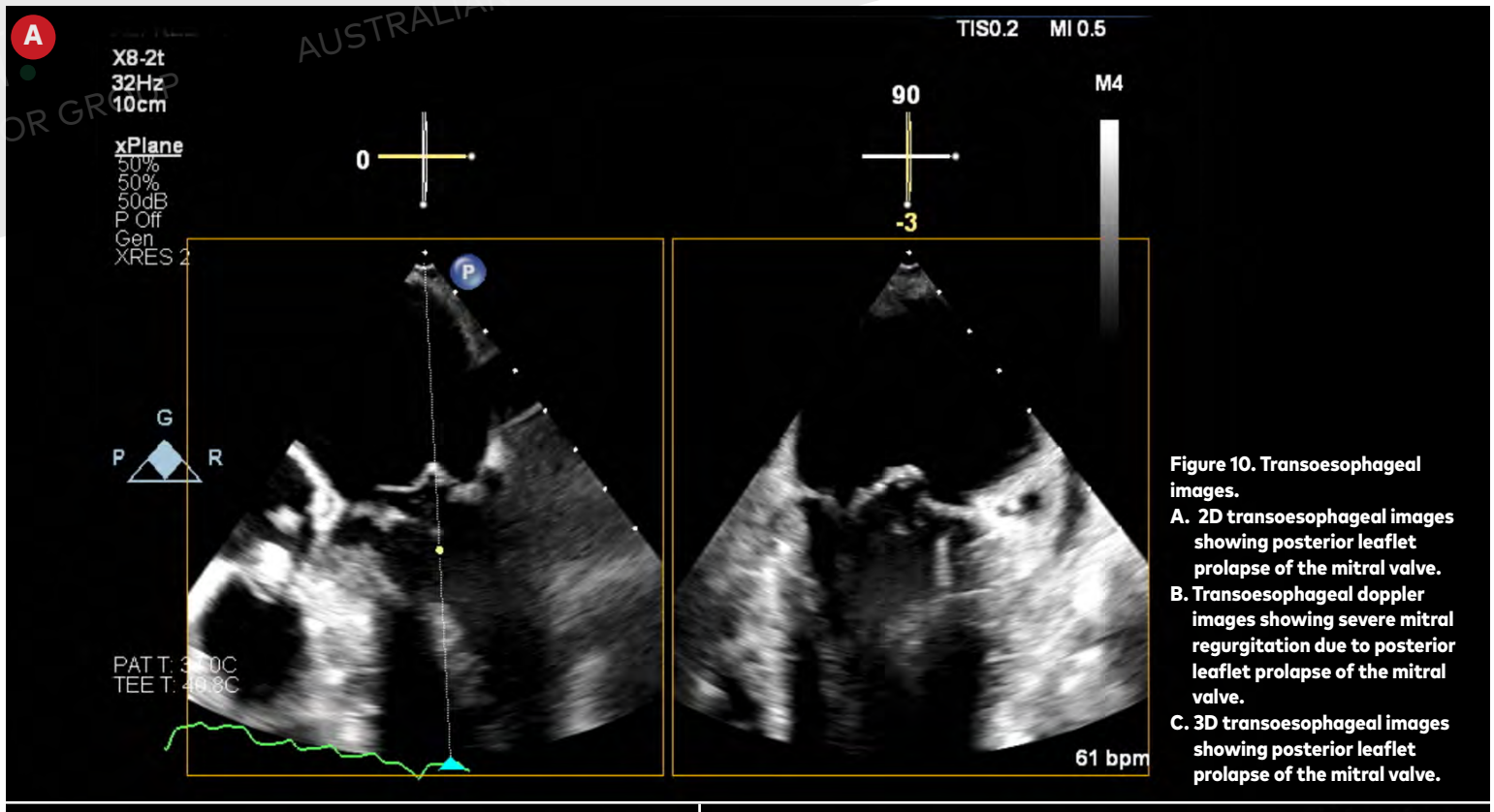


Figure 10. Transoesophageal images.
A. 2D transoesophageal images showing posterior leaflet prolapse of the mitral valve.
B. Transoesophageal doppler images showing severe mitral regurgitation due to posterior leaflet prolapse of the mitral valve.
C. 3D transoesophageal images showing posterior leaflet prolapse of the mitral valve.

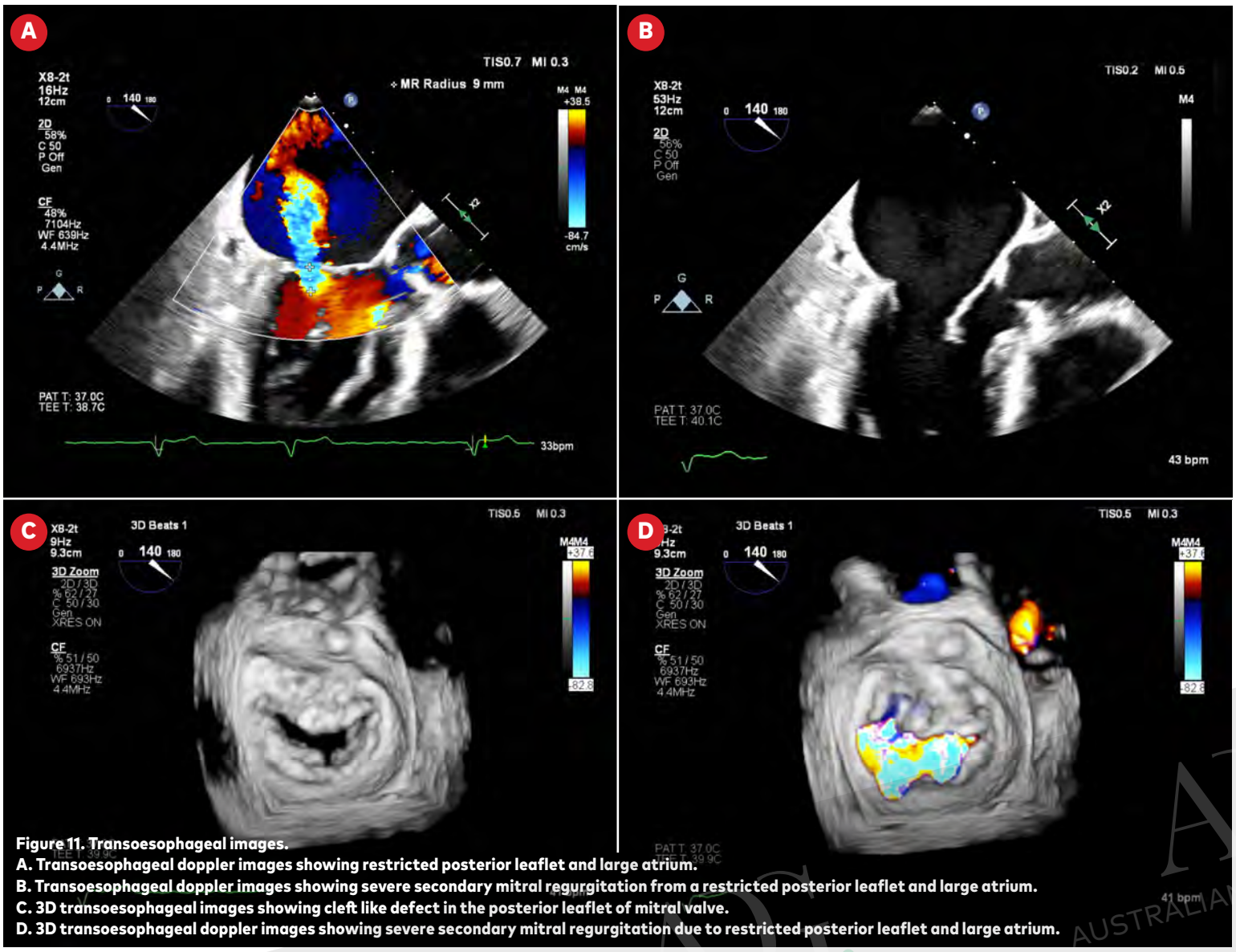


Figure 11. Transoesophageal images.
A. Transoesophageal doppler images showing restricted posterior leaflet and large atrium.
B. Transoesophageal doppler images showing severe secondary mitral regurgitation from a restricted posterior leaflet and large atrium.
C. 3D transoesophageal images showing cleft like defect in the posterior leaflet of mitral valve.
D. 3D transoesophageal doppler images showing severe secondary mitral regurgitation due to restricted posterior leaflet and large atrium.

PAGE 20 small cleft of the posterior leaflet (see figure 11). The aetiology is thought to be mixed degenerative and functional, but the small cleft makes the transcatheter edge-to-edge technique challenging.

His right heart catheterisation shows normal pulmonary pressures, and his left heart catheterisation does not show any significant coronary artery disease. Lung function tests are performed, given his history of lobectomy and smoking, and these show severely reduced lung capacity. Blood tests show a creatinine that is at his baseline level, with a haemoglobin at the upper levels of normal. There are no significant abnormalities in the remaining blood work.

Trevor is referred to the heart team. Given his age, comorbidities (especially the reduced lung capacity from smoking and previous surgery), he is deemed unsuitable for cardiac surgery. His TOE is also reviewed to assess for anatomical suitability.

The mixed nature of the aetiology, and the cleft in the posterior mitral leaflet, render Trevor anatomically unsuitable for percutaneous mitral edge-to-edge repair. The consensus from the heart team meeting is to optimise his medical therapy and refer him for a trial based on new MV devices.

CONCLUSION

MITRAL valve disease is a common disease that requires accurate and detailed assessment. The mitral valve apparatus is very complex; all components, either in isolation or combination, may cause MR.

Thus, a detailed examination is important for decision making and guiding therapeutic options. Echocardiogram, both transthoracic and transesophageal, remains the cornerstone of evaluation of MV.

When intervention is required, surgery is the first-line therapy, but transcatheter options are viable in high-risk surgical patients. Early referral to specialised centres is essential for best outcomes.

A comprehensive history-taking coupled with a routine heart auscultation by GPs can play a pivotal role in early diagnosis and timely referral to specialised centres, ensuring optimal patient outcomes.

RESOURCES

- **Vahanian A, et al. 2021 ESC/EACTS Guidelines for the management of valvular heart disease: Developed by the Task Force for the management of valvular heart disease of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J* 2022;43(7):561–632**
  bit.ly/49SsQK1
- **Otto CM, et al. 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circ* 2021;143:e72–e227**
  bit.ly/3Tgy9NU

References
Available on request from howtotreat@adg.com.au

How to Treat Quiz.

MITRAL REGURGITATION

GO ONLINE TO COMPLETE THE QUIZ **ausdoc.com.au/how-to-treat**

1. **Which THREE statements regarding MR are correct?**
   - **a** It is the most common valvular heart disease in Western countries.
   - **b** MR is defined as backward flow of blood from the left ventricle into the left atrium during diastole.
   - **c** Untreated, the disease can lead to heart failure and death.
   - **d** The US prevalence is greater than 10% in those older than 75 years.

2. **Which TWO may cause primary MR?**
   - **a** Ventricular dilatation.
   - **b** Rheumatic heart disease.
   - **c** Endocarditis.
   - **d** Chronic atrial fibrillation.

3. **Which THREE statements regarding MR are correct?**
   - **a** A recognised cause of MR in the young population is mitral annular calcification.
   - **b** Rheumatic heart disease is a common cause of primary MR in developing countries.
   - **c** Primary MR is defined as a structural abnormality in any component of the MV apparatus causing MV leak.
   - **d** Secondary MR is defined as disease of the left atrium or ventricle that interferes with the integrity of the MV apparatus thus causing a leak in the MV.

4. **Which THREE may be features of MR?**
   - **a** Exertional shortness of breath.
   - **b** Some patients may be asymptomatic.
   - **c** Systemic hypertension.
   - **d** Fatigue and palpitations in severe MR.

5. **Which TWO may be present on examination in MR?**
   - **a** Secondary MR murmurs, generally best heard at the right sternal border.
   - **b** S3 plus a short diastolic murmur in significant primary MR.
   - **c** Murmurs from the posterior leaflet generally directed towards the axilla.
   - **d** A displaced apex beat, loud systolic murmur and cardiomegaly.

6. **Which THREE statements regarding the investigation of MR are correct?**
   - **a** TTE is the initial assessment, given its non-invasive nature and easy availability.
   - **b** TOE is mostly needed for a full assessment of MR.
   - **c** Other useful modalities include cardiac MRI and right and left heart catheterisation.
   - **d** Cardiac MR is generally less accurate and reproducible for quantitating MR volume, LV volume and LV ejection fraction.

7. **Which TWO statements regarding the management of primary MR are correct?**
   - **a** Diuretics and inotropic agents may be indicated in acute MR.
   - **b** Medical therapy has no role in acute MR.
   - **c** Patients with MR and reduced LVEF require guideline directed heart failure therapy only.
   - **d** There is no evidence to support the use of medical therapy in chronic stable MR and preserved LVEF.

8. **Which THREE statements regarding intervention in primary MR are correct?**
   - **a** Intervention is indicated in all asymptomatic patients with severe MR.
   - **b** Valve replacement is associated with a greater life expectancy than valve repair.
   - **c** When intervention is indicated, surgery remains the first line of therapy.
   - **d** Percutaneous MV edge-to-edge repair is safe and effective in patients with primary MR and a prohibitive surgical risk.

9. **Which TWO statements regarding the management of secondary MR are correct?**
   - **a** Treatment focuses on the mechanism of the MR.
   - **b** Guideline-directed medical therapy in patients with heart failure and secondary MR improves mortality and quality of life.
   - **c** Consider intervention before guideline-directed medical therapy.
   - **d** Given the homogenous aetiology, outcomes post intervention are comparable.

10. **Which THREE statements regarding the intervention in secondary MR are correct?**
    - **a** TEER is not recommended in secondary MR.
    - **b** Decisions for intervention are based on the recommendations of a multidisciplinary heart team.
    - **c** Consider mitral valve repair or replacement with bypass surgery in functional MR associated with coronary artery disease.
    - **d** Evidence for surgery in isolated secondary MR is limited.

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