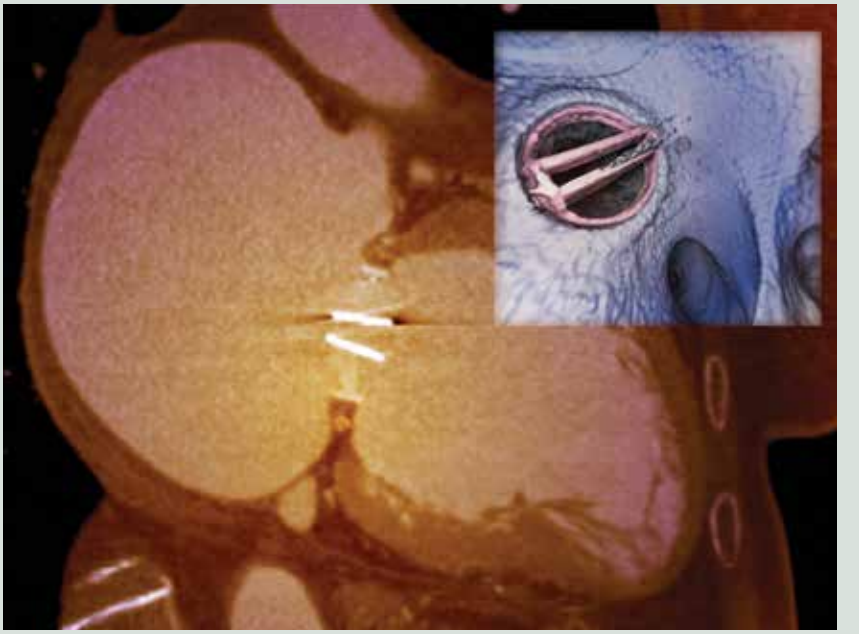
Mechanical valves: a silent worry

AUSTIN N. MAY MB BS(Hons)
JAMES F. ROGERS MB BS, FRACP, FCSANZ
GORDIAN FULDE MB BS, FRACS, FRCS(Ed), FRCS/FRCP(A&E)(Ed), FACEM

Articles in this section use cases to illustrate the emergency management of patients presenting in general practice with cardiac problems. They are inspired by, but not based on, real patient situations.

CARDIOLOGY TODAY 2016; 6(4): 28-30

Dr May is a Cardiology Advanced Trainee at Gosford Hospital, Gosford; and Conjoint Associate Lecturer at UNSW Australia, Sydney.
Dr Rogers is a Consultant Cardiologist and Director of the Clinical Trials Unit in the Central Coast Local Health District, NSW; and Senior Lecturer at the University of Newcastle, Newcastle.
Professor Fulde is Director of the Emergency Department at St Vincent's Hospital; Associate Professor in Emergency Medicine at UNSW Australia; and Professor in Emergency Medicine at the University of Notre Dame, Sydney, NSW.



Mr KD is a 63-year-old schoolteacher who sees you regularly to monitor his warfarin therapy and cardiovascular risk profile. He takes warfarin for atrial fibrillation (AF) and the mechanical mitral valve replacement that he received 25 years ago for rheumatic mitral stenosis, with a target INR of between 2.5 and 3.5. He takes regular metoprolol for heart rate control, which was shown to be adequate on Holter monitoring a few years ago. He is overweight and has hypertension, as well as a family history of premature coronary artery disease. He has no other medical problems. He is married with three adult children and enjoys playing cards at his bridge club.

Today, Mr KD asks to see you because he has developed breathlessness on exertion. He first noticed this two months ago. The breathlessness occurs on mild to moderate exertion outside the house (corresponding to NYHA Class II symptoms). He denies experiencing chest pain, orthopnoea, leg swelling or palpitations. There is also no sputum production, fever or rigors. He is not usually an anxious person but reports being worried about his heart valve. In particular, one of his bridge partners noticed that the valve has fallen silent, having lost its regular 'click'. Mr KD cannot recall this happening before and thought it should be checked.

What does the clinical examination show?

Mr KD's heart rate is 90 beats per minute and irregularly irregular. His blood pressure is 155/80 mmHg, and his peripheral oximetry results and temperature are normal. There is no conjunctival pallor or haemorrhages, and his jugular venous pressure is not elevated. His first heart sound is reduced, with no audible click from the

mechanical valve, and his second heart sound is normal. There are no obvious murmurs. Auscultation of his lung fields reveals medium paninspiratory crackles in the lower zones only, with no wheeze. There is no peripheral oedema or splinter haemorrhages. His ECG shows AF with a normal ventricular rate and without ischaemic changes. His spirometry results are within normal limits.

What are the potential causes of the dyspnoea?

The important considerations for Mr KD's dyspnoea include cardiac, respiratory, haematological and metabolic causes. Firstly, the onset of symptoms that Mr KD describes is subacute, making the chronic causes of dyspnoea such as emphysema unlikely. Secondly, you think pulmonary embolism is unlikely given his warfarin therapy, and you confirm that his INR readings have been in the therapeutic range. Thirdly, there are no infective symptoms or signs to suggest bacterial endocarditis. The remaining causes of dyspnoea are possible, including malfunction of his heart valve, so you plan to refer Mr KD to a cardiologist once you have the results of some simple tests.

Should you send Mr KD to a hospital emergency department?

The threshold to send a patient such as Mr KD to a hospital emergency department (ED) for new-onset dyspnoea can be based on the presence of:

- symptoms, such as dyspnoea during activities of daily living (corresponding to NYHA Class III) or dyspnoea at rest (corresponding to NYHA Class IV)
- vital sign abnormalities, such as hypoxia or tachypnoea
- clinical signs, such as paninspiratory crackles to the mid-zones of the lung or diffuse wheeze
- abnormal laboratory or radiology findings (e.g. acute pulmonary oedema on chest x-ray or marked anaemia on full blood count).

What additional tests are required?

You provide Mr KD with a request for a chest x-ray, mainly to examine for cardiomegaly, oedema or pulmonary infiltrates. You also provide a pathology request including:

- a full blood count and measurement of C-reactive protein level to examine for possible anaemia or inflammation
- biochemistry tests to examine for renal or liver dysfunction
- INR to confirm it is in the range 2.5 to 3.5.

The chest x-ray shows small pleural effusions and mild interstitial fluid in the absence of cardiomegaly. The blood test results are normal. You telephone and arrange an appointment for Mr KD to see a cardiologist urgently within a week, and at the advice of the cardiologist ask him to trial low-dose furosemide (frusemide) in the interim. You also instruct Mr KD to call an ambulance or present to hospital if his symptoms worsen.

How does the cardiologist approach this case?

Mr KD is a nonsmoker who has a two-month history of exertional dyspnoea in the absence of chest pain, and in the context of a previous mechanical mitral valve prosthesis and permanent valvular AF. With normal blood test and spirometry results, and given his therapeutic warfarin levels, the cause of his dyspnoea is likely to be cardiac. Broadly, cardiac causes of dyspnoea are systolic or diastolic heart failure, valvular pathology, pericardial pathology, pulmonary hypertension, tachyarrhythmia or coronary artery disease.

Mr KD undergoes transthoracic echocardiography, which reveals normal left and right ventricular size and systolic function, a left ventricular ejection fraction over 60%, severely increased mean pressure gradient across the mechanical mitral valve prosthesis (15 mmHg, normal ≤5 mmHg) with increased peak velocity (2.7 m/s, normal <1.8 m/s), mild valvular mitral regurgitation, and mildly elevated pulmonary pressures (peak 37 mmHg, normal ≤30 mmHg).

These results explain Mr KD's symptoms and are concerning for degeneration or malfunction of the mechanical mitral valve.

What are the causes of prosthetic valve dysfunction?

Dysfunction of prosthetic valves can be differentiated into early or late complications. Early complications may result from technical challenges during surgery, patient–prosthesis mismatch, conduction abnormalities requiring permanent pacemaker implantation, or early infection.[1] The incidence and nature of late complications depend on the type, durability and thrombogenicity of the valve prosthesis. The different types of prosthetic heart valves are summarised in the Box. Late complications include dehiscence, pannus formation (a reaction between the prosthesis and host, causing fibrous ingrowth), thrombus formation, late infection, and valve degeneration causing stenosis and/or regurgitation. In a study of 2680 patients with mechanical heart valves over 27 years, the incidence of re-operation was 9%, mainly because of dehiscence (53%), pannus (19%) and thrombosis (12%).[2]

Types of prosthetic heart valves as classified by the American Society of Echocardiography[1]

Biologic

- Stented
  - Porcine xenograft
  - Pericardial xenograft
- Stentless
  - Porcine xenograft
  - Pericardial xenograft
  - Homograft (allograft)
  - Autograft
- Percutaneous

Mechanical

- Bileaflet
- Single tilting disc
- Caged-ball

How can we further assess a mechanical heart valve?

Mr KD undergoes transoesophageal echocardiography with two and three-dimensional image acquisition, which identifies no evidence of valvular or paravalvular thrombus. The valve is well seated in the mitral annulus with no evidence of dehiscence. However, as commonly happens, assessment of the prosthetic leaflets on echocardiography is limited by metal-induced shadowing (Figure 1). Consequently, Mr KD undergoes fluoroscopy of the valve

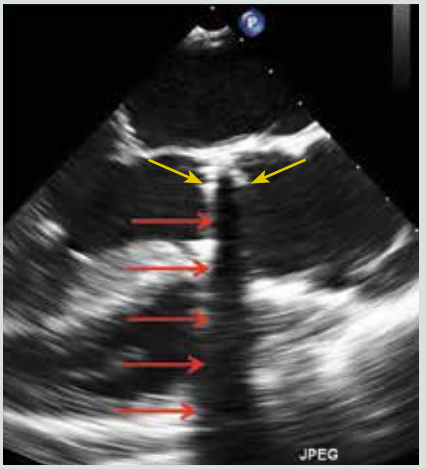


Figure 1. Transoesophageal echocardiography image showing a metal-induced acoustic shadow (red arrows) obscuring assessment of a mechanical valve. The valve in this patient (not Mr KD) is a mechanical aortic valve, which is hidden in the top section of the acoustic shadow (yellow arrows).

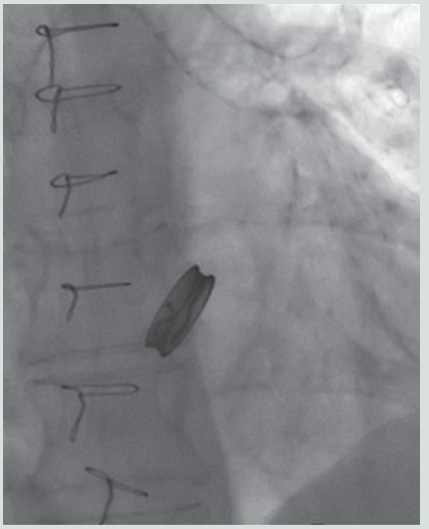


Figure 2. Still image showing fluoroscopy being used to assess the function of the mechanical valve prosthesis in the same patient as in Figure 1. Note the sternal wires on the left of the image.

prosthesis (Figure 2). This reveals tethering of the prosthetic leaflets and explains the elevation in pressure gradients across the valve seen on transthoracic echocardiography.

When is referral for cardiothoracic surgery required?

The decision to re-operate on a heart valve replacement is significant. The onset or worsening of symptoms such as dyspnoea usually indicates the need for an opinion from a cardiothoracic surgeon. If a patient has no symptoms then re-operation would be considered only in the presence of objective evidence of severity, such as severe valve malfunction, dehiscence, infection or left ventricular dysfunction.

Outcome: Mr KD was reviewed by a cardiothoracic surgeon, who also examined the echocardiography and fluoroscopy images provided by the cardiologist. The surgeon considered Mr KD had an appropriate indication for 'redo mitral valve replacement', and he underwent elective surgery four weeks after he initially presented to your practice. Intraoperatively, the cardiothoracic surgeon found significant pannus obstructing the opening of the prosthetic leaflets. Given Mr KD's relatively long life expectancy and need for anticoagulation for permanent AF, he received another mechanical mitral valve prosthesis rather than a bioprosthetic valve. Furosemide was ceased after the surgery. Mr KD required three months' leave from work to recover, during which time he enjoyed playing bridge. His postoperative recovery was otherwise uneventful. **CT**

References

1. Zoghbi WA, Chambers JB, Dumesnil JG, et al. Recommendations for evaluation of prosthetic valves with echocardiography and doppler ultrasound: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Task Force on Prosthetic Valves. J Am Soc Echocardiogr 2009; 22: 975-1014.
2. Rizzoli G, Guglielmi C, Toscano G, et al. Reoperations for acute prosthetic thrombosis and pannus: an assessment of rates, relationship and risk. Eur J Cardiothorac Surg 1999; 16: 74-80.

COMPETING INTERESTS: None.