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EDUCATIONAL PAPER

Acute valvular emergencies

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Acute valvular emergencies represent an important cause of cardiogenic shock. However, their clinical presentation and initial diagnostic testing are often non-specific, resulting in delayed diagnosis. Moreover, metabolic disarray or haemodynamic instability may result in too great a risk for emergent surgery. This review will focus on the aetiology, clinical presentation, diagnostic findings, and treatment options for patients presenting with native acute left-sided valvular emergencies. In addition to surgery, options for medical therapy, mechanical circulatory support, and novel percutaneous interventions are discussed.

Keywords Acute aortic regurgitation • Acute mitral regurgitation • Aortic stenosis • Cardiogenic shock • Mitral stenosis

Introduction

Acute valvular emergencies comprise approximately 8% of coronary care unit admissions.[1] Unfortunately, given their non-specific clinical presentation and subtle examination findings, diagnosis is often elusive. In light of a relative paucity of data to guide management, recommendations are based primarily on expert opinion in combination with small case series and retrospective analyses.[2,3] Most importantly, prompt recognition of acute valvular disease in combination with surgical (or percutaneous) intervention portends the best prognosis. For patients with multiple comorbidities or profound metabolic disarray in whom immediate intervention is considered too high risk, medical therapy in combination with mechanical circulatory support (MCS) may be used until stability is achieved.

This review aims to provide a brief overview of acute valvular emergencies with a focus on the diagnosis and management of native left-sided valvular regurgitation and stenosis (given they are most frequently the cause of haemodynamic decompensation). Multiple aspects of management will be discussed, including medical therapy, MCS, surgery, and the role of percutaneous interventions, such as transcatheter edge-to-edge repair (TEER) and transcatheter aortic valve replacement (TAVR).

Acute valvular regurgitation

There are fundamental differences in the pathophysiology underlying acute versus chronic regurgitant lesions. In chronic regurgitant lesions, left ventricular (LV) remodelling occurs, primarily characterized by chamber enlargement. This augments forward stroke volume and allows for maintenance of overall cardiac output without elevations in left ventricular end-diastolic pressure (LVEDP).[4]

In acute mitral and aortic valvular regurgitation, sudden rises in LV end-diastolic volumes lead to rapid elevations in LVEDP, left atrial (LA) pressure and pulmonary venous pressures with resultant pulmonary congestion (*Figure 1*). While there is some augmentation in total stroke volume due to the Frank–Starling mechanism, high regurgitant volumes result in decreased forward flow and a low effective cardiac output. This is compounded in acute aortic regurgitation (AR), wherein severely elevated LVEDP may cause premature closure of the mitral valve and diastolic mitral regurgitation (MR), resulting in further impairment of effective cardiac output and greater elevations in pulmonary venous pressures (*Figure 2*).[5,6] Compensatory tachycardia is common to both acute valvular lesions, however, is often inadequate to preserve forward stroke volume and may be detrimental in those with a Type A aortic dissection or concomitant ischaemia.

It is worth noting that acute mitral or aortic regurgitation is better tolerated in patients with pre-existing valvular regurgitation and normal LV function, compared with those with impaired systolic or diastolic LV function, ischaemia, or aortic dissection.

Aetiology

While causes of acute regurgitation vary based on the valve affected, there are multiple shared aetiologies (*Table 1*). Notably, acute severe MR can be divided into primary and secondary causes (i.e. structural

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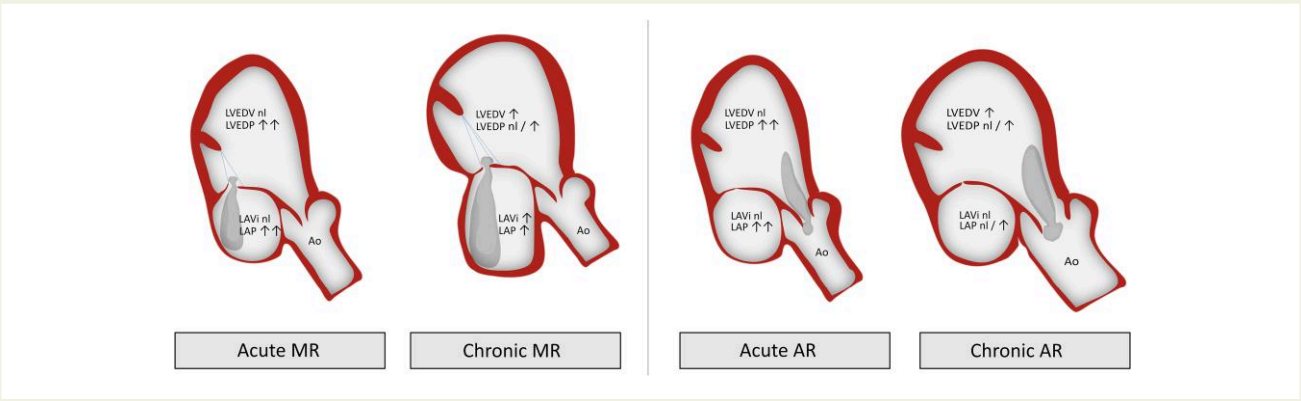

Figure 1 Haemodynamic changes in acute valvular regurgitation. LAP, left atrial pressure; LAVi, left atrial volume index; LVEDV, left ventricular end-diastolic volume; LVEDP, left ventricular end-diastolic pressure; nl, normal.

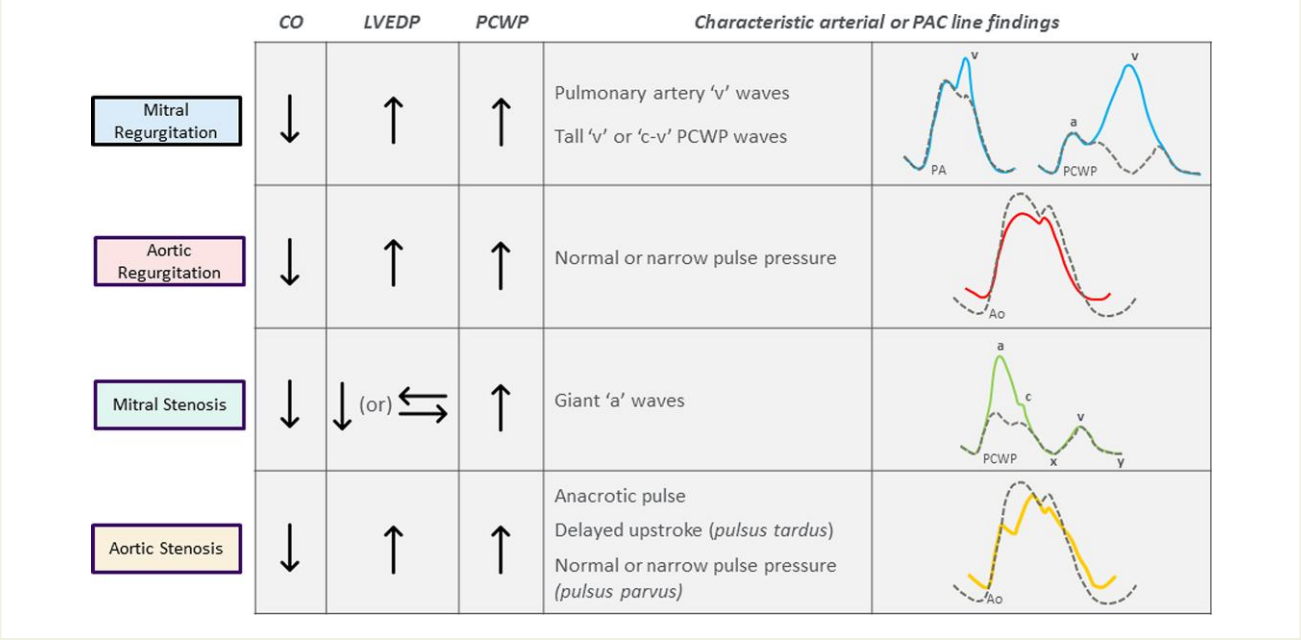

Figure 2 Haemodynamic findings in acute valvular emergencies. *Note:* solid lines indicate the abnormalities observed with the associated valvular emergency, whereas dashed lines represent normal arterial or PAC tracings. CO, cardiac output; LVEDP, left ventricular end-diastolic pressure; PA, pulmonary artery; PAC, pulmonary artery catheter; PCWP, pulmonary capillary wedge pressure; Ao, aorta.

abnormalities of the mitral valve or chordal structures versus left ventricular dysfunction with an intact mitral valve apparatus, respectively). Ultimately this distinction is critical, as it factors heavily into the management strategy.

Clinical presentation

Patients with acute valvular regurgitation will typically present with dyspnoea and cardiogenic shock, owing to rapid and sudden elevations in LVEDP. Physical examination will reflect the aforementioned with tachypnoea, inspiratory crackles, and hypoxaemia. Tachycardia, altered mental status, hypotension, and cool extremities are expected findings, with an associated *narrow* pulse pressure (although severe AR has been associated with a wide pulse pressure, this is typically seen in patients with chronic, rather than acute aortic insufficiency). Other clinical symptoms may be present owing to the aetiology of regurgitation (e.g. fever or peripheral emboli in endocarditis, or chest pain secondary to ischaemia or aortic dissection).

While 'classic' (and eponymous) auscultatory findings associated with severe mitral and aortic valvular regurgitation are characteristic for *chronic* valvular regurgitation, findings in acute valvular regurgitation are different and often elusive (*Figure 3*).[4] Notably, in up to 30% of patients with acute myocardial infarction and acute severe MR, no audible murmur is noted at all.[7] Furthermore, the physical

Table 1 Causes of acute mitral and aortic regurgitation

Mitral regurgitation	Aortic regurgitation	
	Both	
[a]Chord rupture	[a]Endocarditis	[a]Type A aortic dissection
Papillary muscle rupture	Trauma/deceleration injury	• Sinotubular dilation
Acute rheumatic fever	Peri-procedural complications	• Dilated sinus(es) of Valsalva
Papillary muscle dysfunction (ischaemia)	• Balloon valvuloplasty	• Aortic cusp prolapse
Takotsubo's cardiomyopathy	• TAVR	• Intimal flap prolapse through aortic valve
• SAM with LVOT obstruction	• Post-catheterization	Rupture of congenital fenestration
• Mitral valve leaflet tethering		
Acute cardiomyopathy		

LVOT, left ventricular outflow tract; SAM, systolic anterior motion; TAVR, transcatheter aortic valve replacement.
[a]Most common causes of acute valvular regurgitation.

examination may be obscured by the environment in which the patient is examined. Indeed, important contributors to the delayed diagnosis of acute valvular regurgitation are (i) conflation of 'classic' physical examination findings in acute versus chronic valvular regurgitation and (ii) overestimation of the sensitivity of the physical examination in diagnosis.

Given the delays in diagnosing acute valvular regurgitation (and the associated morbidity and mortality), cardiogenic shock in combination with certain clinical scenarios should herald rapid evaluation for the aforementioned (*Table 2*).

Diagnostic evaluation

Transthoracic echocardiography (TTE) is considered the first-line modality for evaluation of valvular regurgitation given its availability, portability and diagnostic yield in patients presenting with acute-onset dyspnoea. Notably, however, TTE may be limited by poor patient windows, concomitant lung disease, or respiratory distress. Even with good imaging quality, highly eccentric regurgitant lesions may be qualitatively underestimated and the underlying mechanism may be difficult to discern. In these circumstances, there should be a low threshold to utilize transesophageal echocardiography (TEE) to better delineate the degree and mechanism of acute valvular regurgitation. Moreover, if surgical planning is required, TEE can be a useful surgical adjunct. Of note, while point-of-care ultrasound has become a mainstay of patient evaluation in a multitude of hospital settings, incomplete or inexperienced evaluation can lead to a delay in diagnosis or misdiagnosis. Therefore, corroboration of findings (or lack thereof) should always be confirmed with a complete echocardiogram.

Echocardiographic findings in the setting of acute mitral or aortic regurgitation are described in *Figures 4 and 5* and *Table 3*. Two-dimensional (2D) and three-dimensional (3D) echocardiography can demonstrate the mechanism of acute valvular regurgitation such as ruptured chordae tendinae, papillary muscle rupture, prolapsed or flail leaflets, or an ascending aortic dissection flap. LV and LA sizes are often normal given the acute nature of the presentation. LV function is typically normal or hyperdynamic, however, regional wall motion abnormalities may suggest concomitant acute ischaemia (e.g. in the setting of ascending aortic dissection with coronary involvement, papillary muscle rupture), acute myocarditis or Takotsubo's cardiomyopathy. Premature mitral valve closure is a hallmark of acute severe AR and represents a rapid rise in LV diastolic pressure above LA pressure.

The application of colour Doppler is most likely to clue the clinician in to acute valvular regurgitation as the primary aetiology of decompensation. While large areas of flow convergence with a prominent vena contracta are typically seen, very severe lesions may lack turbulent flow owing to rapid equalization of chamber pressures. Eccentric jets should be well delineated to avoid underestimation of the degree of severity. Of note, quantitative measures for valvular regurgitation (e.g. effective regurgitant orifice area, regurgitant volume) should not be used given their decreased accuracy in the context of tachycardia and altered loading conditions secondary to cardiogenic shock.[4]

Pulsed and continuous-wave Doppler interrogations may corroborate the severity of acute valvular regurgitation. Flow reversal in the pulmonary veins and abdominal aorta have high specificities for severe mitral and aortic regurgitation, respectively.[9] Continuous-wave Doppler profiles of acute MR will typically be densely echogenic, have a low peak velocity (< 5 m/s) and triangular in shape, reflecting rapid equalization of pressures between the LV and LA. Analogously, continuous-wave Doppler profiles of acute AR will also be densely echogenic with a short pressure half-time and low-end diastolic velocity (reflecting rapid equalization of aortic and LV pressures).

While frequently obtained, the electrocardiogram (ECG) and chest X-ray (CXR) lack sensitivity and specificity to establish the diagnosis of acute valvular regurgitation. Nonetheless, ECGs can still provide important clues to the aetiology of acute valvular regurgitation (e.g. ST-segment elevation or Q waves). Typical CXR findings are that of pulmonary oedema with a normal cardiac silhouette. Of note, pulmonary oedema can be unilateral in acute eccentric MR with flow directed towards a single pulmonary vein.[10] This finding may be easily confused with pneumonia as it is frequently accompanied by leukocytosis and may occur concomitantly with other systemic illness such as endocarditis. As such, it must be carefully interpreted in the clinical context so as not to delay diagnosis.

Coronary angiography is typically not required either routinely or pre-operatively *unless* it is suspected that treatment of acute

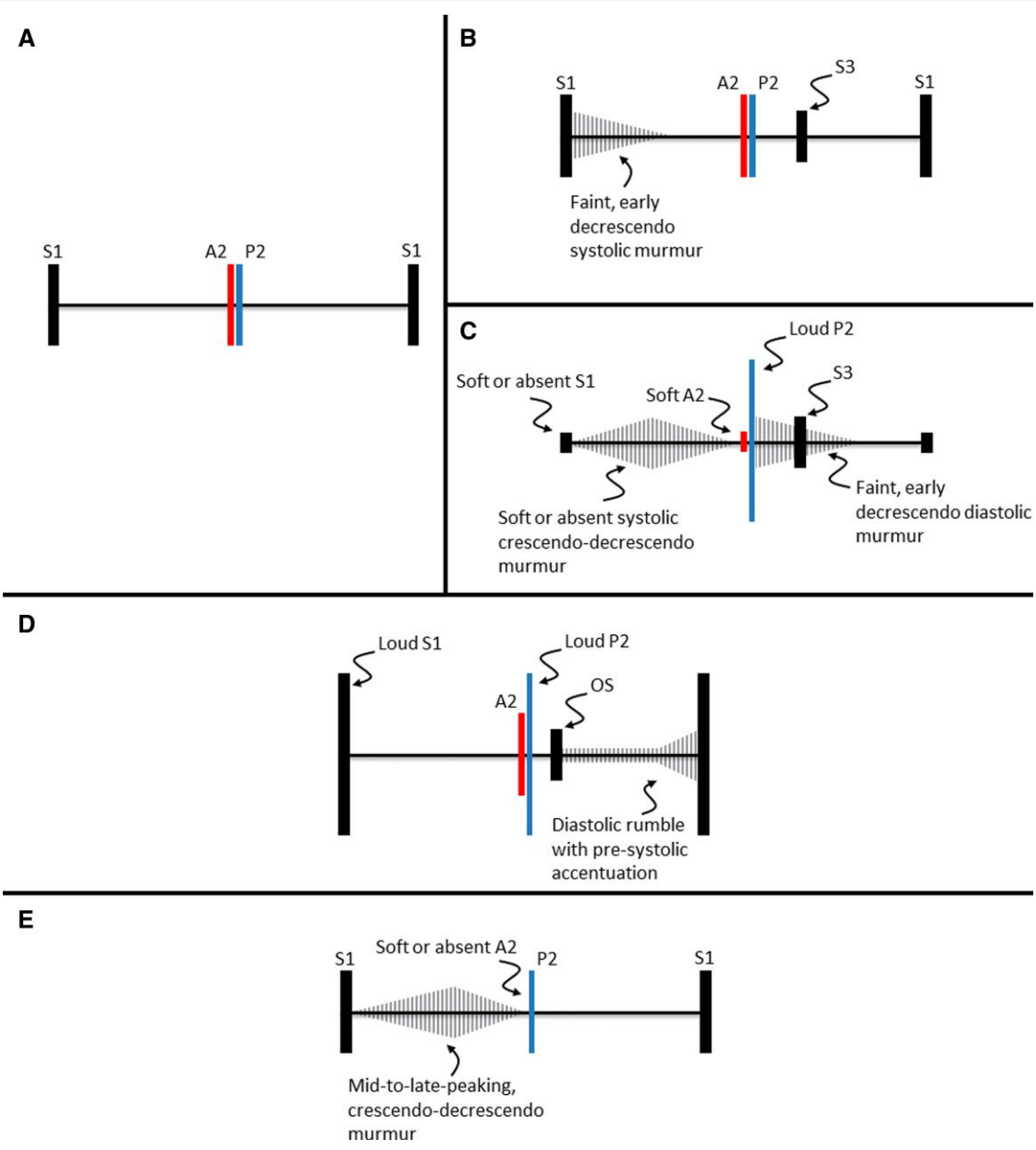

Figure 3 Auscultatory findings in acute valvular emergencies. Normal (*A*) when compared with acute severe mitral regurgitation (*B*), acute severe aortic regurgitation (*C*), chronic severe rheumatic mitral stenosis (*D*), and chronic severe aortic stenosis (*E*). OS, opening snap.

ischaemia will reverse acute MR. Delays in surgery or percutaneous valvular intervention may otherwise only serve to worsen cardiogenic shock.

Management

Much like complex coronary artery disease and chronic structural heart disease, comprehensive management of acute valvular regurgitation requires a heart-team approach (*Figure 6*).[2] Primary treatment of acute valvular regurgitation is surgical. However, for patients with haemodynamic instability, a significant number of comorbidities or profound metabolic disarray, surgery may be delayed. In this setting, medical therapy combined with the use of MCS may be used as a 'bridge' for the critically ill patient. For those patients where the risk of surgery is too great, structural interventions such as TAVR and TEER have emerged as potential treatment options. As always, patient goals of care are paramount and should guide all management decisions as made by the treating team.

Surgery

Treatment of acute primary MR and acute AR is surgical in nature and (in accordance with European Society of Cardiology and American Heart Association guidelines) should therefore be treated as a *surgical emergency* with prompt inclusion of the cardiothoracic surgery team.[2,3] Knowing the aetiology plays a critical role in what surgical intervention is chosen. For those with acute MR, mitral valve

Table 2 Clinical scenarios associated with the presence of acute valvular regurgitation

Acute onset of shortness of breath OR cardiogenic shock OR sudden decompensation	PLUS
Mitral regurgitation	
History of mitral valve prolapse	
STEMI *or* NSTEMI	
At presentation (*papillary muscle dysfunction*)	
2–7 days after presentation (*papillary muscle rupture*)	
Bacteraemia (particularly staphylococcal, streptococcal, or enterococcal species)	
Acute neurologic event (e.g. intracranial haemorrhage and ischemic stroke)	
Recent intense emotional or physical stress	
Other criteria for acute rheumatic fever (migratory polyarthritis, chorea, erythema marginatum, and subcutaneous nodules)	
Recent procedure (e.g. BMV, TAVR, and cardiac catheterization)	
Trauma/deceleration injury	
Aortic regurgitation	
Diagnosis OR signs/symptoms of aortic dissection	
Bacteraemia (particularly staphylococcal, streptococcal, or enterococcal species)	
Trauma/deceleration injury	

repair may only be possible in patients with chordal rupture, whereas mitral valve replacement may be required in patients with endocarditis or papillary muscle rupture. Acute AR often requires aortic valve replacement and may necessitate intervention on the ascending aorta or its associated branch vessels. In some cases, however, a valve-sparing root replacement or an isolated repair of the ascending aorta is sufficient.[11]

While surgery is considered first-line therapy for the management of acute valvular regurgitation, outcomes are worse compared with intervention on patients with chronic regurgitant lesions. In one cohort of 279 patients with acute severe MR (the aetiology being acute myocardial infarction > degenerative > infective endocarditis), 30-day operative mortality was 22.5% with cardiogenic shock representing one predictor of early death.[12] Similarly, in a retrospective analysis of 1342 patients undergoing surgery for papillary muscle rupture, 30-day (or in-hospital) operative mortality was 20.0%, with cardiogenic shock and emergent salvage status representing adverse predictors.[13] Notably, however, in smaller studies, post-operative mortality with papillary muscle rupture has climbed to as high as 53%.[14] Patients with acute AR face similar challenges. Surgical mortality rates for Type A aortic dissection are 18%, with AR representing one major significant risk factor.[15,16] Finally, the ESC-EORP European Endocarditis registry noted in-hospital mortality rates of 17.1% for all patients diagnosed with endocarditis, with two important independent predictors of mortality being congestive heart failure and failure to undertake surgery when indicated.[17]

The decision to proceed with surgical intervention is complex, often hinging on the assessment of perioperative risk relative to the risk of non-operative management. The Society of Thoracic Surgeons risk score may be helpful in this regard as are some other prognostic algorithms that have been developed.[18,19] Ultimately, however, ongoing multidisciplinary discussions are crucial in the assessment of timing for surgical intervention. As discussed below, medical therapy and MCS can sometimes establish periods of stability facilitating a safer transition to the operating room for definitive correction.

Percutaneous structural intervention

Percutaneous structural interventions such as TEER and TAVR have gained an increasing role in the management of chronic progressive valvular disease. However, for patients with severe acute regurgitation and a prohibitively high surgical risk, percutaneous intervention has been suggested as a possible alternative treatment. TEER has been successfully employed in patients with acute MR due to chordal rupture, acute myocardial infarction, and even papillary muscle rupture.[14,20,21] However, these interventions will often be technically challenging and cannot be performed in the setting of concomitant endocarditis. TAVR has a narrower range of utility for acute AR for several reasons. Primarily, current devices need to be seated in a rigid annulus, which may not be the case for acute AR. Device selection is also performed in conjunction with comprehensive testing, including a gated multidetector computed tomography (MDCT) scan to assist with TAVR sizing. Finally, TAVR cannot be used in either of the two most common reasons for acute AR—endocarditis and aortic dissection.

Traditional medical therapy and mechanical circulatory support

Medical therapy for acute mitral and aortic regurgitation is limited and primarily focused on diuresis and afterload reduction (*Figure 7*). Reduction in systemic afterload promotes forward flow in both regurgitant lesions and therefore is a critical aspect of management. Similarly, diuretic therapy decreases overall pulmonary congestion. While heart rate has not been investigated to play a major role in the management of acute MR, lower heart rates are essential when the aetiology of severe MR is systolic anterior motion of the mitral valve. For patients with acute AR, increased heart rates shorten diastole and have been demonstrated to decrease regurgitant volumes and increase stroke volume.[22] For patients with low heart rates despite inotropic or chronotropic agents, transvenous pacing should be considered.[23]

While nitroprusside has been recommended as the afterload agent of choice in both lesions, this is likely of somewhat historical context given its evaluation in initial studies in the 1970s.[24,25] It is likely that most intravenous afterload reducing agents will work in these scenarios, including isoproterenol for acute AR with low heart rates, intravenous β-blockers in Type A aortic dissection and intravenous nitroglycerin for patients with concomitant ischaemia. For patients with persistent hypotension, inotropes should be considered. While there is no clear 'drug of choice' should additional vasopressors be required, norepinephrine is associated with fewer arrhythmias and is often utilized as a first-line pressor in cardiogenic shock. Dopamine and epinephrine may also be considered depending on the clinical scenario, although notably, both have been

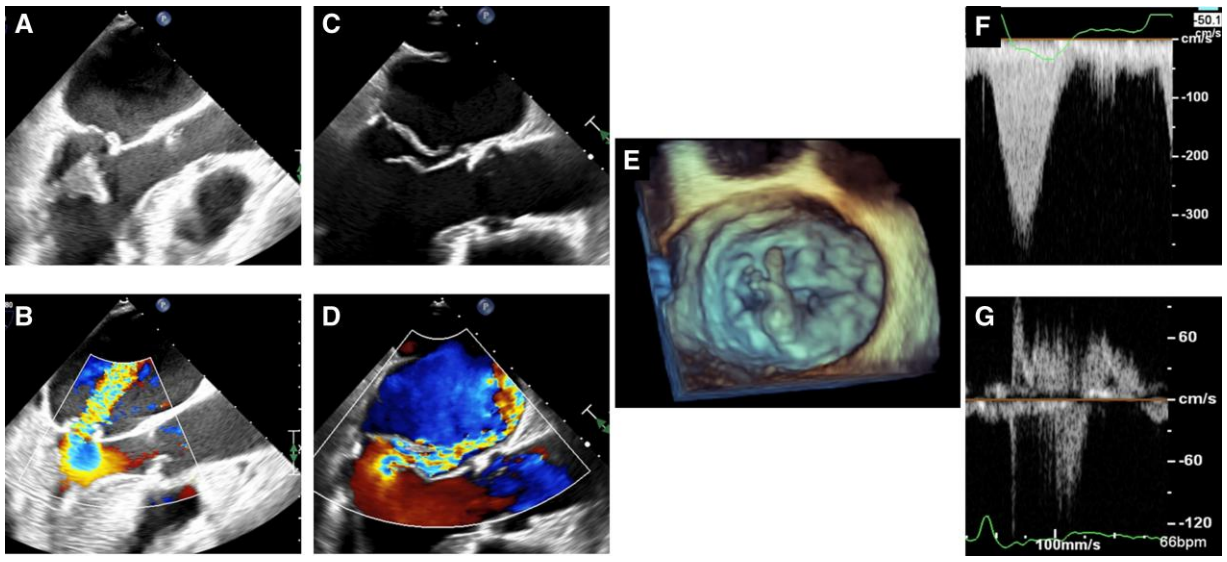

Figure 4 Echocardiographic findings in acute mitral regurgitation. Two-dimensional and colour Doppler imaging of papillary muscle rupture (*A, B*) and a flail posterior mitral valve leaflet (*C, D*) with associated three-dimensional imaging (*E*). Continuous-wave Doppler in acute mitral regurgitation (*F*) and pulsed-wave Doppler demonstrating systolic pulmonary vein reversal (*G*).

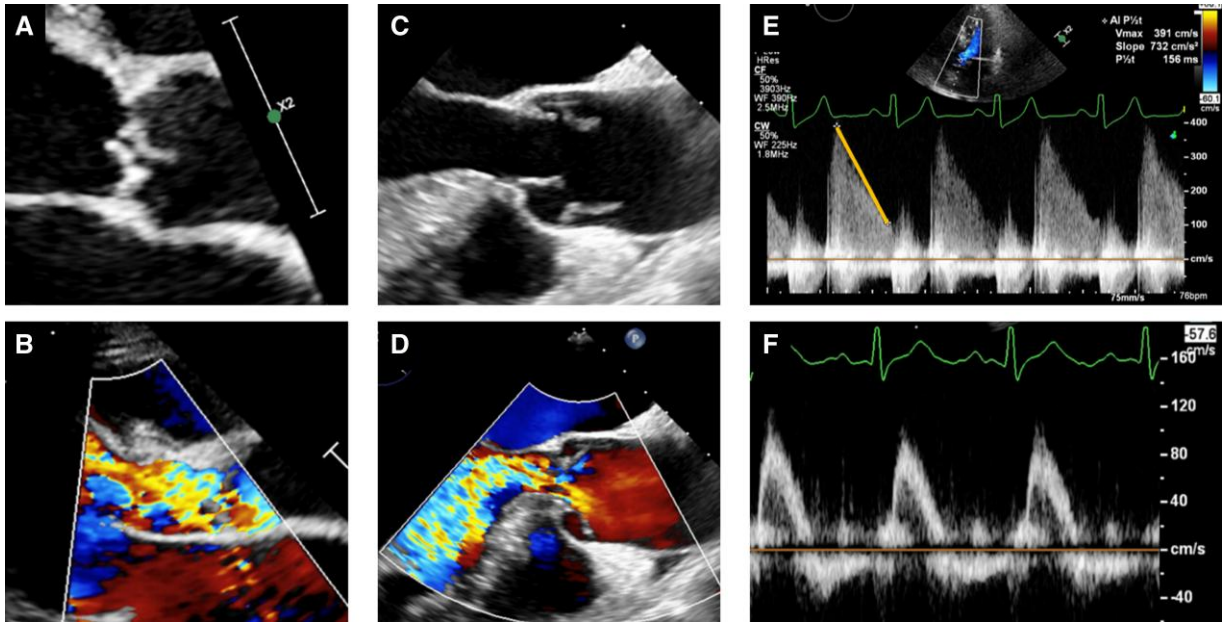

Figure 5 Echocardiographic findings in acute aortic regurgitation. Two-dimensional and colour Doppler imaging of a flail aortic valve leaflet (*A, B*) and Type A dissection with intimal flap prolapse (*C, D*). Continuous-wave Doppler demonstrating a short pressure half-time with low end-diastolic velocity (*E*) and pulsed-wave Doppler showing diastolic flow reversal in the abdominal aorta (*G*).

associated with adverse outcomes in subgroups of patients with cardiogenic shock.[23,26,27] Of note, no clear data have established a 'target heart rate' in patients with acute AR, particularly for patients with a Type A aortic dissection. As such, intravenous β-blockers are not strictly contraindicated in this setting.

MCS has emerged as an option to haemodynamically and metabolically optimize patients who are initially at too high a surgical risk (*Figure 8*). Multiple devices are available, including intra-aortic balloon pump (IABP), Impella (Abiomed, Danvers, MA, USA), TandemHeart (LivaNova, Pittsburgh, PA, USA), veno-arterial extracorporeal

Table 3 Characteristic echocardiographic findings in acute mitral regurgitation and acute aortic regurgitation

	Mitral regurgitation	Aortic regurgitation	
		Both	
2D/3D	Torn mitral valve chordae Ruptured papillary muscle Leaflet tethering SAM	Normal LV size Normal LV function Leaflet flail/prolapse	Dissection flap Premature mitral valve closure
Colour Doppler	Vena contracta ≥ 7 mm Colour Doppler splay[8]	Large flow convergence Jets may appear *very* eccentric *Avoid* quantitative metrics (e.g. EROA)	Vena contracta ≥ 6 mm Regurgitant jet width ≥ 65% LVOT diameter
Pulsed- or Continuous-Wave Doppler	Triangular-shape profile Peak velocity < 5 m/s Pulmonary vein systolic flow reversal	Densely echogenic regurgitant profile	Pressure half-time < 200 ms Low end-diastolic velocity Holodiastolic flow reversal in the abdominal aorta

2D, two-dimensional; 3D, three-dimensional; EROA, effective regurgitant orifice area; LV, left ventricular; LVOT, left ventricular outflow tract; SAM, systolic anterior motion of the mitral valve leaflets; TAVR, transcatheter aortic valve replacement.

membrane oxygenation (VA-ECMO), and left atrial veno-arterial extracorporeal membrane oxygenation (LAVA-ECMO).

Acute MR is well served by most of the available MCS options. IABP provides the least support but is the most widely and readily available and can rapidly decrease LV afterload. Similarly, Impella can result in direct LV unloading, however, should be used with caution (or avoided) in patients with papillary muscle rupture and may be difficult to position/alarm frequently in the setting of normal LV size. TandemHeart and LAVA-ECMO are more technically challenging to insert given the need for transseptal puncture across the interatrial septum, however, have the benefits of direct LA unloading. Finally, VA-ECMO may be used, but is often done so in conjunction with TandemHeart, IABP or Impella to prevent pulmonary oedema from increased LV afterload.

On the other hand, acute AR represents either a relative or absolute contraindication to MCS, especially in the presence of concomitant aortic dissection. Diastolic balloon inflation of the IABP results in an *increased* regurgitant volume. The forward flow effects of the Impella are mitigated by severe AR, of which the latter may be worsened due to the device precluding aortic valve coaptation. AR and pulmonary oedema may be worsened in the setting of increased afterload with VA-ECMO. If MCS is absolutely necessary, TandemHeart or LAVA-ECMO could be considered due to concomitant LA unloading.[29]

Acute valvular stenosis

Native mitral and aortic valve stenosis do not occur acutely, and therefore by definition are chronic lesions. However, patients may present with acute decompensated heart failure or cardiogenic shock in the context of a precipitating event, resulting in acute elevations of LA and pulmonary venous pressures, and potentially a decrease in left ventricular ejection fraction (LVEF) or cardiac output (*Figure 2* and *Table 4*).

Aetiology

Rheumatic and calcific valve disease are the two most common causes of both native mitral and aortic valve stenosis (of which rheumatic MS and calcific AS are most frequent). Congenital mitral valve disease (e.g. parachute mitral valve, supramitral ring) is rare and often associated with other congenital cardiac abnormalities. Bicuspid aortic valves are the most common congenital cardiac malformation and an important cause of AS, typically involving premature fibrosis, stiffening, and calcification.[30] Unicuspid aortic valves are associated with AS, but are rare.[31]

Clinical presentation

Patients with acute decompensation in the setting of severe valvular stenosis will present with dyspnoea due to a rapid rise in LA pressure ± LVEDP. Some patients will have cardiogenic shock depending on the precipitant and remainder of cardiac function. Other clinical symptoms may be present depending on the cause of deterioration (*Table 4*).

Like patients with acute valvular regurgitation, the physical examination will reflect elevated pulmonary pressures and (if present) cardiogenic shock. As such, tachypnoea, scattered inspiratory crackles and hypoxaemia are expected. Tachycardia, altered mental status, hypotension, cool extremities, and a narrow pulse pressure are likely manifestations for those in cardiogenic shock. The remainder of auscultatory findings is consistent with that of severe valvular stenosis (*Figure 3*). Like acute valvular regurgitation, care should be taken in overly relying on the physical examination to diagnose acute valvular stenosis, particularly with MS wherein auscultatory findings are subtle.

Diagnostic evaluation

Similar to acute valvular regurgitation, TTE remains the first-line diagnostic modality for severe valve stenosis given its portability, availability and diagnostic yield. Particularly in patients with severe AS, TTE has the added benefit of facilitating the assessment of valvular gradients via multiple different windows and using the dedicated PEDOF (pulse echo Doppler flowmeter) probe. However, in the event where TTE imaging is suboptimal, TEE should be pursued.

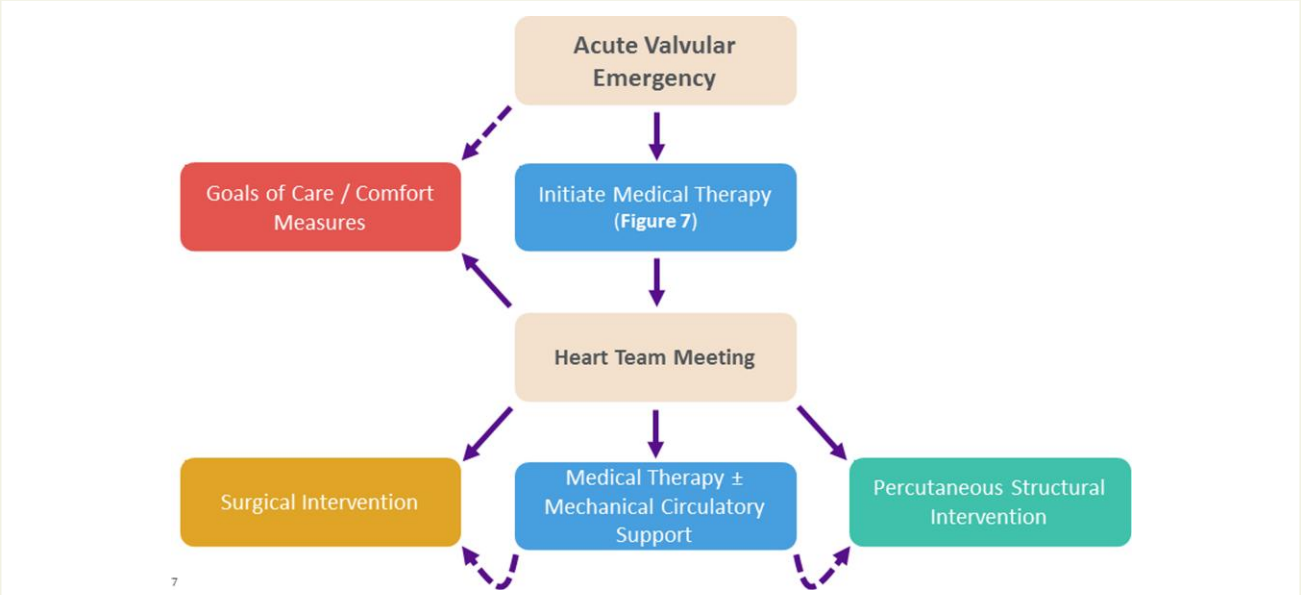

Figure 6 Heart-team approach to the management of acute valvular emergencies.

Mitral Regurgitation	Aortic Regurgitation	*Treatment*	Mitral Stenosis	Aortic Stenosis
✓	✓	*Diuresis*	✓	✓
↓	↓	*Afterload*	⇆	↓
⇆ (↓ if SAM)	↑	*Heart Rate*	↓	⇆
✓	✓	*Inotropy*	✗	✓ (if ↓ LVEF)
Norepineprhine Dopamine Epinephrine	Norepineprhine Dopamine Epinephrine	***Vasopressors (if needed)***	Phenylephrine Vasopressin	Phenylephrine Vasopressin
–	Isoproterenol	***Other Vasoactive Agents***	IV β-blockers Amiodarone Digoxin	–
Angiography + PCI (if ischemia causing MR)	Transvenous pacing	***Other temporizing, non-MCS adjuncts***	BMV (rheumatic)	BAV

Figure 7 Medical therapy for the management of acute valvular emergencies. BAV, balloon aortic valvuloplasty; BMV, balloon mitral valvuloplasty; IV, intravenous; LVEF, left ventricular ejection fraction; MR, mitral regurgitation; PCI, percutaneous coronary intervention; SAM, systolic anterior motion of the mitral valve.

2D and colour Doppler typically suggest the presence of severe valvular stenosis (*Figures 9 and 10* and *Table 5*). Native valve leaflets will have significantly restricted leaflet motion with turbulent flow at the stenotic orifice. Rheumatic changes of the mitral valve will demonstrate the classic 'hockey-stick' appearance and may have associated leaflet thickening, calcification or thickening of the subvalvular apparatus. Calcific valvular stenosis will show severe leaflet (and annular) calcification. Long-standing valvular stenosis will typically result in LA enlargement, with LV hypertrophy common to those patients with AS.

Pulsed and continuous-wave Doppler are critical for the assessment of the degree of valvular stenosis and as previously noted, should

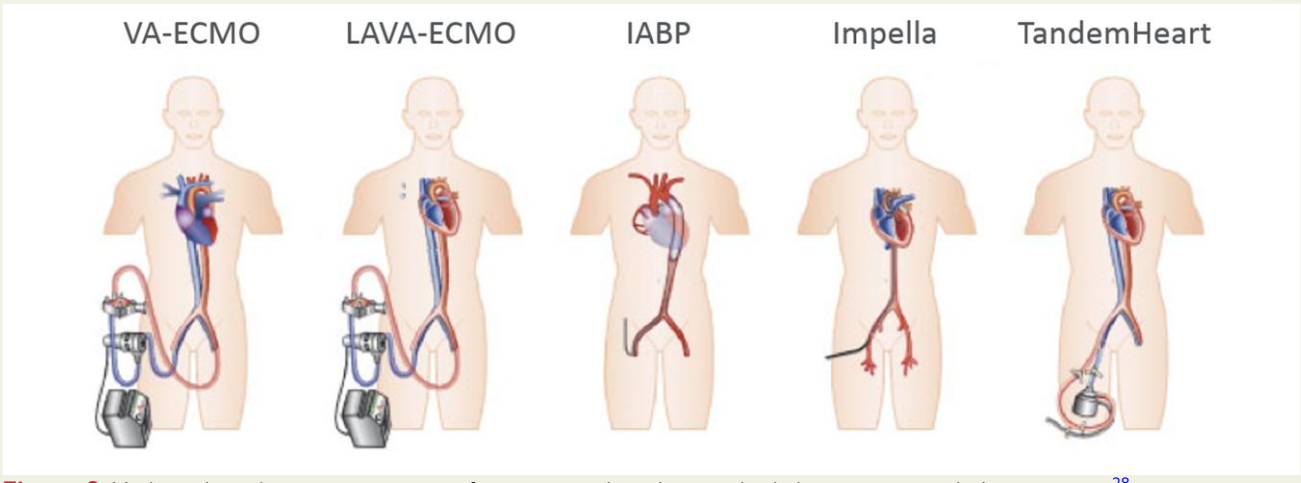

Figure 8 Mechanical circulatory support options for patients with cardiogenic shock due to an acute valvular emergency.[28]

Table 4 Causes of acute deterioration in the context of severe valvular stenosis

Atrial fibrillation
Fever/infection/sepsis
Hypertensive urgency/emergency
Ischaemia
Pregnancy
Pulmonary embolism
Emotional stress
↓ LVEF

LVEF, left ventricular ejection fraction.

be assessed from multiple different acoustic windows (*Table 5*). In patients where there is a discordance between aortic valve area and aortic valve gradients, low-flow low-gradient severe AS should be considered and reassessed with further imaging if needed.

MDCT can be a valuable adjunct in patients with native valvular AS (particularly in those with low-flow low-gradient severe AS), wherein LVOT dimensions, valvular calcium score and direct valvular area measurements can be directly obtained. Notably, MDCT does not provide haemodynamic assessment of valvular stenosis and therefore is an *adjunct*, not a replacement, for echocardiography. MDCT also requires ECG-gating, which may be limited by tachycardia or irregular cardiac rhythms.

Management

Like that of acute valvular regurgitation, management of patients with acute decompensation in the face of severe valvular stenosis (*particularly* in the setting of cardiogenic shock), requires a heart-team approach (*Figure 6*).[2] Definitive therapy is indeed surgical; however, there is a greater role for percutaneous interventions (i.e. valvuloplasty) in temporization of patients with profound instability or metabolic disarray. For those who do not qualify for valvuloplasty (or remain in cardiogenic shock despite intervention), medical therapy combined with MCS may also be used as a 'bridge' to eventual surgical intervention. TAVR and transcatheter mitral valve replacement (TMVR) also represent potential treatment options.

Surgery

While patients with severe native valve stenosis with cardiogenic shock should be promptly evaluated by cardiothoracic surgery, there are more established roles for medical therapy and percutaneous intervention in the stabilization of these patients (as described below). This is particularly true in patients who are haemodynamically stable or in those with severe rheumatic MS who are candidates for balloon mitral valvuloplasty (BMV). Peri-operative mortality is high, with up to 21% operative mortality demonstrated in patients with severe AS and cardiogenic shock.[32] Mitral annular calcification is known to be associated with high peri-operative morbidity and mortality in the *non-emergent* setting and portends a poor prognosis in combination with severe MS.[2] As such, while surgical evaluation should be expeditious, surgical replacement may not need to occur emergently.

Similar to acute valvular regurgitation, timing of operative intervention should be decided in the broader context of the risks of operative versus non-operative intervention, including haemodynamic and metabolic status, available alternative (percutaneous) options and other surgical risk factors.

Percutaneous intervention

BMV and balloon aortic valvuloplasty (BAV) represent important percutaneous interventions in critically ill patients with severe rheumatic MS and severe AS, respectively.[2] While BMV may represent a definitive therapy for patients with severe rheumatic MS, BAV should be considered a *bridge* given its generally modest efficacy and suboptimal haemodynamic effects.[33] Notably, BMV cannot be performed in patients with calcific MS, nor in patients with an established LA appendage thrombus, more than mild MR or an unfavourable valve morphology. Similarly, BAV is contraindicated in patients with moderate or greater AR, infectious endocarditis or in patients with poor vascular access. For both procedures, patients can develop severe regurgitant lesions with the progression of haemodynamic instability.

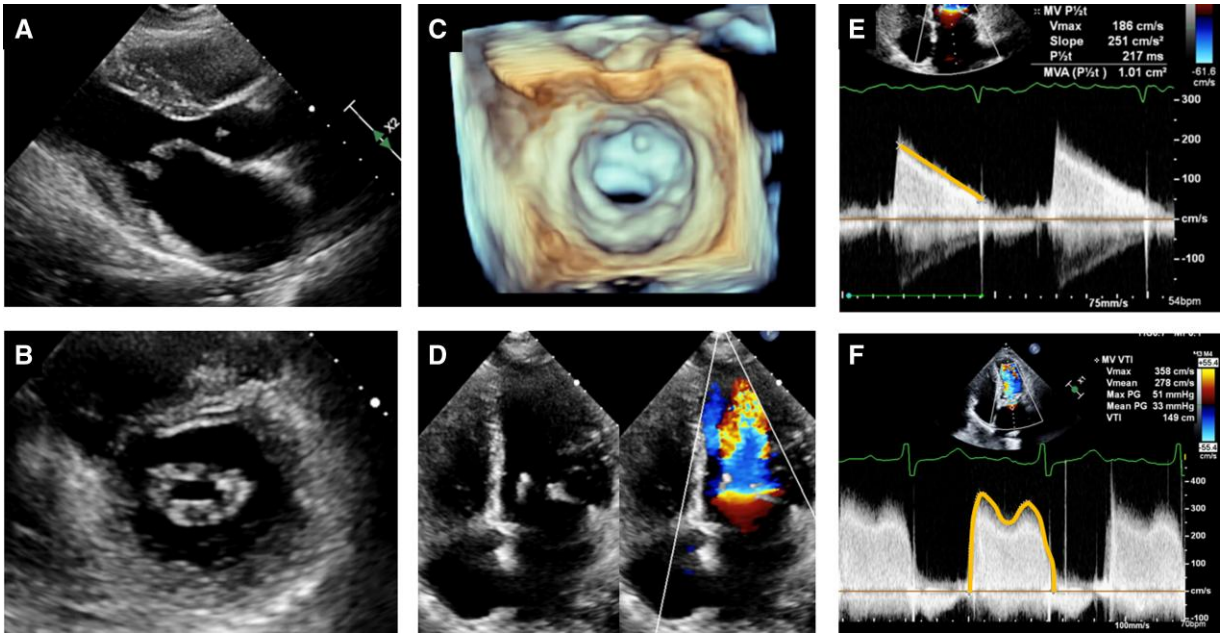

Figure 9 Echocardiographic findings in severe rheumatic mitral stenosis. 'Hockey-stick' deformity with mitral valve leaflet thickening, commissural fusion and flow acceleration on colour Doppler imaging (*A*–*D*). Continuous-wave Doppler demonstrating pressure half-time method for assessment of mitral valve area (*E*) and mean mitral valve gradient (*F*).

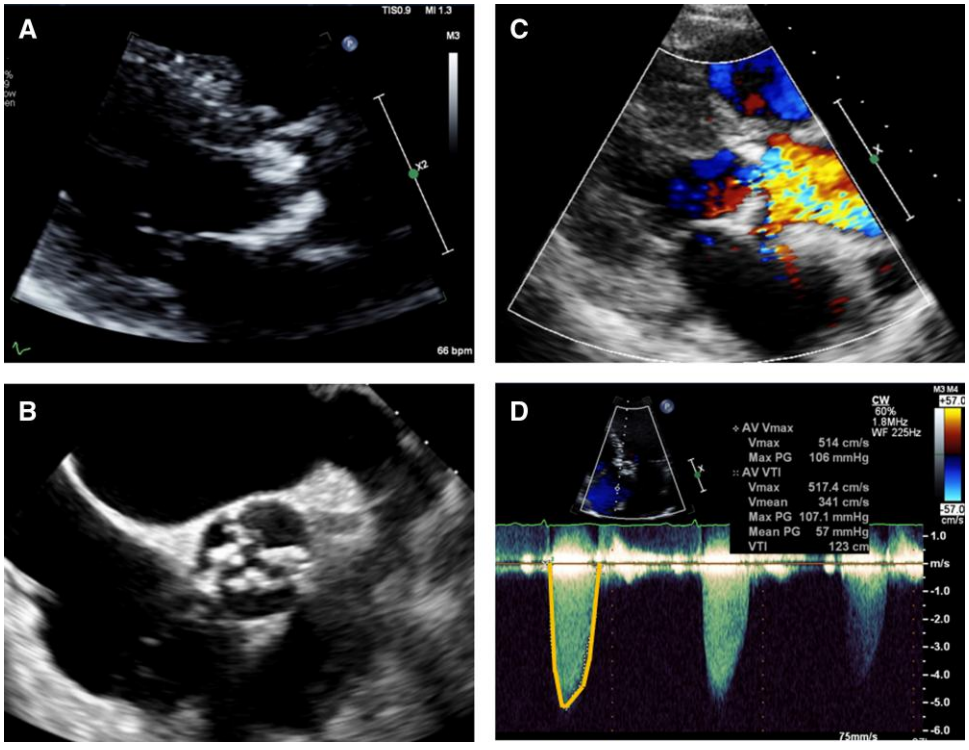

Figure 10 Echocardiographic findings in severe aortic stenosis. Two-dimensional parasternal long- and short-axis imaging demonstrating severe aortic valve leaflet and annular calcification (*A*, *B*). Colour Doppler shows turbulent flow (*C*) with a peak aortic valve velocity >5 m/s on continuous wave Doppler evaluation (*D*).

Table 5 Characteristic echocardiographic findings in severe mitral stenosis and severe aortic stenosis

	Mitral stenosis	Both	Aortic stenosis
2D/3D	'Hockey-stick' leaflet morphology Subvalvular thickening and calcification	Thickened/calcified valve leaflets Decreased leaflet motion Annular calcification	
Colour Doppler		Turbulent flow (at the stenotic orifice)	
Pulsed- or Continuous-Wave Doppler	MVA < 1.5 cm^2 (planimetry or PHT if rheumatic) Mean diastolic mitral valve gradient ≥ 5 mmHg		AVA ≤ 1.0 cm^2 or AVAi ≤ 0.6 cm^2/m^2 • High-grade AS • Peak velocity ≥ 4.0 m/s or mean gradient ≥ 40 mmHg • Low-flow low-gradient AS[a] • Peak velocity < 4.0 m/s or mean gradient < 40 mmHg • Stroke volume index ≤ 35 mL/m^2

2D, two-dimensional; 3D, three-dimensional; AS, aortic stenosis; AVA, aortic valve area; AVAi, aortic valve area index; LVEF, left ventricular ejection fraction; MVA, mitral valve area; PHT, pressure half-time.
[a]Low-flow low-gradient AS can be seen in the setting of a low LVEF (<50%) or preserved LVEF (≥50%).

Whereas TAVR has a narrow range of utility for patients with acute AR, it appears to be a burgeoning option for patients with cardiogenic shock and critical AS. In a large retrospective registry analysis, TAVR was successfully performed on 2220 patients in cardiogenic shock with a resultant 19.1% mortality (compared with 4.9% mortality in high-risk patients).[34] While there are multiple important limitations to urgent TAVR (most notably the inability to obtain a gated MDCT for assessment of annular size and iliofemoral access) and mortality remains high, it is nonetheless feasible. TMVR has been evaluated in non-critically ill patients with calcific MS, however, is far more challenging to implement. Procedural planning with MDCT is vital for mitral valve annular sizing, understanding the distribution of annular calcium and to avoid LVOT obstruction. TMVR is technically challenging and given the (often) high-risk patient population associated with severe calcific MS, 30-day mortality tends to be high (up to 25%).[35]

Traditional medical therapy and mechanical circulatory support

Medical therapy for acutely decompensated valvular stenosis is limited (*Figure 7*). For patients with pulmonary oedema, diuresis is a mainstay of therapy. To maximize LV filling in patients with severe MS, efforts should be focused on slowing heart rates and restoring normal sinus rhythm using medications such as β-blockade (e.g. esmolol), digoxin, or amiodarone. As such, prioritizing the use of pressors that avoid chronotropy (e.g. vasopressin or phenylephrine) is preferred.

Afterload reduction is critical is the management of patients with severe AS due to the already significant load imposed upon the LV from the stenotic aortic valve. Nitroprusside has been studied in AS patients with cardiogenic shock and a reduced LVEF and has demonstrated improvements in cardiac index, systemic vascular resistance (SVR) and pulmonary capillary wedge pressure.[36] Somewhat counterintuitively, phenylephrine has classically been recommended as the pressor of choice for AS with cardiogenic shock with the three primary reasons being (i) LV afterload is relatively 'fixed' by the stenotic valve (and therefore increasing SVR will have a smaller effect on myocardial work), (ii) higher diastolic blood pressure will translate to increased coronary perfusion pressure, and (iii) reflex bradycardia will decrease overall myocardial work.[37] Vasopressin has also been recommended in this setting.[23] Inotropes may be considered in the setting of a reduced LVEF, but should be used with caution given the risk of tachycardia and arrhythmia.

MCS can be used to support patients with severe valvular stenosis. In cardiogenic shock accompanying MS, LVEDP is generally low owing to poor flow from the LA into the LV due to significantly elevated mitral valve gradients with potential contributions from right ventricular failure and severe pulmonary hypertension. As such, with concomitant right heart failure, patients would be best served by VA- or LAVA-ECMO (particularly the latter for decompression of both atria). Otherwise, however, a TandemHeart (with direct LA drainage) is a reasonable alternative.

In the case of severe AS (or decompensation after rapid ventricular pacing during valvuloplasty), most MCS options may be used for haemodynamic support.[38,39] One consideration would be feasibility of Impella placement, currently contraindicated in patients with an aortic valve area <0.6 cm^2 due to the risk of complete valvular occlusion or inability to insert the device.[40]

How we manage acute valvular emergencies

Even before the diagnosis of an acute valvular emergency is established, patients often will (or should) receive diuretics for the

management of pulmonary oedema and volume overload (*Figure 6*). Once the diagnosis is made, further appropriate medical therapy should be initiated (*Figure 7*) and a heart team meeting convened. Incorporating the patient's haemodynamic and metabolic status with medical comorbidities and goals of care, a decision should be made regarding continuation of medical management alone, initiation of MCS, emergent surgery, or emergent percutaneous structural intervention. Notably, acute valvular regurgitation often requires emergent surgery given the highly unstable nature of the lesions and poor response to medical therapy, whereas acute valvular stenoses are more amenable to percutaneous interventions and medical temporization prior to surgery.

Conclusions

Recognizing an acute valvular emergency is often delayed due to non-specific clinical symptoms and subtle physical examination findings. Diagnosis is rapidly established through comprehensive echocardiography. A heart-team approach is critical to best identify next steps in management, including the timing for definitive surgical or transcatheter intervention.

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No new data were generated or analysed as part of this research.

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