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Mechanical Circulatory Support in Cardiogenic Shock due to Structural Heart Disease

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Mechanical Circulatory Support in Cardiogenic Shock due to Structural Heart Disease

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KEYWORDS

• Cardiogenic shock • Mechanical circulatory support • Aortic stenosis • Aortic regurgitation • Mitral regurgitation • Mitral stenosis

KEY POINTS

- Early recognition and escalation of care with mechanical circulatory support are crucial for patients presenting with cardiogenic shock due to structural heart disease.
- Selection of mechanical circulatory support methods should be based on device availability, familiarity of the multidisciplinary team with the device, and specific needs of the patient.
- Surgical or transcatheter repair of structural heart disease should be done using appropriate mechanical support with a "heart team" approach after the patient has improved.

Tremendous advances in all forms of cardiovascular care[1] have developed over the past decade, with remarkable and dramatic declines in cardiovascular mortality (between 60% and 70%).[2] Despite such advances in cardiovascular disease therapies, cardiogenic shock (CS) is a common cause of mortality, and management of CS remains challenging. Acute coronary syndrome accounts for more than 80% of cases of CS.[3] As a result, interest in CS has predominantly focused on managing acute coronary syndrome, including revascularization. Few studies to date have explored the role of structural heart disease (SHD) in the pathogenesis of CS. In the SHOCK (should we emergently revascularize occluded coronaries for cardiogenic shock) trial registry of 1190 patients with CS, 8% of patients had SHD that caused or worsened their hemodynamic status, with a mortality close to 100%.[4] Similar poor outcomes have been observed in other observational studies.[5–7]

Temporary mechanical circulatory support (MCS) is an attractive and intuitive option to use when other medical therapies have been insufficient. Many exciting developments in MCS methods have occurred in the past few years, including the development of smaller portable pumps.[8] Although the field is a growing one, patients with SHD are often excluded from randomized trials, and the role of mechanical therapies in this specific population remains controversial and not well established.

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SHD refers to non–coronary heart disease for which some therapy, surgical or percutaneous, exists. Examples include valvular heart disease, congenital disorders, mechanical complication of acute myocardial infarction, and cardiomyopathies.[9] Although the treatment of SHD often requires pharmacologic and surgical intervention, established and emerging device-based interventions in the setting of CS offer exceptional promise for revolutionizing the practice of cardiovascular medicine. Nevertheless, when patients with SHD present with CS, treatment becomes more challenging and complex.

Currently, there are no published guidelines for using MCS in patients with SHD. The focus of this review is on MCS device selection, specifically, selection pathways for patients with CS from SHD. The objective is to provide the reader with an understanding of general considerations, based on current evidence and institutional experience, for determining the appropriateness of MCS for SHD.

HEART TEAM

With the number of therapeutic options increasing, the "heart team" has become an increasingly important strategy for evaluating SHD, whereby comprehensive decision making may result in a change of diagnostic or therapeutic strategies and promote improved outcomes. Several guidelines have highlighted the effect of heart teams for managing valvular heart disease and heart failure.[10–14] Determining an optimal treatment strategy for patients with complex SHD and CS requires assessing each patient's presenting illness, clinical stability, anatomy, comorbidities, and goals of care. Implementing the scarce guidance available to guide SHD-CS management to the nuances of real-world practice can be challenging; thus, supporting an interdisciplinary model of care is key.

DEFINITION OF CARDIOGENIC SHOCK

The American Heart Association defines CS as a state in which ineffective cardiac output caused by a primary cardiac disorder results in both clinical and biochemical manifestations of inadequate tissue perfusion and dysfunction. In addition to severe systolic and diastolic cardiac dysfunction compromising macrocirculation and microcirculation, systemic inflammatory response syndrome and even sepsis may develop, which could result in multiorgan dysfunction syndrome and biochemical manifestations of inadequate tissue perfusion, such as elevated arterial lactate. The most common hemodynamic criteria for CS include a systolic blood pressure less than 90 mm Hg, a cardiac index less than 2.2 L/min/m,[2] a pulmonary capillary wedge pressure greater than 18 mm Hg, or a right ventricular (RV) end diastolic pressure greater than 10 to 15 mm Hg. Although myocardial infarction with left ventricular (LV) failure remains the most common cause of CS, any acute cause of severe LV or RV dysfunction may lead to CS.[15]

HEMODYNAMIC MONITORING

Although not mandatory in clinical practice, assessing objective hemodynamic parameters, such as reduced cardiac index and elevated pulmonary capillary wedge pressure, is helpful for diagnosing CS, and other hemodynamic parameters are essential for defining RV function in CS.[16] A large national US registry showed that assessing premature atrial contractions in patients with CS is an effective strategy, and using this method is associated with improved outcomes, which may reflect better selection of patients or better use of the information to guide therapies.[17] To improve patient outcomes, the authors recommend assessing hemodynamic parameters using premature atrial contractions in patients with SHD-CS for monitoring guiding treatment effectiveness.

MEDICAL THERAPY

Disease management in patients with CS should focus on maintaining adequate cardiac output for vital end-organ perfusion. For patients with acute coronary syndrome with CS, therapy for patient-specific cause should focus on coronary reperfusion and treatment of the underlying SHD that is causing the CS or is a consequence of myocardial infarction. Urgent revascularization and surgical/transcatheter therapies remain the gold standard of care for CS; however, patients often are unstable with increased mortality to receive a definitive therapy. SHD-specific definitive interventions will not be discussed because they are beyond the scope of this review.

Pharmacotherapies, such as inotropes and vasopressors, are used to enhance contractility and modulate vascular tone. Maximal medical therapy (volume resuscitation, vasodilators, inotropic agents) is not considered a justifiable endpoint for refractory CS, at least in well-resourced health settings.[18] The lack of clear evidence on the effectiveness of pharmacologic

inotropic support and the limited (or adverse) effect of catecholamine therapy on survival in patients with CS from acute myocardial infarction[19,20] are the driving forces behind exploration of mechanical means of circulatory support.[21]

The authors believe that physicians treating patients with SHD should adhere to recommendations similar to those recently proposed by the European Acute Cardiovascular Care Association for patients with acute coronary syndrome complicated by CS, such as the following: (1) When severe SHD is diagnosed and is contributing to instability, the patient should be admitted or transferred to a hospital that has 24/7 MCS capability to treat the impending cardiovascular crisis; (2) catecholamine and inotropes should be administered at the lowest possible dose and for the shortest possible duration; (3) the routine use of intra-aortic balloon pump is not recommended, whereas the use of percutaneous MCS devices should be restricted to cases of refractory CS, with treatment being guided by individual physician experience in dedicated centers; and (4) in addition to the general principles of RV dysfunction management, the use of MCS devices with dedicated RV support or venous arterial extracorporeal membrane oxygenation (VA-ECMO) may be considered for certain patients with refractory CS.[22]

MECHANICAL CIRCULATORY SUPPORT OPTIONS

Temporary selection of MCS should be based on device availability, familiarity of the multidisciplinary team with the device, and specific patient needs.[8] In the United States, temporary percutaneous mechanical options for drug-refractory CS have included the following methods: intra-aortic balloon pump, counterpulsation, and percutaneous LV assist devices. Specific LV assist devices include the TandemHeart percutaneous system (Cardiac Assist, Inc, Pittsburgh, PA, USA), the Impella (Abiomed Europe GmbH, Aachen, Germany), and VA-ECMO.[23] A variant of VA-ECMO is left atrial venoarterial extracorporeal membrane oxygenation (LAVA-ECMO), which is a novel technique that involves transseptal placement of a single multistage drainage venous femoral cannula to simultaneously drain both atria in patients with severe LV systolic dysfunction.[24] For RV failure, right-sided support devices, such as Impella RP and the TandemHeart RA-PA, are available options in addition to ECMO.[23] Fig. 1 shows schematic drawings of current percutaneous mechanical support devices for CS, including technical features.

AORTIC STENOSIS

Aortic stenosis is the most common valvular heart disease causing LV outflow tract obstruction. Pressure gradient and LV pressure overload are the hallmarks of severe aortic stenosis that cause leaflet stretch, fluid shear stress, bending stresses, and pressure forces. This tissue damage results in elevated left atrial pressure and pulmonary capillary wedge pressure. Over time, some patients may develop LV dysfunction because of increased wall stress secondary to inadequate wall thickening, potentially resulting in "afterload mismatch."[25,26] Patients with severe aortic stenosis, LV dysfunction, and unrevascularized coronary artery disease are particularly susceptible to hemodynamic decompensation owing to limited myocardial reserve.[27]

The incidence of CS in patients with aortic stenosis is low (close to 6%),[28] but mortality in patients who have developed CS can be considerably high, up to 70%, if no durable intervention is performed.[29] Therapeutic interventions for CS related to aortic stenosis are challenging because of a paucity of data. Whereas medical treatment alone is an unreliable option, and surgery is often deemed prohibitive, it is unclear whether direct transcatheter aortic valve replacement (TAVR) or balloon aortic valvuloplasty (BAV) followed by elective TAVR or surgical aortic valve replacement (SAVR) after medical stabilization should be performed. Medical therapy for patients with aortic stenosis and CS should include optimal ventilatory and inotropic support, and every effort should be made to identify and treat the precipitating factors. Treatment options for aortic stenosis with CS include surgery or urgent TAVR.[30] Despite recent advances in therapies, caring for patients with severe aortic stenosis who go on to develop systolic dysfunction and CS remains an important clinical challenge, and this condition is associated with increased morbidity and mortality.[30,31]

Over the past decade, Impella has become commercially available for providing circulatory support in patients with aortic stenosis.[32] A relative contraindication for using the Impella device is a concern about potential compromise of blood flow in the remaining valvular orifice from the presence of a catheter, which could lead to worsened hemodynamics through a severely stenotic aortic valve orifice. Regardless,

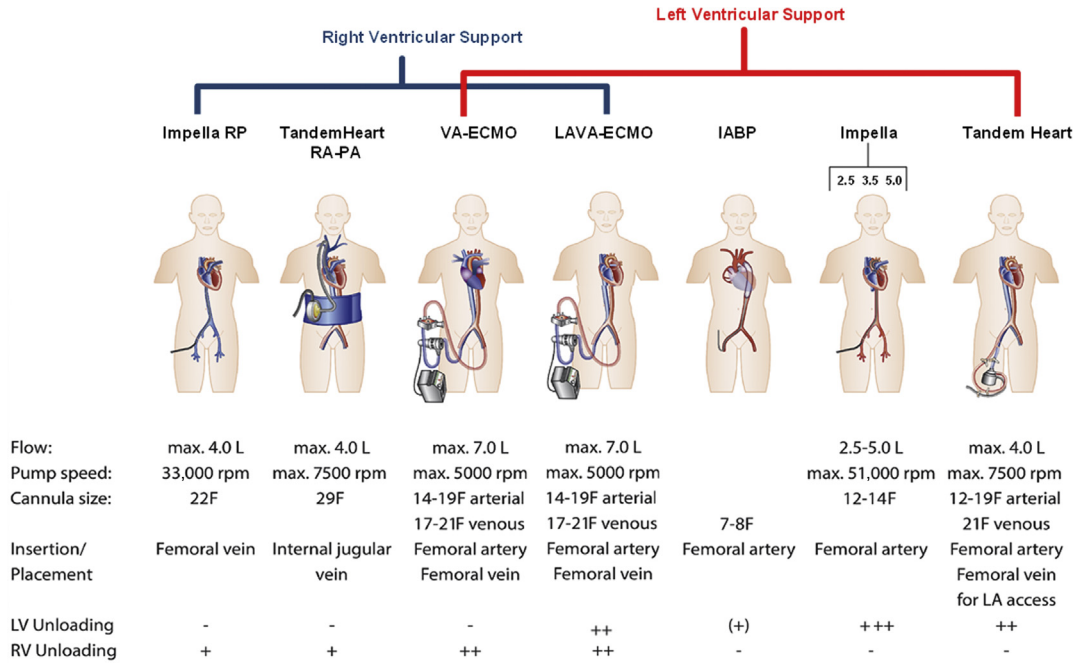

Fig. 1. Current percutaneous mechanical support devices for CS. IABP, intra-aortic balloon pump. (*Adapted from* Thiele H, Ohman EM, de Waha-Thiele S, Zeymer U, Desch S. Management of cardiogenic shock complicating myocardial infarction: an update 2019. *Eur Heart J.* 2019;40(32):2671-2683; with permission.)

the use of Impella has been shown to be feasible, with promising results seen in selected patients with severe aortic stenosis.[33] The Impella device directly aspirates blood from the LV into the aorta in series with the native cardiac blood flow. Owing to the unique design, this device effectively unloads the LV and simultaneously stabilizes the patient's hemodynamics and augments cardiac output. Implantation of the 2.5, 3.5, and 5.0 left-sided Impella seems to be feasible in patients with severe aortic stenosis, and a balloon-assist technique may be used to facilitate device implantation when initial unassisted attempts have failed. Improved hemodynamic stability may enhance the tolerability of lengthy and complex procedures by unloading the LV.[34,35] In cases of CS with concomitant coronary artery disease, the risk of the decompensation is higher,[36] but MCS with Impella can improve distal coronary pressure and coronary perfusion pressures in the presence of critical coronary stenosis.[37] **Fig. 2**A shows schematic drawings of current percutaneous mechanical support devices for aortic stenosis in CS.

Several single-center studies have demonstrated the feasibility of using BAV as a bridge to TAVR and SAVR in patients with acute presentations and as a way to triage select high-risk patients who are not good candidates for aortic valve replacement.[38,39] When BAV is performed and the ventricles are paced at high rates, a sudden decrease in stroke volume and cardiac output causing ischemic and hemodynamic strain may result during the procedure, and these results may be due to periods of hypotension with subsequent systemic and coronary hypoperfusion. BAV can be done with the Impella device in place to minimize interruption of blood flow during balloon inflation and during high-risk BAV[40,41] (**Fig. 3**). Furthermore, evidence exists that points to a similar cerebrovascular risk in patients undergoing BAV or TAVR, with the central venous access device registry reporting an adverse event rate of 1.72% at 30 days following Impella-assisted BAV.[36]

Peri-interventional CS is associated with high mortality and can occur during TAVR in a variety of scenarios, such as coronary obstruction, refractory ventricular arrhythmia, annular rupture, and hemodynamic collapse.[33,42] In a study of 54 patients who required an MCS device during TAVR, Impella was used in only 7 patients: 3 elective cases and 4 emergency rescues. The overall in-hospital mortality in this study was 11% for elective cases and 53% for emergency rescue. CS was the cause of death in 50% of cases, and all-cause mortality at 1 year was

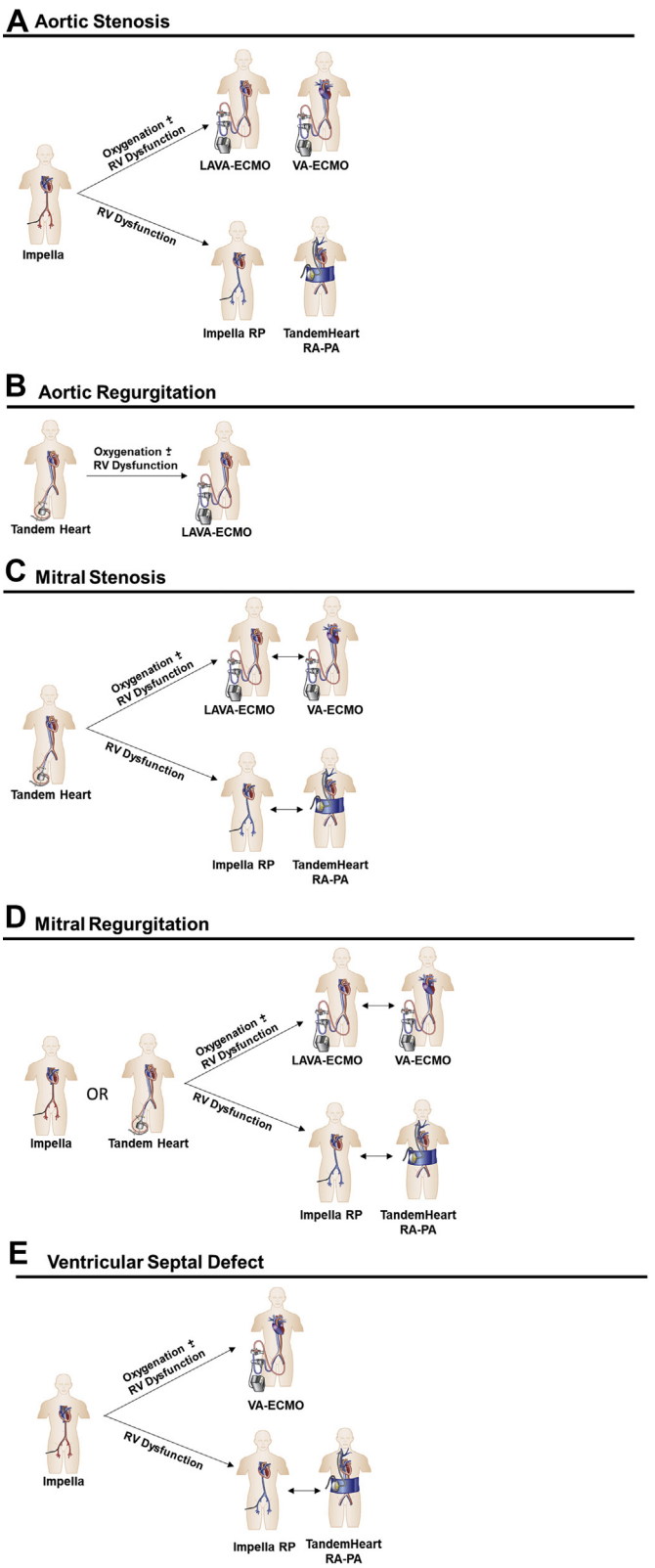

Fig. 2. Recommended algorithm for MCS utilization for SHD: (*A*) aortic stenosis, (*B*) AR, (*C*) mitral stenosis, (*D*) mitral regurgitation, and (*E*) VSD.

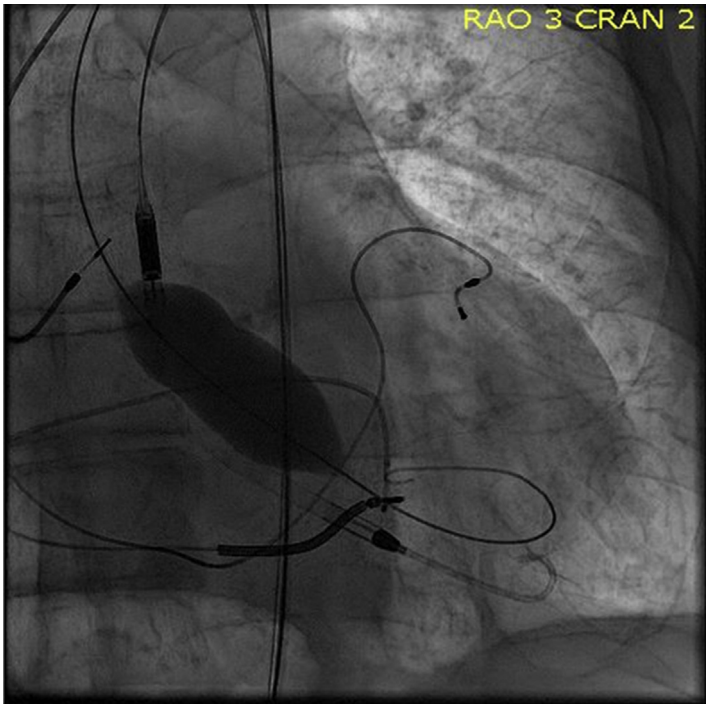

Fig. 3. Impella CP device-assisted BAV in a patient with CS and severe aortic stenosis. (*Courtesy of* ABIOMED Inc., Danvers, MA.)

19% for elective cases and 71% for emergency cases.[43] In situations wherein Impella has not been available, ECMO has been used for bailout in TAVR use complicated by CS[44] (Fig. 4).

AORTIC REGURGITATION

Aortic regurgitation (AR) causes volume overload of the LV. Over time, LV end-diastolic volume continues to increase; the ejection fraction

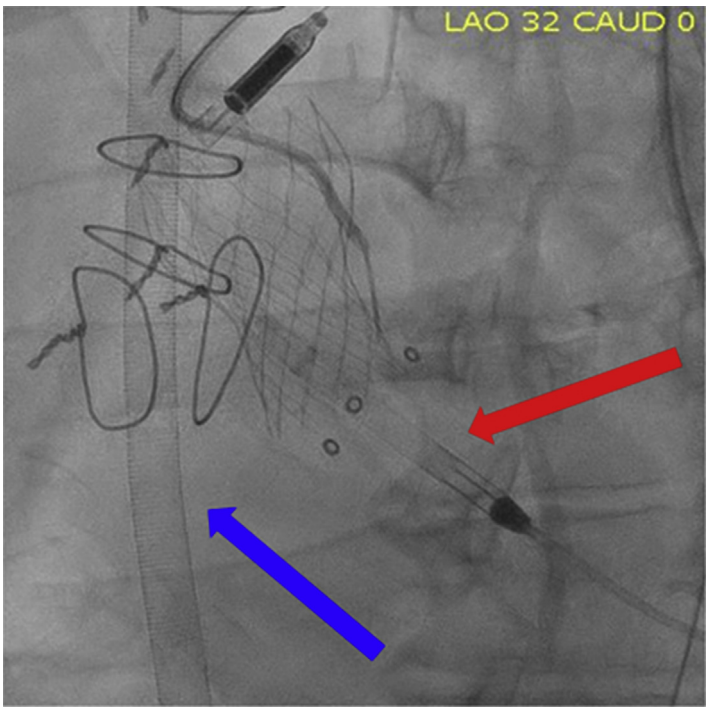

Fig. 4. Combined use of percutaneous ECMO and Impella CP in a case of periprocedural coronary occlusion in a valve-in-valve TAVI (*blue arrow*: venous ECMO cannula; *red arrow*: Impella CP in the left ventricle). (Impella CP courtesy of ABIOMED Inc., Danvers, MA.)

drops, and these changes may precede the appearance of clinical symptoms. Acute AR can be life threatening, as LV dilatation and other compensatory mechanisms cannot develop rapidly enough to prevent hemodynamic deterioration. Regarding the medical management of AR in the setting of CS, stabilization with airway intubation and hemodynamic support may be required, especially before intervention. The use of vasodilators, such as nitroprusside, in conjunction with inotropic therapy may help with hemodynamic stabilization.[45] Pacing after BAV and TAVR has been adopted as temporary or permanent therapy to mitigate perivalvular leak in patients affected by moderate to severe AR.[46] This approach is based on the concept that a shorter diastolic phase reduces the time available for blood to flow back into the ventricle, thus diminishing the ventricular overload. Prompt SAVR remains the standard of care for operable patients; however, TAVR has been used in selective cases.[47]

Unfortunately, given the pathophysiology of the disease, most (if not all) MCS have a relative contraindication in the setting of severe AR. Management of CS with acute AR with an Impella or intra-aortic balloon pump device would not provide adequate circulatory support or mitigate aortic insufficiency. If MCS is mandated, the TandemHeart device could be considered, although it indirectly unloads LV volume by actively unloading the left atrium; however, the AR may remain unaffected or could be worsened because of pressurized blood in the aorta, increasing the retrograde flow. At the authors' center, they have used TandemHeart as a bridge to surgery, considering the limitations mentioned above. Case reports describing use of TandemHeart with off-label use of an Amplatzer occluder device to limit AR have reported mitigation of the acute phase as a bridge to surgery.[48] Another possibility for treating severe AR is the LAVA-ECMO, as it might be better for unloading the LV than the standard VA-ECMO because it offers sufficient biventricular decompression.

MITRAL STENOSIS

The incidence of CS in patients with mitral stenosis is unknown but is probably very low in wealthier nations, despite being a highly prevalent condition worldwide. It occurs mainly in patients who have not received treatment until the mitral stenosis is very advanced, with CS being the final manifestation. Mortality can reach close to 25% if it is not treated accordingly. The key hemodynamic consequence of mitral stenosis is the development of a

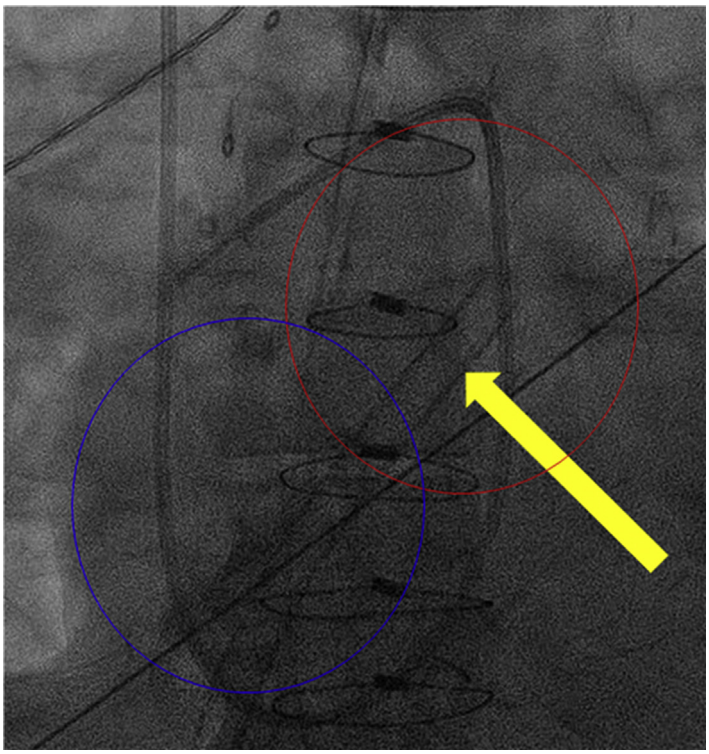

Fig. 5. LAVA ECMO in a patient with biventricular failure and severe mitral regurgitation. The multistage cannula (*arrow*) drains in both the left atrium via end hole (*red circle*) and right atrium via side holes (*blue circle*).

pressure gradient between the left atrium and LV, which is transmitted to the pulmonary circulation and results in an increase in both pulmonary pressures and pulmonary vascular resistance, resulting in pulmonary edema, RV failure, and CS. A rapid diagnosis of this condition is important because emergency interventions, such as valve replacement or percutaneous balloon mitral valvuloplasty (PBMV), are effective and readily available. Only a few case series have reported successful treatment of CS with PBMV.[49–51] The most commonly encountered occurrence is an underlying mitral stenosis affected by septic or hypovolemic shock, which may trigger CS.

Medical treatments to stabilize patients with mitral stenosis include optimal ventilatory and inotropic support. Excessive tachycardia in these patients shortens diastole and causes an undesired increase in pressure gradients across the mitral valve. When a patient's condition remains unstable despite treatment of precipitating factors, emergent mechanical relief of mitral stenosis should be done as soon as possible with either PBMV or surgery. If inappropriate valvular anatomy precludes PBMV or surgery, or if a contraindication for PMBV exists, the device of choice should be TandemHeart. This MCS facilitates hemodynamic stabilization by directly unloading the left atrium and promoting decongestion of the lungs, which facilitates a bridge to mitral valve surgery. If there is RV failure and hypoxemia, the CS mitral stenosis can be treated with VA-ECMO, with the preferred use of the LAVA-ECMO modality that decompresses both atriums and pulmonary filling pressures.

MITRAL REGURGITATION

Acute mitral regurgitation is a rare but lethal condition that often results in CS and high mortality, especially in the setting of acute coronary syndrome (10% to 40% with surgery; 80% without surgery).[4,52,53] Urgent surgical mitral repair or replacement is the current standard of care; however, a significant portion of patients do not receive surgery because of prohibitive operative risk or inability to be stabilized before surgery.[4,54] As a result, large randomized studies of this phenomenon are difficult to perform, and evidence of treatment is limited to case reports, even for the current gold standard of surgery.[53,55] MitraClip has been previously reported for treating acute mitral regurgitation after myocardial infarction or with CS, with most cases being poor LV function.[56–60]

Recently improved MCS could potentially stabilize patients with acute mitral regurgitation and serve as a bridge to definitive treatment.[61] The preoperative implantation of MCS seems to improve outcomes in patients with CS who are

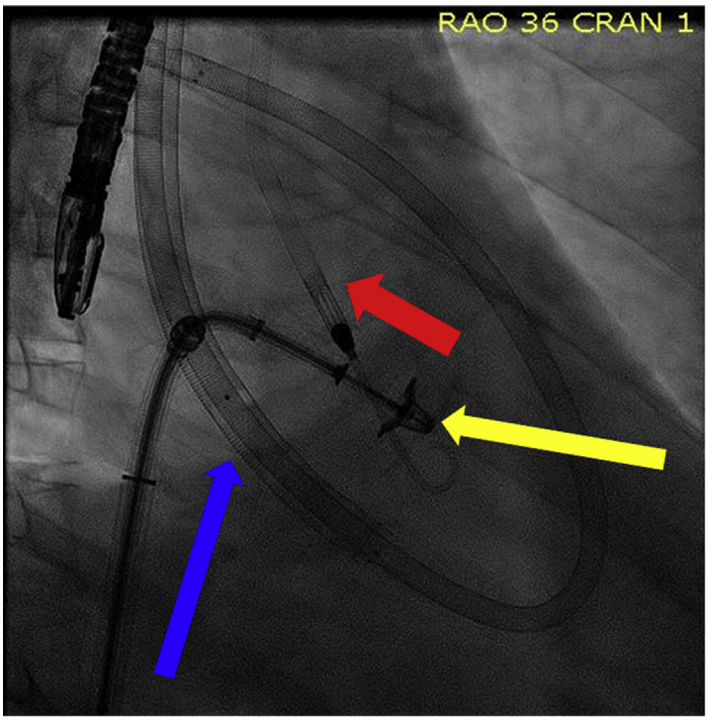

Fig. 6. Combined use of percutaneous TH-RAPA and Impella CP in a case of severe mitral regurgitation secondary to chordae rupture post myocardial infarction treated percutaneously with edge-to-edge repair (*blue arrow*: venous TH-RAPA cannula; *red arrow*: Impella CP in the left ventricle; *yellow arrow*: MitraClip system). (TandemHeart RAPA courtesy of TandemLife; LivaNova, London, UK; Impella CP courtesy of ABIOMED Inc., Danvers, MA; MitraClip courtesy of Abbott Vascular, Santa Clara, CA, USA).

suitable for urgent surgery[62,63] and is generally accepted as the standard of care until emergent mitral valve surgery can be performed. The intra-aortic balloon pump has been a commonly used device, although it offers the least cardiac output augmentation; however, it is widely available and can decrease afterload, thereby supporting adequate mean arterial pressure and potentially decreasing the mitral regurgitation. The Impella device, used alone or together with ECMO (ie, ECPELLA), offers more significant cardiac output augmentation and directly unloads the LV. On the other hand, ECMO has been less commonly used alone, as it may increase total peripheral vascular resistance, potentially worsening the mitral regurgitation. In cases whereby only ECMO is available, physicians should consider the LAVA-ECMO modality to unload the left atrium (Fig. 5). The TandemHeart device can directly unload the left atrium and potentially offer the best hemodynamic effect in patients with acute mitral regurgitation. However, MCS use has not been without risk, and it has been reported to directly cause chordal rupture and acute mitral regurgitation after myocardial infarction.[64]

POSTMYOCARDIAL INFARCTION VENTRICULAR SEPTAL DEFECT

Ventricular septal defect or rupture (VSD) is an infrequent but lethal complication of acute myocardial infarction.[65] When VSD is associated with CS, the mortality is greater than 80%.[66] The definitive therapy for VSD is surgical repair or use of percutaneous closure devices for eligible patients.[67] Inotropes and vasopressors worsen left-to-right shunting, whereas vasodilators decrease shunting at the expense of worsening hypotension. Frequently, very ill patients with VSD and CS will need hemodynamic stabilization with MCS to improve systemic perfusion. Most of the available MCS devices, including intra-aortic balloon pump,[68] ECMO,[69,70] TandemHeart,[71,72] and Impella (including 5.0 support), have been used to treat unstable patients with VSD and CS.[73,74] Despite widespread use of percutaneous MCS, guidelines for optimal use have not been defined because the low incidence and high acuity of VSD have made randomized clinical studies almost impossible to conduct. The European Society of Cardiology Guidelines categorize VSD with CS as a class IIa recommendation (level of evidence C) and suggest using short-term MCS therapy as a bridge to recovery or surgery; however, the guidelines do not specify a preferred form of support.[75]

A computer-simulation model assessing hemodynamic effects of MCS in VSD showed that no form of MCS could normalize hemodynamics in the setting of VSD whereby blood flow through the pulmonary artery (PA) was always markedly elevated. This hemodynamic

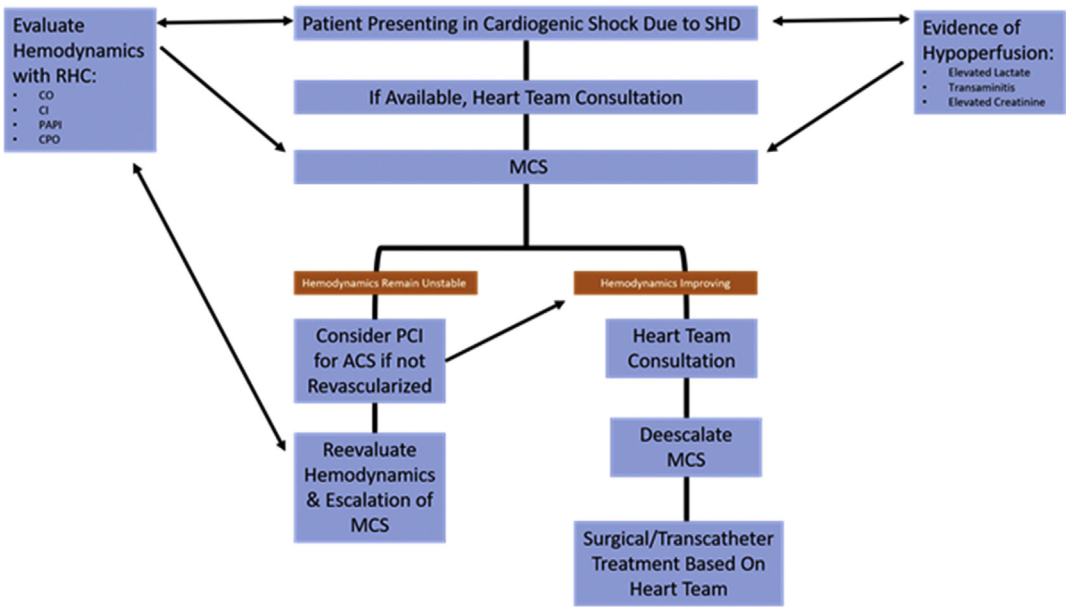

Fig. 7. Proposed algorithm for hemodynamic monitoring and initiation of MCS in those patients presenting with CS due to SHD. ACS, acute coronary syndrome; CI, cardiac index; CO, cardiac output; CPO, cardiac power output; PAPI, pulmonary artery pulsatility index; PCI, primary cutaneous intervention; RHC, right heart catherization.

phenomenon may occur because of increased left-to-right shunting through the VSD or increased right-sided venous return from increased systemic flow in the presence of left-sided support provided by the MCS device. However, this model showed that a combination of 2 devices can provide the greatest degree of overall circulatory support while simultaneously unloading the LV (ie, ECPELLA), with the Impella 5.0 being the most effective MCS and the intra-aortic balloon pump being the least effective MCS.[76] One clinical feature that favors use of an ECMO approach is significant hypoxemia.

RIGHT-SIDED STRUCTURAL HEART DISEASE

CS secondary to isolated right-sided SHD is rare, as this disease can be well tolerated over time. However, left-sided SHD commonly manifests with right RV failure, which increases short-term mortality.[77,78] Diagnosing acute RV failure remains a major clinical challenge. Physical examination, echocardiography, and laboratory tests are helpful tools; however, assessment of premature atrial contractions with other well-established indexes of RV failure can help to confirm diagnosis. Intra-aortic balloon pumps are commonly used to treat RV failure but are not optimally suited for this purpose. Recent advances in percutaneous technology have brought multiple devices into practice that allow rapid deployment of percutaneous RV mechanical support. These devices are categorized according to their mechanism of action, such as direct RV bypass or indirect RV bypass systems. The Impella RP and the TandemHeart RAPA (TH-RAPA) displace blood from the right atrium to the PA, directly bypassing the RV[79,80] (Fig. 6). In contrast, VA-ECMO displaces and oxygenates blood from the right atrium to the femoral artery, thereby indirectly bypassing the RV. As a result, these systems have distinct hemodynamic effects, depending on whether the patient has isolated RV failure or biventricular failure. Because RV MCS device options have been recently introduced, no specific guidelines for optimal device selection and management exist.

FUTURE DIRECTIONS: PROPOSED ALGORITHM

Based on the existing literature and the authors' clinical experience, they have proposed and recommended an algorithm for the use of MCS devices for each SHD discussed in this review (Fig. 2). To guide the management of CS owing to SHD, the authors encourage adopting an early consultation with the heart team to determine an optimal management strategy on a case-by-case basis. They advocate the recognition of CS and early use of percutaneous MCS when indicated based on objective hemodynamic and perfusion parameters to prevent progressive deterioration and organ hypoperfusion. Defining which MCS is the best option should be determined by the main underlying condition and the presence or absence of RV failure and/or hypoxemia. MCS may be considered a temporizing therapy for transcatheter options or potentially as a bridge to surgery or transplant after patient stabilization (Fig. 7).

SUMMARY

Treatment of SHD in the setting of CS remains challenging. Many advances have improved diagnosis and therapy for SHD in CS. Early use of MCS devices instead of dose escalation of inotropes and vasopressors might prevent disease progression and reduce mortality in patients with SHD complicated with CS. Appropriate device selection is still a complex decision-making process, and the authors expect that ongoing studies that take into account the severity of CS, goals of care, patient-specific risks, technical limitations, and assessment for futility of care will help develop better recommendations for MCS choice. Local expertise and comfort with specific measures may dictate MCS device preference, given the lack of evidence demonstrating superiority of 1 method over another. Future advances in CS management are likely to affect the usefulness of the MCS discussed here. Therefore, it is important to stay up-to-date on emerging technologies while maintaining a grasp on older forms of monitoring in an ever-evolving field.

CLINICS CARE POINTS

- Early invasive hemodynamic monitoring.
- Center expertise.
- Early adoption of MCS based on pathophysiology.

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DISCLOSURE

Dr Eng is a clinical proctor for Edwards Lifesciences, Medtronic and Boston Scientific. Dr Frisoli is a clinical

proctor for Edwards Lifesciences, Abbott, Boston Scientific, and Medtronic. Dr B O'Neill has served as a consultant and received research support from Edwards Lifesciences. Dr Lee is a consultant for HeartFlow. Dr W. O'Neill has served as a consultant for Abiomed, Edwards Lifesciences, Medtronic, Boston Scientific, Abbott Vascular and St. Jude Medical; and serves on the Board of Directors of Neovasc Inc. Dr Wang is a consultant to Edwards Lifesciences, Boston Scientific, receives research grant support from Boston Scientific assigned to employer Henry Ford Health System, is a member of the Edwards CLASP IITR, Steering Committee, and Abbott PARADIGM Steering Committee. All other authors report no relevant financial disclosures.

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