SCAI/EAPCI/ACVC Expert Consensus Statement on Cardiogenic Shock in Women

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Abstract
Cardiovascular disease is the leading cause of death for women worldwide, with mortality rates due to cardiogenic shock (CS) remaining exceedingly high. Sex-based disparities in the timely delivery of optimal CS treatment contribute to poor outcomes; addressing these disparities is a major priority to improve women’s cardiovascular health. This consensus statement provides a comprehensive summary of the current state of treatment of CS in women across the spectrum of relevant cardiovascular disease states and identifies important gaps in evidence. As sex-based data are limited in contemporary literature, clinicians may use this document as a resource to guide practice. Further investigations are necessary to inform best practices for the diagnosis and treatment of women with CS.
Abbreviations

AMI, acute myocardial infarction
CS, cardiogenic shock;
IABP, intra-aortic balloon pump
LVEF, left ventricular ejection fraction
PAC, pulmonary artery catheter
PPCM, peripartum/postpartum cardiomyopathy
SCAD, spontaneous coronary artery dissection
tMCS, temporary mechanical circulatory support
VA-ECMO, veno-arterial extra-corporeal membrane oxygenation
VHD, valvular heart disease
Introduction

Cardiovascular disease is the leading cause of death for women worldwide, claiming 8.94 million lives and representing a global age-standardized mortality rate of 204 deaths per 100,000 in 2019 alone. While cardiovascular disease mortality rates have decreased over the past 2 decades, there has been no meaningful improvement in the dismal 30-40% in-hospital mortality rate of patients who experience cardiogenic shock (CS). The burden of CS is recognized as one of the most relevant within cardiovascular disease and identified as a priority to reduce women’s cardiovascular disease burden by 2030. Current evidence points to significant sex-based disparities in the timely delivery of optimal treatment for CS in women, which contributes to persistent poor outcomes. Not only do women encounter delays in treatment, but they are less likely to receive coronary interventions or device therapies compared to men, independent of disease severity. Furthermore, there are limited data to guide management of CS in women despite biologic and pathophysiologic differences in disease presentation. Clinical research and randomized trials in CS pose significant ethical challenges, and women are consistently underrepresented, limiting our ability to evaluate the risks and benefits of cardiovascular drugs or devices in women (Supplemental Table 1). Accordingly, current society practice guidelines do not have sex-specific recommendations and do not highlight instances when evidence is insufficient for diagnostic or management recommendations or outcomes in women with CS. Therefore, the purpose of this consensus statement is to provide a comprehensive summary of the available evidence on CS in women, to identify knowledge gaps, and suggest directions for future clinical investigation.

Methodology

This statement has been developed according to Society for Cardiovascular Angiography & Interventions (SCAI) Publications Committee policies for writing group composition,
disclosure and management of relationships with industry, internal and external review, and
organizational approval. The writing group has been organized to ensure diversity of
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Cardiovascular Care (ACVC) that appointed authors within their associations according to their
expertise. Literature searches were performed by group members designated to lead each section,
and initial section drafts were authored primarily by the section leads in collaboration with other
members of the writing group. Recommendations were discussed by the full writing group until
a majority of group members agreed on the text and qualifying remarks. All advisements are
supported by a short summary of the evidence or specific rationale. The draft manuscript was
peer reviewed in March and April 2024, and the document was revised to address pertinent
comments. The writing group unanimously approved the final version of the document. The
SCAI Publications Committee and Executive Committee endorsed the document as official
Society guidance in [MONTH, YEAR]. SCAI statements are primarily intended to help
clinicians make decisions about treatment alternatives. Clinicians also must consider the clinical
presentation, setting, and preferences of individual patients to make judgements about the
optimal approach.

Sex-based differences in cardiogenic shock: etiology and presentation

Certain etiologies of CS are more common in women as compared with men. While acute
myocardial infarction (AMI) caused by atherosclerotic disease remains an important common causes of CS in men and women, spontaneous coronary artery dissection resulting in CS (SCAD-CS) is an important consideration, particularly for young women. Non-ischemic etiologies of CS are more prevalent in women compared with men and include heart failure-related CS (HF-CS), valvular heart disease-related CS (VHD-CS), stress-induced cardiomyopathy, myocarditis, and peripartum/postpartum cardiomyopathy-related CS (PPCM-CS). Hormonal differences between the sexes may account for some of these observed differences in CS etiologies and outcomes. Estrogen may have anti-inflammatory effects that are protective against cardiac cell death, oxidative damage from ischemic/reperfusion injury, endothelial dysfunction, and adverse cardiac remodeling; however, these hormonal differences may have paradoxical harmful effects by decreasing ischemic pre-conditioning in women compared with men. Furthermore, variation of estrogen levels through reproductive development and life transitions (ie, pregnancy, menopause) can contribute to disease states that may progress to CS such as PPCM and SCAD.

Beyond the different underlying CS etiologies, studies have shown important differences in the clinical presentation of CS in women compared with men. For example, women with AMI complicated by CS (AMI-CS) tend to present with higher left ventricular ejection fraction (LVEF) and similar or lower rates of renal/liver insufficiency as compared with men. Despite this, hemodynamic studies have shown that women have worse cardiac contractility (lower cardiac index or cardiac power output) and a higher risk of death with AMI-CS, and this is substantiated by the Society of Thoracic Surgeons (STS) Mortality Scores. Together, these factors can lead to a mischaracterization of women in AMI-CS as being clinically stable despite on-going hypoperfusion and therefore lead to delays in the initiation of appropriate advanced
care. Sex-based differences in HF-CS are less well characterized, but available studies show that women are more likely to present with cardiac arrest, higher vasopressor requirements, and advanced SCAI SHOCK stages D-E.7

Contemporary shock management

The cornerstones of CS treatment include (1) early identification of CS with timely initiation of hemodynamic support to maintain systemic perfusion and end-organ function, and (2) identification and targeted treatment of the underlying cause of CS. The SCAI SHOCK classification, initially released in 2019 and updated in 2021, provides a 3-axis model that integrates shock severity, clinical phenotype, and risk modifiers across both men and women.8 Building on the SCAI SHOCK classification, we provide a consensus on best practice pathways of care to optimize evidence-based diagnosis, monitoring, and treatment recommendations for women with CS (Figure 1).

General diagnosis of cardiogenic shock in women

Early assessment of end-organ damage and perfusion states is essential to the diagnosis of CS. Lactate is an objective biomarker that correlates with mortality in all types of shock and helps appropriately risk stratify patients. Despite universal society and expert guidelines recommendations for frequent measurement of lactate levels in patients in CS or at risk of going into CS,9–12 women are consistently less likely to have lactate levels measured prior to percutaneous coronary intervention (PCI) (26% vs 38%), likely resulting in delays in diagnosis of AMI-CS. Additionally, invasive hemodynamic monitoring provides important diagnostic and clinical information in the setting of CS to guide phenotyping (uni- or bi-ventricular shock, with
or without congestion), characterize severity, and guide temporary mechanical circulatory support (tMCS escalation (Table 1). Characterizing CS phenotypes can improve short-term outcomes by guiding management earlier, accelerating end-organ perfusion, and potentially slowing progression of CS.13 While we acknowledge that randomized trials of pulmonary artery catheters (PAC) in acute heart failure and critical illness have failed to show a reduction in mortality, it is important to note that these trials evaluated routine, unselected use of PAC, and excluded patients in whom clinicians thought a PAC was required for treatment.14 Thus, selected patients may benefit from PAC monitoring, including those with persistent symptoms or worsening end-organ function despite initial treatment.9 This approach is supported by retrospective observational evidence that targeted PAC use in CS prior to initiating tMCS is associated with lower mortality across all SCAI SHOCK stages.15 Women with CS remain less likely to receive PAC monitoring15 despite several observational studies reporting that PAC-guided CS management improves survival when used to implement standardized pharmacologic and tMCS treatment protocols.16

Consensus Tips for Contemporary Shock Management

- Early and frequent assessments of end-organ function including lactate measurements (ie, serial testing every 2-6 hours) are useful to improve CS diagnosis and risk stratification, aiming to reduce current sex-based disparities in CS care by increasing early interventions.
- Early PAC use in women may assist in CS diagnosis and management.
- PAC should be placed in most patients on tMCS.

General management of cardiogenic shock in women
Inotropes and vasopressors are considered first-line treatment in CS based on rapid onset of action and ease of use. Societal recommendations suggest using norepinephrine or dobutamine as first-line vasoactive support in hypotensive patients.\textsuperscript{10,11} Ino-dilators (milrinone, dobutamine, levosimendan) may be considered in patients who have low cardiac output and are normotensive. A study comparing dobutamine to milrinone in CS showed no difference in outcomes between the 2 medications and no sex-based difference in outcomes.\textsuperscript{17} Levosimendan, where available, may reduce mortality compared with placebo in lower severity CS but showed no benefit when compared with dobutamine.\textsuperscript{18} Therefore, the optimal agent for hemodynamic support for CS in women is unknown, and sex-based data are sparse. Additionally, aggressive escalation of vasopressors and inotropes at the expense of delays in tMCS should be done with caution in women with CS, as evidence suggests women are more prone to their toxic effects, including increased myocardial oxygen consumption, arrhythmias, and reduced end-organ microcirculatory perfusion.\textsuperscript{12,19}

Beyond pharmacologic support for CS, several tMCS options are available, including the intra-aortic balloon pump (IABP), the Impella family of pumps, TandemHeart devices, and veno-arterial extra-corporeal membrane oxygenation (VA-ECMO). Since VA-ECMO significantly increases afterload, which leads to increased LV filling pressures, concurrent afterload reducing or “venting” strategies with concomitant Impella (ECPELLA or ECMELLA) or IABP support can be considered.\textsuperscript{20} Support strategies and their differential hemodynamic and physiologic effects are summarized in Table 2. Device selection is based on the extent of hemodynamic compromise, whether the etiology of CS is univentricular or biventricular, oxygenation status, anatomic considerations, and institutional device availability and expertise. While these devices generally offer more cardiac output support without the risk of arrhythmias...
and toxicity of inotrope pharmacotherapy, each tMCS device is associated with specific considerations and potential complications such as access site bleeding and/or limb ischemia complications. As experience with large-bore access devices increases, vascular complication rates have improved but remain a major barrier to tMCS support in women.7

Although the use of MCS in CS has been increasing over the past 2 decades, no prospective randomized controlled trials have clearly established the clinical benefit of any tMCS device in CS.9,10,22,23 It should be noted, however, that most of these studies were small or terminated early due to inability to randomize patients in extremis and that women represent an even smaller minority of enrolled subjects.22,23 With limited RCT evidence, observational studies represent the best evidence available at this time and have suggested that early initiation of tMCS prior to escalation of inotropes/vasopressors improves mortality.25–27 Single-center and large national and international registries mainly evaluating Impella, ECMO, or the combination of the 2, have consistently found that shorter shock-to-MCS times are associated with improved in-hospital mortality.25–27 In contrast, delays in tMCS implantation of even 1-8 hours reduced survival to hospital discharge.25 Despite this, women with CS are consistently less likely than men to receive tMCS of any kind, more likely to have delays to tMCS, and more likely to receive IABP prior to Impella support escalation. Accordingly, earlier initiation of tMCS in women with CS may serve to close the sex gap in outcomes after CS.

Consensus tips for the General Management of CS in Women

- Early tMCS is advised in women on inotropes and/or vasopressors, particularly those with persistent low cardiac output, rising lactate levels, or other signs of end organ hypoperfusion.

Evidence gaps in the General Management of CS in Women
Studies are needed to inform optimal tMCS device selection in women and to identify sex differences in device complications and outcomes.

Specific Etiologies and Management of Cardiogenic Shock in Women

Acute Myocardial Infarction Cardiogenic Shock

A common cause of CS is AMI, with an analysis by the Critical Care Cardiology Trials Network indicating that women comprised 45% of patients presenting with AMI-CS.\textsuperscript{1} Significant clinical differences exist in comorbidities and early management of AMI-CS in women compared with men. In observational and randomized studies, women with AMI-CS are older with a higher prevalence of hypertension, diabetes, prior heart failure, atrial fibrillation, cerebrovascular, and renal disease; and are more likely to present with non–ST-elevation myocardial infarction (NSTEMI).\textsuperscript{28–31} Women are also more likely to have other nontraditional cardiac risk factors such as autoimmune disease or breast cancer treatment with chest irradiation,\textsuperscript{32} the latter being associated with higher risk disease, more proximal lesions, and higher morbidity and mortality.\textsuperscript{33} Sex-specific atherosclerotic risk factors that significantly increase the risk of AMI in women include early menopause (before 40 years old), early menarche (before 11 years old), history of hypertensive disorders of pregnancy (ie, pre-eclampsia), history of gestational diabetes, history of delivery, history of low or high birth weight infant, or current oral contraceptive use.\textsuperscript{34}

Women with AMI-CS also tend to be at higher risk for less aggressive care due to differences in presentation and diagnosis delays. AMI-CS in women is characterized by more profound hypotension, lower cardiac output, and more acute complications such as acute severe mitral regurgitation and ventricular septal defects compared with men.\textsuperscript{35} Delays in the diagnosis
of ST-elevation myocardial infarction (STEMI) further contribute to the higher frequency of AMI-CS in women. In particular, young women with STEMI compared with age-matched men present to the hospital later, have more atypical anginal symptoms, and experience delays in MI diagnosis even when they do have typical chest pain. Patients with NSTEMI, regardless of sex, have longer delays to PCI or coronary artery bypass grafting (CABG) as compared with patients presenting with STEMI; however, this likely affects women disproportionately as they are more likely to present with CS due to NSTEMI. These factors lead to delays in performing critical diagnostic tests, including electrocardiograms and catheterization, and ultimately contribute to lower rates of primary PCI and higher MI-related complications and mortality in women compared with men.

With regards to treatment, early revascularization with PCI or CABG is the mainstay of therapy in AMI-CS and improves mortality in selected patients of both sexes. Current data suggests that there is no benefit to early multivessel revascularization in the setting of AMI-CS in either men or women, however, it should be noted that these studies tended to include <25% women. As such, further studies that are adequately powered for sex-specific differences are needed to determine whether a multivessel revascularization approach and its timing is warranted in women with AMI-CS.

Sex-specific data on the use of tMCS in AMI-CS is limited. The SHOCK registry showed that IABP use was less common in women compared with men, despite lower cardiac index in women. Subgroup analysis of the IABP-SHOCK-II trial failed to show a sex-specific benefit for 12-month all-cause mortality with IABP use in AMI-CS. Similarly, there is no evidence of differential outcomes of TandemHeart or VA-ECMO in the treatment of AMI-CS in women compared with men. A sex-specific analysis of the
RECOVER registry showed that women with AMI-CS requiring ≥2 inotropes had significantly higher adjusted mortality compared with men (odds ratio [OR], 3.03; 95% CI, 1.26-7.29 vs OR, 1.18; 95% CI, 0.89-1.56, respectively). This finding is supported by observational data evaluating the timing of Impella in AMI-CS, which showed that delays in initiating tMCS after escalation of inotropes resulted in worse survival in women compared with men, whereas, early use of tMCS was not associated with sex-based difference in mortality. Further research is needed to determine whether these findings can be replicated on a larger scale for women with AMI-CS.

**Special consideration: Spontaneous Coronary Artery Dissection**

SCAD is an important underlying cause of non-atherosclerotic MI in women, especially the young. In an analysis of the United States National Readmission Database, women presenting with SCAD were much younger (57 years [IQR 48-68] vs 71 years [IQR 60-81]; p < 0.01) and had fewer comorbidities as compared with patients without SCAD. SCAD has also been shown to be associated with higher rates of CS compared with non-SCAD AMI (9% vs 5%; p < 0.01) even after adjusting for baseline comorbidities (adjusted OR, 1.5; 95% CI, 1.2-1.7). Because SCAD is uncommon in men, there are no data available on outcomes of SCAD-related CS in women compared to men.

The approach to revascularization in SCAD-CS is different from atherosclerotic AMI-CS. In SCAD-CS, percutaneous revascularization outcomes are less predictable due to high rates of complications (ie, iatrogenic dissection, abrupt vessel occlusion). Furthermore, the majority of SCAD will heal within 30 days with conservative management; however, revascularization should still be considered for SCAD patients with ongoing ischemia, extremely high-risk lesions (ie, left main involvement), or multivessel disease. As a consequence, SCAD patients with
STEMI who undergo revascularization are selectively more likely to be in shock or have left main coronary artery or left anterior descending artery culprit lesions, but have better 3-year survival compared to patients with STEMI due to atherosclerosis (98% for STEMI-SCAD vs 84% for STEMI-atherosclerosis; p<0.001). This finding was supported by another analysis of SCAD patients, which showed an early 5% mortality hazard with no further deaths at 2.3 year follow-up\(^5\) despite an early PCI failure (residual stenosis) rate of 53%. At 5-year follow-up, 30% of revascularized vs 19% of conservatively managed patients required repeat revascularization, and CABG graft patency rates were low (5 of 16 grafts patent; median follow-up 3.5 years),\(^5\) suggesting that a conservative revascularization approach is generally well tolerated and preferred, when possible.

**Consensus tips for the treatment of AMI-CS in women**

- Early revascularization with PCI and/or CABG is the mainstay of therapy in AMI-CS.
- In patients presenting with SCAD-CS, tMCS support to recovery and selective revascularization strategies in high-risk lesions may be appropriate.

**Evidence gaps in the treatment AMI-CS in women**

- Addressing local barriers and delays to care access in women with AMI-CS are institutional imperatives.
- Studies are needed to determine whether a complete revascularization approach and its timing improve outcomes in women with AMI-CS.
- Evidence is needed to confirm the clinical benefits of early tMCS before percutaneous revascularization and inotrope escalation in women with AMI-CS.
Cardiogenic Shock in the Pregnant/Post-Partum Patient

CS is rare in pregnancy and occurs in 3.8 out of 100,000 antepartum and postpartum hospitalizations; however, CS in this context is associated with high maternal mortality (18.81% in peripartum CS vs 0.02% peripartum without CS) and higher rates of intrauterine fetal death (1.38% in peripartum CS vs 0.10% peripartum without CS).\textsuperscript{51} Peripartum cardiomyopathy is the most common cause of shock related to pregnancy, accounting for 56% of cases during pregnancy and 82% of cases postpartum. Other etiologies include acute coronary syndrome (either from plaque rupture or SCAD), pre-existing dilated cardiomyopathy, pulmonary arterial hypertension, severe VHD, and amniotic fluid embolism.\textsuperscript{52,53}

Similar to the non-pregnant CS patient, invasive hemodynamics are critical to early identification of shock in the setting of pregnancy and, when identified, hemodynamic support is a priority (\textbf{Figure 2}). Levosimendan, where available, is considered the preferred inotropic agent, as it does not increase myocardial oxygen demand. Otherwise, dobutamine and norepinephrine may be used as first-line inotropic/vasopressor support agents.\textsuperscript{54} tMCS should be considered early after starting intravenous therapy because medical therapy may be insufficient. Registry data suggests that early use of tMCS in pregnancy-related CS (defined as ≤6 days from onset) is associated with greater survival (18% mortality with support ≤6 days vs 38% with >6 days).\textsuperscript{51} Successful tMCS support during pregnancy have been described with IABP, temporary percutaneous or surgical left ventricular assist devices (Impella, TandemHeart, CentriMag), and VA-ECMO, but there is little evidence about which device is preferred.\textsuperscript{54} Need for tMCS support during birth further complicates device selection, with anticoagulation considerations (discussed below) and obstetric recommendations for assisted vaginal delivery (necessitating flexion at the hips) or cesarian section both contributing towards device and access site selection.
Targeted therapies for the specific condition underlying the CS should be considered.

PPCM is discussed in further detail below. SCAD is the most common cause of MI in pregnancy, and patients with AMI-CS from SCAD more frequently have multivessel disease or high-risk lesions (left main or proximal left anterior descending coronary arteries), which increases the likelihood of needing emergent revascularization with either PCI or CABG. For severe symptomatic VHD, especially stenotic left-sided lesions, surgery is an option. Cardiac surgery during pregnancy has been associated with high rates of fetal mortality of up 30%. Catheter-based approaches can be considered (eg, mitral balloon valvuloplasty, aortic balloon valvuloplasty, transcatheter aortic valve replacement), although data in this population is limited to case reports and case series.

Care of the pregnant patient with CS during cardiac procedures poses unique challenges. In the supine position, the gravid uterus may cause aortocaval compression, which can further reduce preload and cardiac output. Placing the patient with a slight left lateral tilt can help relieve this and is especially important if tMCS is being used. Meticulous attention to anticoagulation is imperative, as pregnancy is a hypercoagulable state with increased risk of thromboembolism compared with the nonpregnant state. Both unfractionated heparin (UFH) and low-molecular weight heparin (LMWH) can be used during pregnancy; however, presence of anticoagulation at the time of delivery affects candidacy for epidural or spinal anesthesia, and close coordination with obstetrical anesthesia is required. Additionally, UFH use may be associated with higher rates of postpartum hemorrhage compared with LMWH. Thus monitoring to maintain therapeutic anticoagulation is critical—UFH doses should be adjusted to within a therapeutic aPTT range (aPTT 1.5-2.5 times control) and LMWH doses should be adjusted to maintain anti-Xa levels of 0.6-1.0 units/mL. Measures should be taken to reduce fetal radiation exposure.
include using external abdominal shielding, reducing fluoroscopy time, lower magnification and frame rates, and careful collimation. \textsuperscript{60} Iodinated contrast is also associated with potential risk of fetal congenital hypothyroidism but does not preclude its use. \textsuperscript{61} All measures to reduce fetal exposure are warranted, but these should not take precedence over procedures to preserve maternal life.

Most importantly, a multidisciplinary team collaboration between cardiology, obstetrics, anesthesiology, and critical care are paramount to maternal and fetal/neonatal safety. \textsuperscript{62} A cardio-obstetrics team is recommended for the evaluation and management of high-risk cardiac disease in pregnancy and is required for rapid decision-making in pregnant patients with CS, \textsuperscript{63} especially in conditions with high maternal mortality where pregnancy termination may be considered. \textsuperscript{64} Other considerations such as choice of medications and anesthesia should be made based on the individual clinical situation, maternal benefit, and fetal exposure. Managed anesthesia care improves maternal airway and hemodynamic control while limiting maternal and fetal anesthetic exposure. Continuous fetal monitoring may be considered if the gestational age is at ex-utero viability (typically $\geq 23$ weeks of gestation) and emergent C-section is an option, thus decision to implement fetal monitoring should be made in collaboration with obstetrics. \textsuperscript{65} Timing and mode of delivery depends on maternal stability and fetal status and requires multidisciplinary coordination between cardiac and obstetrical teams.

**Special Consideration: Peripartum/postpartum Cardiomyopathy Complicated by Cardiogenic Shock**

CS complicates $\sim 4\%$ of PPCM, which is defined as idiopathic left ventricular (LV) dysfunction (ejection fraction [EF] $\leq 45\%$) that presents towards the end of pregnancy or in the months following delivery. \textsuperscript{66} The etiology of PPCM is thought to be multifactorial, with
contributions from genetic factors, auto-immune responses, fetal microchimerism, and excessive prolactin production.\textsuperscript{66}

In addition to the general principles of CS treatment with typical pharmacologic therapies, bromocriptine may have a role as targeted treatment of PPCM-CS. Bromocriptine is a dopamine agonist that inhibits prolactin release and has been associated with higher rates of LV recovery in mostly pilot and observational studies.\textsuperscript{67} While the European Society of Cardiology (ESC) guidelines include a modest recommendation (Class IIb, level evidence B) to consider its use,\textsuperscript{62} bromocriptine is considered experimental the United States and Canada. Accordingly, its clinical benefit is being investigated in a randomized double-blind, placebo-controlled clinical trial (REBIRTH; Randomized Evaluation of Bromocriptine in Myocardial Recovery Therapy; ClinicalTrials.gov identifier: NCT05180773), comparing bromocriptine therapy vs placebo in women with PPCM (LVEF ≤35\%).\textsuperscript{67} If used, bromocriptine has been associated with thrombotic complications and should be accompanied by at least prophylactic anticoagulation.\textsuperscript{62,67}

As with other CS etiologies, tMCS may be considered in patients with PPCM-CS who cannot be stabilized on medical therapy alone. A small study reported excellent short-term survival (100\% at 30 days and 80\% at 6 months) with early use of tMCS and bromocriptine therapy.\textsuperscript{68} Increased prolactin levels during ECMO treatment have been reported, which may be detrimental in PPCM-CS, and higher bromocriptine doses can be considered if used.\textsuperscript{54} Because many patients have at least partial LV recovery, a bridge-to-recovery strategy is the preferred approach\textsuperscript{15}; however, the evaluation for long-term advanced heart failure therapies—durable MCS (surgical LV or biventricular assist devices) and/or cardiac transplantation—should be initiated soon after implantation of tMCS, with plans to transition to one of these long-term strategies if temporary support cannot be weaned after 7-10 days. Surprisingly, LV recovery with
durable MCS is uncommon, with 1 study showing only 6% recovery with explant rate in PPCM patients, which may be due to variability in patient selection or recovery protocols between centers. Cardiac transplantation is considered for patients for whom durable MCS is not an option or who do not exhibit substantial LV recovery on durable MCS after 6-12 months. Nevertheless, it should be noted that PPCM patients have worse heart transplant outcomes when compared with transplant patients with CM of other etiologies as evidenced by lower overall survival rates, increased allograft rejection and graft failure, and higher rates of re-transplantation. Special considerations for the management of cardiac arrest in the pregnant or post-partum patient are in Supplement.

Consensus tips for treatment of pregnant patients with cardiogenic shock, including PPCM:

- An established multidisciplinary cardio-obstetrics team, including cardiology, obstetrics/maternal fetal medicine, anesthesiology, critical care, and nursing is paramount to rapid decision making in pregnant patients with CS.

- Early invasive hemodynamics assessment and consideration for early tMCS are critical to maternal survival.

- Measures to reduce fetal exposure to radiation and medications are warranted but should not take precedence over treatments to preserve maternal life.

- For patients with PPCM-CS, a bridge-to-recovery strategy is the preferred approach because of high rates of at least partial LV recovery.

Evidence gaps in the treatment of pregnant patients with cardiogenic shock, including PPCM:

Further data is needed to clarify the safety and efficacy of bromocriptine on LV recovery
Heart Failure-Related Cardiogenic Shock

Cardiogenic shock due to heart failure (HF-CS) is now the most common etiology of CS in the modern cardiac intensive care unit, with women representing a third of these patients.\textsuperscript{70,71} The most common etiology of HF-CS is acute decompensation of chronic HF, accounting >70\% of HF-CS cases in women. De novo HF causes such as myocarditis and stress-induced cardiomyopathy are also more likely to occur in women as compared with men (26.3\% vs 19.3\%)\textsuperscript{7} and are reviewed separately below (see Supplement for Acute and Fulminant Myocarditis).

A sex-based analysis by the Cardiogenic Shock Working Group showed that women with HF-CS have higher baseline SCAI SHOCK stage as compared with men (stage E 26\% vs 21\%), and have worse survival at discharge (69.9\% vs 74.4\%).\textsuperscript{7} This is, in part, related to the fact that women with chronic HF are undertreated when compared with men—they are less likely to received evidence-based pharmacologic therapy and implanted device (internal cardiac defibrillator and cardiac resynchronization) therapy.\textsuperscript{72} Furthermore, women with HF-CS are more likely to be older and have more cardiovascular comorbidities (hypertension, diabetes mellitus) and tend to present with worse initial hemodynamics.\textsuperscript{6} Despite presenting with higher clinical acuity, women with HF-CS have been shown to be less likely to receive pulmonary artery catheterization (52.9\% vs 54.6\%), more likely to be treated without tMCS support (26.2\% vs 18.8\%), and less likely to receive heart replacement therapy with durable LVAD (7.8\% vs 10\%) or cardiac transplantation (6.5\% vs 10.3\%) when compared with men.\textsuperscript{7}

While the majority of CS research has been conducted in AMI-CS, there is increasing data specific to HF-CS that highlight important differences in tMCS use and efficacy. Overall,
tMCS use for HF-CS has increased over the past 2 decades\textsuperscript{73} and is most commonly used as a bridge to advanced HF therapies (durable LVAD or cardiac transplantation); however, in contrast to AMI-CS, where IABP use has been decreasing, IABP remains the most commonly used device in HF-CS, and rates of use are increasing.\textsuperscript{71,74} However, a recent analysis by the Cardiogenic Shock Working Group registry showed that while IABP is the most common tMCS used in HF-CS, it was more often used in combination with other tMCS strategies and likely used as part of a step-wise escalation strategy.\textsuperscript{71} There is little clarity as to which devices are more effective in HF-CS, although hemodynamic profiling remains critical to tMCS device selection.\textsuperscript{15} The notion of the “IABP super-responder” phenotype based on patient characteristics and initial hemodynamic values, which may be associated with significant improvement in CI and/or MAP with IABP support, has been more intensely studies in HF-CS patients, especially after the SHOCK II trial failed to show a mortality benefit for IABP in AMI-CS patients.\textsuperscript{41} However current studies around this concept have focused on physiologic analysis,\textsuperscript{75} and there is conflicting data as to whether improved hemodynamics with IABP in HF-CS results in better outcomes (ie, survival or need for MCS escalation).\textsuperscript{76,77} Furthermore, the majority of patients in these studies were men, and no sex-specific analysis has been performed, so it is unclear if similar parameters are applicable to women with HF-CS.

Most survivors of HF-CS should be initiated and maintained on HF guideline-directed medical therapy and appropriate device therapy to encourage myocardial recovery, optimize functional capacity, reduce the rate of subsequent hospitalizations, and maximize survival.\textsuperscript{9} Although data specific to survivors of CS is limited, women are generally less likely to be adequately prescribed or up-titrated on guideline-directed medical therapy medication or receive cardiac device therapy when indicated,\textsuperscript{78} and even less likely to receive HF discharge
Advanced Heart Failure Therapies: Limitations in Care for Female Survivors of Cardiogenic Shock

Patients with CS who fail to recover with medical therapy or tMCS should be considered for advanced heart failure therapies (LVAD and cardiac transplant). While pivotal LVAD trials have shown a benefit for patients with chronic end-stage HF, women have been underrepresented in these trials, so evidence regarding sex-specific differences in outcomes is murky. For example, in the recent MOMENTUM 3 trial, which compared the HeartMate III and HeartMate II devices, only ~20% of enrollees were women. While early generation pulsatile flow durable LVADs were associated with higher mortality for women (OR, 2.13; 95% CI, 1.45-3.10; p<0.0001), current generation continuous flow LVADs show similar survival between the sexes. There have also been specific concerns about an excess risk of neurologic events in women receiving durable MCS. In a HeartMate II cohort, the risk of hemorrhagic stroke was greatest in women <65 years of age, whereas the risk of thromboembolic events was greatest in women >65 years of age. With the contemporary HeartMate III LVAD, risk of stroke overall is much lower, but women continue to be at higher risk. A recent sex-specific analysis of the MOMENTUM 3 trial showed that women had an increased risk of stroke (adjusted incidence rate ratio [aIRR], 1.52; p = 0.12) in addition to higher risk of major bleeding (aIRR, 1.28; p <0.0001) and infection (aIRR, 1.14; p = 0.01); however, this analysis also showed that there were no sex-based differences in overall survival or in the primary outcome (survival free of disabling stroke or need for pump replacement or removal at 2 years post-implant). These findings highlight the need for further sex-specific studies regarding the outcomes associated with durable LVADs.
Cardiac transplantation remains the gold standard treatment option for patients who
develop end-stage HF and prolonged CS; however, women remain less likely to undergo
transplant compared with men, accounting for only 23% of heart transplant patients. In a
United Network for Organ Sharing (UNOS) analysis, women receiving a durable LVAD as a
bridge to transplantation were found to have lower rates of heart transplantation (55.1% vs
67.5%), greater waitlist mortality (7.0% vs 4.2%), and more delisting for clinical deterioration
(8.5% vs 4.7%) at 2 years of LVAD support, as compared with men (all p<0.001). Another
sex-based analysis evaluating patients at the highest heart transplant urgency strata (Status 1)
found similar trends of women with lower rates of transplant and higher rates of delisting for
death or clinical deterioration. Contributing factors identified in these studies include higher
allosensitization in women (which makes finding suitable donors more difficult) and/or MCS-
related complications, but precise mechanisms underlying lower transplant rates in women
remain unclear. Importantly, women who proceed to cardiac transplantation have a similar
post-transplant survival rate compared to men. These findings underscore the importance of
developing sex-specific best practices in post-CS care for women with HF to help safely support
women to transplant.

Special Consideration: Stress-induced cardiomyopathy / Takotsubo Syndrome

Takotsubo syndrome (TTS) is a specific, acute, non-ischemic cardiomyopathy that can
present as CS in 5-10% of cases. TTS classically follows an intense emotional or physical stress
and tends to present similar to MI but without plaque rupture. Approximately 90% of TTS occur
in women, and it is particularly prevalent in post-menopausal women. Younger women (<50 years
old) with TTS are more likely to present with CS and have higher in-hospital mortality. When
TTS presents with CS, mortality rates are substantially higher compared to TTS without CS
(23.5% vs 2.3%), with the majority of mortality occurring in the first 24 hours after presentation when patients are most severely hypotensive. The development of CS in TTS is likely multifactorial—LV systolic dysfunction may be exacerbated by RV dysfunction, and left ventricular outflow tract obstruction (LVOTO) due to hyperkinetic basal segments may contribute to poor cardiac output. As a result, the administration of catecholamines should be avoided in TTS due to the underlying mechanism and their potential to exacerbate instability, and tMCS is often considered first line for this population. Indeed, a propensity analysis consisting of data predominantly from the International Takotsubo Registry showed lower in-hospital mortality for patients with CS who received tMCS (OR, 0.34; 95% CI, 0.12–0.95; p=0.04). Since there is an observed high rate of rapid recovery from TTS, tMCS is usually only needed as a bridge to recovery.

Consensus tips for the treatment of HF-CS and use of Advanced Heart Failure Therapies in Women

- Development of strategies to address lower transplant rates and limitations in access to durable LVAD support in women are needed at both the individual patient level and systems level.

Evidence gaps in the treatment of HF-CS and use of Advanced Heart Failure Therapies in Women

- Further studies to understand and overcome sex-specific disparities in treatment of women with HF-CS are a priority.
- While a specific phenotype of patients with HF-CS may respond well to IABP therapy
Valvular Heart Disease-Related Cardiogenic Shock

Aortic Stenosis

Cardiogenic shock associated with severe aortic stenosis (AS) occurs in up to 12% of patients and has been associated with an extremely high mortality rate in the absence of a corrective valve procedure. Often, patients are treated with MCS or percutaneous valvular intervention (either balloon aortic valvuloplasty or transcatheter aortic valve replacement [TAVR]) to stabilize CS, as immediate surgical intervention portends a higher risk of mortality in this context. TAVR remains an important treatment option in patients presenting with AS-related CS. While mortality of AS-CS patients who receive TAVR have high 30-day mortality rates of 13-20%, this is still considerably lower than the reported mortality of 35-70% in conservatively managed patients. Furthermore, a recent analysis of the Transcatheter Valve Therapy (TVT) registry showed that for patients who survive the initial 30 days, there was no difference in 1-year mortality between propensity-matched CS and non-CS patients. Furthermore, risk of death from acute CS before TAVR was strongly related to degree of shock pre-procedure, highlighting the importance of early recognition in this population.

There are no data regarding sex-specific treatment or outcomes of AS patients presenting with CS. Sex was not an independent predictor of outcome in the recent TVT registry, although no sex-specific analysis was done. Nevertheless, sex-specific analyses in the non-shock population may offer some insights into sex differences in diagnosis, presentation, and treatment considerations in the CS population with AS. Pre-clinical studies have demonstrated that women

("IABP-Responders"), sex-specific evidence of the selection of tMCS on outcomes is lacking.
with severe AS tend to have less valvular calcium\textsuperscript{99} and higher degrees of valvular fibrosis compared with men.\textsuperscript{100} Furthermore, women have been shown to have more concentric LV remodeling, with evidence of greater diffuse intramyocardial fibrosis,\textsuperscript{101} which can result in lower stroke volumes despite preserved ventricular function. Accordingly, use of sex-specific cut-offs for women with aortic valve calcification (\textgreater{}1300 Agatston Units) as well as stroke volume index (\textless{}32 mL/m\textsuperscript{2}) may be considered for the timely diagnosis of severe AS to help ensure that this condition is not missed in the setting of cardiogenic shock.\textsuperscript{102,103} In terms of treatment, while studies using first- and second-generation TAVR prostheses demonstrated higher rates of bleeding or vascular complications in women treated with TAVR,\textsuperscript{104,105} recent studies have demonstrated no sex-specific differences in survival or stroke in patients,\textsuperscript{106,107} which may reflect the changing demographic of the patient population being treated with TAVR (eg, lower risk) as well as advances in device technology and procedural techniques. Hence, TAVR should be considered as a viable treatment option for women with severe AS presenting with cardiogenic shock.\textsuperscript{108} 

\textbf{Aortic Regurgitation} 

As for AS, there are no sex-specific data on outcomes of acute aortic regurgitation in the setting of cardiogenic shock. For additional information see \textsuperscript{Supplement.} 

\textbf{Mitral Regurgitation} 

Mitral regurgitation (MR) is frequently seen in patients presenting with CS and has been associated with worse outcomes when compared to those patients without CS.\textsuperscript{109} While no randomized trials have evaluated the role of mitral valve intervention in the CS population,
multiple registry studies have demonstrated the feasibility and potentially improved outcomes associated with transcatheter mitral edge-to-edge repair therapy in CS patients.\textsuperscript{110,111} No sex-specific analyses have been reported on treatment or outcomes of mitral valve disease presenting with CS; however, data from the non-shock population may offer some insight into sex differences in treatment and outcomes in the CS population with MR.

Women with MR have been shown to have worse long-term outcomes when compared with men. Women are less likely to be referred for mitral valve interventions compared with men,\textsuperscript{112} and when they do present for mitral valve evaluation, studies have demonstrated that women have higher rates of comorbid conditions (eg, hypertension, diabetes, chronic kidney disease).\textsuperscript{113} At 2 years after mitral valve surgery, women not only have higher all-cause mortality as compared with men (27.1\% vs 17.4\%), but women also had worse quality of life and poorer functional capacity.\textsuperscript{113} Possible reasons for this observation include greater prevalence of comorbidities (ie, diabetes, hypertension, chronic kidney disease) and later presentation to medical attention. Differences in LV remodeling between sexes may also contribute to variations in outcomes. A recent analysis showed that women undergoing mitral valve repair had increasing mortality rates at lower indexed LV systolic dimensions (LVEDi) as compared with men (1.8 cm/m\(^2\) for women vs 2.1 cm/m\(^2\) in men). Women also demonstrated a sharper rise in mortality with LVEF <60\% as compared with men.\textsuperscript{114} Thus, sex-specific indexed imaging measurement cut-offs (ie, lower LVEDi and not waiting for LVEF to decrease <60\% in women) may help risk stratify women with MR more effectively. With regard to percutaneous therapies, the data suggest that transcatheter edge-to-edge repair (TEER) is equally safe in men and women with functional MR\textsuperscript{115} but that women may derive less benefit than men.\textsuperscript{116} That being said, it is notable that women comprised the minority of chronic secondary MR patients enrolled in these
studies,\textsuperscript{115,116} so further larger scale studies are needed to determine whether there is any sex difference in therapeutic benefits with TEER.

**Consensus tips for treatment of VHD-CS in Women**

- Earlier referral of women with VHD to specialists may decrease the likelihood of undertreatment of these patients.

**Evidence Gaps in the treatment of VHD-CS in Women**

- Inclusion of adequate subset of women in percutaneous valve intervention trials to understand sex-specific benefits and complications.

**Barriers to Care for Women with Cardiogenic Shock**

The fear of vascular complications has likely contributed to the underutilization of cardiac treatments in women. Historically, women undergoing PCI are up to 2 times more likely than men to have major access site vascular or bleeding complications,\textsuperscript{117} likely due to smaller vessel sizes and higher incidence of peripheral arterial disease.\textsuperscript{118} Similarly, due to the large-bore access required for tMCS, early studies using IABP suggested women had higher rates of access-related complications; however, improvements in vascular access techniques and device innovation over the past decade have reduced bleeding and vascular complications in both men and women.\textsuperscript{117,116} This improvement may be due, in part, to changes in technique. It is now commonplace to use a combination of techniques such as palpation, fluoroscopy, ultrasound, and micropuncture techniques to guide access and then use vascular closure devices with or without balloon tamponade for large-bore closure.\textsuperscript{119} Similarly for tMCS, more recent sex-based studies of IABP, Impella, and VA-ECMO have found vascular and bleeding complication rates to be lower than historical controls.\textsuperscript{35,43,44} Large-bore femoral access is now safer due to improved
techniques in access, closure, and complication management.\textsuperscript{120} Axillary implantation sites are also considered if large-bore device or prolonged support duration is being considered.\textsuperscript{120} Women, though, are still more likely to experience bleeding or vascular injury requiring surgery, vascular complications requiring surgery, or limb ischemia with tMCS as compared with men.\textsuperscript{7} Nevertheless, it is important to emphasize that these potentially lifesaving procedures should not be avoided in women, especially since the evidence above suggests that improved techniques with vascular access and device innovation can mitigate risks of bleeding and vascular injury.

Second, low enrollment of women in clinical trials of CS spanning revascularization,\textsuperscript{29,39} tMCS,\textsuperscript{41} and advanced HF therapies\textsuperscript{80} remains a major impediment to establishing best practices in this high-risk population. Figure 3 shows the percentage of women enrolled in contemporary CS randomized clinical trials. It is notable that none of the listed trials enrolled women at a level representative of the general population. Best practices associated with improved enrollment of women in clinical trials ought to be established, adopted, and disseminated to better determine risks and benefits of novel treatments in women.

Lastly, standardization of shock treatment can also help improve diagnostic accuracy and reduce sex-based disparities. Multidisciplinary shock teams (inclusive of a HF cardiologist, a cardiothoracic surgeon, an interventional cardiologist, and a cardiovascular intensive care unit physician)\textsuperscript{121} can quickly identify and define the severity of CS, establish the etiology, rapidly implement measures for hemodynamic support, and initiate etiology-specific treatments. Indeed, initiation of shock teams/algorithms has been associated with faster and more appropriate treatments for patients with CS and improvements in survival in multiple centers.\textsuperscript{16,121,122} A standardized team-based CS treatment protocol including mandatory hemodynamic assessment, timely diagnosis, and early, appropriate tMCS use may reduce sex disparities in CS outcomes.\textsuperscript{6}
Consensus tips to address Barriers to Care for Women with CS

- Fear of vascular complications should not deter the use of potentially lifesaving tMCS; rather, risks should be mitigated with improved techniques for vascular access and follow best practices for indwelling devices.

- A standardized, team-based CS treatment protocol including mandatory hemodynamic assessment, timely diagnosis, and early, appropriate tMCS use may reduce sex disparities in CS outcomes.

Evidence Gaps in addressing Barriers to Care for Women with CS

- Improve enrollment by setting a prespecified quota of women in ongoing and future CS clinical trials to determine risks and benefits of novel treatments in women.

- Device innovation for smaller profile devices to reduce vascular complications should be a priority.

- While SCAI SHOCK classification has been validated in a variety of clinical settings, there has not been sex-based analysis to correlate SCAI stage with mortality in women specifically.

Future Directions and Conclusions

Early identification of CS and its etiology and early referral for mechanical support are paramount to improving mortality outcomes in women. Standardization of CS diagnosis and treatment as we have proposed (Figure 1) will help address disparities in current clinical care; however, to have a long-term impact on mortality in women with CS, we must continue to recognize and address barriers to early diagnosis with a goal of early treatment. With this in mind, future observational and randomized trials should seek to enroll an appropriate population of women to inform the balance of risk and benefit in this population. Beyond this, dedicated
powered studies addressing etiologies and treatment specifically in women will help to address
the disparities women face in access to care.

This consensus provides a comprehensive summary of the current state of treatment of
CS in women in relevant disease states and identifies important evidence gaps. As there are
limited sex-based data in contemporary literature, clinicians may use this document as a resource
to guide practice. Further investigations are necessary to inform best practices for women with
CS.

**Peer review statement**

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regarding its peer review.

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doi:10.1016/j.hlc.2014.06.010


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Figure 1. Cardiogenic shock algorithm

STEMI, ST-elevation myocardial infarction; HR, heart rate; MAP, mean arterial pressure. TTE, transthoracic echocardiogram; PAC, pulmonary artery catheter; UOP, urine output; LFTs, liver function tests; MCS, mechanical circulatory support; LVAD, ventricular assist device; GOC, goals of care; HF-CS, heart failure-related cardiogenic shock; AMI-CS, acute myocardial infarction-related cardiogenic shock; valvular-CS, valvular-heart disease related cardiogenic shock; PPCM-CS, peripartum cardiomyopathy-related cardiogenic shock
Figure 2. Cardiogenic shock in pregnancy

NTG, nitroglycerin. PPCMP, peripartum cardiomyopathy. MCS; mechanical circulatory support. RV; right ventricle. LV; left ventricle. P-SCAD; pregnancy-related spontaneous coronary artery dissection.
Figure 3. Rates of women enrollment in randomized clinical trials of cardiogenic shock

Orange: studies evaluating all causes of cardiogenic shock (CS); blue: studies evaluating acute myocardial infarction complicated by CS (AMI-CS); red: studies evaluating non-AMI causes of shock; green: post-surgical CS
Table 1. Invasive cardiac hemodynamics and indicators of cardiogenic shock

<table>
<thead>
<tr>
<th><strong>Left Ventricular Metrics</strong></th>
<th><strong>Calculation</strong></th>
<th><strong>Indicator of cardiogenic shock</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac index (CI)</td>
<td>CO / body surface area</td>
<td>≤ 2.2 L/min/m²</td>
</tr>
<tr>
<td>Cardiac power output (CPO)</td>
<td>(MAP × CO)/451</td>
<td>&lt; 0.6 watts</td>
</tr>
<tr>
<td>Cardiac power index (CPI)</td>
<td>(MAP × CI)/451</td>
<td>&lt; 0.4 watts/m²</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>Systolic – diastolic blood pressure</td>
<td>&lt; 25 mm Hg</td>
</tr>
<tr>
<td>Systemic vascular resistance (SVR)</td>
<td>[(MAP – CVP) / CO] × 80</td>
<td>variable</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Right Ventricular Metrics</strong></th>
<th><strong>Calculation</strong></th>
<th><strong>Indicator of RV dysfunction</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrial pressures (RAP)</td>
<td></td>
<td>&gt; 10/15 mm Hg</td>
</tr>
<tr>
<td>RAP / PCWP ratio</td>
<td></td>
<td>&gt; 0.86 (in AMI)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt; 0.63 (after LVAD)</td>
</tr>
<tr>
<td>Pulmonary artery pulsatility index (PAPi)</td>
<td>(PASP – PDP) / RAP</td>
<td>≤ 0.9 (in AMI)</td>
</tr>
<tr>
<td>Right ventricular stroke work index (RVSWI)</td>
<td>0.0136 × SVI × (mPAP – RAP)</td>
<td>&lt; 1.85 (after LVAD)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt; 6 g/m/beat/m²</td>
</tr>
</tbody>
</table>

CO, cardiac output; MAP, mean arterial pressure; CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; PASP, pulmonary artery systolic pressure; PAP, pulmonary artery diastolic pressure; mPAP, mean pulmonary artery pressure; AMI, acute myocardial infarction; LVAD, left ventricular assist device; Svi, stroke volume index. Adapted from Circulation. 2022;146:e50–e68.
# Table 2. Summary of temporary mechanical circulatory support strategies

<table>
<thead>
<tr>
<th></th>
<th>RV support</th>
<th>LV support</th>
<th>Biventricular support</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mechanism</strong></td>
<td>Axial-flow continuous pump (RA to PA)</td>
<td>Centrifugal-flow continuous pump (RA to PA)</td>
<td>Balloon inflation-deflation (aortic counterpulsation)</td>
</tr>
<tr>
<td><strong>Support</strong></td>
<td>RV</td>
<td>LV</td>
<td>LV</td>
</tr>
<tr>
<td><strong>Insertion/placement</strong></td>
<td>Femoral vein</td>
<td>IJ vein</td>
<td>Femoral artery or axillary artery (2.5, CP)</td>
</tr>
<tr>
<td><strong>Cannula size</strong></td>
<td>22F venous</td>
<td>29F/31F venous</td>
<td>7F–8F arterial</td>
</tr>
<tr>
<td><strong>Flow, L/min</strong></td>
<td>2–4</td>
<td>Maximum 4.5</td>
<td>0–1</td>
</tr>
<tr>
<td><strong>Maximum pump speed, rpm</strong></td>
<td>33 000</td>
<td>7500</td>
<td>NA</td>
</tr>
<tr>
<td><strong>LV unloading</strong></td>
<td>…</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td><strong>RV unloading</strong></td>
<td>↑</td>
<td>↑</td>
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<tr>
<td><strong>Coronary perfusion</strong></td>
<td>↑</td>
<td>↑</td>
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<tr>
<td><strong>CVP</strong></td>
<td>↓</td>
<td>↓</td>
<td>↔ or ↓</td>
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<tr>
<td><strong>MAP</strong></td>
<td>↑</td>
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<tr>
<td><strong>LVEDP</strong></td>
<td>↑</td>
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<tr>
<td><strong>PCWP</strong></td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
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<tr>
<td><strong>Myocardial oxygen demand</strong></td>
<td>↓</td>
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</tr>
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</table>

**Surgical tMCS considerations**

Pump options include Centrimag (Abbott), Cardiohelp (Getinge), and Rotaflow (Getinge). These can be used with or without an oxygenator in multiple configurations, including the following:

1. A temporary RVAD can have a drainage cannula in the femoral vein or RA with a return cannula from the IJ into the PA;
2. A temporary central RVAD can have a drainage cannula in the RA or RV with a return cannula into the PA;
3. A temporary central LVAD can have a drainage cannula in the LA or LV with a return cannula into the aorta; or
4. Multiple central and percutaneous BiVAD configurations are possible.

AO indicates aorta; BiVAD, biventricular assist device; CS, cardiogenic shock; CVP, central venous pressure; FA, femoral artery; IABP, intra-aortic balloon pump; IJ, internal jugular; LA, left atrium; LV, left ventricle; LVAD, left ventricular assist device; LVEDP, left ventricular end-diastolic pressure; MAP, mean arterial pressure; NA, not applicable; PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; RA, right atrium; RV, right
Adapted from Circulation. 2022;146:e50–e68.

<table>
<thead>
<tr>
<th>RV support</th>
<th>LV support</th>
<th>Biventricular support</th>
</tr>
</thead>
<tbody>
<tr>
<td>ventricle; RVAD, right ventricular assist device; tMCS, temporary mechanical circulatory support; and VA-ECMO, venoarterial extracorporeal membrane oxygenation.</td>
<td>* Other percutaneous cannulation sites and multiple cannulation sites can be used: arterial access (axillary, subclavian or carotid) or venous access (IJ). Central configurations are also possible.</td>
<td></td>
</tr>
</tbody>
</table>

Adapted from Circulation. 2022;146:e50–e68.