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REVIEW

Percutaneous mechanical circulatory support for acute right heart failure: A practical approach

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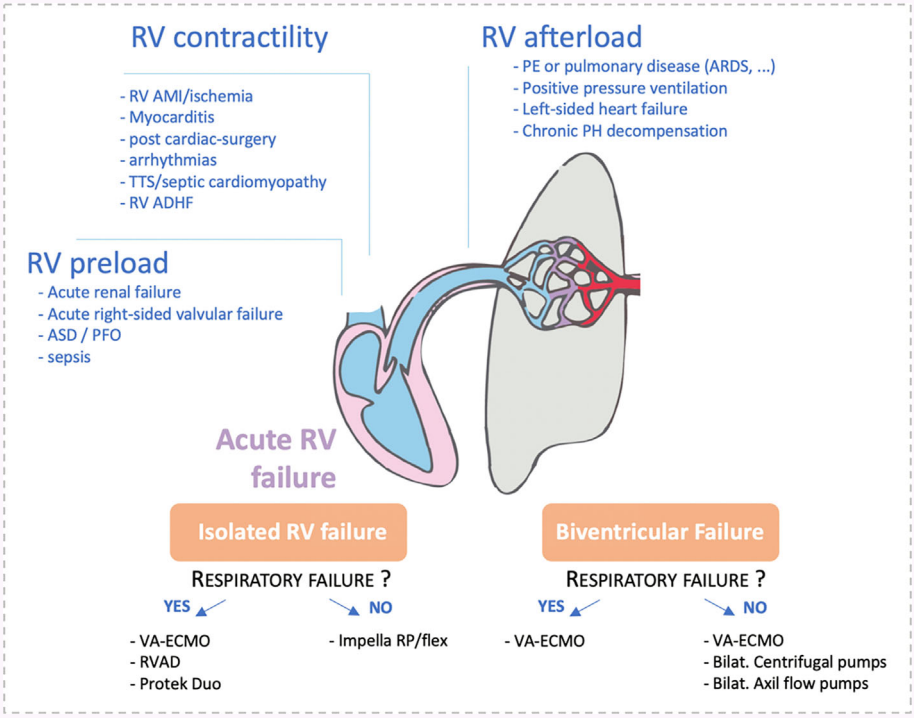
Abstract

Acute right heart failure (RHF) represents a critical entity with significant morbidity and mortality. This review examines the role of percutaneous right ventricular assist devices (pRVADs) as a cornerstone of therapy in cases refractory to conventional management. Devices such as the Impella RP and dual-lumen cannulas provide targeted haemodynamic support, with indications in various clinical scenarios, including acute myocardial infarction, post-cardiac surgery, myocarditis, and after left ventricular assist device (LVAD) implantation. Successful implementation requires meticulous haemodynamic assessment, including parameters derived from pulmonary artery catheterization and echocardiography, to guide patient selection, optimize device placement, and monitor therapeutic response. The manuscript highlights contemporary weaning strategies, emphasizing recovery of right ventricular function, stabilization of systemic haemodynamics, and restoration of end-organ perfusion. While no universal protocols exist, this review presents a pragmatic framework informed by available evidence and expert consensus. Furthermore, the potential complications of pRVAD use—ranging from thromboembolism and haemolysis to device-specific issues such as migration and tricuspid valve damage—are discussed alongside preventive and management strategies. Key challenges in RHF management, including the interplay between right and left ventricular function, the impact of pulmonary vascular resistance, and the use of adjunctive pulmonary vasodilators, are addressed. The review underscores the absence of durable right ventricular assist devices and the need for innovation to close this therapeutic gap. Multidisciplinary collaboration among intensivists, cardiologists, and cardiac surgeons is critical to optimizing outcomes. This review provides actionable insights to assist clinicians in navigating the complexities of acute RHF, fostering a tailored and evidence-based approach to this high-risk population.

Graphical Abstract

This is an overview of percutaneous mechanical circulatory support options for acute right heart failure, illustrating key clinical scenarios, device types, and principles of tailored device selection based on haemodynamics and underlying pathology.



Keywords pRVAD; Impella; ECMO; Cardiogenic shock

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Introduction

Right heart failure (RHF) is a significant clinical challenge in the intensive care setting, particularly due to its strong association with high in-hospital mortality rates, reported between 70% and 75%, and considerable short-term morbidity.[1,2] This condition can manifest either in isolation or in combination with left ventricular (LV) failure, especially in contexts such as post-cardiac surgery, myocardial infarction, or following the implantation of a left ventricular assist device (LVAD). Managing RHF is inherently complex and demands meticulous haemodynamic assessment along with timely therapeutic intervention. Furthermore, the RV's close interaction with the left ventricle (LV) adds another layer of complexity, as dysfunction in one ventricle can exacerbate failure in the other.

Intensivists must remain particularly vigilant in high-risk patients, ensuring the early initiation of appropriate medical and mechanical support strategies. These strategies are crucial for improving outcomes and often require the use of mechanical circulatory support (MCS) devices specifically designed to accommodate the RV's unique anatomy and functional demands.

Despite a significant increase in the use of short-term percutaneous left ventricular assist devices (pLVADs), the utilization of percutaneous right ventricular assist devices (pRVADs) has not seen a corresponding rise.[3] MCS is a potential option for selected patients in the acute setting where conventional therapies fail. Acute RHF can occur due to several underlying pathophysiological mechanisms, including increased afterload (e.g. pulmonary embolism or acute left-sided heart failure), increased venous return causing volume

overload (e.g. LVAD implantation or sepsis), and sudden reductions in contractility (e.g. RV infarction or myocarditis). These mechanisms often coexist, further complicating the clinical picture (graphical abstract, *Table 1*).

Symptoms and signs of acute RHF can be non-specific, encompassing features of systemic/venous congestion as well as reduced cardiac index (CI). Early recognition with clinical imaging, laboratory, and invasive haemodynamic parameters represents an appropriate approach[4,5] to determine potentially reversible causes and guide management including pRVAD.[1,6]

The selection of the appropriate pRVAD can be challenging, and the choice should be carefully weighed on a case-by-case basis. This narrative review focuses on a practical approach to the selection and management of pRVAD in acute RHF when conventional therapies fail.

Diagnosis of acute right heart failure

Diagnosing acute RHF requires a comprehensive approach that considers the clinical context, echocardiographic findings, and haemodynamic data. Clinicians should begin by evaluating the patient's clinical situation. For instance, conditions such as infero-posterior acute myocardial infarction (AMI), post-LVAD implantation, acute respiratory distress syndrome (ARDS), or other underlying factors that predispose a patient to RHF should be closely monitored. An initial clinical assessment should focus on identifying signs and symptoms indicative of RHF, such as jugular venous distension, peripheral oedema, and hepatomegaly.

Table 1 Clinical conditions predisposing acute right heart failure

Physiopathologic features of RH failure	Clinical condition
Increased after-load	Pulmonary embolism
	Pulmonary diseases (e.g. ARDS; pneumonia)
	Hypercarbia
	Acute on chronic pulmonary hypertension
	Left-sided heart failure
Reduced contractility	RV myocardial infarction/ischaemia
	Myocarditis
	Post-cardiac surgery (included LVAD insertion)
	Supraventricular or ventricular tachycardia; AV dyssynchrony
	Septic cardiomyopathy
	RV acute decompensated heart failure
	Post trauma
Increased preload	Acute renal failure (severe fluid retention)
	Acute right-sided valvular insufficiency
	Unrepaired atrial septal defect/PFO
	Sepsis
	Perioperative volume overload

ACT, activated clotting time; ARDS, acute respiratory distress syndrome; ASD, atrial septal defect; AV, atrio-ventricular; DVT, deep vein thrombosis; ECMO, extracorporeal membrane oxygenation; Fr, French; IVC, inferior vena cava; PA, pulmonary artery; PFO, patent foramen ovale; PFO, patent foramen ovale; PTT, partial thromboplastin time; RA, right atrium; RV, right ventricle; RV, right ventricle; SVC, superior vena cava; TTS, Takotsubo syndrome.

Once RHF is suspected, echocardiographic evaluation becomes essential. This non-invasive tool provides crucial information about the right ventricle's size, function, and morphology.[7] Key parameters to assess include tricuspid annular plane systolic excursion (TAPSE) less than 14 mm, tissue Doppler imaging (TDI) with an S' velocity <9.5 cm/s at the tricuspid annulus, right ventricular fractional area change (RVFAC) less than 35%, new regional wall motion abnormality, and the presence of interventricular septal flattening. Estimation of RV size is also essential and can be supported by measuring RV basal diameter and area in an RV-focused four-chamber view. In ICU intubated patients, transesophageal echocardiogram is often preferred, but it limits TAPSE and RVFAC assessment due to poor alignment and lack of a dedicated RV view. These parameters are also load dependent, requiring cautious interpretation in variable haemodynamic states. Emerging trends emphasize ventriculo-arterial coupling, integrating RV function and afterload metrics (e.g. TAPSE-to-pulmonary artery systolic pressure [PASP] ratio), which may provide deeper insights into RV adaptation in acute RHF. Nonetheless, echocardiography may not always provide a complete picture. In cases where echocardiographic findings are inconclusive or when there is a strong suspicion of RHF despite normal echocardiographic results, haemodynamic monitoring using a pulmonary artery catheter (PAC) is crucial.[8] The PAC provides critical haemodynamic data essential in the diagnosis and prognosis of RHF. Pulmonary artery pressure (PAP) is the initial parameter to assess, as it provides critical information regarding the presence and severity of pulmonary hypertension (PH), a major cause of RHF. Pulmonary capillary wedge pressure (PCWP) should then be interpreted to distinguish between isolated RHF and biventricular failure. Additional indices include the ratio of RA pressure to PCWP >0.86, which is associated with pathological evidence of RV infarction and increased mortality risk.[9] The pulmonary artery pulsatility index (PAPi)—calculated as (PASP − PA diastolic pressure [PADP])/right atrial pressure (RAP)—quantifies the right ventricle's capacity to generate pulsatile pressure relative to preload. A PAPi value <1.85 serving as a sensitive predictor of RH failure following LVAD implantation and values <1.0 representing a highly sensitive marker of RH failure in the context of AMI[10] (*Table 2*). In addition to these indices, the PAC plays a pivotal role in differentiating pre-capillary from post-capillary PH, thereby enhancing diagnostic precision. Other haemodynamic measures, including cardiac output CO, stroke volume (SV), and mixed venous oxygen saturation (SvO_2), provide vital insights into systemic oxygen delivery and patient prognosis. Decreased CO and SV are strongly associated with poor

Table 2 Haemodynamic cut-offs for pulmonary artery catheter Monitoring in Right heart failure

Parameter	Cut-off value	Clinical significance
Central venous pressure (CVP)	<14 mmHg	Indicates optimal RV preload
Pulmonary capillary wedge pressure (PCWP)	<18 mmHg	Reflects left ventricular preload and pulmonary congestion risk
Pulmonary artery systolic pressure (PASP)	<25 mmHg	Indicates lower PVR and reduced RV afterload
Cardiac index (CI)	>2.2 L/min/m^2	Ensures adequate systemic perfusion
CVP/PCWP ratio	>0.86	Associated with RV AMI and mortality risk
Pulmonary artery pulsatility index (PAPi): (PASP − PADP)/RAP	>1.85 (post-LVAD) >1.0 (AMI)	Reliable marker of RHF in LVAD patients and AMI

AMI, acute myocardial infarction; LVAD, left ventricular assist device; PADP, pulmonary artery diastolic pressure; PASP, pulmonary artery systolic pressure; PVR, pulmonary vascular resistance; RAP, right atrial pressure; RHF, right heart failure; RV, right ventricle.

outcomes in conditions such as PH and chronic thromboembolic hypertension (CTEPH). Similarly, SvO_2 levels <65% serve as a critical guide for therapeutic adjustments aimed at optimizing tissue perfusion and systemic oxygenation during treatment. Moreover, the possibility of using a PAC with RV waveform can provide additional valuable insights for diagnosis.[11] It is important to note, however, that cut-off values for parameters like PAPi may vary across different clinical contexts and patient populations.[12,13] Clinicians should therefore interpret these metrics within the specific clinical scenario to make informed decisions. Elevated central venous pressure (CVP) also plays a central role as the primary determinant of impaired renal and hepatic function in acute RHF, further emphasizing the importance of detailed haemodynamic monitoring.

By understanding the clinical context and systematically using these diagnostic tools, clinicians can diagnose acute RHF early and accurately. This comprehensive approach is crucial for managing patients with MCS, leading to better patient outcomes and improved care management.

Cardiac pharmacological support

The RV, a low-pressure system, is highly sensitive to changes in preload, afterload, and contractility. Initial management focuses on careful volume assessment to prevent both hypovolemia and fluid overload. Reversible increases in afterload—due to hypoxia, acidosis, hypercarbia, or pain—must be addressed. When mechanical ventilation is required, it should be tailored to RV needs with low tidal volumes, low PEEP, and, if possible, spontaneous breathing to reduce pulmonary vascular resistance (PVR).[14]

Pharmacologic support is considered only after these measures fail. Although adrenergic agents may be necessary, epinephrine is generally discouraged, as trials like OptimaCC have linked it to worse outcomes in cardiogenic shock. Instead, epinephrine should be reserved for scenarios where other options are insufficient.[15,16]

Low-dose norepinephrine is preferred to maintain systemic perfusion without notably raising PVR, but higher doses may worsen RV afterload.[4] In such cases, vasopressin can be introduced early to increase systemic vascular tone without exacerbating PH. Dobutamine can improve RV function by enhancing contractility and lowering afterload via β2-mediated vasodilation. However, its pulmonary effects may be unpredictable in patients on chronic beta-blockers, occasionally causing vasoconstriction. Hence, cautious titration and close monitoring are essential.

Milrinone, a phosphodiesterase-3 inhibitor, offers inotropic and vasodilatory benefits without acting on adrenergic receptors, making it favourable for the pulmonary vasculature.[17] Although recent trials in cardiogenic shock did not show outcome differences between milrinone and dobutamine, data are limited in RV failure.[18] Levosimendan, a calcium sensitizer, may help patients without SIRS or high vasopressor needs, improving RV-PA coupling and easing withdrawal from adrenergic support or MCS.[19] Both milrinone and levosimendan are particularly beneficial in patients on chronic beta-blockade due to their non-adrenergic mechanisms. Pulmonary vasodilators can further aid in managing RV dysfunction with high PVR. Inhaled nitric oxide selectively reduces pulmonary pressures without systemic effects. Inhaled iloprost, a prostacyclin analogue, also selectively vasodilates the pulmonary circulation and may enhance gas exchange. Phosphodiesterase-5 inhibitors like sildenafil can further lower PVR, especially in chronic settings or as a bridge to more definitive therapy. These agents are considered when systemic support is insufficient or during weaning from mechanical support.

All these strategies should be exhausted before considering pRVAD placement.

Patient and device selection for percutaneous right mechanical circulatory support

The timing and type of MCS for acute RHF should be determined by evaluating the patient's haemodynamic data, the device's risk-to-benefit ratio, and the healthcare centre's specific capabilities and equipment availability. In all cases, the escalation of either pharmacological and/or mechanical therapies should be promptly discussed with a multidisciplinary shock team (MDT) composed by a heart failure cardiolo-

gist, a cardiothoracic surgeon, an interventional cardiologist, and a Cardiovascular Intensive Care Unit attending physician. Depending on the underlying/precipitating pathology the shock MDT may require additional expertise for specific situations, such as a pulmonary embolism response team (PERT), PH specialists, or specialists in congenital heart disease.[20,21]

A critical component of this evaluation process is phenotyping patients with RHF, which involves assessing haemodynamic profiles, markers of systemic perfusion, end-organ function, and comorbid conditions. Key phenotypic haemodynamic characteristics of RHF include elevated CVP $>$ 15 mmHg, low CI $<$ 2.0 L/min/m^2, and, most of the time, stable or decreased PAP as the failing RV becomes unable to maintain adequate pressure generation. The combination of rising CVP, falling CI, and reduced PAP is a critical indicator of worsening RV dysfunction and impending haemodynamic collapse. Markers of systemic perfusion, including low mixed venous oxygen saturation ($<$60%) and elevated lactate levels ($>$2 mmol/L), indicate compromised oxygen delivery and end-organ perfusion, highlighting the need for prompt initiation or escalation of MCS. The SCAI shock pyramid,[22] originally developed for LV cardiogenic shock, provides a useful framework for assessing shock severity in RV failure. Patients in SCAI stage D (deteriorating) or stage E (extremis) require urgent MCS implantation to reverse haemodynamic collapse, whereas early intervention in SCAI stage C (classic shock) may prevent further decompensation. While precise evidence-based thresholds for RV MCS are lacking, the decision should incorporate the trajectory of clinical decline and response to conservative measures.

Comorbid conditions, such as the presence of LVAD, ARDS, and AMI, significantly impact the management strategy and prognosis. Depending on the patient phenotype, MCS selection may be driven by the need for mono-ventricular or biventricular support to relieve congestion and boost the cardiac output. However, in cases of biventricular failure, particularly when LV support is already in place, the management becomes more complex. In these scenarios, the timing of RV support initiation, how to balance inotropic therapy, and flow optimization between the LV and RV must be carefully considered. LV support (e.g. LVAD) can indirectly help the RV by unloading the LV and reducing the post-capillary component of pulmonary pressures, but this requires precise flow titration and close monitoring of RV function to prevent RV distension and failure. Of particular importance is the understanding of the concomitant degree of respiratory failure and the degree of additional oxygenation requirements. When initiating MCS, the exit strategy (recovery, durable assist device, or transplant) should be planned in advance, when possible, but in emergencies, MCS also acts as a bridge to decision-making and patient evaluation. For patients with irreversible right ventricular dysfunction requiring long-term support, durable RVAD options remain limited. In clinical practice, although there are no durable systems specifically approved for isolated RV support, options such as the Syncardia Total Artificial Heart (TAH) and biventricular assist devices (BiVADs) using LVAD technology (e.g. HeartMate 3 in off-label configurations) can be considered for end-stage RV failure when recovery is not achievable, and transplantation is either contraindicated or delayed.

Device selection should take into consideration: RV preload/afterload, LV function and eventually respiratory support (graphical abstract). Key is the understanding of ventricular interdependency in either the selection of primary device and assessment of RH failure in the presence of LVAD (*Figure 1* and *Table 3*). RV dilation leads to a decoupling of LV/RV interaction, resulting in reduced LV preload and an overall decreased CI.[23] The identification of patients' phenotype and underlying pathophysiology relies on multiparametric approaches which are detailed in the following sections.

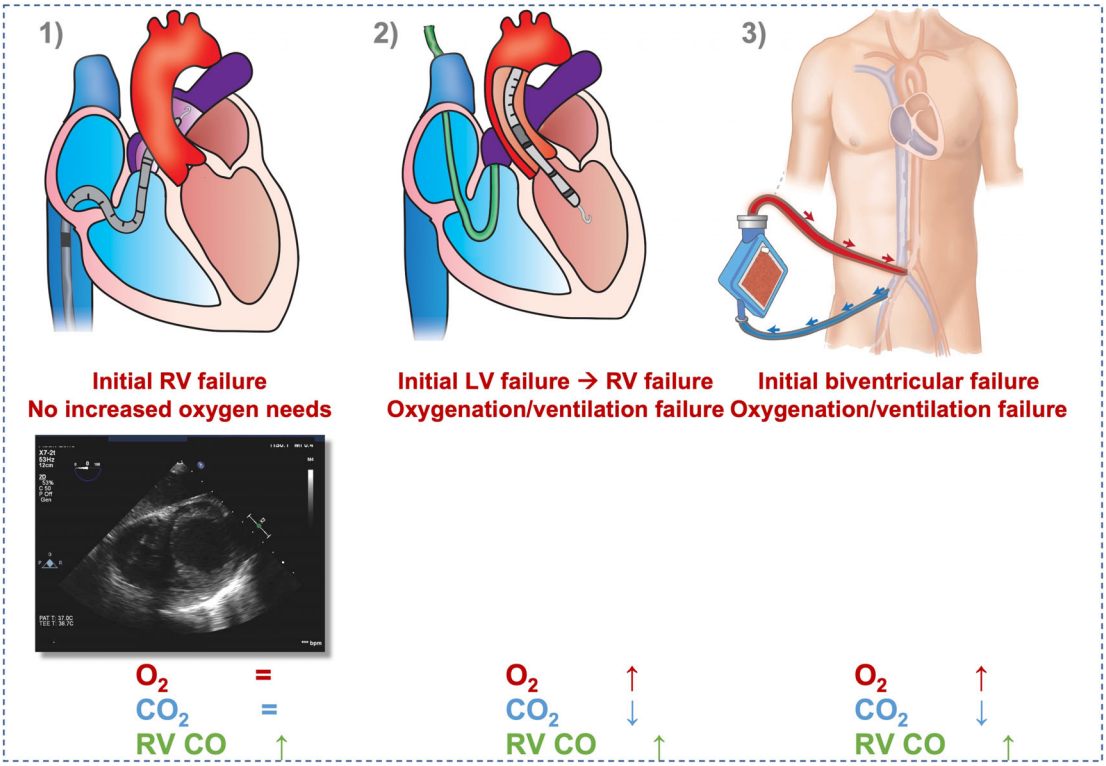
Percutaneous right ventricular mechanical circulatory support

pRVADs can be divided into intracorporeal micro-axial flow pumps (Impella RP and Impella RP Flex—Abiomed Inc, Danvers, MA) and extracorporeal centrifugal flow systems (TandemHeart RVAD/ProtekDuo—TandemLife, Pittsburgh, PA; veno-arterial extracorporeal membrane oxygenation [V-A-ECMO]).[5] Impella RP and TandemHeart (TH) RVAD/Protek Duo are direct RV bypass devices that improve forward pulmonary flow by unloading the RV through peripheral decongestion (*Table 4*). However, if the LV is not able to cope with the increased preload, no CI changes occurs although LV filling pressure may significantly rise (RV–LV uncoupling), leading to pulmonary oedema and eventually the need of LV unloading.

V-A-ECMO is an indirect RV bypass systems that drains the RV by reducing total cardiac preload, instead of directly redirecting blood flow into the PA.

These systems utilize two different configurations: bypass of the RV (e.g. Impella RP and TH-RVAD/ProtekDuo) and bypass of the RV and lung circulation (e.g. V-A-ECMO) (*Table 4*). Each of them has different haemodynamic effects which vary further according to the presence of isolated RHF versus biventricular failure (*Figure 2*). The degree of concomitant LV dysfunction is a major factor modulating the efficacy of pRVAD in augmenting CI. Of note, initially inapparent LV failure may become unmasked with pRVAD-mediated restoration of LV preload. Predicting this scenario can be challenging, but regular echocardiographic evaluations and haemodynamic monitoring using a PAC can be helpful. Indicators include elevations in left atrial pressure (LAP) as estimated by echocardiography and PCWP measured via PAC, alongside evidence of pulmonary congestion on imaging studies.

Figure 1 Potential use of pRVAD in a clinical scenario. Clinical scenarios refer to the clinical cases described in *Table 3*. LV, left ventricle; RV, right ventricle.



In cases of biventricular failure, particularly when LV support is already in place, the management becomes more complex. In these scenarios, the timing of RV support initiation, how to balance inotropic therapy, and flow optimization between the LV and RV must be carefully considered. LV support with an LVAD can reduce LAP and pulmonary congestion, lowering RV afterload, but it may also increase RV preload and cause leftward septal shift, potentially impairing RV function.

In isolated acute RHF, such as RV infarction, pRVAD is often sufficient to bridge patients to recovery. RV bypass based MCS reduce RAP, increase pulmonary artery (PA) pressures, and increase LV preload, with increase of LV filling pressure, resulting in a rise of CI. (*Figure 2* and graphical abstract).

Impella RP (right ventricle bypass device)

The Impella RP is a percutaneous, catheter-based, microaxial-flow pump with flow rates of up to 4 L/min. The Impella RP is composed of a 22-Fr impeller mounted onto an 11-Fr catheter with the inflow positioned at the level of the inferior vena cava (IVC) and the outflow in the PA, bypassing the RV. The device is inserted via a 23-Fr venous peel-away sheath through a single transfemoral venous access. The Impella RP does not allow ambulation of the patient due to the femoral access and is licensed/approved for use for up to 14 days. Recently, Impella RP Flex has received US Food and Drug Administration (FDA) pre-market approval, to treat acute RHF for up to 14 days.[24] Impella RP Flex is implanted through a single venous access via the internal jugular vein and 11 Fr indwelling catheter, which enables patient mobility.

In 2015, the Impella RP became the first pRVAD to be approved by the FDA. The RECOVER RIGHT trial was a prospective, multicentre, randomized study designed to evaluate the feasibility and safety of the Impella RP.[25] In 30 patients with RHF (18 post-LVAD and 12 post-cardiotomy or acute AMI), the device was promptly and safely implanted, with immediate haemodynamic benefit and 73.3% survival at 30 and 180 days. The most common adverse events were bleeding (60%) and haemolysis (13.3%). More recently, a prospective cohort of 60 patients with RHF (31 after LVAD implantation and 29 after cardiotomy or AMI) showed similar results, with rapid haemodynamic improvement and favourable survival (72%) after 30 days or at the time of discharge.[26]

The use of the Impella RP has been reported in multiple scenarios: malignant ventricular arrhythmias, after pulmonary thrombectomy in massive pulmonary embolism, AMI with unsuccessful right coronary artery revascularization or in combination with a LVAD.[27–31]

Table 3 Case vignettes illustrating the importance of device selection in right heart failure

Case presentation	Clinical signs of shock and biomarkers	Echo parameters	PA-catheter parameters	SCAI stage
1 A 68-year-old male admitted with an inferior ST-segment elevation myocardial infarction due to an acute thrombotic occlusion of the proximal RCA. After RCA revascularization, TIMI flow was 3 with persistent thrombotic occlusion of the mid-posterior interventricular branch. Consecutively, the patient underwent to cardiogenic shock	- Wet and cold - Agitation - Anuria - Lactates 5 mmol/L	RV dilatation: 51 mm (mid) TAPSE: 12 mm S'TDI: 7 cm/s sPAP: 40 mmHg Dilated IVC without collapse LVEF: 45%	Missing	C
2. 28-year-old female patient admitted with severe LV failure due to severe viral myocarditis (confirmed on LV biopsy); the patient was intubated due to agitation and supported by a left ventricular micro-axial flow pump in axillary position (Impella™ 5.5) after rising lactate levels under milrinone and noradrenaline	- Cold and wet, extensive rales - Mottled - Agitation - Lactate 4 mmol/L - Loss UO	Initial: • RVOT VTI 19.2 cm; TAPSE 2.3 • LVOT VTI 6.8 cm • LVED After ARDS (on LV Impella): • RVOT VTI 6.2 cm; TAPSE 1.3 • LVOT VTI 9 cm	Initial: • PAPi 1.8 • CI 1.9 • PCPWP 25 mmHg AFTER ARDS (on LV Impella): • PAPi 0.7 • CI 1.7 • PCPWP 16 mmHg	D
3. 60-year-old female patient admitted with a massive pulmonary embolism complicated by cardiogenic shock. After the administration of thrombolysis, the patient underwent to refractory cardiac arrest, so she was intubated and V-A ECMO was placed	- Wet and cold - Lactate 9 mmol/L - Cardiac arrest - Mean arterial pressure 55 mmHg	RV dilatation: 55 mm (mid) TAPSE: 9 mm S'TDI: 3 cm/s Massive TR Dilated IVC without collapse	Absence of pulsatility at the beginning of the V-A ECMO support	E

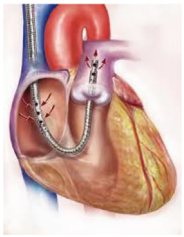
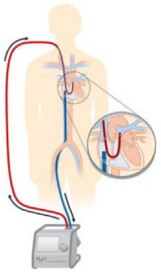
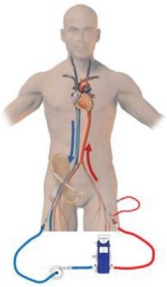
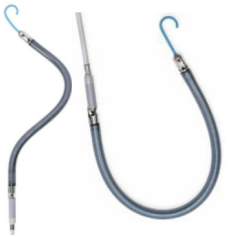
ARDS, acute respiratory distress syndrome; CI, cardiac index; IVC, inferior vena cava; LV, left ventricle; LVED, left ventricular end-diastolic diameter; LVOT, left ventricular outflow tract; PAPi, pulmonary artery pulsatility index; PCWP, pulmonary capillary wedge pressure; RCA, right coronary artery; RVOT, right ventricular outflow tract; SCAI, Society for Cardiovascular Angiography and Interventions; sPAP, systolic pulmonary artery pressure; TAPSE, tricuspid annular plane systolic excursion; VA-ECMO, venoarterial extracorporeal membrane oxygenator; VTI, velocity time integral.

Table 3 (continued)

Case presentation	Oxygenation failure	Device selection	Outcome	Commentary
1 A 68-year-old male admitted with an inferior ST-segment elevation myocardial infarction due to an acute thrombotic occlusion of the proximal RCA. After RCA revascularization, TIMI flow was 3 with persistent thrombotic occlusion of the mid-posterior interventricular branch. Consecutively, the patient underwent to cardiogenic shock	• Positive pressure ventilation with low oxygen need (max Fi02 35%) • Type I failure, no type II failure	• Impella™ RP device	• Isolated RH failure without significant oxygenation failure • Extubated after 4 days • Device removed after 7 days; complete recovery of the RV	• Isolated RV failure with minimal oxygenation failure is an ideal setting for pRVAD use. Early intervention stabilized the patient, leading to a successful recovery
2. 28-year-old female patient admitted with severe LV failure due to severe viral myocarditis (confirmed on LV biopsy); the patient was intubated due to agitation and supported by a left ventricular micro-axial flow pump in axillary position (Impella™ 5.5) after rising lactate levels under milrinone and noradrenaline	• Initially no oxygenation/ ventilation failure. • Evolution to severe ARDS (P/F-ratio 75) and thus: escalation needed • Combined Type I and Type II failure	• Initial left-sided Impella™ 5.5 • Protek Duo™ device for escalation after ARDS/RH failure	• Initial LV failure without oxygenation failure: start Impella™ 5.5 device • Evolution to ARDS and RH failure: escalation by ProtekDuo™ cannula (alternative: VA-ECMO) • ProtekDuo was weaned after 5 days, Impella 5.5 after 12 days; patient had a full recovery	• This case highlights the importance of tailored escalation strategies for combined LV and RV failure. pRVAD + pLVAD provided effective dual unloading and oxygenation
3. 60-year-old female patient admitted with a massive pulmonary embolism complicated by cardiogenic shock. After the administration of thrombolysis, the patient underwent to refractory cardiac arrest, so she was intubated and V-A ECMO was placed	• Positive pressure ventilation with Fi02 100%	• V-A ECMO™	• Initial RH failure complicated by cardiac arrest: V-A ECMO placement to maintain perfusion. After the pulmonary thrombectomy with FlowTriever T24™ the patient gained pulsatility and the V-A ECMO supported the RV dysfunction up to its recovery after 72 h	• Demonstrates the critical role of VA-ECMO in massive PE with cardiogenic shock. Early placement ensured perfusion while definitive therapy (thrombectomy) was performed

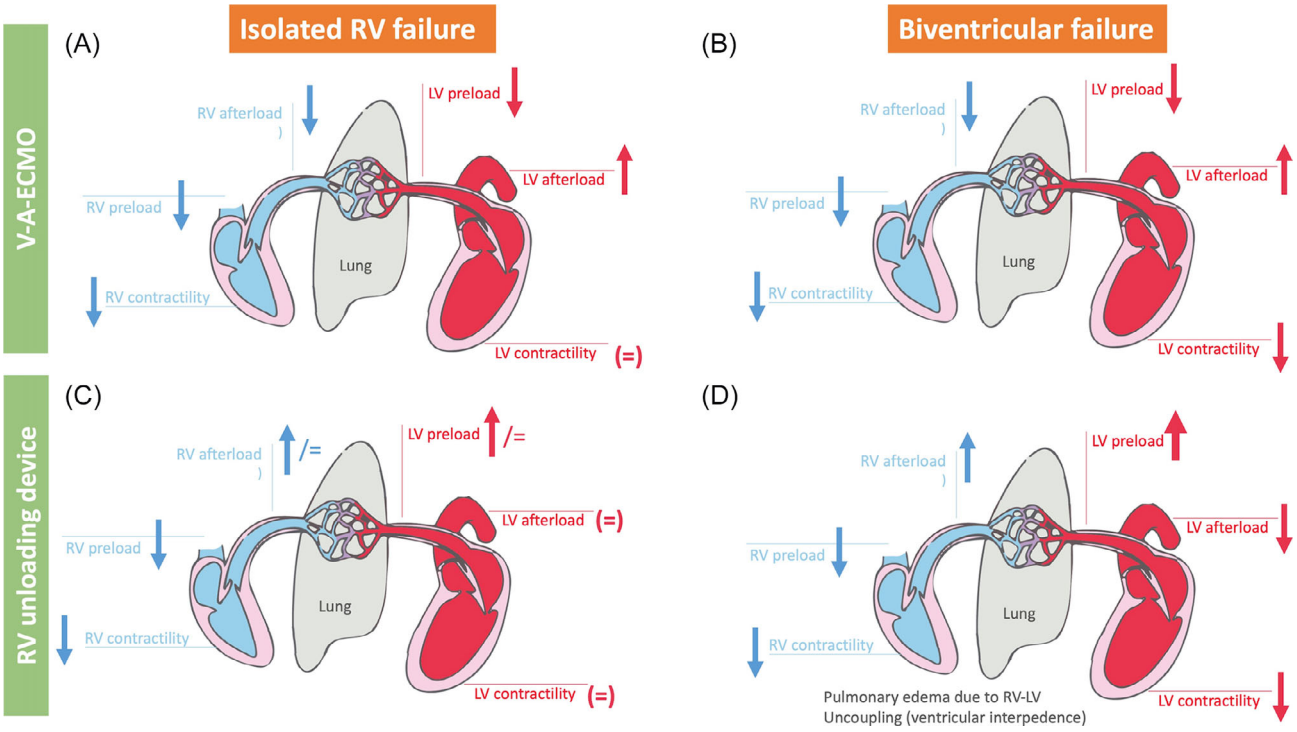
ARDS, acute respiratory distress syndrome; CI, cardiac index; IVC, inferior vena cava; LV, left ventricle; LVED, left ventricular end-diastolic diameter; LVOT, left ventricular outflow tract; PAPi, pulmonary artery pulsatility index; PCWP, pulmonary capillary wedge pressure; RCA, right coronary artery; RVOT, right ventricular outflow tract; SCAI, Society for Cardiovascular Angiography and Interventions; sPAP, systolic pulmonary artery pressure; TAPSE, tricuspid annular plane systolic excursion; VA-ECMO, venoarterial extracorporeal membrane oxygenator; VTI, velocity time integral.

Table 4 Comparison of short-term percutaneous right ventricular mechanical circulatory support devices

Device	Dual-lumen RA-PA cannula (or ProtekDuo)	Dual-lumen RV-PA or RA/RV-PA cannula (or Spectrum)	VPa-ECMO	VA-ECMO	Impella RP/flex
					
Size (outer)	31 or 29 Fr	31, 27, or 24 Fr	15–19-Fr PA line 21–25-Fr vein	15–19-Fr artery 21–25-Fr vein	23 Fr
Pump type	Extracorporeal	Extracorporeal	Extracorporeal	Extracorporeal	Intracorporeal
Oxygenator allowance	Yes	Yes	Yes	Yes	No/yes
Max flow	4.5 L/min	4.5 L/min	7 L/min	7 L/min	4 L/min
Inlet location	Right atrium	Right ventricle	Right atrium	Right atrium	Inferior/superior vena cava
Outlet location	Pulmonary artery	Pulmonary artery	Pulmonary artery	Femoral artery, axillary artery or aorta	Pulmonary artery
Mobilization compatible	Yes	Yes	Yes, with specialized physical therapy	Yes, if outflow axillary artery	No/yes (flex)
Contraindications: Device specific	RA, RV, or PA[a] thrombus, pulmonic valve disease, anatomic abnormalities precluding insertion, PA conduit, severe pulmonary hypertension, inability to tolerate anticoagulation	RA, RV, or PA[a] thrombus, pulmonic valve disease, anatomic abnormalities precluding insertion, PA conduit, severe pulmonary hypertension, inability to tolerate anticoagulation	RA, RV, or PA[a] thrombus, pulmonic valve disease, anatomic abnormalities precluding insertion, PA conduit, severe pulmonary hypertension, inability to tolerate anticoagulation	Aortic dissection, aortic insufficiency, severe peripheral arterial disease	RA, RV, or PA[a] thrombus, pulmonic valve disease, documented DVT or presence of IVC filter, anatomic abnormalities precluding insertion, PA conduit, severe pulmonary hypertension
Contraindications: Patient specific	Evidence of acute neurologic injury, active infection, inability to tolerate anticoagulation	Evidence of acute neurologic injury, active infection, inability to tolerate anticoagulation	Evidence of acute neurologic injury, active infection, inability to tolerate anticoagulation	Inability to tolerate anticoagulation, unrecoverable condition	Evidence of severe end-organ injury, evidence of acute neurologic injury, active infection

ACT, activated clotting time; ASD, atrial septal defect; DVT, deep vein thrombosis; ECMO, extracorporeal membrane oxygenation; Fr, French; IVC, inferior vena cava; PA, pulmonary artery; PFO, patent foramen ovale; PTT, partial thromboplastin time; RA, right atrium; RV, right ventricle; SVC, superior vena cava.
[a]May be considered in highly selective cases where the benefits outweigh the risks.

Figure 2 Haemodynamic effects of pRVADs. LV, left ventricle; RV, right ventricle.



There is a lack of large real-world studies on Impella RP, and further data from larger patient cohorts are urgently needed to validate its clinical use.

TandemHeart/ProtekDuo (right ventricle bypass devices)

TandemHeart-RVAD is an extracorporeal continuous flow centrifugal pump that provides direct RV bypass with flows up to 4 L/min and oxygenation support. Dual-lumen cannulas provide access and return of the blood flow through a single vascular insertion. Depending on the location of the inlet and the output ports, these cannulas can provide right-sided support by bypassing the RV.

Currently, there are two dual-lumen cannulas available that are specifically designed for providing RV support: RA-PA dual-lumen cannula (ProtekDuo) and RA/RV-PA dual-lumen cannula (Spectrum).[32]

Both are flexible, dual-lumen cannulas that require a single right internal jugular vein access point. Blood is removed from the proximal port (either RA or RA/RV) and returned distal from the pulmonic valve bypassing the RV.[33] This configuration allows patient ambulation. It also allows for the insertion of an additional external oxygenator to provide veno-venous extracorporeal oxygenation (V-V-ECMO). In case dual-lumen cannulas are not available, two separate cannulas as RV bypass device can be used (one from internal jugular vein and one from femoral vein).

The first successful percutaneous implantation of a TH-RVAD was reported in 2006.[34] Clinical data demonstrated both the feasibility and effectiveness of the TandemHeart in RHF.[35,36]

In 2014, the THRIVE study (TandemHeart in Right Ventricular Support) retrospectively studied 46 patients receiving a TH-RVAD with percutaneous or surgical implantation.[36] The TH-RVAD was associated with improved haemodynamic status, including increased CI and reduced CVP and PAP, within 48 h. In-hospital mortality was 57%, with the lowest mortality in the setting of AMI, chronic left heart failure, or post-LVAD implantation. Comparably, in a smaller cohort of ProtekDuo-supported patients, the mortality rate was 41%.[37]

Although precise data on the number of cases are not available, the use of the TH-RVAD and ProtekDuo cannula has been reported in several clinical scenarios. Specifically, in the setting of post-cardiotomy acute RHF, TH-RVAD can be used safely and in an easy way to step down from a cardiopulmonary bypass to an isolated RV support, further allowing the weaning of the oxygenator.[38]

Extracorporeal membrane oxygenation

V-A-ECMO provides both biventricular support and oxygenation, and it has been used in the setting of biventricular and

isolated RHF on specific aetiologies.[39] While V-A ECMO rapidly unloads the venous system, increased LV afterload may eventually lead to pulmonary stasis and increased RV afterload. This can be mitigated with adequate inotropic support to maintain low LVEDP and, if needed, venting strategies like intra-aortic balloon pump or Impella, targeting LVEDP around 15 mmHg.[40] In cases of elevated PVR due to PH or CTEPH, V-A-ECMO may be the preferred option to bypass both the RV and pulmonary circulation. This strategy is typically used as a bridge to lung transplantation or pulmonary endarterectomy in specialized centres.[41] In addition, ECMO is often the preferred strategy in RHF or circulatory collapse due to massive pulmonary embolism, given the combined impairment of pulmonary circulation and gas exchange. However, small case series have reported the use of Impella RP and ProtekDuo in this setting.[42]

Transitioning to V-V-ECMO may be considered only in cases of respiratory failure without ongoing RV support requirements. V-V-ECMO offers respiratory support by oxygenation/CO_2 removal by the artificial lung. In patients with severe acute respiratory failure leading to acute cor pulmonale, V-V-ECMO can be an effective strategy to revert the RV dysfunction related to perfusion mismatch (hypoxaemia) and increased afterload by reducing hypercarbia and ventilatory pressures.

A hybrid configuration with an additional venous cannula (V-AV configuration in case of Harlequin syndrome or VV-A configuration for further RV unloading) is generally less favoured and should be reserved for select cases where other strategies fail.[43]

Intensive care unit management of pulmonary right ventricular assist devices

Managing acute RHF in the ICU involves a comprehensive approach that includes early recognition, appropriate use of pRVADs, and continuous monitoring. To provide practical guidance for clinicians, we have summarized key 'tips and tricks' for managing acute RHF using pRVADs. These recommendations are based on clinical experience and available evidence and are designed to help optimize patient outcomes in the setting of acute RHF. The detailed tips are presented in *Table 5*.

Haemodynamic monitoring

PAC allows a comprehensive haemodynamic evaluation of patients with RHF, and it is recommended (class II, level B) according to the International Society for Heart and Lung Transplantation/Heart Failure Society of America Guideline on acute MCS.[44] PAC allows for continuous monitoring and provides detailed haemodynamic data that are crucial for adjusting MCS settings, preventing complications, and guiding timely interventions. Regular assessment is essential to ensure the effectiveness of the MCS and to detect any early signs of complications (*Table 2*). Depending on the cause of RHF, various haemodynamic parameters have been linked to specific outcomes. Any rise in central venous and/or wedge pressures

Table 5 Tips and tricks for managing acute right heart failure with pRVADs

Tips and tricks for managing acute RHF with pRVADs
Early recognition and intervention: Be vigilant for early signs of RV dysfunction, such as rising CVP and decreasing CI. Early identification and timely intervention with pRVAD can significantly improve outcomes.
Optimizing pRVAD position: Proper pRVAD placement is crucial for effective support. Utilize echocardiography to confirm optimal positioning.
Regular haemodynamic monitoring: Continuous monitoring with a PAC allows for real-time adjustments in pRVAD settings. Aim to maintain target haemodynamic parameters: CVP $<$ 14 mmHg, PCWP $<$ 18 mmHg, and CI $>$ 4 L/min.
Echocardiographic assessments: Focus regular evaluations on RV size, function, and interventricular dependence. Key parameters include TAPSE $>$ 14 mm, tricuspid annular peak systolic velocity (S') $>$ 9.5 cm/s, and FAC $>$ 35%.
Managing interventricular dependence: Monitor for changes in interventricular septal position and function. RV dilatation can impact LV filling and function. Adjust pRVAD settings to optimize both RV and LV performance.
RV pulse pressure optimization: Maintain RV pulse pressure through inotropes (e.g. dobutamine, milrinone) or vasodilators (e.g. inhaled nitric oxide, SNP) to reduce RV afterload and improve unloading. Especially when an indirect support strategy is employed.
Pulmonary vascular resistance (PVR) management: Use vasodilators (e.g. inhaled nitric oxide, SNP, PDE5 inhibitors) to lower PVR and enhance RV function.
Weaning strategies: Gradual weaning from pRVAD should be guided by haemodynamic and echocardiographic assessments. Ensure stable haemodynamics and adequate RV function before reducing support.
Anticoagulation management: Maintain appropriate anticoagulation to prevent thromboembolic complications. Regularly monitor coagulation parameters and adjust anticoagulation therapy as needed.
Multidisciplinary approach: Engage a multidisciplinary team, including cardiologists, intensivists, and cardiac surgeons, to optimize patient management and decision-making.

AMI, acute myocardial infarction; CI, cardiac index; CVP, central venous pressure; FAC, fractional area change; LV, left ventricle; LVAD, left ventricular assist device; PAC, pulmonary artery catheter; PDE5, phosphodiesterase-5; pRVAD, percutaneous right ventricular assist devices; PVR, pulmonary vascular resistance; RHF, right heart failure; RV, right ventricle; SNP, sodium nitroprusside; TAPSE, tricuspid annular plane systolic excursion.

during MCS should always prompt a comprehensive evaluation. Although there are no studies that evaluate specific haemodynamic targets prospectively, reasonable targets include central venous pressure (CVP) $<$ 14 mmHg, PCWP $<$ 18 mmHg, and a CI matching the individual perfusion targets, which can be assessed through parameters such as $SvO_2 > 65\%$ and lactate <2 mmol/L.[45] Along with the CVP/PWCP index, PAPi has been demonstrated as reliable marker of RHF in acute myocardial infarction and predictor of RH failure during LVAD.[5]

While continuous monitoring with a PAC is essential during pRVAD therapy, the interpretation of parameters such as PAPi may be unreliable due to altered haemodynamics. Instead, CVP remains particularly useful in isolated RHF, while CVP combined with PCWP provides valuable insights in biventricular failure. These parameters are most informative during the weaning process, where trends guide the tapering of support.

Thermodilution measurements of CI may be also unreliable due to altered haemodynamics. Indeed, during ECMO support, several challenges arise that can affect haemodynamic measurements. In V-V-ECMO, blood recirculation can cause errors in CI measurements via thermodilution, while in V-A-ECMO, retrograde flow in the aorta can distort the thermal indicator distribution.[46,47] For this reason, CI via thermodilution should be interpreted with caution due to ECMO influences. For accurate evaluation of cardiac function and estimation of CI and stroke volume (SV), echocardiography is recommended, while SvO_2 provides insights into oxygen balance. Baseline measurements should be established before pRVAD, and it is crucial to correlate PAC data with clinical findings and monitor trends rather than relying on single values.

In the context of an indirect strategy (e.g. V-A-ECMO), maintaining RV pulse pressure is crucial for optimal RV function. This can be achieved by careful management of preload and afterload, as well as targeted therapies to reduce PVR. Effective PVR management can include vasodilators, optimizing fluid balance, and ensuring adequate oxygenation. These interventions should be employed before weaning begins and adjusted throughout the process to ensure the RV remains stable as mechanical support is reduced.

Advanced techniques such as pressure–volume (P-V) loops are highly informative, their routine clinical use remains limited due to technical and interpretative challenges, though future developments may enhance their applicability.

Echocardiography

Echocardiography is an essential tool for device selection, positioning, monitoring, and weaning. Echocardiographic interpretation begins with understanding the phenotype of cardiac failure (mono- vs. biventricular) and MCS configuration (VA-ECMO vs. RA-PA devices). Multiparametric assessment interpreting haemodynamics alongside echocardiography is the first step to rule out pathophysiological conditions that may change the indication and/or affect the function of an RVAD (i.e. pericardial diseases, including causes of constrictive or restrictive physiology). Determining the optimal cannula position should be judged also with echocardiography (especially in RA-PA configuration), as no accurate x-ray data regarding the cannula direction and interference with the RV valve apparatus are validated. Loading conditions affect ventricular performance considerably; the unloaded ventricle may appear contractile, but the degree of dysfunction only becomes clear after reloading.[48] During RA/RV-PA-unloading, the RV free wall can be splinted, giving the appearance of RV impairment on echocardiography and potentially leading to delays in weaning. Interventricular septal contribution to RV function largely impacts on efficiency[1] and the alteration of interventricular dependence (RV dilatation) during weaning, which is highly predictive of failure to wean in V-A ECMO patients.[49] Longitudinal function (assessed by TAPSE and S' at tissue Doppler imaging) is the most commonly used parameter although it may not always represent global RV function.[50] Conversely, fractional area change offers a more global evaluation but suffers from greater interobserver variability. RV and pulmonary haemodynamics (including CI and cardiac power, stroke volume, and right heart pressures) should be charted when possible (i.e. ECMO) and ideally interpreted with PA-catheter measurements in parallel. Echo assessment of tricuspid regurgitation (TR) may be unreliable due to cannula configuration and RV function. Additionally, TR was not associated with weaning outcome in V-A ECMO. No data on TAPSE/sPAP are available to further characterize RHF in acute setting and/or predict weaning yet. Development of restrictive RV physiology with diastolic antegrade flow across the pulmonary valve is an early sign of ventricular dysfunction.[51]

In conclusion, regular echocardiographic evaluations should focus on RV size, function, and interventricular dependence. Monitoring these parameters helps in adjusting the MCS settings to optimize haemodynamic support and prevent complications. Load-dependent parameters such as TAPSE and FAC may not reliably represent intrinsic RV function, and their interpretation should be adjusted accordingly. Instead, serial assessments during the weaning process provide the most reliable insights into RV recovery and readiness for tapering support.

Pharmacological support

Anticoagulation

pRVAD are prone to thrombotic complications due to the low flow and necessitate proper anticoagulation management, although they are frequently managed safely without antico-

agulation, in particular where no oxygenator is required.[10] The major bleeding rate in the RECOVER-RIGHT trial was up to 60%,[25] underpinning the difficult balance between bleeding and thrombosis.

Heparin, due to its ideal profile for supporting critically ill patients in the intensive care unit (parenteral administration, short-acting, readily reversible, low cost, low renal excretion), is the most often used anticoagulant worldwide. Nevertheless, other anticoagulants (e.g. direct thrombin inhibitors, anticoagulant device coatings, etc.) are available or under development.[52] Presently, therapeutic UFH anticoagulation is the preferred therapy of choice for many centers, although studies assessing the ideal UFH level are lacking.[53] To avoid bleeding complications in this precarious patient population, a standardized anticoagulation approach should be strongly considered. Recently, an anti-Xa guided UFH-titration protocol with APTT measurements in parallel has been proposed.[54]

Antiplatelet therapy

Certain patients with acute RHF will have an additional indication for mono or dual antiplatelet therapy because of PCI with stent implantation. Here, UFH should be combined with low-dose aspirin and/or P2Y12 inhibitors depending on the individual bleeding risk of the patient.[3] However, it is important to note that data regarding impaired absorption of oral agents such as ticagrelor under mechanical ventilation remain limited and inconclusive.[55] In critically ill patients, the IV antiplatelet agent cangrelor—when available—offers multiple advantages including rapid onset of action, rapid return of platelet function after cessation, and ease of administration in intubated patients mostly affected by impaired gastrointestinal absorption.[56] However, its large volume requirements for dilution can pose challenges in managing patients with RV failure.

Weaning or device escalation

The decision to initiate weaning from pRVAD should prioritize signs of end-organ function recovery, such as improved renal output, normalization of lactate, and stabilization of SvO_2.

Successful weaning, defined as removal from RV support and survival, is reported in 20–75% of patients,[57] depending on aetiology and device used.[58,59] Currently, there are no validated weaning protocols for acute RV mechanical support, and evidence is based on local algorithms or experience. Regular assessment of CVP, PAPi, and overall haemodynamic trends is crucial for guiding weaning decisions. CVP should ideally remain below 14 mmHg during the weaning process, as a rise above 15 mmHg may indicate failure and necessitate reassessment. Additionally, echocardiography should be employed to evaluate RV size, function, and overall performance. Trends in haemodynamic data, rather than absolute values, are important. The approach to assessing RV recovery differs significantly depending on whether the failure is isolated or secondary to biventricular failure.

Generally, at pRVAD support reduction, the improvement of RV systolic function without size increase, improvement of PAPi and stability of CVP, and systemic haemodynamic with low or no pharmacological support along with gas exchange improvement are favourable signs of successful weaning (*Figure 3*).[25]

While the criteria outlined in *Figure 3* provide a structured approach to weaning, it is important to recognize that not all patients will meet every criterion. In such cases, clinical judgement is essential to determine the appropriate timing and strategy for weaning.

In cases of biventricular support, weaning requires balancing LV and RV support to maintain stability. Weaning typically begins with the ventricle showing greater recovery, allowing the other to adapt. Synchronized recovery of both ventricles is essential, and adjusting the flows between support devices is vital to prevent overload, particularly during the early stages of weaning.

For indirect RV offloading, such as with LV support, careful reloading of the RV is crucial to avoid overloading.

The use of inotropes differs depending on whether RHF is isolated or part of biventricular failure. In isolated cases, early weaning from inotropes is typically pursued as the RV recovers, whereas in biventricular failure, prolonged inotropic support may be necessary to balance flows and prevent RV overload during weaning.

If a weaning trial is unsuccessful, the cause of weaning failure has to be identified and addressed before a new weaning trial can be attempted (usually after 24 h).

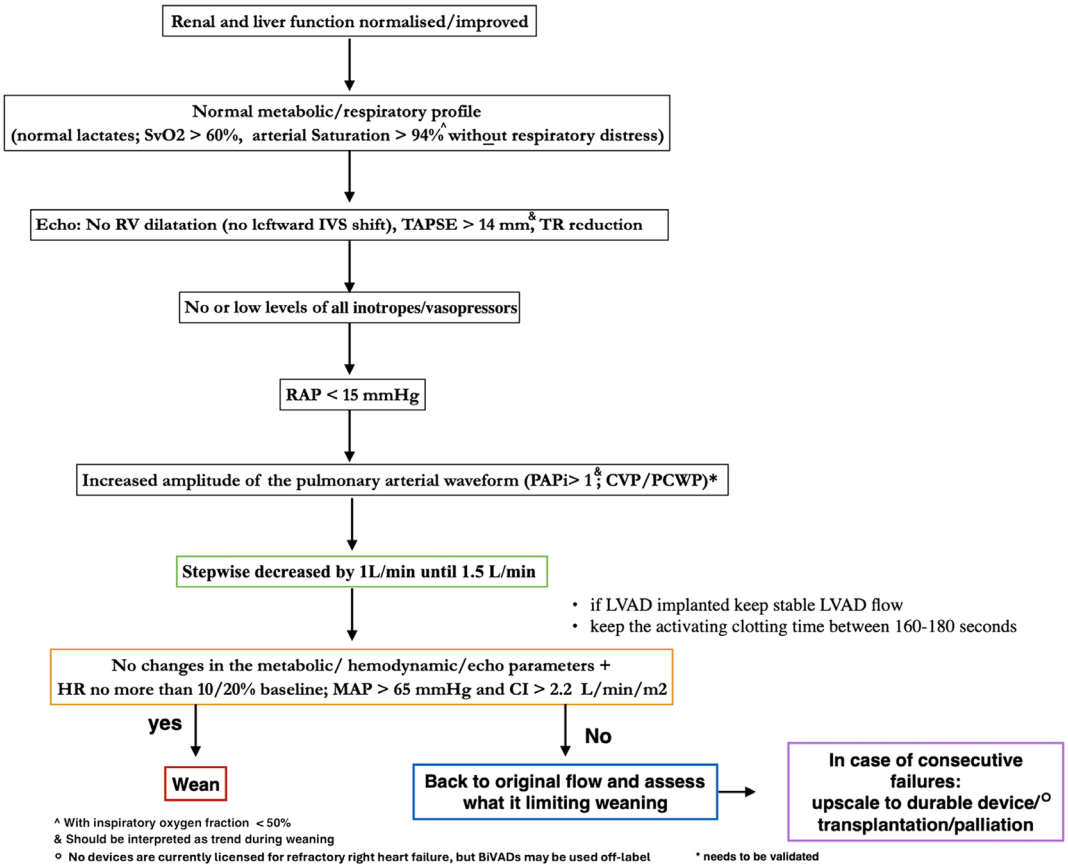
In cases where RV recovery is not achieved despite optimized percutaneous support and medical therapy, escalation to long-term MCS or heart transplantation should be considered. The decision must be individualized based on the underlying aetiology of RHF. In patients with acute isolated RV failure, temporary support may be sufficient to bridge recovery. However, if recovery is incomplete despite optimized medical therapy and pRVAD support, transitioning to durable BiVADs (e.g. HeartMate 3 in off-label configurations) or a TAH (e.g. Syncardia Total Artificial Heart) may be necessary. Early consideration of heart transplantation is particularly important in patients with irreversible RV dysfunction and no evidence of recovery, especially in those with biventricular failure. By evaluating transplant candidacy early, the risk of complications from prolonged mechanical support can be minimized.[60]

A suggested weaning protocol is described in *Figure 3*.

Complications

MCS device-related complications are significant. Complications are related to insertion (e.g. vascular access complica-

Figure 3 Suggested weaning protocol for pRVAD. CI, cardiac index; CVP, central venous pressure; HR, heart rate; IVS, interventricular septum; LVAD, left ventricular assist device; MAP, mean arterial pressure; PAPi, pulmonary artery pulsatility index; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure; RV, right ventricle; TAPSE, tricuspid annular plane systolic excursion; TR, tricuspid regurgitation.



tions, cardiac perforation), the device itself (e.g. dislocation, haemolysis, or valvular damage) or alteration of homeostasis/organ function. Among the most critical complications is RV perforation, which can occur during device implantation or because of mechanical interaction between the device and the fragile RV wall. This condition requires immediate recognition and intervention, typically necessitating surgical repair or percutaneous management depending on the severity and clinical context.

Severe tricuspid regurgitation may occur due to mechanical interaction with devices such as Impella, ProtekDuo, or Spectrum, potentially impairing right ventricular function and hindering successful weaning.

Other potential complications include thromboembolism, which can lead to device malfunction or systemic embolization, and haemolysis, often due to high shear forces within the device. Residual thrombus or clot formation after device removal is another significant concern, potentially necessitating aspiration or further intervention. Additionally, clinicians must be vigilant for device-related infections, particularly in patients with prolonged device use, and for mechanical issues such as device malposition or migration, which can significantly impair function and lead to haemodynamic instability. Signs of device malfunction may include unexplained changes in haemodynamic parameters such as decreased CI, increased CVP, or worsening organ function despite stable medical therapy. Alarm notifications from the device should prompt immediate investigation. In case of suspected malfunction, clinicians should initiate a diagnostic evaluation, including imaging, device alarm checks, power consumption assessment, and haemodynamic response. Adjustments to anticoagulation therapy may be necessary for thrombus formation. Involvement of the multidisciplinary team is crucial for troubleshooting, recalibration, or repositioning of the device. Device replacement should be considered when malfunction cannot be corrected or in cases of ongoing haemodynamic instability, such as irreversible pump failure, significant thrombosis, or cannula kinking. The decision to replace the device must balance procedural risk with the patient's need for continued circulatory support.

Bleeding and thrombotic complications are the far most frequent complications and are related to therapeutic

anticoagulation, consumptive coagulopathy, and large-bore vascular cannulation.[10] The major bleeding rate in the RECOVER-RIGHT trial (specifically looking at the Impella RP) was up to 60%,[25] again underpinning the difficult balance between bleeding and thrombosis. Patients treated with MCS are at risk of high shear-induced haemolysis as erythrocytes pass through the pump. The main consequence of haemolysis is anaemia, plasma-free haemoglobin-induced thrombosis, and kidney failure.[61] However, the direct impact of haemolysis to excess mortality in MCS remains unclear.[62] Therefore, a standardized anticoagulation protocol should strongly be considered to improve bleeding outcomes.[54]

More specific complications related to V-A-ECMO include differential hypoxaemia (north–south syndrome)[63] and limb ischaemia.

Future directions and conclusions

Acute RHF is a difficult diagnostic and therapeutic challenge. Prompt aetiology diagnosis, device selection, and timely implant are crucial to successfully treat a failing RV refractory to maximal medical therapy. Consistent data on reliable and reproducible parameters for RV evaluation in the context of mono-/biventricular cardiogenic shock, predictors of RHF in LV support, and timing for exit strategy are lacking. Specific studies on device configuration and related treatment to improve organ related injuries, representing the commonest reason of mortality, also need to be implemented. Consequently, adequately powered randomized clinical trials and large multicentre registries are needed to better investigate pRVADs' efficacy/safety and patient selection criteria.

The development of durable RVADs remains a priority to address the lack of long-term support options for patients with refractory RHF, particularly as a bridge to transplantation or destination therapy.

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References

1. Konstam MA, Kiernan MS, Bernstein D, Bozkurt B, Jacob M, Kapur NK, *et al*. Evaluation and management of right-sided heart failure: a scientific statement from the American Heart Association. *Circulation* 2018;**137**:e578-e622. doi:10.1161/CIR.0000000000000560
2. Houston BA, Brittain EL, Tedford RJ. Right ventricular failure. *N Engl J Med* 2023;**388**:1111-1125. doi:10.1056/NEJMra2207410
3. Chieffo A, Dudek D, Hassager C, Combes A, Gramegna M, Halvorsen S, *et al*. Joint EAPCI/ACVC expert consensus document on percutaneous ventricular assist devices. *EuroIntervention Europa Group* 2021;**17**:E274-E286. doi:10.4244/EIJY21M05_01
4. Harjola VP, Mebazaa A, Čelutkiene J, Bettex D, Bueno H, Chioncel O, *et al*. Contemporary management of acute right ventricular failure: a statement from the Heart Failure Association and the Working Group on Pulmonary Circulation and Right Ventricular Function of the European Society of Cardiology. *Eur J Heart Fail* 2016;**18**:226-241. doi:10.1002/ejhf.478
5. Kapur NK, Esposito ML, Bader Y, Morine KJ, Kiernan MS, Pham DT, *et al*. Mechanical circulatory support devices for acute right ventricular failure. *Circulation* 2017;**136**:314-326. doi:10.1161/CIRCULATIONAHA.116.025290
6. Kanwar MK, Everett KD, Gulati G, Brener MI, Kapur NK. Epidemiology and management of right ventricular-predominant heart failure and shock in the cardiac intensive care unit. *Eur Heart J Acute Cardiovasc Care* 2022;**11**:584-594. doi:10.1093/ehjacc/zuac063
7. Price S, Platz E, Cullen L, Tavazzi G, Christ M, Cowie MR, *et al*. Expert consensus document: echocardiography and lung ultrasonography for the assessment and management of acute heart failure. *Nat Rev Cardiol* 2017;**14**:427-440. doi:10.1038/nrcardio.2017.56

8. Garan AR, Kanwar M, Thayer KL, Whitehead E, Zweck E, Hernandez-Montfort J, *et al*. Complete hemodynamic profiling with pulmonary artery catheters in cardiogenic shock is associated with lower in-hospital mortality. *JACC Heart Fail* 2020;**8**:903-913. doi:10.1016/j.jchf.2020.08.012
9. Lopez-Sendon J, Coma-Canella I, Gamallo C. Sensitivity and specificity of hemodynamic criteria in the diagnosis of acute right ventricular infarction. *Circulation* 1981;**64**:515-525. doi:10.1161/01.cir.64.3.515
10. Balthazar T, Vandenbriele C, Verbrugge FH, Den UC, Engström A, Janssens S, *et al*. Managing patients with short-term mechanical circulatory support: JACC review topic of the week. *J Am Coll Cardiol* 2021;**77**:1243-1256. doi:10.1016/j.jacc.2020.12.054
11. Couture EJ, Grønlykke L, Denault AY. New developments in the understanding of right ventricular function in acute care. *Curr Opin Crit Care* 2022;**28**:331-339. doi:10.1097/MCC.0000000000000946
12. Lim HS, Gustafsson F. Pulmonary artery pulsatility index: physiological basis and clinical application. *Eur J Heart Fail* 2020;**22**:32-38. doi:10.1002/ejhf.1679
13. Yim IHW, Khan-Kheil AM, Drury NE, Lim HS. A systematic review and physiology of pulmonary artery pulsatility index in left ventricular assist device therapy. *Interdiscip Cardiovasc Thorac Surg* 2023;**36**:ivad068. doi:10.1093/icvts/ivad068
14. Dayer N, Ltaief Z, Liaudet L, Lechartier B, Aubert JD, Yerly P. Pressure overload and right ventricular failure: from pathophysiology to treatment. *J Clin Med* 2023;**12**:4722. doi:10.3390/jcm12144722
15. Levy B, Clere-Jehl R, Legras A, Morichau-Beauchant T, Leone M, Frederique G, *et al*. Epinephrine versus norepinephrine for cardiogenic shock after acute myocardial infarction. *J Am Coll Cardiol* 2018;**72**:173-182. doi:10.1016/j.jacc.2018.04.051
16. Riccardi M, Pagnesi M, Chioncel O, Mebazaa A, Cotter G, Gustafsson F, *et al*. Medical therapy of cardiogenic shock: contemporary use of inotropes and vasopressors. *Eur J Heart Fail* 2024;**26**:411-431. doi:10.1002/ejhf.3162
17. Takeda K, Takayama H, Colombo PC, Yuzefpolskaya M, Fukuhara S, Han J, *et al*. Incidence and clinical significance of late right heart failure during continuous-flow left ventricular assist device support. *J Heart Lung Transplant* 2015;**34**:1024-1032. doi:10.1016/j.healun.2015.03.011
18. Mathew R, Di SP, Jung RG, Marbach JA, Hutson J, Simard T, *et al*. Milrinone as compared with dobutamine in the treatment of cardiogenic shock. *N Engl J Med Massachusetts Medical Society* 2021;**385**:516-525. doi:10.1056/NEJMoa2026845
19. Bouchez S, Fedele F, Giannakoulas G, Gustafsson F, Harjola VP, Karason K, *et al*. Levosimendan in acute and advanced heart failure: an expert perspective on posology and therapeutic application. *Cardiovasc Drugs Ther* 2018;**32**:617-624. doi:10.1007/s10557-018-6838-2
20. Saif K, Kevane B, Áinle FN, Rosovsky RP. *The role of the PERT team in 2021*. Vol. **6**. Thromb Updat Elsevier; 2022:100092.
21. Taleb I, Koliopoulou AG, Tandar A, Mckellar SH, Tonna JE, Nativi-Nicolau J, *et al*. Shock team approach in refractory cardiogenic shock requiring short-term mechanical circulatory support: a proof of concept. *Circulation Lippincott Williams and Wilkins* 2019;**140**:98-100. doi:10.1161/CIRCULATIONAHA.119.040654
22. Naidu SS, Baran DA, Jentzer JC, Hollenberg SM, van Diepen S, Basir MB, *et al*. SCAI SHOCK stage classification expert consensus update: a review and incorporation of validation studies: this statement was endorsed by the American College of Cardiology (ACC), American College of Emergency Physicians (ACEP), American Heart Association (AHA), European Society of Cardiology (ESC) Association for Acute Cardiovascular Care (ACVC), International Society for Heart and Lung Transplantation (ISHLT), Society of Critical Care Medicine (SCCM), and Society of Thoracic Surgeons (STS) in. *J Am Coll Cardiol* 2022;**79**:933-946. doi:10.1016/j.jacc.2022.01.018
23. Naeije R, Badagliacca R. The overloaded right heart and ventricular interdependence. *Cardiovasc Res* 2017;**113**:1474-1485. doi:10.1093/cvr/cvx160
24. Han JJ, Impella RP. Flex with SmartAssist receives FDA pre-market approval. *Artif Organs* 2023;**47**:10-11. doi:10.1111/aor.14461
25. Anderson MB, Goldstein J, Milano C, Morris LD, Kormos RL, Bhama J, *et al*. Benefits of a novel percutaneous ventricular assist device for right heart failure: the prospective RECOVER RIGHT study of the Impella RP device. *J Hear Lung Transplant (Elsevier)* 2015;**34**:1549-1560. doi:10.1016/j.healun.2015.08.018
26. Anderson M, Morris DL, Tang D, Batsides G, Kirtane A, Hanson I, *et al*. Outcomes of patients with right ventricular failure requiring short-term hemodynamic support with the Impella RP device. *J Hear Lung Transplant (Elsevier USA)* 2018;**37**:1448-1458. doi:10.1016/j.healun.2018.08.001
27. Heinzmann D, Schibilsky D, Gramlich M, Klingel K, Kandolf R, Rosenberger P, *et al*. Ablation of an electrical storm in a patient with giant cell myocarditis using continuous flow left ventricular assist device and percutaneous right ventricular assist device. *Int J Cardiol (Elsevier Ireland Ltd)* 2016;**209**:84-86. doi:10.1016/j.ijcard.2016.02.060
28. Gramegna M, Beneduce A, Bertoldi LF, Pagnesi M, Marini C, Pazzanese V, *et al*. Impella RP support in refractory right ventricular failure complicating acute myocardial infarction with unsuccessful right coronary artery revascularization. *Int J Cardiol* 2020;**302**:135-137. doi:10.1016/j.ijcard.2019.12.024
29. Botti G, Gramegna M, Burzotta F, Masiero G, Briguori C, Trani C, *et al*. Impella RP for patients with acute right ventricular failure and cardiogenic shock: a subanalysis from the IMP-IT registry. *J Pers Med* 2022;**12**:1481. doi:10.3390/jpm12091481
30. Chieffo A, Ancona MB, Burzotta F, Pazzanese V, Briguori C, Trani C, *et al*. Observational multicenter registry of patients treated with IMPella mechanical circulatory support device in ITaly: the IMP-IT registry. *EuroIntervention* 2019;**15**:e1343-e1350. doi:10.4244/EIJ-D-19-00428
31. Agarwal S, Saum J, Chanamalou S, Cole W, Patel SM. When Harry met Sally: single-session INARI FlowTriever and Impella RP. *J Cardiol Cases* 2020;**23**:57-60. doi:10.1016/j.jccase.2020.10.002
32. Usman AA, Spelde AE, Olia SE, Cevasco M, Bermudez C, Haddle J, *et al*. First-in-man successful use of the SPECTRUM percutaneous dual-stage right ventricle and right atrium to pulmonary artery ventricular assist device. *J Card Surg* 2022;**37**:3403-3407. doi:10.1111/jocs.16753
33. Oliveros E, Collado FM, Poulin MF, Seder CW, March R, Kavinsky CJ. Percutaneous right ventricular assist device using the TandemHeart ProtekDuo: real-world experience. *J Invasive Cardiol* 2021;**33**:E407-E411. doi:10.25270/jic/20.00490
34. Atiemo AD, Conte JV, Heldman AW. Resuscitation and recovery from acute right ventricular failure using a percutaneous right ventricular assist device. *Catheter Cardiovasc Interv* 2006;**68**:78-82. doi:10.1002/ccd.20691
35. Kapur NK, Paruchuri V, Korabathina R, Al-Mohammdi R, Mudd JO, Prutkin J, *et al*. *Effects of a percutaneous mechanical circulatory support device for medically refractory right ventricular failure*. Vol. **30**. HEALUN (Elsevier Inc); 2011:1360-1367. doi:10.1016/j.healun.2011.07.005
36. Kapur NK, Paruchuri V, Jagannathan A, Steinberg D, Chakrabarti AJ, Pinto D, *et al*. Mechanical circulatory support for right ventricular failure: the TandemHeart in right VEntricular support (THRIVE). *JACC Hear Fail* 2013;**1**:127-134. doi:10.1016/j.jchf.2013.01.007
37. Ravichandran AK, Baran DA, Stelling K, Cowger JA, Salerno CT. Outcomes with the tandem Protek duo dual-lumen percutaneous right ventricular assist device. *ASAIO J* 2018;**64**:570-572. doi:10.1097/MAT.0000000000000709
38. Lang SA, O'Neill B, Waterworth P, Bilal H. Can the temporary use of right ventricular assist devices bridge patients with acute right ventricular failure after cardiac surgery to recovery? *Interact*

Cardiovasc Thorac Surg 2014;**18**:499-510. doi:10.1093/icvts/ivt472
39. Guglin M, Zucker MJ, Bazan VM, Bozkurt B, El Banayosy A, Estep JD, *et al*. Venoarterial ECMO for adults: JACC scientific expert panel. *J Am Coll Cardiol (Elsevier USA)* 2019;**73**:698-716. doi:10.1016/j.jacc.2018.11.038
40. Petroni T, Harrois A, Amour J, Lebreton G, Brechot N, Tanaka S, *et al*. Intra-aortic balloon pump effects on macrocirculation and microcirculation in cardiogenic shock patients supported by venoarterial extracorporeal membrane oxygenation*. *Crit Care Med* 2014;**42**:2075-2082. doi:10.1097/CCM.0000000000000410
41. Savale L, Benazzo A, Corris P, Keshavjee S, Levine DJ, Mercier O, *et al*. Transplantation, bridging, and support technologies in pulmonary hypertension. *Eur Respir J* 2024;2401193. doi:10.1183/13993003.01193-2024
42. Abdulaziz S, Kakar V, Kumar PG, Hassan IF, Combes A, Brodie D, *et al*. Mechanical circulatory support for massive pulmonary embolism. *J Am Heart Assoc* 2025;**14**:e036101. doi:10.1161/JAHA.124.036101
43. Grant C, Richards JB, Frakes M, Cohen J, Wilcox SR. ECMO and right ventricular failure: review of the literature. *J Intensive Care Med* 2021;**36**:352-360. doi:10.1177/0885066619900503
44. Bernhardt AM, Copeland H, Deswal A, Gluck J, Givertz MM. The International Society for Heart and Lung Transplantation/Heart Failure Society of America guideline on acute mechanical circulatory support. *J Heart Lung Transplant* 2023;**42**:e1-e64. doi:10.1016/j.healun.2022.10.028
45. Mathew R, Fernando SM, Hu K, Parlow S, Di SP, Brodie D, *et al*. Optimal perfusion targets in cardiogenic shock. *JACC Adv Elsevier BV* 2022;**1**:100034. doi:10.1016/j.jacadv.2022.100034
46. Haller M, Zöllner C, Manert W, Briegel J, Kilger E, Polasek J, *et al*. Thermodilution cardiac output may be incorrect in patients on venovenous extracorporeal lung assist. *Am J Respir Crit Care Med* 1995;**152**:1812-1817. doi:10.1164/ajrccm.152.6.8520741
47. Norris SL, King EG, Grace M, Weir B. Thermodilution cardiac output--an in vitro model of low flow states. *Crit Care Med* 1986;**14**:57-59. doi:10.1097/00003246-198601000-00013
48. Combes A, Price S, Slutsky AS, Brodie D. Temporary circulatory support for cardiogenic shock. *Lancet (Publishing Group)* 2020;**396**:199-212. doi:10.1016/S0140-6736(20)31047-3
49. Aissaoui N, Caudron J, Leprince P, Fagon JY, Lebreton G, Combes A, *et al*. Right-left ventricular interdependence: a promising predictor of successful extracorporeal membrane oxygenation (ECMO) weaning after assistance for refractory cardiogenic shock. *Intensive Care Med* 2017;**43**:592-594. doi:10.1007/s00134-016-4657-0
50. Bleakley C, Singh S, Garfield B, Morosin M, Surkova E, Mandalia MS, *et al*. Right ventricular dysfunction in critically ill COVID-19 ARDS. *Int J Cardiol* 2021;**327**:251-258. doi:10.1016/j.ijcard.2020.11.043
51. Tavazzi G, Bergsland N, Alcada J, Price S. Early signs of right ventricular systolic and diastolic dysfunction in acute severe respiratory failure: the importance of diastolic restrictive pattern. *Eur Heart J Acute Cardiovasc Care* 2020;**9**:649-656. doi:10.1177/2048872619883399
52. Vandenbriele C, Vanassche T, Price S. Why we need safer anticoagulant strategies for patients on short-term percutaneous mechanical circulatory support. *Intensive Care Med* 2020;**46**:771-774. doi:10.1007/s00134-019-05897-3
53. Kanji R, Vandenbriele C, Arachchillage DRJ, Price S, Gorog DA. Optimal tests to minimise bleeding and ischaemic complications in patients on extracorporeal membrane oxygenation. *Thromb Haemost* 2021;**122**:480-491. doi:10.1055/a-1508-8230
54. Vandenbriele C, Arachchillage DJ, Frederiks P, Giustino G, Gorog DA, Gramegna M, *et al*. Anticoagulation for percutaneous ventricular assist device-supported cardiogenic shock: JACC review topic of the week. *J Am Coll Cardiol* 2022;**79**:1949-1962. doi:10.1016/j.jacc.2022.02.052
55. Kanic V, Kompara G, Suran D. GP IIb/IIIa receptor inhibitors in mechanically ventilated patients with cardiogenic shock due to myocardial infarction in the era of potent P2Y12 receptor antagonists. *J Clin Med MDPI* 2022;**11**:7426. doi:10.3390/jcm11247426
56. Bhatt DL, Stone GW, Mahaffey KW, Gibson CM, Steg PG, Hamm CW, *et al*. Effect of platelet inhibition with cangrelor during PCI on ischemic events. *N Engl J Med* 2013;**368**:1303-1313. doi:10.1056/NEJMoa1300815
57. Cheung AW, White CW, Davis MK, Freed DH. Short-term mechanical circulatory support for recovery from acute right ventricular failure: clinical outcomes. *J Hear Lung Transplant (Elsevier)* 2014;**33**:794-799. doi:10.1016/j.healun.2014.02.028
58. Lampert BC, Teuteberg JJ. Right ventricular failure after left ventricular assist devices. *J Heart Lung Transplant* 2015;**34**:1123-1130. doi:10.1016/j.healun.2015.06.015
59. Kremer J, Farag M, Brcic A, Zubarevich A, Schamroth J, Kreusser MM, *et al*. Temporary right ventricular circulatory support following right ventricular infarction: results of a groin-free approach. *ESC Hear Fail (Wiley-Blackwell)* 2020;**7**:2853-2861. doi:10.1002/ehf2.12888
60. Mulaikal TA, Nakagawa S, Prager KM. Extracorporeal membrane oxygenation bridge to no recovery. *Circulation* 2019;**139**:428-430. doi:10.1161/CIRCULATIONAHA.118.036304
61. Loscalzo J, Editor G. Nitric oxide insufficiency, platelet activation, and arterial thrombosis. *Circ Res (Lippincott Williams & Wilkins)* 2001;**88**:756-762. doi:10.1161/hh0801.089861
62. Badiye AP, Hernandez GA, Novoa I, Chaparro SV. Incidence of hemolysis in patients with cardiogenic shock treated with impella percutaneous left ventricular assist device. *ASAIO J* 2016;**62**:11-14. doi:10.1097/MAT.0000000000000290
63. Wilson J, Fisher R, Caetano F, Soliman-Aboumarie H, Patel B, Ledot S, *et al*. Managing harlequin syndrome in VA-ECMO - do not forget the right ventricle. *Perfusion* 2021;**37**:526-529. doi:10.1177/02676591211020895