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A Review of percutaneous mechanical support devices and strategies

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The technology available to offer acute hemodynamic support to critically ill patients has evolved exponentially in recent years. As our experience grows, and our armamentarium of mechanical therapies expands, devices are becoming increasingly important to providing acute hemodynamic support. This review article will describe approaches to providing acute support and a concise review of the percutaneous mechanical support devices currently available. In an effort to define how and when to use them in acute situations, we will also describe some advantages and disadvantages of each platform, and highlight the primary limitations in defining safety and efficacy.

Keywords

Cardiogenic shock; Heart failure; Percutaneous mechanical support

1. Introduction

Disorders that impair function of the myocardium, valves, conduction system, or pericardium can result in a state of hemodynamic instability which produces a cardiac output incapable of providing adequate tissue perfusion. In 1976, about 20 years prior to the SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock) trial, it was found that patients whose myocardial infarction resulted in a pulmonary capillary wedge pressure greater than 18 mm Hg and a cardiac index less than 2.2 L/min/m^2 had a mortality of 51% (Forrester et al., 1976; Hochman et al., 1999). Since the pre-reperfusion era, our understanding of the pathophysiology of cardiogenic shock has evolved. Unfortunately, despite advances in protocols, reperfusion strategies, percutaneous coronary intervention (PCI), and pharmacotherapy, the mortality for patients with the most severe forms of shock still remains high (Miller et al., 2017).

We now understand that there are different phases of shock, and it has been shown that hospital mortality correlates with the level of inotropic support provided (Atkinson et al., 2016; Samuels et al., 1999; Basir et al., 2017). Patients with cardiogenic shock typically fall into four categories: 1) mild reversible shock that responds to a vasoactive medication, 2) moderate reversible shock that improves after partial support from a device added to high dose medications, 3) severe reversible shock that requires full hemodynamic support, and finally 4) severe mixed shock. Patients with severe mixed shock are typically the most difficult to support because they not only have a component of pump failure, but are also assaulted by acidosis and a toxic milieu, which induces an overwhelming drop in systemic vascular resistance (Thiele et al., 2017). The platform we choose should adequately interrupt this cascade because when we treat patients in the last stages of shock with a percutaneous mechanical support device, or platform, we do it as either a bridge to recovery, as a bridge to transplant, as a bridge to a more durable form of mechanical support, or as a bridge to decision (den Uil et al., 2017).

2. Shifting Our Approach to Support

There are two basic modes to providing hemodynamic support. The first mode can be considered escalation. In this stepwise approach, shock patients are initially treated with medications, such as inotropes (dobutamine or milrinone) or vasopressors (norepinephrine, phenylephrine, or high-dose dopamine) (van Diepen et al., 2017). When those measures fail to adequately support the patient, an intra-aortic balloon pump (IABP) may be inserted, and if their hemodynamic embarrassment continues, more advanced forms of support are initiated. This style has historically been reflected in the guidelines, and reserves the use of IABP or extracorporeal membrane oxygenation (ECMO) for those who "require urgent CABG", who "do not quickly stabilize with pharmacological therapy", or who "have refractory cardiogenic shock" (O'Gara et al., 2013; Peura et al., 2012; Anderson et al., 2011). The introduction of more easily implantable and hemodynamically potent percutaneous devices, however, has led to a paradigm shift that favors a more aggressive approach.

Along with the classic indicators of adequate perfusion, such as lactate, pH, and urine output, some clinicians are increasingly monitoring alternative parameters including the cardiac power output (CPO) and the pulmonary artery pulsatility index (PAPi). There are no randomized trials that prove superiority of one parameter over another. However, the calculations of CPO and PAPi is simple, they have been shown to provide valuable prognostic information, and have been incorporated into recently published shock algorithms.

CPO is the product of cardiac output (CO) and mean arterial

pressure (MAP), divided by 451 (CO × MAP / 451) to allow conversion to watts (W) (Cotter et al., 2003). While it should be noted that the CPO equation does not account for body surface area, in the SHOCK trial, CPO was the hemodynamic variable most strongly associated with in-hospital mortality (normal CPO > 0.6 W). It reflects myocardial reserve adequate to generate flow in the face of high resistance (Fincke et al., 2004). The PAPi score is calculated as the systolic pulmonary artery pressure (sPAP) minus the diastolic pulmonary artery pressure (dPAP) divided by the right atrial pressure (RAP) (sPAP-dPAP/RAP) (normal PAPi > 1.0) (Korabathina et al., 2012). In addition to predicting right ventricular failure in the setting of myocardial infarction, it is one of the predictors of right ventricular failure and the need for right ventricular assist device (RVAD) support in left ventricular assist device (LVAD) recipients (Kang et al., 2016). Physicians adhering to most of the recently developed shock algorithms continue to intensify support until the patient's CPO is greater than 0.6 W and their PAPi is greater than 0.9.

The shift to percutaneous support device delivery from mostly surgical placement has minimized procedural invasiveness, which has helped to reduce the treatment threshold (Stretch et al., 2014). Guidelines are beginning to reflect this more decisive mode of therapy. The 2015 SCAI/ACC/HFSA/STS Clinical Expert Consensus Statement on the Use of Percutaneous Mechanical Circulatory Support Devices in Cardiovascular Care supports the insertion of mechanical support devices "as soon as possible in the cardiogenic shock patient, if initial attempts with fluid resuscitation and pharmacologic support fail to show any significant hemodynamic benefit, and before PCI" (Rihal et al., 2015). There is also a growing body of evidence that earlier, more aggressive, initiation of support with devices improves mortality in cardiogenic shock (Basir et al., 2017; Thiele et al., 2017). However, the financial impact of more widespread use is unclear, there are no large randomized multicenter trials designed to help guide therapy, but there is evidence that this technology should not be used in unselected patients.

As opposed to the escalation model described above, an alternative approach, one rooted in the perfusion experience, is being used to support patients in need of an acute form of hemodynamic support. In the perfusion literature, when placing a patient on cardiopulmonary bypass, one assumes no native contribution to cardiac output with the goal is to achieve a cardiac index (CI) of at least 2.4 L/min/m^2. (McKendry et al., 2004; Kapoor et al., 2008; Smetkin et al., 2009) When addressing a shock patient with this predictive strategy, one calculates the total cardiac output, or flow rates, required to give the patient an adequate CI of 2.4 L/min/m^2. There are easily accessible perfusion calculators that take into account the patient's weight, height, hematocrit, and age to determine the flow rates required to yield a CI of 2.4 L/min/m^2. Essentially, this application allows a clinician to predict the net total CO, either the patient's own, or in combination with a device, to reach a threshold CI of 2.4 L/min/m^2. Therefore, to determine how robust a platform needs to be, the physician must first estimate how much cardiac output the patients own heart can contribute, and then select the optimal device that is capable of providing the balance of flow to reach the 2.4 L/mim/m^2 threshold. Echocardiography and PA catheters can help estimate the patient's native contribution to cardiac output/index. However, if the patient has ongoing CPR or if they are severely acidotic in the setting of cardiogenic shock, the support device one choose in this mode is the device that provides enough flow to yield a CI of at least 2.4 L/min/m^2 without any native contribution.

Rapid diagnosis followed by prompt stabilization and reversal of the underlying cause are fundamental aspects to adequately supporting patients. To facilitate stabilization with the appropriate level of support, one must take many device and patient factors into consideration. The goal of a predictive approach is to avoid borderline support, and subsequently avoid the vicious cascade leading to severe mixed shock and end-organ failure. There are no randomized trials designed to define the optimal support strategy. However, there are reports suggesting that extracorporeal membrane oxygenation is preferable in cases of profound CS, whereas Impella devices seem more appropriate for less severe hemodynamic compromise, such as those who can still generate some native contribution to their cardiac output. Furthermore, the combination of both techniques, discussed later in this article, may help to overcome the limits inherent with each device (Mourad et al., 2017).

3. Devices, Hemodynamics, and Limitations

3.1 Overview

Optimal hemodynamic support results in adequate end-organ perfusion, especially to the brain, kidneys, liver, and heart. An ideal acute percutaneous mechanical support device does not exist. An ideal device would simultaneously 1) provide enough MAP to open end-organ arterioles, 2) provide adequate flow to perfuse tissue after arterioles are opened, 3) unload the ventricle (measured by a reduction in LV pressure and LV volume) to reduce myocardial wall stress and oxygen demand, 4) increase coronary perfusion pressure (determined by the difference between coronary arterial and LV end-diastolic pressure), and 5) provide gas exchange to treat respiratory, in addition to, circulatory failure.

There are two basic platform categories for percutaneous support, pulsatile and continuous flow. Pulsatile support is accomplished with the intra-aortic balloon pump (IABP) and more powerful continuous flow is achieved with Impella (Abiomed) devices, CardiacAssist (makers of TandemHeart, Protek Duo, and TandemLife) devices, or venoarterial (VA) ECMO. Continuous flow can either be intracorporeal and axial with Impella devices, or extracorporeal with cannulas and centrifugal flow pumps.

Extracorporeal platforms can have an oxygenator spliced into their circuit in series. Venovenous (VV) ECMO, while unable to provide circulatory support on its own, can be used in parallel with an Impella device to provide adequate gas exchange (Fig. 1).

3.2 Intra-aortic Balloon Pump

The IABP consists of a double-lumen, 7.5- to 8-F catheter, with a long polyethylene balloon attached to its distal end. The helium inflated balloon, which is coupled to an electrocardiogram, arterial line pressure, and now fiber-optic sensor, can increase cardiac output by two different mechanisms. The first mechanism is afterload reduction and typically results in a predictable 10 % in-

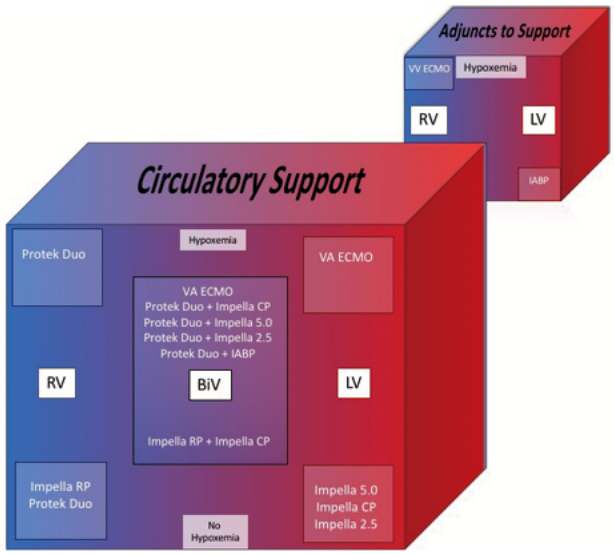


Figure 1. Percutaneous Mechanical Support Options. Devices that offer percutaneous circulatory support and oxygenation capabilities are located at the top of the large box. Since VV ECMO does not provide circulatory support, and because the direct increase in cardiac output from an IABP is small, they are depicted as adjuncts to support in this figure. RV, right ventricular support, BiV, biventricular support, LV, left ventricular support, IABP, intraaortic balloon pump.

crease in cardiac output. The second mechanism is by increasing native cardiac function via increased coronary perfusion pressure from the augmentation wave in early diastole. The impact of this mechanism is highly variable and dependent on the ability of the heart to increase its native contractility. In the setting of diffuse ischemia, this may result in a dramatic increase in cardiac output. However, in the setting of a myocardial infarction resulting in severely stunned or damaged myocardium, the impact will likely be negligible. This may partially explain the lack of significant hemodynamic benefit in clinical trials involving only STEMI patients (Ohman et al., 2005; Vijayalakshmi et al., 2007; Patel et al., 2011).

3.3 Left Ventricular Axial-flow Pump

The Impella device provides intracorporeal support via a continuous trans-aortic valve axial-flow pump. Currently, the two most common left ventricular Impella devices used are the 5.0 (21 Fr), capable of 5.0 liters of flow per minute, and the CP (Cardiac Power) capable of nearly 4 liters per min of flow, averaging less during a long term dwell.

The 5.0 is currently the most ideal Impella device due to its high flow capabilities. However, due to its diameter, malperfusion of the leg can occur if inserted directly into an artery via a sheath. Therefore, surgical cut down and introduction via an anastomosed 8-10 mm Dacron graft is obligatory. This process requires anatomical knowledge of the anastomotic target i.e. axillary, femoral, innominate, and ascending aorta. Due to the vascular surgery component, anesthesia is typically required. While highly variable and institution dependent, deployment of the Impella 5.0 can therefore be delayed at multiple points during this process. This potentially lengthened deployment time limits the 5.0's ability to be used as an acute salvage device. However, the Impella 5.0 is often successfully used as a device to upgrade from Impella CP, or as an avenue to uncouple from veno-arterial ECMO (Mourad et al., 2017; Moazzami et al., 2017).

Conversely, the lower profile Impella CP (14 Fr) can be placed across the aortic valve within minutes with sheath based direct arterial puncture. The Impella CP, after its highest setting or boost mode is reduced, averages 3.4-3.7 liters of flow per minute. Consequently, the Impella CP would most likely provide full support to a 5-foot 5-inch female who weighs less than 125 lbs. However, when there is no native contribution to cardiac output in a larger patient, rendering them incapable of achieving a CI of 2.4 L/min/m^2, changing the platform device should be considered.

Marginal support platforms, those that barely supply the amount of CO needed to provide adequate tissue perfusion, are vulnerable to failure. Supplemental inotropes or vasopressors are often used in patients with marginal support. It is crucial to understand that mechanically supported patients are more susceptible to reductions in mean arterial pressure (MAP = CO $\times$ SVR) due to near fixed mechanical output from the device. This means that for the same given degree of systemic vascular resistance (SVR) reduction, hypotension is more severe for mechanically supported patients because of an inability to augment total cardiac output and balance the equation. Additionally, commonly used medications can induce sustained atrial arrhythmias and ventricular tachycardia. These arrhythmias severely reduce a mechanically supported patient's native contribution, and may place a marginally supported patient in jeopardy (American Thoracic Society et al., 2005).

As mentioned earlier, CPO (CO $\times$ MAP / 451) strongly correlates with in-hospital mortality and reflects myocardial reserve adequate to generate flow in the face of high resistance (Fincke et al., 2004). While it is a valuable predictor of the adequacy of a device, CPO has its limitations. If a patient has a MAP of 60 mmHg with an Impella 5.0 in place, their CPO is calculated to be 0.66 W, indicating that their cardiac power on the device is adequate. However, a MAP of 60 mmHg for that specific patient may not be adequate for multiple reasons. First, as blood passes through a luminal vascular narrowing, mean pressure is reduced (Bernoulli's Equation). Thus, the pre-arteriolar pressure distal to a patient's vascular stenosis may be significantly lower depending on the severity of obstruction, lead to hypoperfusion and can commonly result in non-occlusive mesenteric ischemia or acute tubular necrosis. Moreover, a calculated Cardiac Power Output > 0.6 W may be associated with malperfusion if a patient's total flow yields a less than an acceptable cardiac index. Hence, multiple factors to measure end organ perfusion need to be taken into account such as patient size and degree of peripheral vascular disease.

3.4 Veno-Arterial Extracorporeal Membrane Oxygenation (VA ECMO)

VA ECMO can provide nearly 6 liters of flow in vivo, which meets the cardiac index calculation of 2.4 L/min/m^2 in most patients. The calculated flow requirement to achieve a CI of 2.4 L/min/m^2 helps determine the cannula size given acceptable arterial access; the combination of a 21-F venous cannula/17-F ar-

terial cannula can provide 4-4.5 L/min of flow, and a 25-F venous cannula/19-F arterial cannula can deliver approximately 5-5.5 L/min. Although a VA ECMO platform offers stable perfusion to end organs, there are some disadvantages: 1) ischemia to the limb, 2) Harlequin syndrome 3) cerebrovascular embolization from the device itself or from an atheromatous aorta, 4) need for moderate heparinization, and 5) high left ventricular afterload which may result in left atrial hypertension and/or cardiac thrombosis due to poor forward flow (Samuels et al., 2016).

Leg ischemia is commonly and effectively counteracted by adding a percutaneous distal perfusion line. Ideally, this perfusion line is placed with ultrasound and fluoroscopy prior to cannula insertion.

The watershed phenomenon is also known as Harlequin syndrome, differential hypoxia, North-South syndrome, and two-circulation syndrome. Patients on VA ECMO have varying degrees of native contribution to cardiac output. This anterograde native flow meets the retrograde ECMO flow at a point called the 'watershed' which creates a 'mixing cloud' (Hoeper et al., 2014). The location is dynamic and patient dependent because it is determined by the competition of native cardiac output and ECMO flow (Fig. 2). Improving the native cardiac output will move the cloud more distal, and all tissues distal to the watershed receive adequately oxygenated blood from the ECMO circuit. In contrast, areas proximal to the watershed receive blood from the left ventricle, and the oxygen saturation of this native blood can be low due to pulmonary edema, pneumonia or other pulmonary conditions. Thus, despite a seemingly suitable perfusion pressure and CPO, respiratory failure during VA ECMO can result in anoxic brain injury, myocardial ischemia and weaning failure from ECMO (Choi et al., 2014).

Left atrial hypertension and cardiac thrombosis is often initially prevented by augmenting aortic valve opening with inotropes, which have also been used in conjunction with an IABP. Usually, left atrial hypertension can resolve with time. However, left ventricular venting is preferred in cases of refractory cardiogenic shock to prevent this issue. Addition of an Impella device to the ECMO circuit, or "ECPELLA," has been recognized as a successful venting stragegy (Akanni et al., 2016). This strategy should be used with caution in the setting of significant pulmonary edema, as "ECPELLA" can increase the risk of Harlequin syndrome. Alternatively, a LA drainage cannula can be "Y" adapted into the venous drainage cannula. It is unclear if Impella venting prevents left ventricular thrombosis more adequately than left atrial drainage as there are no randomized trials to compare these strategies.

3.5 Transseptal Extracorporeal Membrane Oxygenation

Unlike VA ECMO, the transseptal TandemHeart (CardiacAssist) platform provides left ventricular unloading by displacing blood from the left atrium to the femoral artery. Potential advantages of this system are shorter circuit tubing length, elimination of an oxygenator, and operator priming without a perfusionist. A 21-F draining cannula has to be inserted through the interatrial septum with fluoroscopic guidance, with or without ultrasound assistance. Similar to VA ECMO, blood is directed outside the body to a centrifugal pump. The femoral artery cannula determines the maximal flow provided, ranging from 3.5 L/min (15 F) to 4.5 L/min (17 F). In the setting of severe hypoxemia, lack of an oxygenator can be limiting.

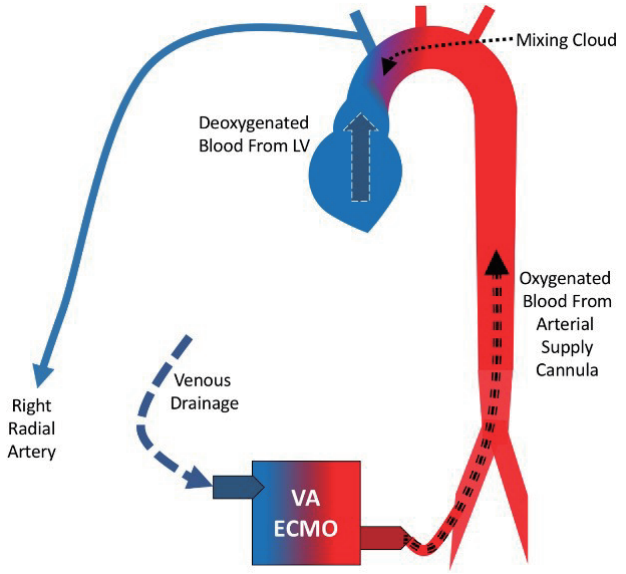


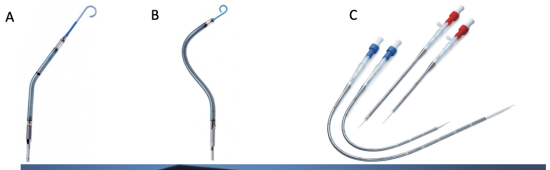
Figure 2. Watershed Phenomenon. Although the coronaries are furthest away from the oxygenated blood supplied by the ECMO circuit, we cannot easily measure oxygen content here without direct left ventricular sampling. Therefore, an arterial blood gas drawn from a right radial arterial line provides the best indicator of cerebral oxygenation.

3.6 Right Ventricular Support, Biventricular Support, and Emerging Configurations

Right ventricular dysfunction prevents adequate drainage of a plethoric venous system, which results in a higher central venous pressure and a narrowed pulse pressure in the pulmonary artery secondary to low stroke volume. Other variables that indicate need for mechanical right ventricular support include a RA: PCWP ratio > 0.8 and PAPi (sPAP-dPAP/RA) < 1.0 (Korabathina et al., 2012; Nagy et al., 2013).

The Proteck Duo by Cardiac Assist is an example of a right ventricular support device. Typically placed in the right or left internal jugular vein, this single 31-F cannula has proximally positioned inflow vents that can direct blood from the right atrium (through an outer draining lumen) to the main pulmonary artery (via an inner 18-F supply lumen). Addition of an oxygenator to this circuit also results in VA ECMO with little recirculation of oxygenated blood. Deployment of the Protek Duo, safest under fluoroscopy, can be performed in locations with a fluoroscopic stretcher/table and portable C-Arm.

An alternative right ventricular circulatory support platform is the Abiomed Impella RP, which typically produces 4.0 – 4.5 L/min of support, equivalent to the Protek Duo (Anderson et al., 2015). Similar to left sided Impella devices, the Impella RP has a flexible pigtail-shaped tip followed by a cannula that contains the pump outlet and inlet areas, motor housing, and pump pressure monitor. In addition to these features, the RP has a three-dimensional shape to help guide placement into the main PA. In contrast to the left sided Impella devices, the inlet area is located proximally, and



	Device	Access
A	Impella CP	14 Fr, arterial
B	Impella RP	22 Fr, venous
C	VA ECMO	17-19 Fr arterial, 21-25 Fr venous
D	Protek Duo	31 Fr, venous

Figure 3. Commonly used percutaneous mechanical support devices and access site requirements.

the outlet area is near the distal tip. This allows for continuous, intracorporeal, axial blood flow from the IVC/RA junction to the main PA. The access site for the Impella RP is typically the right femoral vein. As long as there is no thrombosis, the precedence of an IVC filter does not prohibit use of the RP if its 22-F pump motor can safely transverse the filter. Again, unlike cannula-based extracorporeal devices, gas exchange is not possible with any Impella device, and VV ECMO does not provide circulatory support on its own.

It is not uncommon to use more than one mechanical support device per patient to optimize end-organ perfusion (Mourad et al., 2017; Moazzami et al., 2017; Samuels et al., 2016). Veno-arterial ECMO is frequently initiated outside the cath lab for patients in distress due to severe refractory shock. Due to the risks associated with the VA ECMO platform, uncoupling within 12-24 hours is preferred if possible. Uncoupling is performed by combining right and/or left ventricular support devices in place of a VA ECMO circuit. This may consist of transitioning to Impella devices, which are preferably placed with a transaxillary approach to improve patient mobility and permit more stable cannula positioning (Samuels et al., 2016). This uncoupling allows for lower heparin dosing and even no anticoagulation for some platform configurations (Plush et al., 2016).

4. Conclusions

Although logistical and ethical challenges hinder our ability to perform randomized controlled trials on mechanically supported patients in acute situations, technology will continue to improve, and we need to learn how to utilize it more effectively. The acuity of the situation and its setting influence which mechanical support device can be used. Time to initiation is a significant factor, especially when there is an element of CPR. A simple transthoracic echocardiogram can help with rapid assessment for severe valvular disease, estimation of the patient's native contribution to cardiac output, and calculation of the required flows to maintain adequate end-organ perfusion. When presented with evidence of severe respiratory compromise, one should consider a device platform that can provide gas exchange. For patients with severely deranged laboratory values, such as pH and lactate, one should consider providing the most robust platform available. Proactively selecting adequate support platforms, quickly uncoupling to a device with fewer complications once the patient is stabilized, and monitoring the patient in a dedicated support unit are all essential components of a successful support strategy.

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Conflict of Interest

The authors declare no competing interests.

References

Akanni O, Takeda K, Truby L, et al. ECPELLA: combined use of extracorporeal membrane oxygenation and percutaneous microaxial pump left ventricular assist device. *J Heart Lung Transplant.* 2016;35:323.

American Thoracic Society. Infectious Diseases Society of America. Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. Am J Respir Crit Care Med. 2005;171:388-416.

Anderson JL, Adams CD, Antman EM, et al. 2011 ACCF/AHA focused update incorporated into the ACC/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association task force on practice guidelines. *Am Circulation.* 2011;123:426-579.

Anderson MB, Goldstein J, Milano C, et al. Benefits of a novel percutaneous ventricular assist device for right heart failure: the prospective RECOVER RIGHT study of the Impella RP device. *Heart Lung Transplant.* 2015;34:1549-1560.

Atkinson TM, Magnus O, O'Neill WW, et al. A practical approach to mechanical circulatory support in patients undergoing percutaneous coronary intervention: an interventional perspective. *JACC: Cardiovasc Interv.* 2016;9:871-883.

Basir MB, Schreiber TL, Grines CL, et al. Effect of early initiation of mechanical circulatory support on survival in cardiogenic shock. *Am J Cardiol.* 2017;119:845-851.

Choi JH, Kim SW, Kim YU, et al. Application of veno-arterial-venous extracorporeal membrane oxygenation in differential hypoxia. Multidiscip Respir Med. 2014;9:55.

Cotter G, Moshkovitz Y, Kaluski E, et al. The role of cardiac power and systemic vascular resistance in the pathophysiology and diagnosis of patients with acute congestive heart failure. *Eur J Heart Fail.* 2003;5:443-451.

den Uil CA, Akin S, Jewbali LS, et al. Short-term mechanical circulatory support as a bridge to durable left ventricular assist device implantation in refractory cardiogenic shock: a systematic review and meta-analysis. *Eur J Cardiothorac Surg.* 2017;52:14-25.

Fincke R, Hochman JS, Lowe AM, et al. Cardiac power is the strongest hemodynamic correlate of mortality in cardiogenic shock: a report from the SHOCK trial registry. *J Am Coll Cardiol.* 2004;44:340-348.

Forrester JS, Diamond G, Chatterjee K, et al. Medical therapy of acute myocardial infarction by application of hemodynamic subsets (second of two parts). *N Engl J Med.* 1976;295:1404-1413.

Hochman JS, Sleeper LA, Webb JG, et al. Early revascularization in acute myocardial infarction complicated by cardiogenic shock: SHOCK investigators: should we emergently revascularize occluded coronaries for cardiogenic shock. *N Engl J Med.* 1999;341:625-634.

Hoeper MM, Tudorache I, Kuhn C, et al. Extracorporeal membrane oxygenation watershed. *Circulation.* 2014;130:864-865.

Kang G, Ha R, Banerjee D. Pulmonary artery pulsatility index predicts

right ventricular failure after left ventricular assist device implantation. *J Heart Lung Transplant*. 2016;35:67-73.
Kapoor PM, Kakani M, Chowdhury U, et al. Early goal-directed therapy in moderate to high-risk cardiac surgery patients. Ann Card Anaesth. 2008;11:27-34.
Korabathina R, Heffernan KS, Paruchuri V, et al. The pulmonary artery pulsatility index identifies severe right ventricular dysfunction in acute inferior myocardial infarction. *Catheter Cardiovasc Interv*. 2012;80:593-600.
McKendry M, McGloin H, Saberi D, et al. Randomized controlled trial assessing the impact of a nurse delivered, flow monitored protocol for optimization of circulatory status after cardiac surgery. *BMJ*. 2004;329:438.
Miller EP, Solomon MA, McAreavey D. Advanced percutaneous mechanical circulatory support devices for cardiogenic shock. *Crit Care Med*. 2017;45:1922-1929.
Moazzami K, Dolmatova EV, Cocke TP, et al. Left Ventricular Mechanical Support with the Impella during Extracorporeal Membrane Oxygenation. *J Tehran Heart Cent*. 2017;12:11-14.
Mourad M, Gaudard P, De La Arena P, et al. Circulatory Support with Extracorporeal Membrane Oxygenation and/or Impella for Cardiogenic Shock During Myocardial Infarction. *ASAIO J*. 2017;[online] Available at: https://insights.ovid.com/crossref?an=00002480-900000000-98942
Nagy CD, Jumean MF, Pham DT, et al. Percutaneous Circulatory Support for Biventricular Failure. Circ Cardiovasc Interv. 2013;6:12-14.
O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: executive summary. *JACC*. 2013;61:485-510.
Ohman, EM, Nanas, J, Stomel, RJ, et al. Thrombolysis and counterpulsation to improve survival in myocardial infarction complicated by hypotension and suspected cardiogenic shock or heart failure: results of the TACTICS Trial. *J Thromb Thrombolysis*. 2005;19:33-39.
Patel MR, Smalling RW, Thiele H, et al. Intra-aortic balloon counterpulsation and infarct size in patients with acute anterior myocardial infarction without shock: the CRISP AMI randomized trial. *JAMA*. 2011;306:1329-1337.
Peura JL, Colvin-Adams M, Francis GS, et al. Recommendations for the use of mechanical circulatory support: device strategies and patient selection: a scientific statement from the American Heart Association. *Circulation*. 2012;126:2648-2667.
Rihal CS, Naidu SS, Givertz MM, et al. 2015 SCAI/ACC/HFSA/STS clinical expert consensus statement on the use of percutaneous mechanical circulatory support devices in cardiovascular care. *J Card Fail*. 2015;21:499-518.
Plush T, Casanova-Ghosh E, Gnall E, et al. Successful use of extracorporeal membrane oxygenation (ECMO) therapy without intravenous heparin in a patient with acute intracranial hemorrhage and severe ARDS. *Am J Respir Crit Care Med*. 2016;193:A1871.
Samuels L, Gnall E, Casanova-Ghosh E, et al. A hybrid configuration of a left ventricular assist device and venovenous extracorporeal membrane oxygenation. *J Thorac Cardiovasc Surg*. 2016;151:75-76.
Samuels LE, Kaufman MS, Thomas MP, et al. Pharmacological criteria for ventricular assist device insertion following postcardiotomy shock: experience with the Abiomed BVS system. *J Card Surg*. 1999;14:288-293.
Smetkin AA, Kirov MY, Kuzkov VV, et al. Single transpulmonary thermodilution and continuous monitoring of central venous oxygen saturation during off-pump coronary surgery. *Acta Anaesthesiol Scand*. 2009;53:505-14.
Stretch R, Sauer CM, Yuh DD, et al. National trends in the utilization of short-term mechanical circulatory support incidence, outcomes, and cost analysis. *JACC*. 2014;64:1407-1415.
Thiele H, Jobs A, Ouweneel DM, et al. Percutaneous short-term active mechanical support devices in cardiogenic shock: a systematic review and collaborative meta-analysis of randomized trials. *Eur Heart J*. 2017;38:3523-3531.
van Diepen S, Katz JN, Albert NM, et al. Contemporary management of cardiogenic shock: a scientific statement from the American Heart Association. *Circulation*. 2017;136:e232-e268.
Vijayalakshmi, K., Kunadian, B., Whittaker, V.J., et al. Intra-aortic counterpulsation does not improve coronary flow early after PCI in a high-risk group of patients: observations from a randomized trial to explore its mode of action. *J Invasive Cardiol*. 2007;19:339-346.