Perioperative Management of Patients Receiving Short-term Mechanical Circulatory Support with the Transvalvular Heart Pump

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Cardiogenic shock continues to be an unresolved clinical challenge. The initial management of cardiogenic shock includes etiology-specific treatment (*e.g.*, coronary revascularization for acute myocardial infarction), optimization of volume and respiratory status, and administration of inotropic and vasopressor medications. However, inotropes may increase myocardial oxygen consumption, which may further worsen myocardial ischemia, can induce arrhythmias, and may not provide adequate circulatory support. Vasopressors may further decrease tissue perfusion and impair microcirculation.[1]

As a result, interest in the potential role of short-term mechanical circulatory support in cardiogenic shock has grown and clinical uptake of these devices continues to increase.[2,3] Use of the Impella (Abiomed Inc., USA) transvalvular heart pump in particular, has grown significantly.[3–5] As such, anesthesiologists are increasingly likely to care for patients receiving transvalvular heart pump support and should have a thorough understanding of the device and its hemodynamic effects and perioperative considerations. Recent publications have described other short-term mechanical circulatory support devices, including venoarterial extracorporeal membrane oxygenation and intraaortic balloon pump counterpulsation.[6,7] To provide clinicians with a comprehensive review, this article focuses on the perioperative management of a single device, the transvalvular heart pump.

Transvalvular Heart Pump

Device Overview

The Impella family of heart pumps are microaxial, transvalvular ventricular assist devices that provide continuous, antegrade flow. There are five left ventricular assist devices capable of providing various levels of circulatory support and one right ventricular assist device, the Impella RP (table 1). The Impella 2.5, CP, and RP are typically placed percutaneously, while the Impella 5.0, LD, and 5.5 are placed surgically. The surgically placed devices provide the highest levels of maximum pump flow, with the Impella 5.5 capable of flowing up to 6 l/min, and can be used in patients with unsuitable femoral and aorto-iliac anatomy.

The Impella catheter consists of a blood inlet area, cannula, placement sensor area, blood outlet area, and motor housing (fig. 1). Blood is aspirated from the blood inlet area and expelled through the outlet area. In the case of a left-sided transvalvular heart pump, the blood inlet and outlet areas reside in the left ventricle and ascending aorta, respectively; for the RP, they reside in the inferior vena cava and pulmonary artery, respectively. The placement sensor area, which is a fluid-filled pressure lumen in the 2.5 and CP, a differential pressure sensor in the 5.0, LD, and RP, and a fiber-optic sensor in the 5.5, generates a placement signal that is used to assist in proper device positioning. The Impella 2.5, CP, 5.0, and RP have a pigtail that is used to stabilize the device during placement.

The Automated Impella Controller (Abiomed Inc.) is used to monitor and control the Impella catheter. It contains a purge cassette, which delivers purge fluid to the catheter. The heparin-containing purge fluid prevents blood from entering the motor housing by pushing blood away from the motor housing and maintaining a pressure barrier between the blood and motor. The Automated Impella Controller monitor displays alarms, the P-level (flow rate) indicator, transvalvular heart pump flow rates, purge fluid flow rates, and battery power status. It also contains a central display area that changes depending on the "screen" selected. The placement screen, which is used to verify correct device positioning, is the most relevant screen for device placement and will

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Table 1. Impella Heart Pump Characteristics

	Impella 2.5	Impella CP	Impella LD	Impella 5.0	Impella 5.5	Impella RP
Supported ventricle	Left ventricle	Left ventricle	Left ventricle	Left ventricle	Left ventricle	Right ventricle
Insertion depth	3.5 cm into left ventricle from aortic valve annulus	3.5 cm into left ventricle from aortic valve annulus	3.5 cm into left ventricle from aortic valve annulus	3.5 cm into left ventricle from aortic valve annulus	5 cm into left ventricle from aortic valve annulus	4 cm into pulmonary artery from pulmonary valve annulus
Typical insertion technique	Percutaneous	Percutaneous	Surgical	Surgical	Surgical	Percutaneous
Typical insertion location	Femoral artery	Femoral artery	Ascending aorta	Axillary artery, femoral artery	Axillary artery, ascending aorta	Femoral vein
Catheter/motor housing size	9 Fr/12 Fr	9 Fr/14 Fr	9 Fr/21 Fr	9 Fr/21 Fr	9 Fr/19 Fr	11 Fr/22 Fr
Maximum flow rate	~2.5 l/min	~4.3 l/min	~5.0 l/min	~5.0 l/min	~6 l/min	~4.0 l/min
Placement signal	Fluid-filled pressure lumen	Fluid-filled pressure lumen*	Differential pressure sensor	Differential pressure sensor	Fiber-optic sensor	Differential pressure sensor
U.S. Food and Drug Administration–approved indications and emergency use authorizations	• Refractory cardiogenic shock • High-risk PCI • COVID-19 emergency use authorization	• Refractory cardiogenic shock • High-risk PCI • COVID-19 emergency use authorization	• Refractory cardiogenic shock	• Refractory cardiogenic shock • COVID-19 emergency use authorization	• Refractory cardiogenic shock • COVID-19 emergency use authorization	• Acute right heart failure • COVID-19 emergency use authorization

*Impella CP with SmartAssist (Abiomed Inc., USA) uses a fiber-optic sensor to determine the placement signal.

Fr, French; PCI, percutaneous coronary intervention.

be discussed here. When the placement screen is selected, the placement signal and motor current waveforms are displayed.

The placement signal is determined at the placement sensor area of the catheter and is used to confirm whether the device is appropriately positioned (fig. 2; fig. A1). In the Impella 2.5 and CP, the placement signal is a pressure waveform that is measured from the fluid-filled pressure lumen just proximal (closer to the insertion site) to the outlet area. The placement signal will display either a pulsatile aortic waveform (correct position) or pulsatile ventricular waveform (incorrect position) depending on the position of the fluid-filled pressure lumen. In the Impella 5.5, the placement signal is a pressure waveform measured from the fiber-optic sensor just distal (further from the insertion site) to the outlet area. The placement signal will display either an aortic pressure waveform (correct position) or ventricular pressure waveform (incorrect position) depending on the position of the fiber-optic sensor. In the Impella LD, 5.0, and RP, the placement signal is a differential pressure waveform that is measured from the differential pressure sensor. The differential pressure waveform displays the difference in pressure between the outside of the cannula and the inside of the cannula. In left-sided devices, when the Impella catheter is appropriately positioned across the aortic valve, the outside of the differential pressure sensor is exposed to aortic pressures, and the inside of the sensor is exposed to ventricular pressures. In this scenario, the difference in pressure should result in a pulsatile placement signal, with the maximum pressure difference occurring in diastole (when ventricular pressure is lowest relative to aortic pressure) and the minimum pressure difference occurring in systole (when ventricular pressure is closest to aortic pressure). If the blood inlet and outlet areas are in the same chamber, the outside and inside of the cannula will be exposed to the same pressures throughout the cardiac cycle leading to a nonpulsatile placement signal. In contrast to the left-sided devices, the differential pressure sensor on the RP is located by the blood inlet area. When the Impella catheter is appropriately positioned across the pulmonary valve, the outside of the differential pressure sensor is exposed to the inferior vena cava pressure, while the inside of the sensor is exposed to the pulmonary artery pressure. The placement signal will be pulsatile if the transvalvular heart pump is correctly positioned and will be nonpulsatile if the blood inlet and outlet areas are incorrectly located in the same chamber. Notably, the placement signal will also be pulsatile when the outlet is incorrectly positioned in the right ventricle, if the inlet is in the inferior vena cava. In this case, echocardiographic assessment will show the tip of the device in the right ventricle.

The motor current waveform displays the variation in energy use by the transvalvular heart pump motor (fig. 2; fig. A1). Energy use is affected by motor speed and by the pressure difference between the blood inlet and outlet areas. Because the pressure difference between the inlet and outlet changes throughout the cardiac cycle, the motor current waveform should be pulsatile if the device is appropriately positioned. This is particularly useful for left-sided devices to ensure that the device remains properly positioned. Notably, the motor current waveform will also be pulsatile even when the outlet of the RP is incorrectly positioned in the right ventricle, if the inlet is in the inferior vena cava.

Hemodynamic Effects

Transvalvular heart pump flow is directly related to rotations per minute of the device and inversely related to

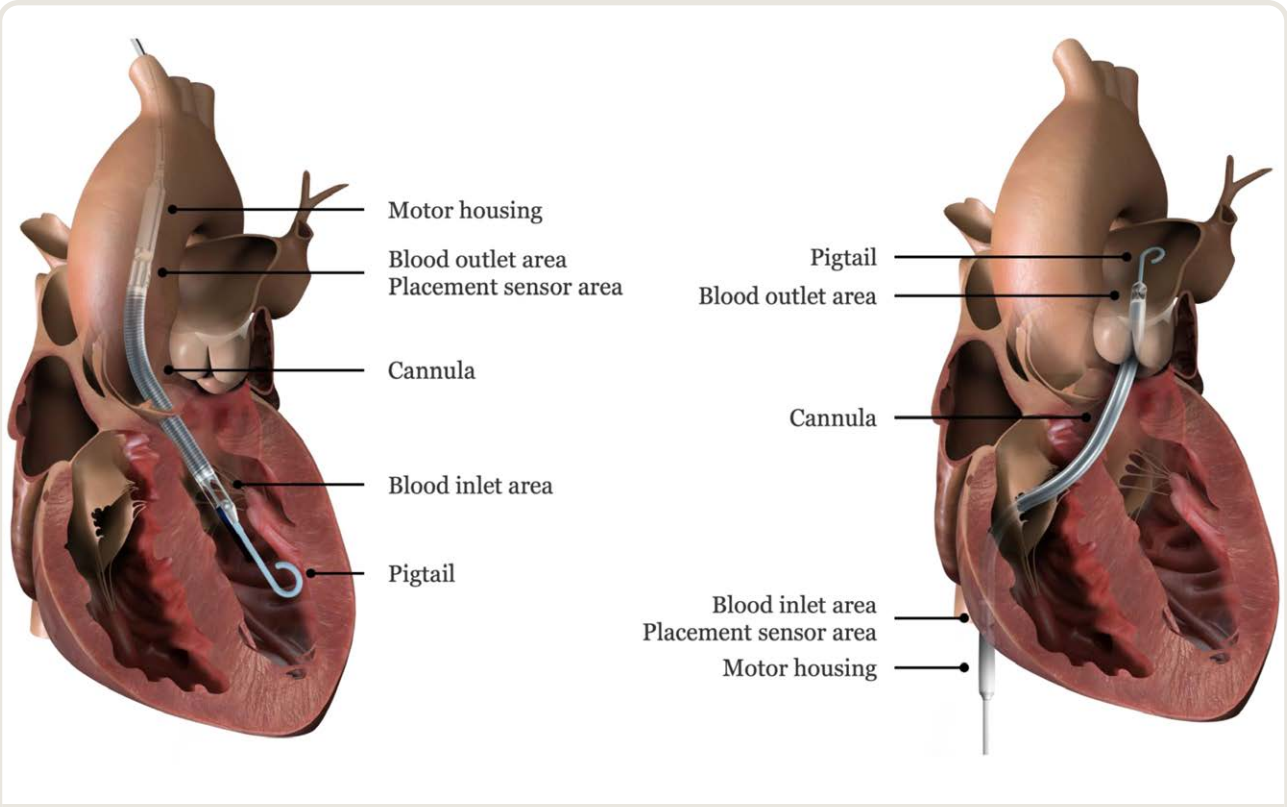

Fig. 1. Impella catheter. The Impella 5.0 (*left*) and Impella RP (*right*) are shown in their appropriate positions. Left-sided devices traverse the aortic valve, while the right-sided Impella RP traverses the tricuspid and pulmonary valves. Adapted and reproduced with permission from Abiomed Inc. (USA).

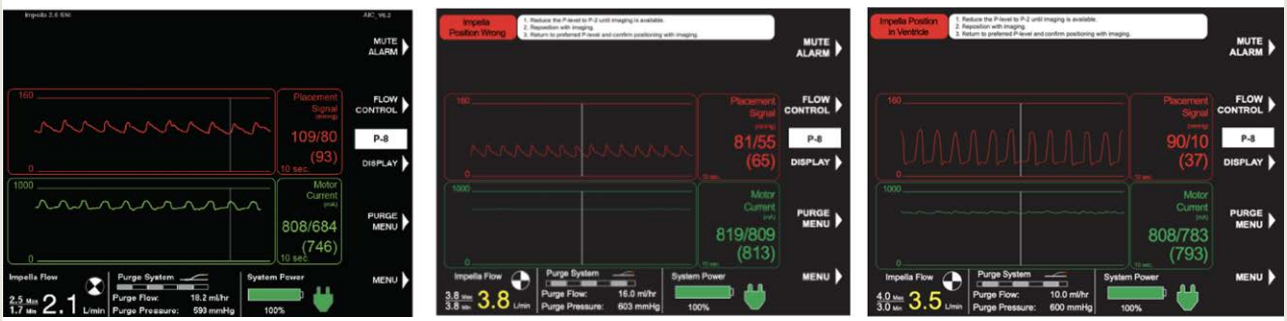

Fig. 2. Placement screen on the Automated Impella Controller. Placement screens generated by a fluid-filled pressure lumen (Impella 2.5 and CP) are shown. (*Left*) The transvalvular heart pump is appropriately positioned and shows an aortic waveform in the placement signal and a pulsatile motor current. (*Middle*) The blood inlet and outlet areas are both in the aorta (aortic waveform placement signal, flat motor current). (*Right*) Both the inlet and outlet areas are in the ventricle (ventricular waveform placement signal, flat motor current). Please see figure A1 for placement screens generated by a differential pressure sensor. Adapted and reproduced with permission from Abiomed Inc. (USA).

the pressure gradient between the blood inlet and outlet areas of the pump. The highest pump flow occurs when the gradient between the left ventricle and aorta (in the case of a left-sided device) or the inferior vena cava and pulmonary artery (in the case of the RP) is minimized. Variation in flow throughout the cardiac cycle is typically more pronounced in a left-sided device compared to the RP because there is greater pressure variation in the left ventricle than in the inferior vena cava and pulmonary artery.[8] Pump flows will decrease with vasopressor administration because the pressure gradient will increase as the systemic pressure (for left-sided devices) and pulmonary

arterial pressure (for the RP) increase. Unlike the intraaortic balloon pump, the transvalvular heart pump functions independently of cardiac rhythm and is not reliant upon on a consistent EKG signal or arterial pressure waveform to function properly.[9]

In left ventricular failure, the transvalvular heart pump is placed retrograde across the aortic valve and pumps blood from the left ventricle into the ascending aorta. This unloads the left ventricle, improves cardiac output, and increases systemic blood pressure.[9,10] Left ventricular unloading decreases peak left ventricular pressure, left ventricular end-diastolic pressure, and left ventricular wall tension, leading to a reduction in stroke work and myocardial oxygen demand. These hemodynamic effects are illustrated by the left ventricular pressure–volume loops in figure 3. Greater ventricular unloading is achieved with increasing pump flow rates, shown by a leftward shift in the pressure–volume loop. A decrease in the area enclosed by the loop reflects a reduction in left ventricular stroke work, while a decrease in the pressure–volume area reflects a reduction in myocardial oxygen consumption. Because the pump continuously unloads the ventricle throughout the cardiac cycle, isovolumic phases are absent, and the pressure–volume loop becomes triangular in shape. Left ventricular unloading also decreases left atrial and pulmonary capillary wedge pressure, which in turn may reduce cardiogenic pulmonary edema and right ventricular afterload.[9,11]

Escalating transvalvular heart pump flow rates induce a widening dissociation between arterial pressure, which increases, and peak left ventricular pressure, which decreases (fig. 3).[11] An increase in arterial diastolic pressure along with a decrease in left ventricular end-diastolic pressure leads to improved coronary perfusion pressure.[12]

In right ventricular failure, the transvalvular heart pump is placed antegrade across the tricuspid and pulmonary valves. Blood is pumped from the inferior vena cava into the pulmonary artery, bypassing the right ventricle. This decreases right atrial pressure while increasing mean pulmonary artery pressure and left ventricular preload.[8] If left ventricular function is normal, right-sided transvalvular heart pump support will result in increased or unchanged left ventricular pressures and improved cardiac output. However, in the setting of concomitant left ventricular dysfunction, right-sided transvalvular heart pump support may lead to a significant increase in left ventricular pressures, worsening left ventricular function, and pulmonary edema, with little improvement in cardiac output.[8] Similarly, initiation of left-sided transvalvular heart pump support in the setting of concomitant right ventricular dysfunction may result in an abrupt increase in right ventricular pressure and size, further exacerbating right ventricular dysfunction and limiting left ventricular preload. These effects underscore the importance of evaluating biventricular function before initiating right- or left-sided transvalvular heart pump support.

Evidence and Clinical Use

An overview of the major studies evaluating transvalvular heart pump use in cardiogenic shock are shown in table A1. While transvalvular heart pump support has been found to provide improved hemodynamics compared to intraaortic balloon pump counterpulsation, randomized controlled trials have not shown a survival benefit.[13,14] However, these trials involve very small numbers of patients and are likely underpowered. Studies have also found higher rates of major bleeding and vascular complications with transvalvular heart pump therapy compared to intraaortic balloon pump counterpulsation.[14,17] Therefore, while transvalvular heart pump use has expanded rapidly in recent years, there is limited evidence to support such robust growth. Larger randomized controlled trials are needed to further clarify the appropriate clinical indications and patient populations that would benefit most from transvalvular heart pump therapy.

The Impella received U.S. Food and Drug Administration approval in 2008 and is currently approved for commercial use in cardiogenic shock and high-risk percutaneous coronary intervention (PCI; table A2). In clinical practice, it is most commonly used for the treatment of acute heart failure—cardiogenic shock after acute myocardial infarction, decompensated heart failure,[22] postcardiotomy cardiogenic shock,[23] myocarditis,[24–26] and peripartum cardiomyopathy[27]—and for ventricular support during high-risk PCI. It has also been placed during procedures with elevated risk for hemodynamic instability, including ventricular tachycardia ablation,[28,29] off-pump coronary artery bypass grafting,[30] high-risk balloon aortic valvuloplasty,[31] and high-risk transcathether aortic valve replacement.[32] In advanced heart failure patients, transvalvular heart pump support has bridged patients to recovery, durable left ventricular assist device, and heart transplantation.[33] Finally, transvalvular heart pump therapy has been used to unload the left ventricle during the increased afterload state of venoarterial extracorporeal membrane oxygenation (ECMO).[34] The transvalvular heart pump is contraindicated in patients with conditions that would preclude safe device placement or use, including the presence of mechanical valves (aortic position for left-sided devices, and tricuspid or pulmonary position for the Impella RP) or cardiac thrombus, and significant valvular stenosis or regurgitation (aortic for left-sided devices, and tricuspid or pulmonary for Impella RP; table A3).

There are a number of known complications associated with transvalvular heart pump support. In a meta-analysis of 671 patients, Vargas *et al.*[35] found that major bleeding was the most common complication (19.9%). Hemolysis (10.5%), limb ischemia (5.0%), and stroke (3.8%) were also common. Hemolysis, which is caused by shear stress from the axial pump, may be exacerbated by higher device flows or obstruction of the blood inlet or outlet areas from improper device positioning or hypovolemia. Hemolysis

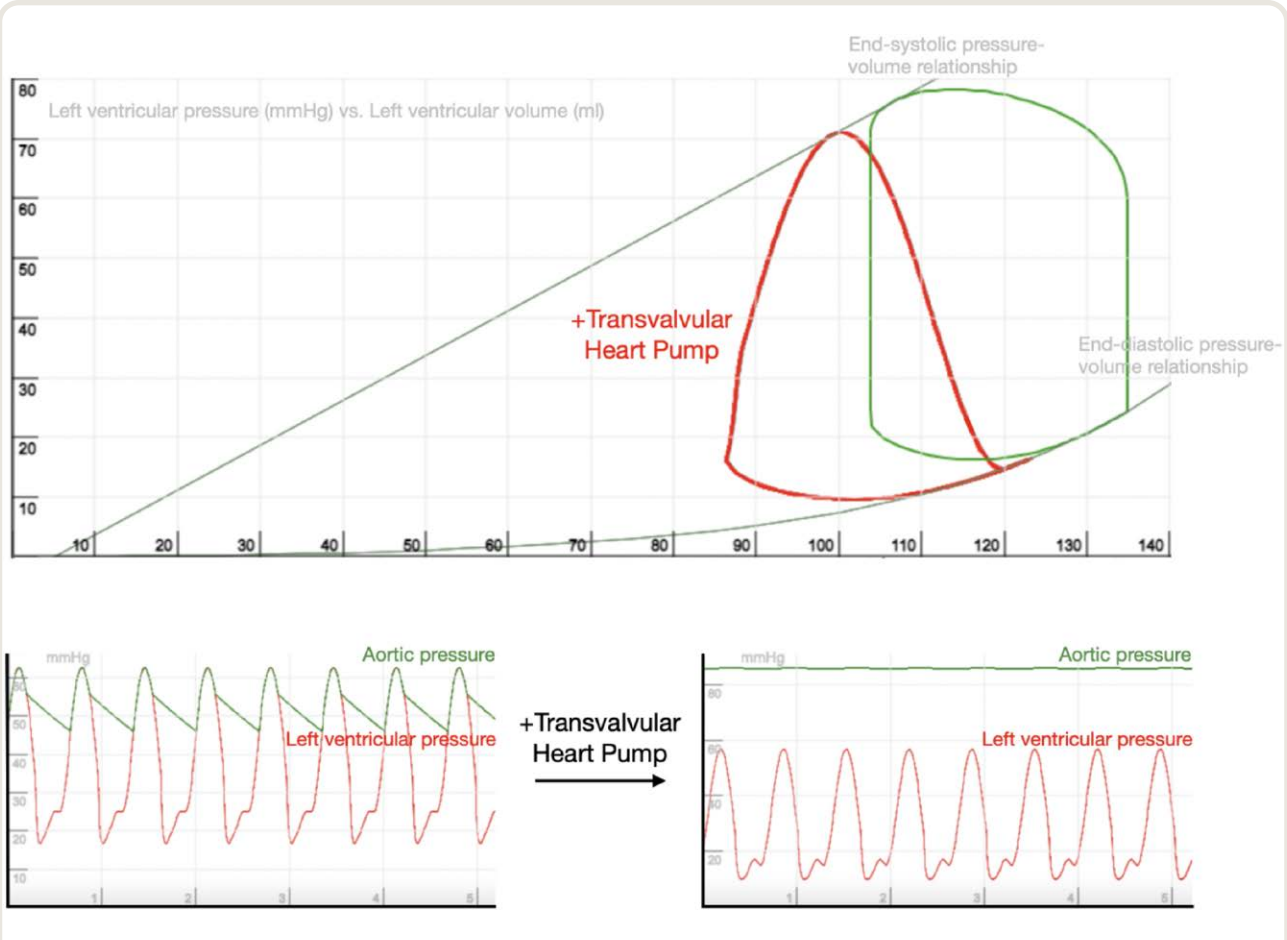

Fig. 3. Hemodynamic effects of the transvalvular heart pump. Pressure–volume relationships with (*red loop*) and without (*green loop*) transvalvular heart pump support are shown (*top*). The triangular-shaped pressure–volume loop reflects continuous left ventricular unloading performed by the pump. A reduction in pressure–volume area (the area enclosed by end-systolic pressure–volume relationship, end-diastolic pressure–volume relationship, and the systolic portion of the loop) with transvalvular heart pump support indicates a decrease in myocardial oxygen consumption. (*Bottom*) Uncoupling of arterial and left ventricular pressures after initiation of pump flow is illustrated. This uncoupling results in flattening of the arterial pressure waveform due to a loss of left ventricular ejection and native cardiac output. Figure created using Harvi.online with permission from PVLoops, Inc., USA (Burkhoff D, Dickstein ML, Schleicher T. Harvi – Online. Available at: https://harvi.online. Accessed December 15, 2021).

may also be an indicator of pump thrombosis. Persistent hemolysis despite proper device positioning and adequate volume is associated with acute kidney injury and may require device removal. In a meta-analysis comparing the transvalvular heart pump to intraaortic balloon pump counterpulsation, rates of major bleeding (relative risk, 3.11; 95% CI, 1.50 to 6.44; $P = 0.002$) and limb ischemia (relative risk, 2.58; 95% CI, 1.24 to 5.34; $P = 0.01$) were higher in the transvalvular heart pump population.[36] Infection, ventricular arrythmias, device migration, device malfunction from thrombosis, acute kidney injury, and thrombocytopenia are also known complications of transvalvular heart pump support.[14,37,38] Finally, aortic and mitral valve injury can occur during insertion or manipulation of left-sided devices, leading to clinically significant increases in aortic or mitral regurgitation.[39,40]

Perioperative Management

Device Placement and Initial Optimization

Standardized intraoperative monitoring requirements for patients who may require or are receiving transvalvular heart pump therapy have not been developed. However, it is appropriate to use standard American Society of Anesthesiologists monitors with continuous intraarterial blood pressure monitoring, pulmonary artery catheter monitoring, and echocardiography. In their analysis of registry data on 15,259 patients who received transvalvular heart pump support for cardiogenic shock, O'Neill *et al.*[16] found a higher survival rate in those who received hemodynamic monitoring with a pulmonary artery catheter than in those who did not (63% *vs.* 49%; $P < 0.0001$). In a separate registry analysis of 1,414 patients in cardiogenic shock, use

of complete pulmonary artery catheter-derived hemodynamic data before initiation of temporary mechanical circulatory support was found to be associated with improved survival.[41]

Anesthesiologists are most likely to encounter patients requiring transvalvular heart pump therapy for cardiogenic shock. The first line treatment for cardiogenic shock includes optimization of volume status and administration of vasopressors and inotropic medications. However, when medical therapy alone does not provide adequate circulatory support, temporary mechanical circulatory support should be considered. The specific mechanical circulatory support strategy will depend on patient factors, including the presence of any contraindications, as well as institutional experience and preference. The transvalvular heart pump is not commonly utilized in refractory cardiac arrest or when there is severe respiratory compromise; venoarterial ECMO should be considered in these situations.[42] Although not used in patients with severe primary pulmonary disease, transvalvular heart pump support may improve oxygenation by unloading the left heart and reducing pulmonary edema. The choice of left-sided transvalvular heart pump will depend in part on the level of support that is required; maximal flow rates range between 2.5 l/min for the Impella 2.5 and 6 l/min for the Impella 5.5 (table 1).

During transvalvular heart pump placement in the operating room, heparin should be administered to achieve an activated clotting time of 250 s or greater. Of note, while an activated clotting time of 250 s or greater is recommended for placement, the recommended activated clotting time for an indwelling device is 160 to 180 s. The optimal anticoagulation strategy for patients with heparin-induced thrombocytopenia is unknown. However, direct thrombin inhibitors have been used for both systemic anticoagulation and as a replacement for heparin in the purge solution.[9] The manufacturer notes that use of a purge solution without heparin has not been tested.[43]

Transesophageal echocardiography (TEE) can be used in combination with fluoroscopy to guide implantation. Successful implantation with TEE guidance alone has also been described when fluoroscopy is unavailable.[44] In the case of a left-sided transvalvular heart pump, the guidewire should be visualized in the lumen of the aorta, crossing the aortic valve and terminating in the left ventricle, with the tip directed toward the apex. The tip of the wire can be visualized crossing the aortic valve and terminating in the left ventricle using the midesophageal long-axis view. Worsening mitral regurgitation may occur with guidewire positioning because of interference with the mitral subvalvular apparatus. The midesophageal aortic valve long- and short-axis views can be used to verify normal aortic valve movement and rule out tethering or aortic valve injury. The descending aorta, aortic arch, and ascending aorta views should also be visualized to rule out aortic dissection. Once proper guidewire position is verified, the transvalvular heart pump is advanced over the wire under echocardiographic and fluoroscopic guidance. For the Impella 5.5, the device should be inserted 5 cm into the left ventricle from the aortic valve annulus (fig. 4). For the remaining left-sided devices, the inlet should terminate 3.5 cm into the ventricle (fig. 4). The tip of each device should be directed toward the left ventricular apex and sit in the middle of the ventricular cavity to prevent suction events. This should be verified using multiple two-dimensional views or a three-dimensional view of the left ventricle. A thorough examination of the aorta, mitral and aortic valves, and pericardial space should be performed after transvalvular heart pump implantation to promptly identify injuries to these structures. Transvalvular heart pump placement may also precipitate arrhythmias, which can often be managed by adjusting device position.

Echocardiographic guidance can also assist with Impella RP placement. The RP is inserted into the femoral vein and advanced antegrade over a guidewire, placed with the aid of a flow-directed catheter, across the tricuspid and pulmonary valves and into the pulmonary artery. As with left-sided device placement, the procedural steps can be visualized with echocardiography; correct positioning of the blood inlet (inferior vena cava) and outlet areas (4 cm into the pulmonary artery from the pulmonary valve annulus) should be confirmed; and surrounding structures, including the pericardial space, pulmonary artery, and tricuspid and pulmonary valves, should be examined to rule out injury.

For the sake of simplicity, we will primarily focus on the initial optimization of left-sided transvalvular heart pumps. Once a left-sided device is appropriately positioned, pump flow is initiated and increased gradually to allow the right ventricle to adapt to the increasing preload provided by the device and to allow for real-time hemodynamic and echocardiographic assessment. Color Doppler imaging should be used to confirm flow into the inlet (located in the left ventricle) and out of the outlet (located in the ascending aorta). Malpositioning of a left-sided transvalvular heart pump (inlet and outlet areas both within the left ventricle or both within the aorta) will cause recirculation and will not provide circulatory support. With correct positioning and optimization of pump flow rates, the interventricular septum should be in a neutral position when visualized in the midesophageal four-chamber view.

Deviation of the interventricular septum toward the right ventricular lateral wall may indicate inadequate left ventricular decompression, which may be due to malposition of the blood outlet area within the left ventricle. This will be associated with inadequate circulatory support and a lack of pulsatility (for devices with a differential pressure sensor) or increased pulsatility (left ventricular pressure waveform for devices with a fluid-filled or fiber-optic pressure sensor) on the placement screen of the Automated Impella Controller (fig. 2; fig. A1; table 1). If the transvalvular

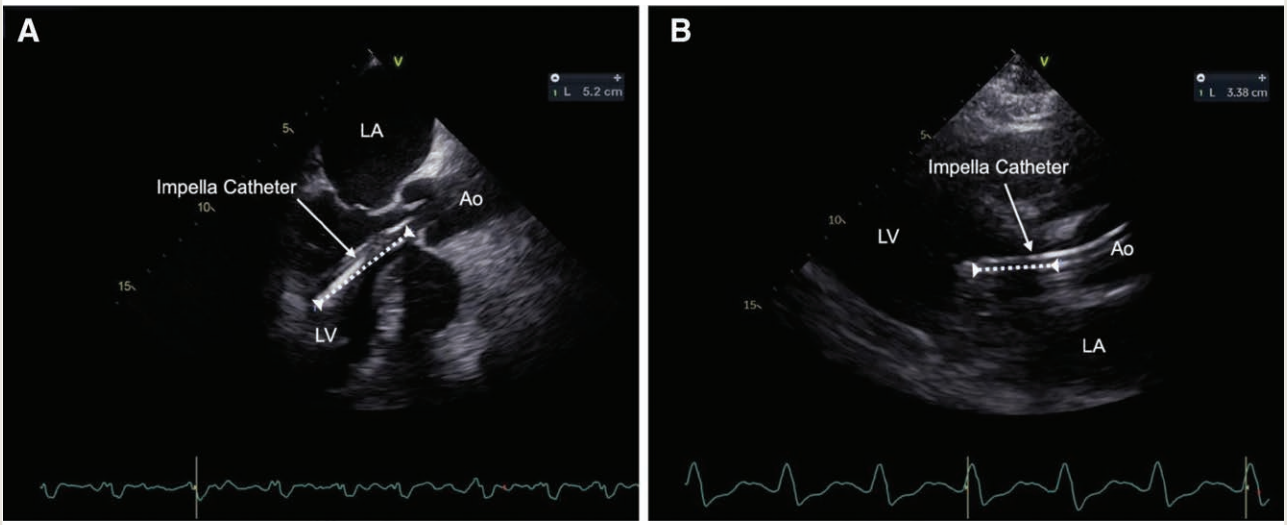

Fig. 4. Impella (Abiomed Inc., USA) placement. (*A*) The Impella 5.5 should be inserted 5.0 cm into the left ventricle, measured from the aortic valve annulus (*dashed line*). A midesophageal long-axis view is shown. (*B*) The Impella CP should be inserted 3.5 cm into the left ventricle (*dashed line*). A parasternal long-axis view is shown.

heart pump is noted to be inappropriately positioned, the device flow rate should be reduced, and the device should be repositioned under echocardiographic and fluoroscopic guidance. If rightward septal deviation remains despite appropriate device position, this can be managed by increasing transvalvular heart pump flows in the setting of inadequate cardiac output.

Deviation of the interventricular septum toward the left ventricular anterolateral wall as visualized in the midesophageal four-chamber view may indicate excessive left ventricular decompression due to pump flows that are too high, right heart failure causing inadequate left ventricular filling, or hypovolemia. Even if leftward septal shift is not caused by right ventricular failure, the resultant right ventricular distension will increase right ventricular wall stress and afterload and can lead to right ventricular decompensation. Device flow rates should be temporarily decreased, and the presence of right ventricular dysfunction or hypovolemia should be identified and treated.

Uriel *et al.*[45] have proposed a framework that can be used to evaluate the cardiovascular response to changing pump speeds after device implantation. This framework may be used to complement TEE evaluation. They propose monitoring the central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), and cardiac index as pump speeds are increased in a stepwise fashion. If achievable, the pump speed that normalizes PCWP, CVP, and cardiac index should be used.[11,45] As an example, patients with isolated left ventricular failure undergoing left-sided transvalvular heart pump placement may present with an elevated PCWP, normal CVP, and reduced cardiac index. As pump speeds are increased, PCWP should fall. If the right ventricle is able to increase right-sided output in response to the increase in venous return, CVP should remain within normal limits, and cardiac index should rise. A brisk rise in CVP and a greater-than-expected reduction in PCWP may indicate the need for additional right ventricular support.

Similarly, escalating pump speeds of a right-sided transvalvular heart pump in isolated right ventricular failure should reduce CVP and increase cardiac index with a minimal change in PCWP. A significant rise in PCWP may indicate the need for additional left ventricular support.

If additional left or right ventricular support is required, this may be achieved with escalation of inotropic support or initiation of pulmonary vasodilators, in the case of concomitant right ventricular failure. When this is inadequate, temporary mechanical circulatory support, including transvalvular heart pump placement, should be considered. In the case of BiPella (both left-sided Impella and Impella RP) support, it is critical to balance pulmonary and systemic blood flow.

If PCWP and CVP are both elevated despite increasing pump speeds and a normal cardiac index, the patient may be fluid overloaded and may benefit from diuretic therapy. On the other hand, if PCWP and CVP are both low, the patient may benefit from fluid administration.

Device Management

Patients receiving transvalvular heart pump support may require surgical intervention. Initial intraoperative management should include confirmation of appropriate device position and assessment of ventricular size and function by echocardiography. The adequacy of circulatory support

should also be continuously evaluated intraoperatively. Blood pressure, cardiac index, urine output, lactate, and mixed venous oxygen saturation are readily available indicators of tissue perfusion that should be monitored by the anesthesiologist. If there are signs of hypoperfusion, echocardiography and pulmonary artery catheter data can be used to elucidate the cause and to optimize pharmacologic and mechanical circulatory support. The potential causes of inadequate circulatory support and options for management are summarized in table 2 and described in the following paragraphs.

Hypoperfusion despite transvalvular heart pump support may be due to improper device positioning or device malfunction.[42] Device positioning was discussed in previous sections of this review and should be assessed when the expected pump flow cannot be achieved or when a suction event occurs. A suction event is a reduction in pump flow due to complete or partial obstruction of the blood inlet area. Suction events can be caused by incorrect device positioning (*i.e.*, blood inlet area abutting the ventricular wall), aspiration of thrombus, hypovolemia, or right ventricular failure leading to inadequate left ventricular preload. If a suction event occurs, device flow should be reduced until the etiology of the suction event is identified and addressed. Pump flows can then be returned to previous levels. Device malfunction is rare but often requires exchange or placement of an alternative mechanical circulatory support device. Malfunction is most frequently caused by aspiration of thrombus into the pump and typically presents with a motor current spike followed by motor current instability and hemolysis.[42]

Inadequate preload is a common cause of inadequate pump flow and may be due to hypovolemia or failure of the unsupported ventricle.[42] Hypovolemia should be suspected if filling pressures decrease in the setting of a constant pump speed, particularly in procedures with significant blood loss or coagulopathy. Assessment of ventricular size on echocardiography will also provide information on the etiology of inadequate preload. In particular, a low cardiac output associated with a reduction in both right and left end-diastolic volumes suggests that the patient is hypovolemic and will respond to volume administration. Fluid responsiveness can also often be assessed by passive leg raise, Trendelenburg positioning, or fluid challenge. In hypovolemia without concomitant dysfunction of the unsupported ventricle, a rapid fluid bolus should improve cardiac output without inducing a rapid significant rise in CVP or PCWP.

Failure of the unsupported ventricle may also cause inadequate preload to the supported ventricle, leading to low pump flow. Optimizing right ventricular function is one of the cornerstones of the management of left-sided transvalvular heart pumps. Poor right ventricular function often presents with an elevated CVP and reduced pulmonary artery pulsatility index.[46] Pulmonary artery pulsatility index is equal to pulmonary artery pulse pressure divided by CVP. A pulmonary artery pulsatility index less than 1.0 was found to be a highly sensitive marker for right ventricular failure in acute myocardial infarction,[47] while a pulmonary artery pulsatility index less than 1.85 was a sensitive predictor of right ventricular failure after left ventricular assist device implantation.[48] Echocardiographic signs of right ventricular dysfunction include worsening right ventricular dilation, a leftward shift in the interatrial or interventricular septum, and a reduction in parameters such as right ventricular fractional area change, tricuspid annular plane systolic excursion, and tissue Doppler-derived tricuspid lateral annular systolic velocity. Right ventricular output may be improved by reducing afterload with pulmonary vasodilators such as inhaled prostacyclin or nitric oxide and increasing contractility with inotropic agents such as epinephrine or milrinone. Right ventricular afterload may also be minimized by optimizing mechanical ventilation to achieve the lowest mean airway pressures and positive end-expiratory pressure that effectively avoids hypercarbia, atelectasis, and hypoxemia.[49] Left-sided transvalvular heart pump flows should also be adjusted to avoid leftward shift of the interventricular septum, with the goal of keeping the interventricular septum midline. Increasing systemic vascular resistance can reduce flattening of the interventricular septum by inducing a net increase in the pressure gradient between the device inlet and outlet areas. If this occurs, and rotations per minute are held constant, pump flow will be reduced, and left ventricular volumes will increase. In addition to decreasing leftward septal shift, increasing systemic vascular resistance can also be helpful in acutely reversing a suction event. Right-sided mechanical circulatory support, including use of the Impella RP should be considered in refractory right ventricular failure.

In the setting of an Impella RP, insufficient support may be caused by concomitant left ventricular failure. Under these conditions, cardiac output will remain unchanged or increase slightly with increasing RP flows, but the incremental improvement may not be enough to provide adequate circulatory support.[8] Left ventricular volumes and pressures may also increase significantly with escalating Impella RP flow rates, leading to pulmonary edema.[8] As such, biventricular failure must be identified and managed when initiating Impella RP support. Signs of left ventricular failure include an elevated PCWP, as well as worsening mitral regurgitation, left ventricular dilation, and poor ventricular contractility on echocardiography. Additionally, a cardiac power output less than 0.6 W is an indicator of ongoing cardiogenic shock.[50,51] Cardiac power output is equal to mean arterial pressure times cardiac output divided by 451. Management of left ventricular failure should include afterload optimization and consideration for additional inotropic or left ventricular mechanical support.

Table 2. Intraoperative Causes of Inadequate Circulatory Support in Transvalvular Heart Pump Therapy

Causes	Corroborating Findings	Treatment Options
Device-related complications		
Malposition	• Improper placement on echocardiography • Automated Impella Controller (Abiomed Inc., USA) showing inappropriate placement signal	• Reposition device
Malfunction	• Presence of thrombus on echocardiography • Motor current spike and instability • Abrupt cessation of pump flow	• Exchange device
Inadequate preload		
Hypovolemia	• Reduced biventricular filling on echocardiography • Improvement in systemic blood flow with fluid challenge • Reduction in CVP and PCWP • Presence of intraoperative bleeding	• Administer crystalloid, packed red blood cells • Treat coagulopathy
Dysfunction of unsupported ventricle	• Reduced ventricular function on echocardiography • No improvement in systemic blood flow with fluid challenge • *Right ventricular dysfunction*: elevated CVP and low/normal PCWP, reduced pulmonary artery pulsatility index • *Left ventricular dysfunction*: elevated PCWP and low/normal CVP, pulmonary edema	• Administer/escalate inotropic support • Optimize ventricular afterload* • Adjust device flows to optimize ventricular shape† • Place mechanical circulatory support device in unsupported ventricle • Rule out/treat arrhythmia, tamponade
Increased metabolic demand	• Inadequate depth of anesthesia • Hyperthermia	• Treat underlying cause • Increase depth of anesthesia • Avoid hyperthermia • Consider muscle paralysis, tracheal intubation
Insufficient maximum device flow	• Persistent hypoperfusion despite maximum device flow	• Place higher flow device • Place additional mechanical circulatory support device

*Pulmonary vasodilators/adjustment of ventilator settings to reduce right ventricular afterload. Reduction in vasopressor support to reduce left ventricular afterload. †Transvalvular heart pump flows should be adjusted to obtain a neutral interventricular septum (*e.g.*, reducing left-sided device flows can decrease flattening of the interventricular septum in the setting of excessive left ventricular decompression).

CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure.

Ventricular function may be influenced by factors outside of intrinsic myocardial contractility, such as arrhythmia or cardiac tamponade. Depending on the hemodynamic status and precise rhythm abnormality, an arrhythmia may be managed medically or electrically, with pacing, cardioversion, or defibrillation. If cardiopulmonary resuscitation is indicated, device flow rates should be reduced while resuscitation is performed. After recovery of cardiac function to preresuscitation levels, pump flow rates may be restored. Because chest compressions may result in transvalvular heart pump displacement,[52] correct pump placement should be confirmed with imaging and assessment of the placement signal on the Automated Impella Controller. Tamponade in the setting of left-sided support presents with an elevated CVP without equalization of pressures,[42] and pericardial effusion with chamber collapse on echocardiography. The effusion should be drained to restore ventricular function and cardiac preload.

Inadequate circulatory support may also be due to increased metabolic demand,[42] which should be managed by treating the underlying cause. Intraoperatively, this may also require deeper levels of anesthesia, tracheal intubation, muscle paralysis, and avoidance of hyperthermia. Despite minimizing metabolic demand, confirming appropriate device function and position, and optimizing volume status and ventricular function, transvalvular heart pump therapy still may not provide sufficient support despite maximum pump flow. This should prompt consideration for escalation to a device capable of higher maximum flow or placement of an additional mechanical circulatory support device.

Weaning and Explantation

Due to a lack of standardized practice guidelines, the timing and management of weaning from transvalvular heart pump support is often determined by institution-specific protocols. In general, weaning should be considered once patient hemodynamics have stabilized (mean arterial pressure of at least 65 mmHg and heart rate less than 100 beats/min) on less than moderate doses of pharmacologic support, systemic arterial pulsatility (left-sided device), and pulmonary arterial pulsatility (right-sided device) have increased, indicators of tissue perfusion have improved (lactate less than 2 mmol/l), and end-organ dysfunction is resolving.[53] Under these conditions, transvalvular heart pump flows can be gradually reduced in a stepwise fashion while monitoring hemodynamics, mixed venous oxygen saturation, cardiac index, and ventricular function with echocardiography. Real-time calculated cardiac power output, which is available with SmartAssist (Abiomed Inc.) technology on the Impella CP and 5.5 devices, should not decrease

significantly during the weaning process.[53] If the transvalvular heart pump is removed in the operating room, the same monitors and parameters used during device implantation, including pulmonary artery catheterization and echocardiography, may be used to ensure adequate ventricular function and end-organ perfusion. Low doses of inotropes and vasopressors, including epinephrine and norepinephrine, are frequently administered to facilitate the weaning process and device explant. The transvalvular heart pump is typically removed once the activated clotting time is less than 150 to 160 s. In cases of inadequate myocardial recovery, a decision should be made regarding patient candidacy for escalation to durable ventricular assist device or heart transplantation. If irreversible multiorgan failure has occurred, withdrawal of mechanical circulatory support may also be considered.

Future Directions

With advancements in device technology,[54] the clinical uptake of transvalvular heart pump therapy will likely continue to grow. Larger, high-quality randomized controlled trials are needed to further clarify optimal clinical indications, patient selection criteria, and outcomes compared to standard therapy and alternative mechanical circulatory support devices. One such ongoing study is the Danish–German cardiogenic shock trial (ClinicalTrials.gov NCT01633502), a randomized controlled trial comparing Impella CP to conventional circulatory support in patients with acute myocardial infarction complicated by cardiogenic shock.[55] As use of the transvalvular heart pump in the treatment of cardiogenic shock increases, anesthesiologists will increasingly be called upon to care for patients who are receiving or may require mechanical support. A comprehensive understanding of the transvalvular heart pump, its mechanics, hemodynamic effects, and considerations for perioperative management will allow anesthesiologists to provide optimal patient care and contribute to the perioperative decision-making process.

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Competing Interests

Dr. Naka is a consultant for Abbott (Abbott Park, Illinois), Biomet-Zimmer (Warsaw, Indiana), and CryoLife (Kennesaw, Georgia) and a speaker for Nipro (Osaka, Japan). Dr. Dickstein is a consultant for LivaNova (London, United Kingdom) and Abiomed (Danvers, Massachusetts) and is cofounder of PVLoops, Inc. (Remsenburg, New York). The other authors declare no competing interests.

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Appendix

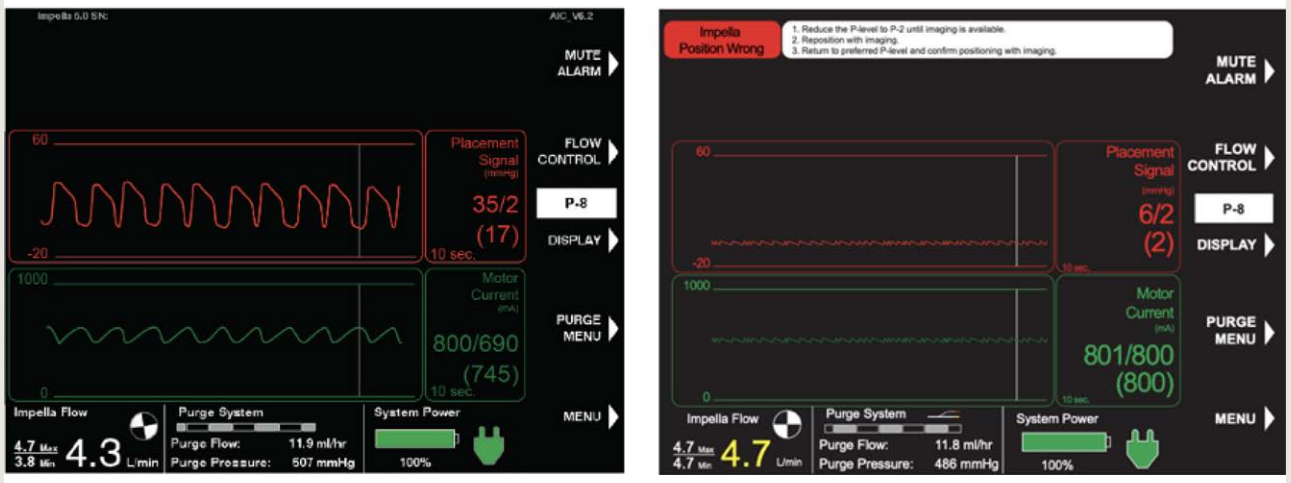

Fig. A1. Placement screen. Placement screens generated by a differential pressure sensor (Impella LD and 5.0) in an appropriately placed transvalvular heart pump (*left*) and inappropriately placed transvalvular heart pump (*right*) are shown. (*Right*) The inlet and outlet areas of the pump are in the same chamber (either the aorta or ventricle), resulting in no pulsatility in the placement signal or motor current. Adapted and reproduced with permission from Abiomed Inc. (USA).

Table A1. Overview of Major Studies

Reference	Design	Patient Population	N	Device(s)	Findings	Limitations
Seyfarth *et al.*[13]	Prospective, randomized	Cardiogenic shock after acute MI	26	Impella 2.5 (Abiomed Inc., USA) *vs.* intraaortic balloon pump	• Greater change in cardiac index from baseline at 30 minutes in Impella group: 0.49 ± 0.46 l/min/m^2 (Impella) *vs.* $0.11 \pm .31$ l/min/m^2 (intraaortic balloon pump), $P = 0.02$ • No difference in 30-day mortality: 46% in both groups	• Small N • Underpowered for mortality analysis
Ouweneel *et al.*[14]	Prospective, randomized	Cardiogenic shock after acute MI*	48	Impella CP *vs.* intraaortic balloon pump	• No difference in 30-day all-cause mortality: 46% (Impella) *vs.* 50% (intraaortic balloon pump), $P = 0.92$ • Major vascular/bleeding complications were higher in the Impella group	• Small N • Underpowered
Ouweneel *et al.*[15]	Meta-analysis	Cardiogenic shock after acute MI	95	Impella 2.5/CP *vs.* intraaortic balloon pump	• No difference in 30-day and 6-month all-cause mortality • No difference in follow-up left ventricular ejection fraction in subset of patients	• Retrospective • Small number of included studies/ total N • Included studies with different inclusion criteria
O'Neill *et al.*[16]	Retrospective analysis	Cardiogenic shock after acute MI	15,259	Impella 2.5/CP/5.0	• 51% survival to explantation • Wide variation in survival rates between institutions • Higher-volume centers, pre-PCI Impella implantation, use of right heart catheter associated with higher survival rate	• Retrospective • No control arm
Schrage *et al.*[17]	Retrospective, matched-pair analysis with Intraaortic Balloon Pump in Cardiogenic Shock II trial patients	Cardiogenic shock after acute MI	237 matched pairs	Impella 2.5/CP *vs.* intraaortic balloon pump or medical therapy	• No difference in 30-day mortality: 48.5% (Impella) *vs.* 46.4% (intraaortic balloon pump or medical therapy), $P = 0.64$ • Higher rates of major bleeding and peripheral vascular complications in Impella group	• Retrospective
Dhruva *et al.*[18]	Retrospective, propensity-matched analysis	Cardiogenic shock after acute MI	1,680 matched pairs	Impella device(s) not specified *vs.* intraaortic balloon pump	• Higher risk of in-hospital mortality in Impella group: absolute risk difference 10.9% (95% CI, 7.1 to 14.2%), $P < 0.001$ • Higher risk of in-hospital major bleeding in Impella group: absolute risk difference 15.4% (95% CI, 12.5 to 18.2), $P < 0.001$	• Retrospective
Griffith *et al.*[19]	Prospective, single-arm	Postcardiotomy shock	16	Impella 5.0/LD	• 94% survival until next therapy • 13% incidence of major adverse events • Improvement in cardiac index, MAP, pulmonary artery diastolic pressure	• Small N • No control arm
Anderson *et al.*[20]	Prospective, single-arm	Right ventricular failure after MI or cardiac surgery	30	Impella RP	• 73% survival to 30 days or hospital discharge • Improvement in cardiac index, reduction in CVP after device initiation	• Small N • No control arm
Anderson *et al.*[21]	Retrospective analysis	Right ventricular failure after MI or cardiac surgery	60	Impella RP	• 72% 30-day survival rate • Improvement in cardiac index, reduction in CVP after device initiation	• Retrospective • Small N • No control arm

*This study included only patients with ST-segment elevation myocardial infarction.

CVP, central venous pressure; MAP, mean arterial pressure; MI, myocardial infarction; PCI, percutaneous coronary intervention.

Table A2. U.S. Food and Drug Administration Approval Indications

Device	Approved Indications	Maximum Duration
Impella 2.5 (Abiomed Inc., USA)	High-risk PCI*	6 h
	Refractory cardiogenic shock†	4 days
	COVID-19 emergency use authorization‡	4 days
Impella CP	High-risk PCI*	6 h
	Refractory cardiogenic shock†	4 days
	COVID-19 emergency use authorization‡	4 days
Impella LD	Refractory cardiogenic shock†	14 days
Impella 5.0	Refractory cardiogenic shock†	14 days
	COVID-19 emergency use authorization‡	14 days
Impella 5.5	Refractory cardiogenic shock†	14 days
	COVID-19 emergency use authorization‡	14 days
Impella RP	Acute right heart failure§	14 days
	COVID-19 emergency use authorization‖	14 days

*Performed electively or urgently in hemodynamically stable patients with severe coronary artery disease. †Because of left ventricular failure in the setting of acute myocardial infarction, cardiac surgery, cardiomyopathy, and myocarditis. ‡In patients with COVID-19 and heart failure from myocarditis while on veno-venous ECMO or with pulmonary edema while on veno-arterial ECMO. §In the setting of acute myocardial infarction, heart transplantation, left ventricular assist device implantation, or cardiac surgery in patients with a body surface area greater than or equal to 1.5 m^2. ‖In patients with acute right ventricular failure caused by complications from COVID-19.

ECMO, extracorporeal membrane oxygenation; PCI, percutaneous coronary intervention.

Table A3. Contraindications

Contraindications to left-sided Impella (Abiomed Inc., USA)
- Presence of a mechanical aortic valve
- Presence of left ventricular thrombus
- Inability to tolerate anticoagulation
- Presence of an atrial or ventricular septal defect
- Severe peripheral arterial disease
- Severe aortic stenosis/≥2+ aortic regurgitation

Contraindications to Impella RP
- Presence of a mechanical tricuspid or pulmonary valve
- Presence of right-sided thrombus
- Presence of vena caval filter
- Severe tricuspid or pulmonary valve stenosis/regurgitation

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