Mechanical Circulatory Support Management

Options beyond the cath lab.

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Temporary mechanical circulatory support (MCS) is often required in patients with cardiogenic shock and selected patients undergoing complex, high-risk percutaneous coronary intervention (PCI). When used in the setting of a reversible cause (eg, fulminant myocarditis), temporary MCS may be a bridge to recovery. Conversely, in the setting of nonreversible or slowly reversible causes, temporary MCS can provide the time needed for patients and physicians to evaluate the course of the underlying disease, weigh the risks and benefits, and decide about long-term MCS, heart transplantation, or palliative care.

The most commonly used MCS devices in clinical practice are the intra-aortic balloon pump (IABP), Impella (Abiomed, Inc.), and venoarterial extracorporeal membrane oxygenation (VA-ECMO). In this article, we discuss the management of temporary MCS beyond the cardiac catheterization laboratory.

MONITORING

Monitoring all types of MCS includes periodic assessment of efficacy (hemodynamics) and safety (anticoagulation and arterial access site).

Hemodynamics

Patient hemodynamics should be continually monitored. In cardiogenic shock patients, this is usually achieved with an arterial line and a Swan-Ganz catheter (the latter is increasingly being used for high-risk PCI as well). Inotrope and vasopressor use should be minimized, if possible, to maximize the likelihood of myocardial recovery.

Anticoagulation

Intravenous anticoagulation is commonly used to prevent thrombus formation and distal embolization. Optimal anticoagulation levels are not well defined; however, for IABP, the target activated partial thromboplastin time is usually 60 to 75 seconds.[1] Although there are significant variations in anticoagulation practices, the manufacturer recommends a target activated clotting time of 160 to 180 seconds for Impella.[2,3] Anticoagulation cannot be administered in some patients (eg, in those with active bleeding or a hemor-

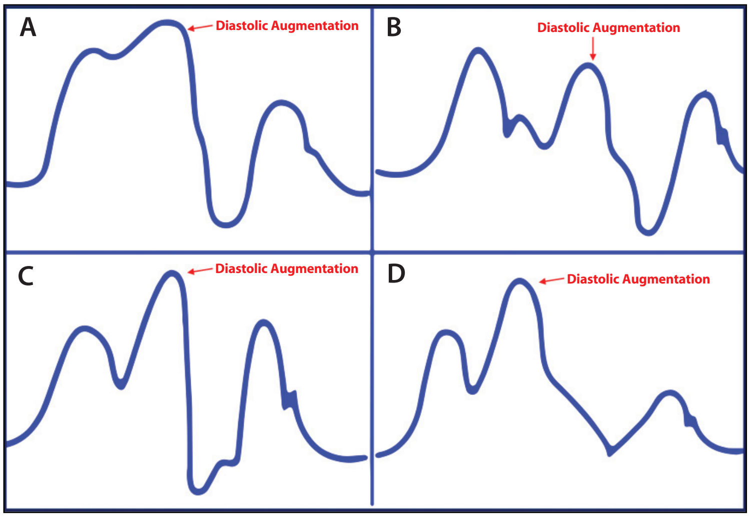


Figure 1. IABP troubleshooting. Early inflation can cause potential premature closure of the aortic valve and increase left ventricular filling pressure and left ventricular wall stress and afterload (A). Late inflation can result in suboptimal coronary artery and peripheral perfusion (B). Early deflation: can cause suboptimal coronary perfusion, potentially causing retrograde coronary and carotid blood flow; it also leads to suboptimal afterload reduction and increased myocardial oxygen demand (C). Late deflation can result in increased afterload and myocardial oxygen consumption because the left ventricle is ejecting against greater resistance (D).

IABP TROUBLESHOOTING

POOR CARDIAC OUTPUT AUGMENTATION

Causes and Treatment

- Incorrect timing of balloon inflation and deflation can be corrected by adjusting inflation and deflation on the console (Figure 1)
- A leak in the balloon catheter requires prompt changing of the balloon catheter
- For a leak in the gas circuit resulting in poor inflation, all connections from the catheter to the gas cylinder should be checked and tightened as necessary; a kinked catheter may require replacement
- In the case of poor cardiac function or low vascular resistance, add positive inotropic and vasoactive agents or escalate therapy, if appropriate
- Dysrhythmias such as atrial fibrillation or ventricular tachycardia can impair IABP function; although contemporary algorithms can help adjust for tachycardia, treating the tachyarrhythmia will improve the IABP efficacy

LOSS OF ELECTROCARDIOGRAM TRIGGER

Causes

- Poor electrode placement, low electrocardiography (ECG) voltage, faulty electrode pads or cables, dysrhythmias, and other equipment's interference with the ECG signal

Treatment

- Switch to the arterial pressure trigger until the underlying course is corrected

LOSS OF PRESSURE TRIGGER

Causes

- Improper transducer setup, occluded arterial line

Treatment

- Ensure that the pressure bag is inflated to 300 mm Hg
- Check the patency of the arterial line by withdrawing blood, then flushing
- Confirm the transducer position and calibration

AUTOFILL FAILURE/NO HELIUM

Causes

- Insufficient amount of gas in the tank or occlusion of the gas outlet

Treatment

- Check the amount of gas in the tank and replace the tank as needed

BLOOD DETECTED

Causes

- IABP balloon rupture

Treatment

- Perform emergent IABP removal to prevent thrombus formation in the balloon

rhagic stroke). MCS can be used in such patients without anticoagulation, at the cost of increased thrombotic risk. In IABP patients who do not receive anticoagulation, 1:1 counterpulsation should be used to minimize the risk of device thrombosis; the pump should never be stopped for > 30 minutes. Performing VA-ECMO without anticoagulation is possible due to developments in equipment, such as heparin-bonded tubing and heparin-coated polymethylpentene oxygenators.[4] However, the possibility of oxygenator thrombosis remains.

Arterial Access

All MCS can lead to vascular access site complications, the risk of which increases with larger arterial sheaths. Access sites should be assessed hourly for bleeding, hematoma, and signs of infection. Moreover, the patient's limbs should be monitored for signs of ischemia (color, sensation, temperature, movement, capillary refill) every 15 minutes during the first hour and then hourly thereafter.

Hemolysis

Hemolysis can occur with any MCS due to increased shear forces on the red blood cells. According to INTERMACS,minor hemolysis is defined as plasma-free hemoglobin > 20 mg/dL or a serum lactate dehydrogenase [LDH] > 2.5 times the upper limit of normal (ULN) range after the first 72 hours postimplantation in the *absence of* clinical symptoms, hemolysis, or abnormal pump function.[5] Major hemolysis is defined as plasma-free hemoglobin > 20 mg/dL or an LDH level > 2.5 times the ULN occurring after the first 72 hours postimplantation and *associated with* clinical symptoms, hemolysis, or abnormal pump function). Major hemolysis requires the presence of one or more of the following conditions: hemoglobinuria (tea-colored urine), anemia (decrease in hematocrit or a hemoglobin level out of proportion to levels explainable by chronic illness or usual MCS care), hyperbilirubinemia (total bilirubin > 2 mg/dL, with predominately indirect component), and MCS malfunction and/or abnormal MCS parameters. If significant hemolysis occurs, the type of MCS may need to be changed.

DEVICES

IABP

In addition to the previously mentioned necessary assessments, there are additional key monitoring aspects for an IABP.

Positioning. Daily chest x-rays should be performed to confirm optimal device positioning in the descending thoracic aorta distal to the left subclavian artery origin. Distal IABP migration is common and may result in renal ischemia and reduce the hemodynamic efficacy of the counterpulsation. Pedal and radial pulses and perfusion should be checked regularly because device migration could affect upper and lower limb perfusion.

Timing. The timing of inflation and deflation should be monitored to ensure optimal IABP function. The IABP should be placed in a 1:2 inflation ratio to ensure that the augmented systolic pressure is higher than the nonaugmented and the postaugmentation diastolic pressure is lower than the diastolic pressure after a nonaugmented beat (Figure 1).

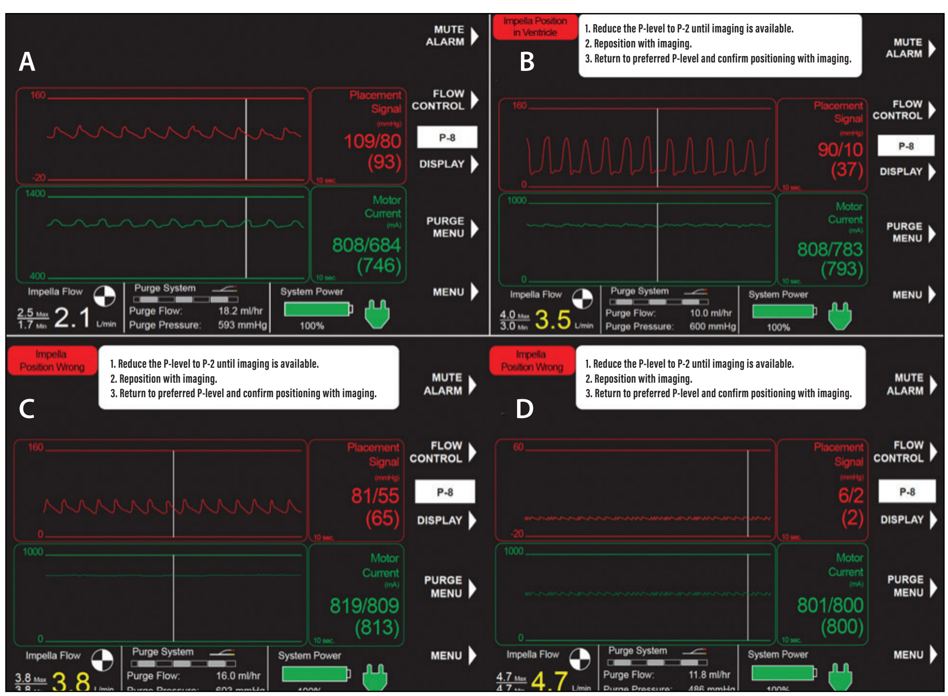
Urine output. IABP distal migration can impair renal artery flow, which can affect renal perfusion and ultimately decrease urine output.

IABP tubing. The IABP tubing should be assessed hourly for any signs of blood or helium leak.

Aortic dissection. Monitor for aortic dissection (see the *IABP Troubleshooting* sidebar).

Impella

In addition to radiography and echocardiography, appropriate Impella placement is verified by the pressure waveform generated from the pressure sensor at the distal end of the pump. The placement signal is used to verify whether the Impella pump is correctly placed by evaluating the pressure differential on a pulsatile wave-



Courtesy of Abiomed, Inc.

Figure 2. Correct position (Impella 2.5 and CP): placement signal, aortic; motor current, pulsatile (A). Device position in the ventricle (Impella 2.5 and CP): placement signal, ventricular; motor current, flat (B). Incorrect pump position (Impella 2.5 and CP): placement signal, aortic; motor current, flat (C). Incorrect pump position (Impella 5.0): placement signal, flat; motor current, flat (D).

IMPELLA TROUBLESHOOTING

POSITION ALARM

Causes

- Device migration

Treatment

- Reduce flow rate to P2
- Reposition the pump under echocardiographic guidance until the inlet area is placed 3.5 cm below the aortic valve annulus; if this fails, return to the cardiac catheterization laboratory and reposition the device under fluoroscopy
- Resume support at previous P level setting

SUCTION ALARM

Causes

- Suboptimal Impella device position or low left ventricular filling pressure (eg, due to hypovolemia or right ventricular failure

Treatment

- Drop 1 or 2 P levels or until suction alarm stops
- Assess volume status
- Evaluate catheter position with imaging; reposition if necessary
- Assess right ventricular function
- Rule out tamponade
- Return the flow rate to the prealarm setting when suction has resolved

PURGE PRESSURE HIGH

Causes

- Kinks in the purge tubing or purge fluid concentration is too high

Treatment

- Check purge tubing for kinks
- Decrease concentration of dextrose in purge solution

PURGE PRESSURE LOW

Causes

- Leaks in the purge system tubing or purge fluid concentration is too low

Treatment

- Check purge system tubing for leaks
- Increase concentration of dextrose in purge solution
- Replace purge cassette

HEMOLYSIS

Causes

- Inflow obstruction due to suboptimal Impella positioning causing the blood to travel slower through the pump, thus increasing the exposure time to the higher shear stresses near the impeller
- Obstruction within the pump (eg, by clot) can create flow disturbances near the impeller, resulting in high shear stress and hemolysis
- If the outflow windows are obstructed by the aortic valve, blood will exit the pump at higher speeds from unobstructed regions and make contact with the aortic valve and other obstructing structures, resulting in high shear force and hemolysis

Treatment

- Confirm optimal positioning of the Impella by echocardiography and/or fluoroscopy
- Intravenous crystalloid administration if hemolysis is accompanied by central venous or pulmonary capillary wedge pressure < 10 mm Hg
- Reduce the P level
- If there is evidence of uncontrolled severe hemolysis, the device may need to be removed

VA-ECMO TROUBLESHOOTING

VENOUS CHATTER (OSCILLATIONS OF THE VENOUS CANNULA DUE TO VENOUS/CAVAL COLLAPSE)

Causes

- Hypovolemia, bleeding, high intrathoracic or intra-abdominal pressures, high VA-ECMO flow, cannula malposition

Treatment

- Decrease VA-ECMO flow rate
- Treat underlying cause

OXYGENATOR FAILURE

Causes

- Excessive clot formation

Treatment

- Optimize anticoagulation
- Replace oxygenator

EXCESSIVE LEFT VENTRICULAR LOADING

Causes

- Increased afterload due to VA-ECMO flow

Treatment

- Increased inotropic support
- Vasodilation with nitroprusside
- Venting with IABP, Impella, or central cannulation

HARLEQUIN SYNDROME

Causes

- Increased afterload due to VA-ECMO flow

Treatment

- Increased inotropic support
- Mechanical ventilation
- Left ventricular venting
- Conversion to veno-veno-arterial ECMO, splitting the outflow between the femoral artery and a new cannula that is inserted in the superior vena cava

form. Appropriate placement can also be assessed on the display using the motor current waveform, which is a measure of the Impella pump's energy intake. The energy used by the Impella device varies with motor speed and the pressure difference between the inflow and outflow areas of the pump. When the Impella device is correctly positioned with the inlet area in the left ventricle and the output in the ascending aorta above the aortic valve, the motor current should be pulsatile because of the pressure difference between the two areas.[3,6] When the intake and output are on the same side of the aortic valve, the motor current will be dampened due to the lack of a pressure differential (Figure 2).

Because of the lower profile of the Impella catheter shaft (9 F) compared with the Impella inflow/outflow, the original peel-away sheath (17-F outside diameter) used for inserting the device can be removed and a repositioning sheath (13-F outside diameter) advanced through the arteriotomy, decreasing the risk of lower extremity ischemia. Downsizing the Impella sheath may, however, lead to bleeding around the sheath that could be minimized by tightening two Perclose ProGlide sutures (Abbott) if they have been placed around the arteriotomy at the time of Impella insertion (see the *Impella Troubleshooting* sidebar).

VA-ECMO

Limb ischemia. Because of the often large size of arterial cannulas used for VA-ECMO, an antegrade perfusion catheter is usually placed in the corresponding limb.[7] Near-infrared reflectance spectroscopy monitoring is used to ensure that tissue oxygen saturation in the lower extremities is maintained at > 50%.

Clot formation in the VA-ECMO circuit and oxygenator. Systematic monitoring of the circuit and oxygenator with a flashlight is recommended to detect thrombus formation, which may lead to oxygenator failure or stroke. The most common causes of clot formation are subtherapeutic anticoagulation and slow pump flow.

Swan-Ganz monitoring and arterial waveform. VA-ECMO increases left ventricular afterload that may adversely affect left ventricular function, causing pulmonary edema and poor gas exchange that can lead to Harlequin syndrome. Monitoring the left ventricular filling pressures (with a Swan-Ganz catheter, showing increasing pressures) and the arterial waveform (showing narrow pulse pressure or absence of dicrotic notch, suggesting the aortic valve is not opening) is critical for determining the need for left ventricular venting, which may be accomplished using an IABP, Impella, or central cannulation.

Harlequin syndrome. Harlequin syndrome occurs when poorly oxygenated blood perfuses the coronary arteries and the cerebral arteries, impeding myocardial recovery and possibly leading to neurologic injury.[8] Monitoring oxygen saturation in the fingers of the right hand can allow early detection of Harlequin syndrome and corrective action (see the *VA-ECMO Troubleshooting* sidebar).

ESCALATION/DE-ESCALATION AND REMOVAL

The need for continuation, escalation, or de-escalation of MCS should be continually assessed, considering the potential for severe complications due to MCS. End-organ function (neurologic, renal) should also be monitored. Cardiac function is assessed using a combination of invasive hemodynamics and echocardiography. Maintenance of systemic blood pressure while turning down the support level is encouraging because it suggests that cardiac function can support hemodynamics without MCS. In VA-ECMO patients, the duration of low pump flow should be minimized due to an increased risk of thrombosis.

CONCLUSION

When the decision is made that MCS is no longer needed, anticoagulation is held until coagulation is adequate for removal. The IABP should be maintained at 1:1 support and VA-ECMO flow should be > 2.5 to 3 L/min during this time to minimize the risk of thrombosis. IABPs are usually removed at bedside using manual compression, whereas Impella and VA-ECMO are often removed surgically, with repair of the arteriotomy sites. ■

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