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Physiology of Continuous-Flow Pumps

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ABSTRACT

The use of mechanical pumps for circulatory support started in the mid-1950s. The evolution of these devices has led to the present-day use of continuous-flow pumps to take over the function of a patient's failing heart. The physiology associated with rotary blood pump use is quite different from normal cardiovascular physiology. Clinicians caring for patients who are supported by rotary blood pumps must have an understanding of the differences in physiology, monitoring methods, and unique complications associated with the use of these pumps.

Keywords: Continuous-flow pumps, mechanical circulatory support, monitoring, physiology, rotary blood pump, ventricular assist devices

The use of artificial pumps for circulatory support is a concept that was first described in the 1950s, when a bubble oxygenator was used to maintain a patient's life during the closure of an atrial septal defect.[1] During the 1960s and 1970s, the field of mechanical circulatory support (MCS) began with the introduction of a variety of methods to support or replace function of the failing heart. Advancements at that time were encouraged and supported by the National Heart, Lung and Blood Institute, which issued a series of requests for proposals for electrically powered mechanical devices to provide circulatory support.[2] By the mid-1980s, the first total artificial heart had been implanted, and patients were successfully being supported by pulsatile ventricular assist devices as a bridge to transplant.[2] The first pulsatile devices were approved for use by the US Food and Drug Administration (FDA) in the early 1990s. The Abiomed BVS 5000 (Abiomed, Inc, Danvers, Massachusetts), Thoratec PVAD (Thoratec Corporation, Pleasanton, Massachusetts), and the HeartMate IP (ThermoCardiosystems, Burlington, Massachusetts) were pulsatile devices that required that patients be attached to a large console and remain in the hospital for an extended period of time.

Home use of an MCS device did not occur in the United States until the trial and FDA approval of the Novacor LVAS (Novacor, Oakland, California) and the HeartMate XVE (ThermoCardiosystems) in 1998. For the first time, patients were able to live at home with an artificial pump providing circulatory support for their failing heart. Due to the size and frequent complication rate of pulsatile devices, the continuous-flow pump (CFP) used for circulatory support has now taken over the field.[3] Cardiovascular physiology related to rotary blood pump function is described in this article as well as pertinent issues unique to the care of patients with CFPs in the critical care setting.

What Is a CFP?

A CFP is a mechanical device that is implanted into a patient with cardiogenic shock or end-stage heart failure to augment or take over the function of the failing ventricle.[4] It is typically attached to the cardiovascular system with an inlet cannula in the left ventricular apex and an outlet cannula attached to the ascending aorta.[4] Other configurations, including right ventricular cannulation to deliver blood to the pulmonary artery (PA), are possible; therefore, clinicians must be aware of the cannulation points of a pump in any patient who is under

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their care. If issues arise with the stability of the patient, knowledge of cannulation sites is imperative for troubleshooting the altered cardiovascular physiology related to the CFP.

Continuous-flow pumps come in the form of axial-flow pumps and centrifugal pumps.[4] Both types of pumps spin at a speed that is set by the clinician who operates the device. Neither type of CFP has valves; thus, both types will allow retrograde blood flow if pump speed is not adequate, if pump function stops, or if inadequate preload or excessive afterload is present.

An axial-flow pump in its basic form is a tube with an impeller (propeller-type component) suspended inside. Typically, a bearing to which the impeller is attached keeps it centered within the tube. The impeller spins within the tube creating a suction, which pulls blood into the tube through the inlet opening and pushes it out through the outlet opening (Figure 1). An axial-flow pump implanted into a patient with end-stage heart failure is attached to the left ventricular apex with an inlet cannula (tube). The cannula is inserted and secured into a hole that is created in the apex of the left ventricle and connected to the pump. The outlet of the pump is connected to another cannula (outlet cannula), which is attached to a hole that is created in the ascending aorta. When the pump begins to spin, blood is pulled into the pump from the left ventricle by the suction created by the impeller and then pushed to the rest of the body via the cannula attached to the ascending aorta (Figure 2). The impeller spins continuously, so the blood flow pattern of a patient who is fully supported by the axial-flow pump is laminar and not pulsatile.[4]

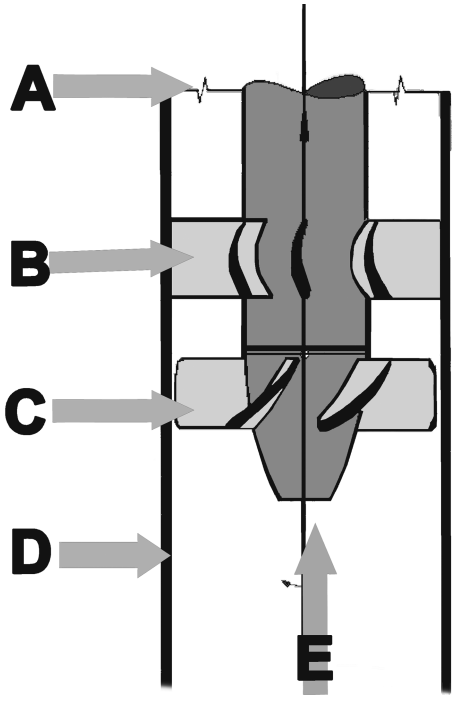

Figure 1: Diagram of a basic axial flow pump. A: Flow straightener. B: Motor stator. C: Impeller. D: Pump external tubing. E: Fluid flow direction.

A centrifugal pump is mechanically different from an axial-flow pump in that the centrifugal pump changes the fluid flow direction to move the blood through the pump. A centrifugal pump has an outer casing that resembles a cone. Inside the pump is an impeller that is typically suspended within the outer casing by a magnetic field or fluid layer. The pump is powered and the impeller spins by using an electromagnetic field. The inlet opening for the pump is at the top of the cone, and blood is pulled into the pump by suction created by the spinning impeller. Once inside the pump, the blood then changes direction and is pushed out through an opening in the side of the pump using centrifugal force (Figure 3).[5] Like the axial-flow pump, the centrifugal pump is attached to the cardiovascular system using inlet and outlet cannulas. The inlet cannula typically feeds from the left ventricle, and the outlet cannula takes blood to the ascending aorta. Centrifugal pumps come in many sizes. Some are manufactured for short-term use and reside outside the body, whereas others are designed for long-term use and are implanted inside the body.

The Cardiovascular System and MCS Devices

Adding an MCS device to the cardiovascular system changes cardiac physiology. To understand MCS therapy, one needs to break the heart down into its basic functional parts: structural, electrical, and mechanical.

Structural System

The structural components of the heart are key in determining the source of heart failure and how the patient will respond to CFP therapy. Depending on the site of failure, blood can flow too slowly (valvular stenosis, heart failure), in the wrong direction (valvular incompetence), or not at all (cardiac arrest). All patients who require the use of a CFP have heart failure from some cause. The overwhelming majority

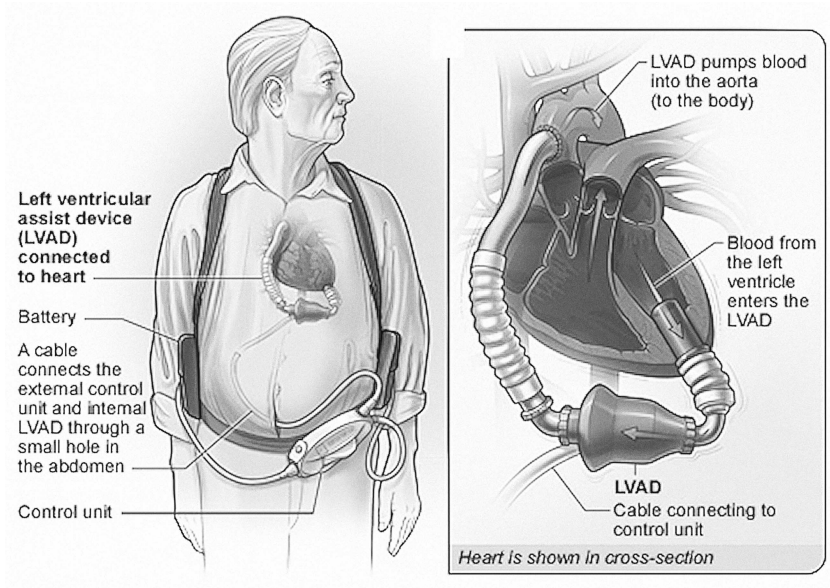

Figure 2: Placement of the axial-flow pump within the patient's body. From http://www.nhlbi.nih.gov/health/health-topics/topics/vad/.

of patients have a systolic dysfunction caused by some injury to the heart on a cellular level (infarction, viral, or chemical). Cardiac myocytes become enlarged and unorganized (apoptosis) and, if infarction has occurred, can be replaced by scar tissue. The change in the cellular structure of the cardiac cells produces a distention in the affected area of the heart and a loss of contractile function that leads to the patient's medical diagnosis of dilated cardiomyopathy. If valvular dysfunction is also present, blood flow patterns and efficiency of flow through the heart are compromised, decreasing cardiac output, which can cause catastrophic consequences for the patient.

Existence of a CFP changes blood flow patterns within the heart. The CFP augments or replaces the function of the failing ventricle in which it was placed. Blood is drawn from the failing ventricle into the pump and returned to the body through the distal great vessel. In the case of a left-sided CFP, blood is drawn from the left ventricular apex and returned to the ascending aorta beyond the aortic valve. Mitral and aortic valve function must be intact for the system to work efficiently. If there is a compromise in either valvular structure, CFP efficiency is affected. Knowing how those patterns are affected is key in the care and troubleshooting of issues surrounding the cardiovascular system of patients with a CFP.

A CFP itself can also produce abnormal physiological strains on the different structures of the heart, including the ventricular walls and valves. Constant backward pressure on the aortic valve can cause aortic insufficiency in patients with a CFP. Constant suction pulling blood from the failing ventricle can cause the ventricular wall to collapse into the inlet cannula of the pump, impeding pump flow. Constant suction in the failing left ventricle produced by the pump can displace the ventricular septum, causing

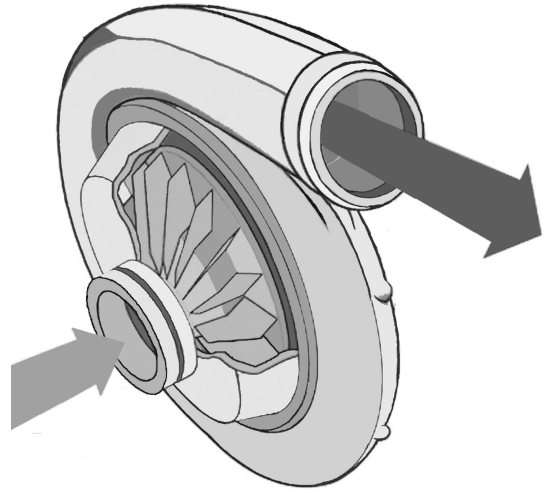

Figure 3: Diagram of a basic centrifugal pump.

distortion of the tricuspid valve, which leads to tricuspid insufficiency. Any abnormal physiological strain created by the CFP can produce structural failure if left unchecked, leading to catastrophic consequences for patients.

Electrical System

The electrical system of the heart acts to produce a mechanical response from the myocytes, which in turn gives the characteristic contraction that we measure through noninvasive and invasive monitoring as blood pressure. The electrical system of the heart is essential to the function of a normal heart. Without an intact electrical system, the myocytes would not contract properly, which in turn would produce a malfunction of the mechanical system of the heart. In patients without a CFP, arrhythmias can produce life-threatening consequences. With a CFP in place, the electrical system of the heart becomes less significant because forward blood flow is maintained by the pump and not by the mechanical response of the myocytes related to the electrical activity within the heart. The CFP operates irrespective of the electrical impulses generated by the heart. Arrhythmias that are lethal to patients who are not supported by a CFP may cause few or no symptoms in patients who are supported by a CFP.[6]

Mechanical System

The mechanical system of the heart plays the leading role for patients with an MCS device. The mechanical function of the heart produces the forward flow of blood that we feel as a patient's pulse and measure in the form of blood pressure. When the ventricles of the heart contract, blood is pushed to the lungs and the body. For patients with cardiogenic shock or end-stage heart failure, the mechanical function of the heart is severely compromised.

Normal heart function is measured in terms of cardiac output. A patient with impaired cardiac function has a low cardiac output, inadequate to sustain metabolic functions. When a CFP device is attached to a compromised heart, forward blood flow is restored. The device either augments or takes over the function of the failing left ventricle, which, in most cases, allows the patient to recover and function similarly to a patient without heart failure.

What About a Pulse?

A CFP moves blood in a constant flow pattern and, therefore, does not, in itself, produce a palpable pulse in the patient in which it is implanted. If the patient is partially supported, the mechanical function of the heart produces a diminished pulse that may or may not be palpable. When a patient is fully supported by a CFP, the patient may not have a palpable pulse, and monitoring blood pressure and oxygen saturation is difficult.

Controversy persists as to whether life can be sustained without a palpable pulse. Currently, a significant amount of data shows that life can be sustained without a pulse, but unique complications, such as arteriovenous malformation in the gastrointestinal system and in the nose, have occurred in this subset of patients.[7] The cause of these complications is still under investigation, and researchers have many theories as to why they may occur.

What Do I Need to Know?

Cardiovascular assessment of patients with a CFP is markedly different from that of patients who are not supported by a CFP. Different assessment techniques need to be used throughout all stages of patient support. Although MCS technology is advanced, assessment techniques require a return to low-technology methods that require basic nursing skills.

What Does the Patient Look Like?

Patients who arrive in the intensive care unit (ICU) after implantation of a CFP have similar hemodynamic monitoring equipment in place as other postoperative heart patients. Typically, an arterial catheter is in place to monitor blood pressure, and a PA catheter is in place to monitor central venous pressure (CVP) and PA pressures. Patients are treated with mechanical ventilation and commonly have several chest tubes to drain blood from their surgical sites. Patients with short-term devices commonly have the pump inlet and outlet cannulas coming from either a femoral insertion site or directly through the sternal wound (Figure 4). The pump then sits outside the patient's body and can be viewed and monitored by nursing staff. Patients with long-term devices commonly have a driveline, which is tunneled under the skin and generally exits on either side of the patient's abdomen. The driveline is attached to a small computer or controller, which, in turn, is attached to an electrical source. Long-term pumps are implanted within the chest cavity or in a pocket made by the surgeon just below the diaphragm on the left side. Surgical wounds in

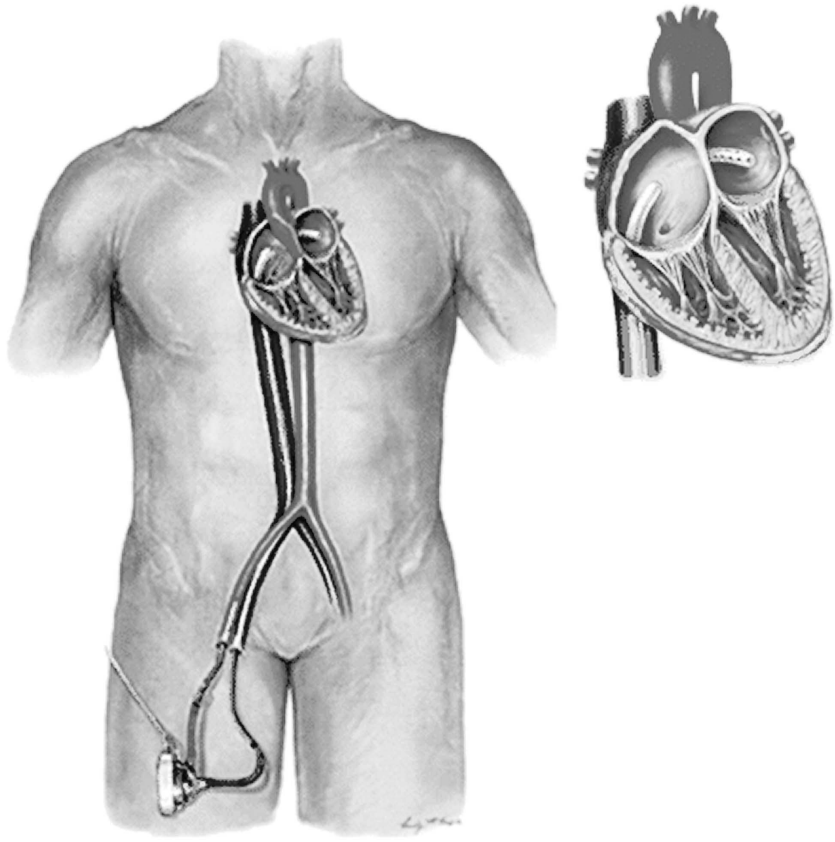

Figure 4: TandemHeart percutaneous centrifugal pump. Used with permission from CardiacAssist Inc.

these patients will consist of a sternal incision site and most likely a drain coming from the pump pocket if one exists. These pumps are not visible but can be palpated (vibration is felt) and auscultated (presence of a humming sound) to grossly determine functionality.

The External Parts

All CFPs have a controller and a power source. Power is provided to the pump either through a wall socket or from batteries. Power is connected to a pump controller that functions as the "brain" of the system. The controller is the computer interface that dictates how fast the impeller spins and monitors the CFP for malfunctions. Integral to the controller is a monitor that provides a visual interface that is used to display the current functioning parameters of the pump and reprogram its settings when necessary. The visual interface gives information about key components that are universal to all CFPs: speed of the pump in revolutions per minute, pump output or flow (either estimated or measured with a flow sensor) in liters per minute, battery status, and alarm status. Pump speed is the only parameter that clinicians can change. In most institutions, only designated personnel change pump speed, and normal pump speeds vary depending on the manufacturer of the CFP being used. The CFP's visual interfaces also report the amount of power (in watts) that is being used to propel the impeller to spin. All of these parameters should be recorded as vital signs in any patient supported by a CFP. An abrupt change in any of these parameters or the presence of an active alarm is indicative of a problem with the patient and/or the pump and needs to be investigated.

Electrocardiograph Monitoring

Electrocardiograph monitoring is essential in any ICU patient, and patients with CFPs are

not any different. Care must be taken to monitor arrhythmias and intervene appropriately. Many arrhythmias are not as emergent in a patient with a CFP as in a patient without a CFP, but urgency should be used to evaluate the cause of the arrhythmia and to prevent future occurrences. Arrhythmias can range from atrial fibrillation to ventricular fibrillation and asystole or anywhere in between. If a patient is experiencing an arrhythmia and remains stable hemodynamically, the situation can be assessed and treated in a less urgent fashion. If hemodynamic compromise occurs, treatment of the arrhythmia should be dealt with immediately.

Hemodynamic Monitoring

Hemodynamic monitoring using invasive catheters postoperatively is essential. Fluid shifts typical of any postoperative cardiac surgery patient occur in patients with CFPs as well and should be anticipated and treated postoperatively. Technical issues related to surgical anastomosis sites, although rare, can occur postoperatively and will be evidenced by increased bleeding as well as inability to hemodynamically stabilize the patient, despite fluid boluses and vasopressor use. Device speed adjustment is also performed frequently postoperatively, and changes in pump performance are reflected in the hemodynamic picture produced through invasive monitoring techniques. Problems related to any of these issues can be common and, if detected early, can be corrected without affecting the outcome of the patient.

Blood Pressure Measurement

Depending on the level of CFP support, an arterial catheter waveform will appear anywhere from normal to flat line (Figure 5). Arterial pressure should be monitored, but no hard-and-fast guidelines exist as to what the pressure value represents or an optimal target range, although a mean arterial pressure of 70 to 90 mm Hg is usually the norm.[8] Speed of the pump does not affect systolic blood pressure as much as it does diastolic blood pressure. Because a CFP provides blood flow during all phases of the cardiac cycle, blood flow that would not be present during a patient's normal cardiac diastole is created by the CFP, which increases the patient's diastolic blood pressure, which in turn narrows the patient's pulse pressure.[9]

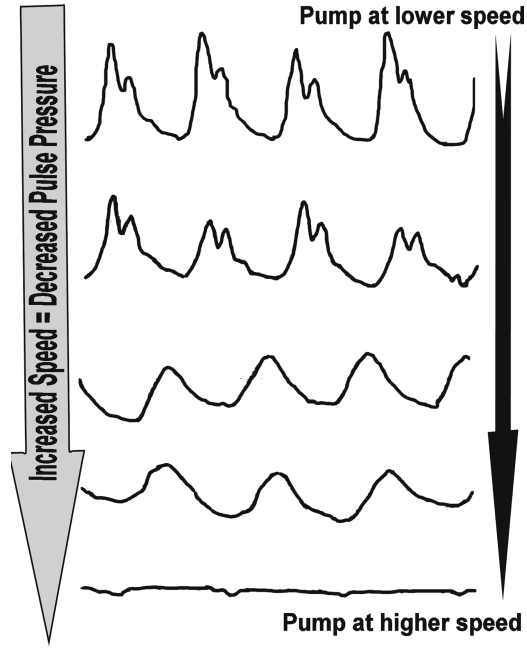

Figure 5: How changes in pump speed affect arterial pulse pressure waveforms.

Automatic blood pressure monitors may or may not provide a systolic, diastolic, or mean value, depending on the pulse pressure of the supported patient. Manual blood pressure readings are difficult to auscultate without the use of a Doppler probe to determine when the flow is returned to the radial artery. Using the Doppler probe on the radial artery, clinicians should record the pressure at which the "swish" of blood is heard, indicating the return of blood flow to the radial artery. Typically, an arterial pressure that allows the patient to have a urine output of at least 1 mL/kg per hour with good peripheral perfusion is adequate. Caution should be exercised for high arterial pressures (>90 mm Hg) with fresh surgical anastomoses because increased surgical site stress and bleeding can occur. Changes in trends of arterial pressures, as well as abrupt differences in waveform appearance when invasive catheters are present, should be investigated. An abrupt change in arterial pressure can indicate blood loss, volume overload, impaired pump efficiency, impaired pump flow, cardiac structure failure, or myriad other issues that require further investigation.

Central Venous Pressure Monitoring

Central venous pressure monitoring is the most sensitive, objective postoperative monitoring

parameter available. Central venous pressure should be kept in a range that provides adequate preload for the CFP without overwhelming the right ventricle. Typically, a CVP of at least 12 mm Hg is necessary to meet this criterion. A CVP greater than 20 mm Hg is an indicator of right ventricular failure that is mechanical in nature, obstructive in nature (tamponade), or due to severe volume overload (rare), and it signals the need for inotropes or placement of a support device for the right ventricle.[10]

Pulmonary Artery Pressures

Pulmonary artery pressures are also important for immediate postoperative management. Pulmonary artery pressures should be kept as low as possible to help support the right ventricle, decreasing the amount of afterload that it must pump against, which, in turn, provides adequate preload to the left side of the heart and the CFP. An abrupt or steady rise in PA pressures could indicate many things, from pulmonary hypertension and pulmonary edema to pump malfunction or structural malfunction of the heart. These complications should be investigated and reported immediately.

Pulse Oximetry

Pulse oximetry is an invaluable tool in the ICU and can be used to monitor both basic and complex patient functions. Unfortunately, pulse oximetry is not always reliable when caring for a patient with little or no pulse.[11] Pulse oximetry readings can be monitored, but in the event of low-oxygen saturation measurements, accuracy should be checked if no other indications of patient instability exist. Blood gas measurements continue to be the criterion standard to determine adequate oxygenation in patients, including those with a CFP.[11]

Nursing Assessment

Assessing the patient with a CFP requires a return to basic assessment skills. As the patient recovers from surgery, the readings obtained from blood pressure cuffs, monitors, and oximeters become less significant and should be combined with an overall assessment of the patient. Patients who are adequately supported by a CFP have good peripheral circulation (their legs and fingers are warm and pink), have brisk capillary refill (<5 seconds), exhibit appropriate mentation (they follow commands appropriately; they can talk to you, have a normal conversation with you, do not fall asleep while talking to you, and remember who you are), and have adequate urine output (unless renal failure is a concern). With all of these variables present and in the absence of alarms from the CFP controller, adequate support is being provided to the patient.

Physiological Factors That Affect Pump Flow

Pump flow is a product of pump speed and, because CFPs do not have valves, the pressure gradient across the pump.[9] For CFPs to function properly, the pressure gradient needs to be adequate enough to allow unimpeded blood flow at whatever speed the pump is programmed to run. For this reason, preload and afterload play a significant role in pump performance.

Preload

A CFP is a machine. It has no intuitive capabilities. The device is programmed to run at a certain speed, which remains fixed until it is changed by a health care provider. Currently, no feedback mechanisms are available for human use that allow a pump to monitor the patient's physiological requirements and decrease or increase speed accordingly.

All MCS devices are preload dependent. If inadequate blood is available for the pump to propel, the output of the pump decreases. This decrease occurs despite the CFP spinning at the programmed speed set by the clinicians. As the CFP continues to run, a negative pressure or suction is produced in the inlet cannula of the pump. When preload is very low, this negative pressure leads to suction events within the ventricle.[12]

Suction Events

A suction event is characterized by the lack of adequate preload to the pump, which in turn increases negative pressure within the left ventricle. With the increase in negative pressure generated by the pump, a part of the ventricular wall is sucked over and covers the pump inlet cannula, which can produce an arrhythmia in the patient.[12] In this situation, the pump will alarm and automatically decrease the speed to release the suction. This type of event is common in patients who have inadequate left-sided heart filling pressures (pulmonary capillary wedge pressure or left atrial pressure), and the cause, whether volume, pressure,

or obstructive in nature, needs to be identified and addressed immediately.

Assessing Inadequate Preload

Decreased preload can be a function of many variables. The easiest to identify and treat is hypovolemia. The most common causes of hypovolemia in patients with a CFP are surgical bleeding, increased urine output from diuretic administration, third spacing of fluids, or inadequate replacement of fluids postoperatively. In patients with a CFP, as decreasing trends in CVP occur (although they may still be >10 mm Hg), pump flow decreases without changes in speed, and overall fluid balance is negative. Hypovolemia is treated with a fluid bolus. Although each institution has its own protocols as to whether to use crystalloid or colloid infusions for fluid replacement, either acts to expand circulating blood volume and therefore treat the hypovolemia, which has caused the CFP's low-flow state.

In the absence of hypovolemia, decreased pump preload is a function of right-sided heart failure, pulmonary hypertension, or some type of flow obstruction into the pump, such as tamponade. All of these causes require an echocardiographic evaluation to determine blood flow patterns through the heart and into the pump inlet cannula.[13] Right-sided heart failure can be treated using inotropes; however, if severe, a second pump to support right-sided heart function is indicated. Pulmonary hypertension can be treated using inhaled nitric oxide[14] or a pulmonary vasodilator such as epoprostenol sodium or sildenafil citrate.[15] Pulmonary hypertension that cannot be managed is a lethal scenario in any MCS patient because preload cannot be adequately supplied to the CFP, causing decreasing output from the pump, which eventually will spiral out of control. Flow obstruction into the pump can come in the form of a clot (rare), incorrect cannula placement within the left ventricle, or tamponade. Any of these scenarios, if symptoms are severe, require that the patient be returned to the operating room for surgical exploration.

Afterload

The amount of blood that is pushed out of a CFP depends on how much pressure the pump has to work against (afterload). Signs of increased afterload include decreasing pump flows with a consequent increase in right-sided pressures, increased systemic vascular resistance, increased arterial pressures, and decreasing power usage (as shown on the pump-monitoring screen). Increased afterload is caused either by a patient's increased vascular tone or by an obstruction of flow on the outlet side of the pump. Elevated vascular tone can be treated using vasodilators either intravenously for the short term or orally for the long term. Care needs to be exercised when administering any of these drugs to ensure adequate circulating blood volume. If overlooked, issues with preload into the pump may arise.

Causes of obstruction to outflow can result from clot formation, twisting of the outflow cannula, and tamponade.[13] Afterload issues arising from obstruction of flow out of the pump require echocardiographic evaluation for diagnosis. All of these causes require that the patient be returned to the operating room for surgical exploration to alleviate the problem.

Abnormal Physiological Effects of a CFP

The presence of a CFP within a patient's cardiovascular system presents abnormal physiological stresses on the structure of the heart and vascular system. Continuous-flow pumps provide a continuous flow of blood to a patient's ascending aorta. Unlike normal heart function in which distinct pressure and flow differences exist between systole and diastole, CFPs provide a continuous flow of blood and a relatively steady amount of pressure to the vascular system. One of the major effects of continuous blood flow is the backward pressure exerted on the aortic valve. If a patient is partially supported and his or her left ventricle is allowed to fill and contract, allowing the aortic valve to open frequently, the amount of abnormal physiological stress exerted on the aortic valve is minimized. If a patient is fully supported with complete decompression of the left ventricle, which does not allow the aortic valve to open, abnormal physiological stress on the aortic valve can become dangerous.

Backward pressure exerted on the aortic valve eventually leads to aortic insufficiency. If allowed to progress, this condition can lead to a circular path of blood from the left ventricle through the pump and back into the left ventricle through the insufficient valve,[16] which reduces forward flow of blood to the body and can lead to lethal complications. Signs of aortic insufficiency in a patient with a CFP include

decreased arterial pressure, increased device flows, increased PA pressures, and elevated CVP. Pulmonary edema can occur because of lack of forward blood flow from the left atrium. Patients with severe aortic insufficiency require echocardiographic examination for diagnosis with aggressive afterload reduction.[13] Aortic valve repair or replacement may be necessary to allow for continued CFP support.

Hematologic effects of a CFP are also characteristic of a rotary blood pump. Von Willebrand's factor is a large protein that is present in the blood and that aids in platelet function and blood clot formation. Continuous-flow pumps have been reported to cause a deficiency in the large-chain monomers associated with von Willebrand's factor as a result of shear stress that is caused when the blood is passed through the CFP. The spinning action of the pump acts to break down the proteins, which in turn creates a hypocoagulable state in the patient.[17] Many of the bleeding complications of patients who have been supported by a CFP have been linked to this deficiency. Current research in this area is focused on finding ways to reduce the shear stress on the blood cells in order to decrease the incidence of this phenomenon.

Summary

Continuous-flow pumps have changed the way that we think about how to care for a patient with heart failure. Many patients, who as recent as 10 years ago would have died, are now being supported by implantable CFPs and living relatively normal lives. Experience with these pumps is still in its infancy. Although unique complications have been identified related to CFP use, these complications continue to be largely manageable. As the use of CFPs continues and newer generations of pumps are introduced, clinicians will gain a better understanding of the long-term physiological effects of these pumps. The ultimate goal is to support a patient with a pump that has minimal complications and the ability to last throughout a patient's lifetime.

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