# Cardiac failure: Mechanical support strategies

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***Objective:*** **Mechanical support of the circulation is necessary when heart failure becomes refractory to medical support and is typically applied when organ dysfunction occurs as a result of hypoperfusion. However, in timing the intervention, it is important to apply mechanical support before multiple organ failure occurs. The objective of this work is to review the current strategies for mechanical circulatory support in patients with refractory cardiac failure.**

***Design:*** **A review of the use of mechanical circulatory support is presented for patients with refractory cardiac failure.**

***Patients:*** **Data are taken from human studies that were selected to best exemplify the results that may be obtained from various forms of mechanical circulatory support.**

***Interventions:*** **Commonly applied forms of mechanical support include mechanical ventilatory support, intraaortic balloon counterpulsation, and hemodialysis or ultrafiltration. If these measures fail, mechanical support of the circulation with ventricular assist devices is possible in specialized centers with expertise in the implantation and management of these devices. The decision to pursue mechanical circulatory support in the critically ill patient is based on the cause of acute decompensation, the potential reversibility of the condition, and the possibility for other treatments to improve the underlying condition or, in highly selected cases, heart transplantation. Newer forms of ventricular assistance that require less surgery are becoming available and may allow use in a broader range of critically ill patients.**

***Main Results:*** **There is a range of means to mechanically support the circulation in patients with advanced heart failure.**

***Conclusions:*** **A variety of means to support the circulation have found application in the treatment of patients with refractory heart failure. More work is required to best identify populations who will benefit from the therapy and to refine the therapy to reduce associated risks. (Crit Care Med 2006; 34[Suppl.]:S268–S277)**

**KEY WORDS: heart failure; ventricular assist devices; intraaortic balloon counterpulsation; cardiogenic shock; continuous positive airway pressure; acute myocardial infarction; multiple organ failure; mechanical ventilation; continuous venovenous hemodialysis; ultrafiltration**

When medical therapy fails to provide sufficient support for patients with refractory heart failure or cardiogenic shock, mechanical means of supporting the circulation are necessary. Patients with refractory heart failure face a very high rate of mortality (1–4). The decision to aggressively support the circulation in this setting is based on the cause of acute decompensation, the potential reversibility of the condition, and the possibility for other treatments, such as revascularization or, in highly selected cases, heart transplantation.

The most common cause of acute refractory heart failure or cardiogenic shock is extensive acute myocardial infarction (4, 5). Although cardiogenic shock with acute myocardial infarction typically occurs in patients with a large anterior myocardial infarction, in patients with preexisting left ventricular systolic dysfunction, a smaller myocardial infarction may precipitate cardiogenic shock. If patients initially stabilize postmyocardial infarction and develop shock late in their course, the cause may be related to infarct expansion or re-occlusion of a previously patent infarct artery (6). In addition to acute loss of functioning myocardium, cardiogenic shock and refractory heart failure may result from mechanical complications of acute myocardial infarction, including acute mitral regurgitation, ventricular septal rupture, or free wall rupture.

Cardiogenic shock may also occur as a result of acute myocarditis, myocardial contusion, and progressive end-stage heart failure. It may occur postcardiotomy, particularly after prolonged cardiopulmonary bypass or when myocardial preservation was suboptimal. Occasionally, septic shock will produce severe myocardial depression and cardiogenic shock. Other causes include left ventricular outflow tract obstruction due to aortic stenosis or hypertrophic cardiomyopathy, obstruction to left ventricular filling by mitral stenosis or a left atrial myxoma, acute mitral regurgitation due to chordal rupture, or acute aortic insufficiency.

In a report from the SHould we emergently revascularize Occluded Coronaries for cardiogenic shocK (SHOCK) trial, predominant left ventricular failure was present in 78.5% of patients in the trial and the registry, severe acute mitral regurgitation in 6.9%, ventricular septal rupture in 3.9%, right ventricular failure in 2.8%, cardiac tamponade or rupture in 1.4%, and other causes in the remaining 6.7% of patients (4). Myocardial infarctions were predominantly anterior, occurring in 55% of patients. Mortality in this registry for all patients was 60.1%, with a mortality of 59.2% in patients with predominantly left ventricular failure.

Among acute myocardial infarction patients who develop cardiogenic shock, it is more likely in those who are elderly, are diabetic, have an anterior myocardial infarction, and have had a previous myocardial infarction (1, 2, 4). They are more likely to have a history of peripheral vascular disease and stroke. Coronary an-

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giography typically demonstrates multivessel disease.

## Pathophysiology of Refractory Heart Failure

The pathophysiology of cardiogenic shock has been described as a cascade of events initiated by myocardial dysfunction, which leads directly to both decreased stroke volume and increased left ventricular filling pressures (5). The decrease in stroke volume and cardiac output produces hypotension, thereby compromising coronary perfusion and exacerbating myocardial ischemia, leading to progressive myocardial dysfunction. At the same time, the decreased systemic perfusion leads to compensatory vasoconstriction and fluid retention, further compromising myocardial function with adverse loading conditions (Fig. 1). Recently, an expansion of the paradigm of progression of cardiogenic shock was proposed by Hochman (7), making note of several observations that seem to implicate an inflammatory component to progressive heart failure. These observations include that patients presenting with cardiogenic shock have only a moderately reduced left ventricular ejection fraction (about 30%), a low systemic vascular resistance on average, often a clinically evident systemic inflammatory response syndrome, and survivors of cardiogenic shock often have little or no chronic heart failure symptoms (New York Heart Association class I).

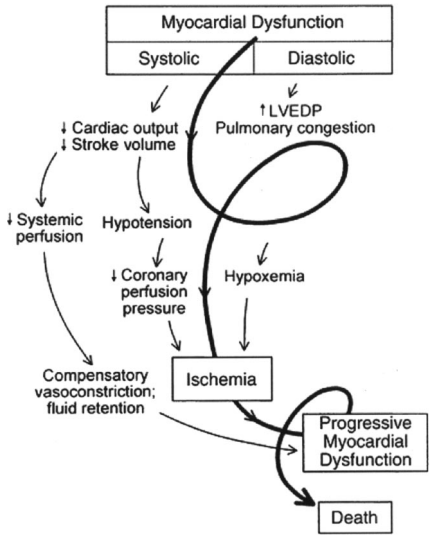


Figure 1. The downward spiral of cardiogenic shock. *LVEDP*, left ventricular end-diastolic pressure.

## Organ Function at Risk

Mechanical support is needed when medical therapy fails to restore adequate blood pressure or organ perfusion. The organs at greatest risk for dysfunction or failure in the setting of cardiogenic shock include the lungs (with pulmonary edema), the kidneys (with oliguria or acute tubular necrosis), the brain, and the liver. Therefore, observation of the function of these organs will typically herald the need for mechanical support of the circulation. However, failure of any of these organs is associated with poor outcomes with mechanical support (8–10). Therefore, intervention is needed in the early stages of organ dysfunction to prevent complete organ failure and death. When blood pressure is not compatible with adequate organ perfusion, despite maximal medical support, one should anticipate organ failure and plan intervention to better support the circulation. Shock is often defined by the absolute blood pressure, such as a mean arterial pressure of $<$60 mm Hg. However, most major organs can autoregulate blood flow as long as organ perfusion pressure is $\geq$50 mm Hg and sometimes as much as 80 mm Hg (11). Organ perfusion pressure is the difference between mean arterial pressure and central venous pressure. If adequate blood pressure cannot be maintained, despite maximally tolerated vasopressor and inotropic support, and there is any clinical indication of organ dysfunction (e.g., oliguria), then prompt intervention is needed.

## Work of Breathing

Acute pulmonary edema creates a number of abnormalities in pulmonary function that greatly increase the work of breathing (12). The lungs become edematous and heavy, decreasing their compliance, and thereby increasing the work required to expand the lungs. The airways are also edematous, causing an acute obstructive pattern, with an increase in the work to both fill and empty the lungs. With progressive edema, some alveoli will become ineffective for gas exchange, and some will be poorly ventilated because of airway edema. Combined with smaller lung volumes because of the restrictive features of pulmonary edema, dead space ventilation increases, creating a need for a high minute ventilation. In all, the work of breathing increases several fold with acute pulmonary edema. This increased work of breathing will create a demand for blood flow in the working muscles of breathing, thereby limiting blood flow to other major organs.

## Renal Perfusion

Renal dysfunction is one of the most common indications to pursue mechanical circulatory support. When pharmacologic therapy fails to produce adequate organ perfusion to maintain renal function, multiple organ failure will rapidly ensue (2, 13). The critical issue is to intervene before permanent organ damage occurs. For example, in the literature of mechanical support with left ventricular assist devices, although renal dysfunction is common before implantation of the device, anuria before implantation is a very poor prognostic feature, and the risk increases with progressive increases in azotemia (8, 10). In addition, the oliguria may lead to progressive volume overload that increases central venous pressure, thereby increasing the preload demand on the heart and mandating a higher mean arterial pressure to maintain organ perfusion.

The kidneys are richly innervated by the autonomic nervous system. Enhanced sympathetic nervous system tone can decrease renal blood flow, even in the presence of adequate cardiac output. However, in the setting of heart failure, the effect of the neural regulation of renal blood flow during exercise is enhanced (14, 15). The increased work of breathing along with the high sympathetic tone associated with refractory heart failure may lead to greatly diminished renal blood flow, even with efforts to restore normal cardiac output.

## Mechanical Ventilation

Although not strictly a form circulatory support, mechanical ventilation, either noninvasive or invasive, can improve a patient's condition with acute, refractory heart failure. A patient with pulmonary edema has a very high work of breathing, which creates demand for blood flow in the working muscles of breathing and diminishes blood flow for major organ functions. The decrease in the work of breathing with mechanical ventilation is most completely accomplished by intubation, full mechanical ventilation, and sedation (16, 17). However, randomized trials of noninvasive positive airway pressure support (continuous positive airway pressure or bilevel positive

airway pressure) in patients with acute pulmonary edema have demonstrated that positive airway pressure decreases respiratory rate while improving ventilation, as demonstrated by a lower $Pco_2$ (17). This has been associated with lower rates of intubation compared with oxygen therapy alone and a trend toward improved survival when compared with medical therapy but not when compared with early intubation.

In addition to the benefit from decreasing the work of breathing, positive pressure will reduce left ventricular afterload (12, 16–18). Left ventricular afterload is defined as the transmural myocardial stress placed on the left ventricular wall. The difference between pressure inside the ventricle during systole (arterial pressure) and outside the ventricle (intrathoracic pressure) determines afterload. Therefore, increased intrathoracic pressure will decrease left ventricular transmural myocardial stress, or afterload. Continuous positive airway pressure has been studied in chronic stable heart failure and seems to have beneficial effects on symptoms of heart failure and left ventricular function. In the normal heart, stroke volume and cardiac output are relatively sensitive to preload, but insensitive to afterload. Therefore, positive airway pressure, which may decrease venous return to the heart, is associated with a decrease in stroke volume, cardiac output, and blood pressure. However, the converse is true in the failing heart, which is relatively insensitive to preload and exquisitely sensitive to afterload. In the acute setting, as long as there is sufficient preload, and in the setting of refractory heart failure there is typically an excess of preload, higher intrathoracic pressures may provide a hemodynamic benefit by reducing afterload. However, caution must be used with relatively high levels of positive airway pressure in the form of continuous positive airway pressure or positive end-expiratory pressure because ventricular filling is eventually compromised.

## Intraaortic Balloon Pump

The design and function of an intraaortic balloon counterpulsation device (or intraaortic balloon pump, IABP) has changed little in the past three decades (19). It is a catheter-based device with two lumens, one of which passes through to the tip of the device and can be used for a guidewire during insertion or to monitor aortic pressure, and the second lumen is connected with a 30- to 50-mL balloon sac. The IABP is typically inserted in the femoral artery and advanced to near the level of the left subclavian artery, being careful not to occlude the artery during inflation (Fig. 2). The proximal aspect of the balloon chamber should be located above the renal arteries to prevent compromise of renal perfusion.

The balloon is designed to inflate with helium during diastole, beginning right after the closure of the aortic valve, using the dicrotic notch of the arterial waveform as a marker for this event. It deflates during isovolumic contraction sufficiently before the onset of ejection to allow diastolic pressure to decrease to levels lower than would occur without pumping. Four pressures are typically monitored during IABP operation, including the systolic pressure, diastolic pressure, and mean pressure. The additional variable is augmented pressure, which is the peak pressure during IABP inflation in diastole. Both systolic and diastolic pressures typically are lower during IABP operation, but the difference is accounted for by the increase in diastolic pressure during augmentation. The augmented pressure is determined by vascular impedence and stiffness. For example, in an elderly patient with atherosclerotic disease of the aorta, one would expect a relatively high augmented pressure, whereas in a young person with a compliant aorta, the augmented pressure would be lower. The mean arterial pressure tends to be slightly higher during IABP operation.

The physiologic effects of IABP operation include increasing coronary perfusion pressure by increasing diastolic pressure and increasing cardiac output primarily by a reduction in left ventricular afterload that occurs after balloon deflation just before systole. However, the increase in systemic perfusion may be modest, $<0.5$ L/min (20, 21). The net effect is to greatly improve the balance between myocardial oxygen supply and demand while creating a modest improvement in systemic perfusion and blood pressure.

*Indications and Clinical Results.* Intraaortic balloon counterpulsation is indicated for medically refractory cardiogenic shock during acute myocardial infarction or after cardiac surgery and for refractory angina (19, 22, 23). It can also be used for other causes of cardiogenic shock and refractory malignant ventricular arrhythmias (24). However, it is contraindicated in the presence of clinically significant aortic insufficiency and aortic dissection. Relative contraindications include severe peripheral vascular disease involving the femoral arteries, iliacs, or aorta, morbid obesity, and abdominal aortic aneurysm.

There have been few randomized studies of the use of the IABP, and the clinical results are implied mainly from observational data. In the SHOCK trial of 1,190 patients, those who were selected for an IABP had lower in-hospital mortality than those who did not receive one (50% vs. 72%, $p < .0001$) (4, 25). In this study, the majority of patients supported with an IABP also underwent revascularization procedures, which further improved outcomes. However, this was not a randomized trial of IABP use, and the results are clearly subject to selection bias.

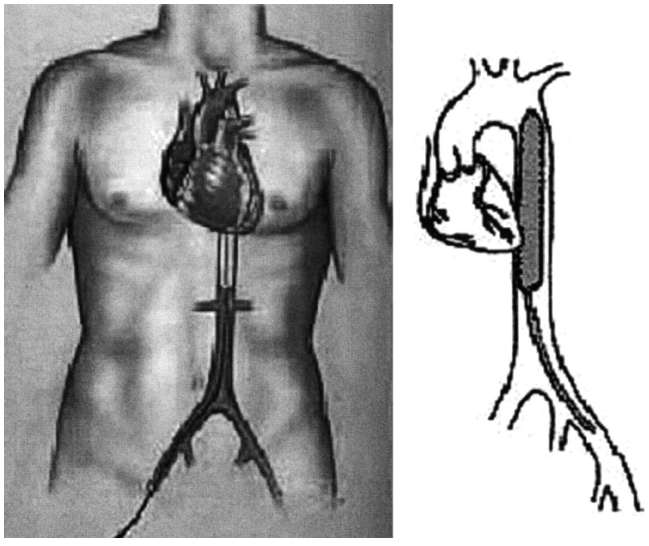

Figure 2. Intraaortic balloon placement.

During the last several years, we have used IABPs surgically placed in the subclavian fossa as a means of supporting selected patients who are awaiting transplant. It is placed in the operating room anterograde into the descending aorta with its distal end just above the renal arteries. Vascular access is obtained with an end-to-side graft placed on the axillary artery. The catheter is tunneled subcutaneously along the anterior chest wall and exits the skin along the lower left sternal border. This approach allows the patient to be ambulatory and can provide up to several months of support, although frequent replacement was necessary due to balloon rupture. To date, we have supported 17 patients for up to 6 months, with 12 being bridged to transplant without the need for additional mechanical support. In general, these patients were bridged with IABP support rather than a left ventricular assist device (LVAD) because of small size and immunologic presensitization, a problem that can be exacerbated by the use of ventricular assist devices (VADs).

*Complications.* Complications of IABP use range from 2% to 47%, with the majority associated with vascular compromise, occurring in 1–20%, and including flow obstruction by the balloon catheter, the need for thrombectomy, or amputation. The rate of complication seems to be decreasing over time, likely reflecting better familiarity with insertion and care of the IABP, along with improvements in design, including smaller-diameter catheters, sheathless insertion techniques, and changes in materials used to construct the device. In a registry of IABP use across a broad range of centers, 2.8% of 16,909 patients treated with an IABP had a major complication and 7.0% had any vascular complication (26). Aortic dissection, aortoiliac laceration or perforation, deep wound infection, catheter infection, and atheroembolic complications are less common complications of IABP use. Fever occurs fairly frequently in patients who are treated with an IABP (27). The fever is often fleeting, although occasionally bacteremia does occur, particularly early after implantation of the pump.

## LVAD

*LVAD Types.* When medical therapy along with mechanical support with an IABP fail to provide sufficient circulatory support, the next means of support is mechanical ventricular assist. This has been evaluated and used extensively in the postcardiotomy shock situation, to bridge patients with refractory heart failure to heart transplantation, and, to a limited extent, in myocardial infarction with cardiogenic shock (28, 29). LVADs have been used in a number of other scenarios with refractory heart failure, including acute myocardial infarction with cardiogenic shock.

Ventricular assist devices are blood pumps and can be divided into several types based on the mechanism of pumping blood. Pulsatile LVADs operate with a sac or diaphragm to create a compressible blood chamber. Valves are used on the inflow and outflow to maintain the forward direction of blood as the blood chamber is emptied by compression and filled as the chamber expands. Two types of pulsatile devices are distinguished by the method of compressing the blood chamber, either by air or by an electric motor and a pusher plate. Examples of these types of pumps are the Thoratec pneumatic LVAD and the HeartMate vented electric LVAD. Both have been widely used successfully for a number of conditions for which mechanical circulatory support was needed. Nonpulsatile blood pumps can be divided into one of two mechanisms used to move blood, axial flow or centrifugal flow (30). Axial pumps use a corkscrew-like impeller that rotates rapidly to propel blood in one direction. Centrifugal pumps introduce blood near the center of a rotating plate, and the spinning action progressively accelerates blood as it moves toward the periphery of the plate. There are multiple advantages and disadvantages of each type of blood pump that will become more apparent as each is discussed. Finally, pumps are also distinguished by their location with respect to the patient. An intracorporeal pump is one in which the pumping mechanism in inside the patient, although energy sources, controllers, and other components may be located outside the patient. An extracorporeal pump is located outside the patient's body. Some extracorporeal pumps are securely located just outside the body and may be referred to as paracorporeal.

*Function.* All LVADs remove blood either directly from the left ventricle or the left atrium and pump it into or near the aorta. In so doing, preload is rapidly removed from the left ventricle. With pulsatile blood pumps, which have valves that separate preload and afterload cleanly, the left ventricle is typically unloaded to the point at which it no longer ejects into the aorta. With nonpulsatile devices, there is typically some degree of native ejection because automatic control of the device is more difficult to accomplish without a clean separation between inflow and outflow of the blood moving through the device. As opposed to the modest increase in flow with an IABP, an LVAD can provide several liters per minute of blood flow to completely replace the work of the left ventricle. As a result, improvement in organ function is often seen after LVAD placement (9) (Fig. 3).

*Indications.* LVADs can be used for short-term support, usually days to a week or two, long-term support, several months to a year or two, and for permanent support. Temporary use is typically indicated for situations in which rapid recovery is anticipated, to stabilize a patient in anticipation of an intervention that may improve cardiac function, or to stabilize a patient until further therapy can be applied. A typical indication is to stabilize a patient with refractory postcardiotomy shock. Long-term use is typical in bridge to heart transplant application. Permanent use has recently become an option for patients with refractory heart failure who are not candidates for heart transplantation.

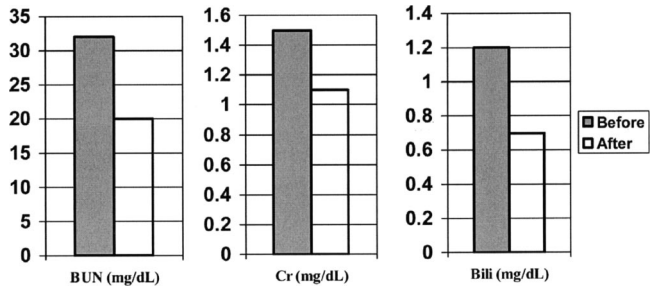


Figure 3. Graph demonstrating improvement in organ function as demonstrated by laboratory values before and after left ventricular assist device placement. *BUN,* blood urea nitrogen; *Cr,* serum creatinine; *Bili,* total bilirubin.

*Pulsatile LVADs.* The Abiomed BVS 5000 is a pulsatile, extracorporeal, pneumatically driven device that is primarily used for conditions that typically require short-term support. It is generally considered technically easy to insert and can be used in the right VAD, LVAD, or biventricular assist device configuration (31, 32). The device is kept at the bedside and consists of two pumping chambers separated by polyurethane valves with cannulas connecting the heart and pumping chambers. The blood chamber acts as a reservoir, and the second blood chamber is compressed pneumatically to eject blood back to the patients. Because of this setup and design, it does not allow for patient mobility as easily as other VADs and is not considered the best option for long-term support. It is primarily used for treatment of postcardiotomy shock and other forms of acute shock, such as that seen with acute myocardial infarction or acute myocarditis (28, 31–35). It has been reported to be used for posttransplant graft failure and refractory ventricular arrhythmias (32, 36, 37). In addition, its use may result in lower survival in patients who are bridged to transplant when compared with patients bridged with the Thoratec VAD. If recovery of ventricular function has not occurred within several weeks and weaning from VAD support is not possible, it can be switched to a Thoratec device or intracorporeal device. It does have the advantage of being less expensive than the Thoratec for short-term support, but, like the Thoratec and Novacor systems, it requires anticoagulation.

The Thoratec VAD is also a pulsatile, paracorporeal, and pneumatically driven VAD. It can be used in the right VAD, LVAD, and biventricular assist device configuration and is typically used for bridging to transplant or recovery and for postcardiotomy shock (38–42). It consists of a single pump with a seamless, smooth sac that is compressed pneumatically during ejection, and a vacuum is used to enhance filling (Fig. 4). The inlet and outlet cannulas each contain a mechanical valve that allow for one-way flow. It is a proven device for more than short-term support and, unlike the Abiomed, allows for patient ambulation and rehabilitation and, with a small, portable pneumatic driver, outpatient management (38–41). In addition to its versatility in configuration, it can be used in small patients as well (43). In centers experienced in its use, the survival in bridge to transplant application seems to be similar to that attained with other pulsatile LVADs (44–46). Because of its mechanical valves and smooth-surface blood-contacting surfaces, it requires anticoagulation to prevent thrombus formation within the device.

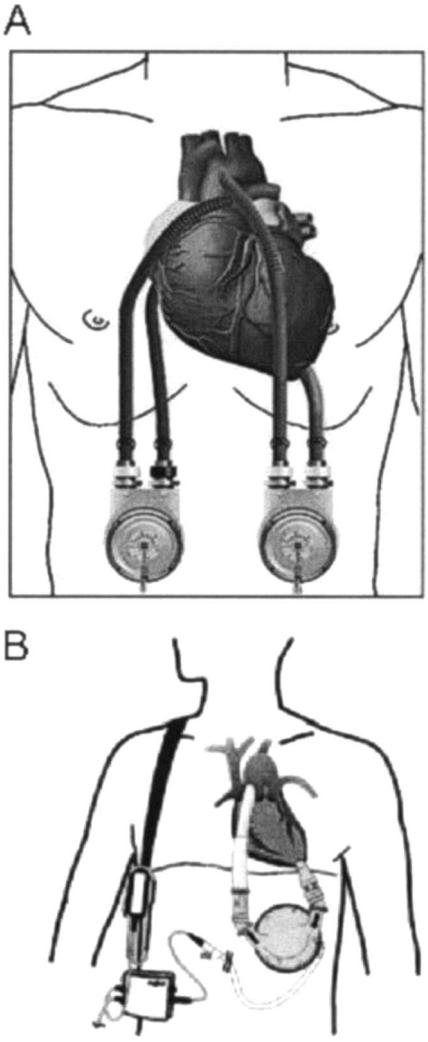


**Figure 4.** Pulsatile left ventricular assist devices. *A*, diagrammatic representation of the placement of the Thoratec ventricular assist device in biventricular support configuration; *B*, the placement of the HeartMate vented electric left ventricular assist device.

The Novacor Left Ventricular Assist System is a pulsatile, intracorporeal, electrically driven device that is used primarily for bridge to transplantation (47, 48). It is used in the LVAD configuration only. The pumping chamber contains two opposing pusher plates and polyurethane blood contacting surface, and the inflow and outflow cannulas contain two porcine pericardial prosthetic valves that allow one-way flow. Like the Abiomed and Thoratec, the inflow and outflow cannulas have connections to the left ventricular apex and aorta, respectively, but instead of passing through the skin to attach to an extracorporeal pumping chamber, they traverse inferiorly within the body to connect to a chamber that is either placed in the abdominal cavity or in a pocket within the rectus muscle. The pump's driveline passes through the skin to connect to its power supply and controller and for providing a vent for air to be shuttled in and out of the device to compensate for the change in volume when the blood sac is compressed. Because of its size and intracorporeal placement, biventricular support is not possible with the Novacor Left Ventricular Assist System, and its placement is difficult in smaller patients. It does allow for long-term outpatient management and requires anticoagulation. The mechanical reliability of the device seems to be very good for ≥2 yrs of use (49). It is currently being studied as destination therapy in patients who are not transplant candidates.

The Thoratec HeartMate vented electric LVAD is an electrically driven, intracorporeal system that is used only in the LVAD configuration (9). Its connections to the heart and site of pump placement and exit of the driveline through the skin are similar to the Novacor device (Fig. 4). Its major difference from the Novacor is a textured blood-contacting surface that resists clot formation. This, combined with the use of prosthetic porcine valves in the inflow and outflow cannulas, negates the need for anticoagulation with warfarin, and patients are generally only prescribed aspirin therapy.

The REMATCH (Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure) trial was a randomized study of 129 patients with end-stage, New York Heart Association class IV heart failure who were not eligible for transplantation for a variety of reasons (50). Sixty-one patients were treated with optimal medical therapy, and the remaining 68 were randomized to placement of an LVAD using the HeartMate vented electric ventricular assist device. The patients in the LVAD group had an improved 2-yr survival, but only 23% of these patients were alive at 2 yrs, compared with only 8% in the medical therapy group. The LVAD-treated patients had a higher rate of adverse events, such as device malfunction, infection, and embolic events, but their overall quality of life was judged to be improved and the absolute number of days out of

hospital was greater. This trial has led to approval of the use of LVADs as destination therapy in select patients, but it has also clearly shown the shortcomings of this therapy with current technology and raised serious questions about cost and payment in an already economically strained healthcare system.

*Nonpulsatile LVADs.* The second-generation VADs have been developed during the last two decades and clinically studied for the last 5 yrs. These devices provide continuous or nonpulsatile flow by using a valveless rotary driven design. Continuous-flow VADs can be further subdivided into two categories: axial and centrifugal flow. The axial flow devices use a propeller or screw-like design to push blood forward, whereas the centrifugal pumps employ a spinning disk to advance blood flow. In general, these devices are smaller than the pulsatile VADs and take less time to implant. In addition, axial flow devices should theoretically consume less power because they do not have to overcome the inertia to a standing column of blood at the onset of systole. However, it is not yet clear that the mechanical advantage of continuous flow has translated into lower energy requirements than that seen with pulsatile devices. Like the pulsatile systems, the inflow cannula is placed at the ventricular apex and the outflow cannula is attached to the aorta or pulmonary artery. All of these devices are placed intracorporeally and are best suited for true ventricular assistance rather than complete ventricular replacement.

There are three axial flow devices currently being studied in the United States: the MicroMed DeBakey VAD, the Heartmate II (Thoratec), and the Jarvik 2000. The MicroMed VAD is being studied as a bridge to transplantation and has proven to be reliable with little device wear or induction of hemolysis (51, 52) (Fig. 5). It generally provides approximately 5–6 L/min of flow but can provide up to 10 L at higher rotational speeds. Worldwide, several hundred patients have been supported with the MicroMed device, with a majority of these being successfully bridged to transplantation. Case reports have also shown a reversal of pulmonary hypertension and high pulmonary vascular resistance in patients supported for months (53, 54). The HeartMate II is of similar size and design as the MicroMed and assumes a similar orientation and connection at implantation. It has not been implanted in as many patients as the

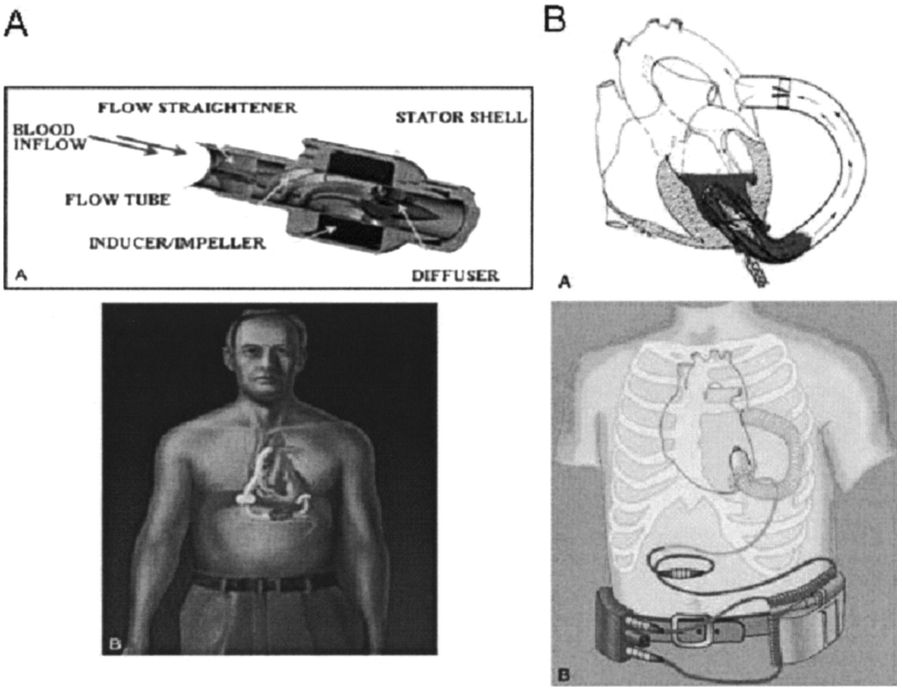


**Figure 5.** Nonpulsatile left ventricular assist devices. *A*, diagrammatic representation of the MicroMed left ventricular assist device along with a diagram of placement of the device; *B*, diagram of the Jarvik 2000 left ventricular assist device and its placement. Note the aortic cannula is anastomosed to the descending aorta, as opposed to most other left ventricular assist devices, which are anastomosed to the ascending aorta.

MicroMed, but recent data have demonstrated success with bridging to transplantation and left ventricular recovery (55). Since their introduction, both of these devices have undergone design changes in the blood-contacting surfaces to reduce thrombus formation and number of thromboembolic events.

The Jarvik 2000 VAD differs from the MicroMed and HeartMate II VADs in that it is placed within the ventricle at the apex, and the outflow cannula in the LVAD configuration is often anastomosed to the descending aorta (56–60). Placement can be performed via a left thoracotomy rather than a sternotomy (Fig. 5). It is also smaller in size and lighter in weight than the other axial flow pumps and therefore better suited for biventricular support where space is more limited. In the univentricular setting, its power supply can be tunneled to the retroauricular region, a setup that seems to have a lower rate of infection than other VADs (57, 58). Hemodynamic variables, such as cardiac output, pulmonary vascular resistance, and pulmonary artery occlusion pressure, measures of renal and hepatic function, and quality of life scores all improve with the device (56, 59). It has been used as a bridge to transplantation and recovery and, in rare cases, as destination therapy. Limited published data have demonstrated it to be mechanically reliable and to have a low prevalence of hemolysis and thromboembolic events. Like the MicroMed and HeartMate II, it requires full anticoagulation with warfarin.

The HeartMate III is an energy-efficient centrifugal flow pump that uses a magnetically levitated rotor to advance blood flow (61). It has been studied in the bovine model with good results and is currently under investigation in human clinical trials (62). It is larger in size than the axial pumps but is smaller than the current pulsatile VADs. Against a blood pressure of 135 mm Hg, it can deliver up to 10 L/min rate of flow but typically delivers approximately 7 L/min flow. Reported rates of hemolysis and thrombogenicity have been low, there have been no recorded mechanical failures with the current version of the device, and indicators of end organ function have remained within normal ranges. Like the axial flow pumps, the HeartMate III requires anticoagulation with warfarin.

*Total Artificial Heart.* In the past several years, complete heart replacement therapy has been studied with the use of totally implantable artificial heart systems. This form of therapy offers several

advantages compared with traditional VAD therapy. Because it replaces both ventricles completely, it is unaffected by ventricular arrhythmias that often hinder univentricular VAD support. It also delivers high cardiac output in the immediate postoperative period and avoids the problem of right heart failure that is commonly seen after LVAD placement. Lastly, its placement is considered by many surgeons to be a technically easier and better tolerated operation than that of traditional biventricular assist devices.

The AbioCor (ABIOMED) total artificial heart has been placed as destination therapy in 14 patients who were critically ill and facing imminent death from heart failure within 30 days. The outcome of 7 patients were published: one lived for >1 yr after implantation, four were able to participate in some outpatient activities, and two were eventually cared for in a transitional care facility (63). Overall, survival was longer than the expected 30 days predicted without mechanical support. The most common adverse events beyond the perioperative period were thrombus formation within the device and embolic events. There were no device failures, infections, or significant hemolysis reported. All 14 patients have since died, with the longest survivor living for >1 yr.

The CardioWest (formerly the Jarvik 7 and Symbion Heart) total artificial heart is now approved by the Food and Drug Administration as a bridge to transplantation (64). It is used as a bridge to heart transplantation in patients who require biventricular support. The results in bridge to transplant application have been published. Although one might expect that a higher-risk group might be selected for a trial of biventricular support, the results were comparable with that of several LVADs in this application. It is clear from the currently available data that more research and technology advancements are needed before more routine use of total artificial heart systems becomes more widespread. Problems with embolic phenomenon and the large size of the current systems are just two of the issues that will need to be overcome.

*Catheter-Based LVADs.* A recent advance in mechanical circulatory support has been the use of very small pumps that can be incorporated into a transvascular catheter. Much of the morbidity associated with LVAD use is the need to perform a major surgical procedure on a critically ill patient. The thoracotomy and cardiopulmonary bypass necessary to complete the implant can compromise already tenuous organ function. Hepatic dysfunction may contribute to a coagulopathy perioperatively and postoperatively, leading to bleeding complications. LVADs placed percutaneously or with limited surgery will likely become a significant advance, particularly in the critically ill patient with shock.

Although there are several such pumps under development, two exemplify the types of pumps under evaluation and are furthest along in development and use. The TandemHeart LVAD is an extracorporeal, centrifugal LVAD that can be placed percutaneously (65–67). Oxygenated blood for inflow is obtained from the left atrium by the femoral vein approach with an atrial transseptal catheterization. Blood is returned high in the iliac arteries with a femoral arterial cannula (Fig. 6). The pump can provide up to 4 L/min blood flow (65). It has been used in postcardiotomy shock, circulatory support to allow high-risk percutaneous coronary intervention, bridge to recovery of acute cardiogenic shock, and for short-term support following acute graft failure after orthotopic heart transplantation. This device is cleared by the Food and Drug Administration and is currently available in the United States and Europe.

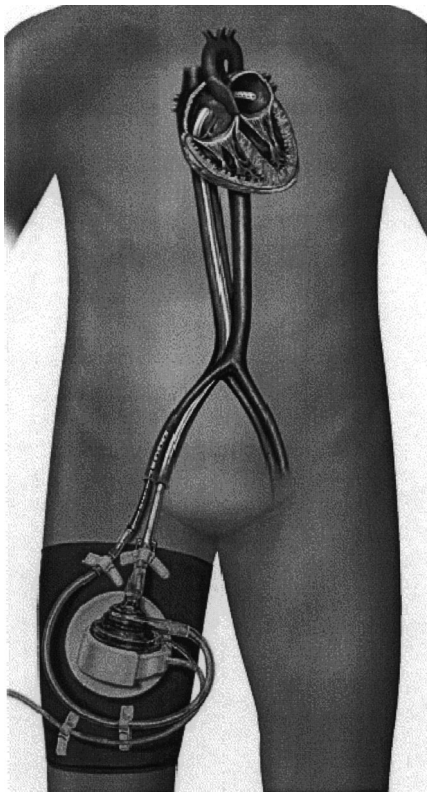

**Figure 6.** The TandemHeart percutaneous left ventricular assist device.

A second device is a catheter-based, axial flow pump with a miniature pump located at the end of the catheter (Impella) (68–76). The catheter is placed across the aortic valve, and the pump draws blood away from the tip of the catheter in the left ventricle and empties it 5 cm proximally into the aorta. The pump is typically inserted under fluoroscopy, and position is confirmed with echocardiography (77–79). The catheter has two diameters, a 24-Fr diameter that forms the pump chamber along with the inlet and outlet and a 9-Fr shaft. Because of the large caliber and differences in diameter, the catheter is placed via a femoral cutdown with an end-to-side graft anastomosed to the femoral artery. It can also be placed via the aorta at the time of thoracotomy.

The pump can deliver 5 L/min flow. This pump has been used for 3–10 days, typically as a bridge to recovery after myocardial infarction or postcardiotomy. One of the major challenges of this mechanical blood pump is the challenge to maintain a stable position across the aortic valve. Because of the direction of blood flow, the device has a tendency to pull itself into the ventricle.

A new concept in the treatment of refractory heart failure is the use of a mechanical blood pump to increase flow in the descending aorta by withdrawing blood from the femoral artery and returning it to the high descending aorta. There is a randomized clinical trial in the United States to evaluate a small centrifugal blood pump for this indication (Cancion System, Orqis Medical, Lake Forest, CA) (80). The concept was originally thought to decrease the resistance of ejection of blood from the heart by reducing the inertia of a standing column of blood in the aorta at the onset of systole. However, the mechanism is likely something different because the improvements in hemodynamics and renal function seen with this technique seem to increase in the course of hours or longer and do not immediately dissipate. Therefore, the mechanism is likely more biological and less mechanical, although the details are not understood at this time.

*Complications.* Complications with LVADs are fairly similar, and differ between devices mainly by degree. Early complications include bleeding, air embolism, right ventricular failure, and all the potential complications that may ac-

company open-heart procedures (81). All mechanical blood pumps can cause hemolysis, and thrombosis may occur within the device. As with any surgical implant, infection is a problem, particularly for those with exposed drivelines. Device malfunction and mechanical failure can occur with any device and is typically more common with longer duration of support. Other common complications associated with LVAD implantation include arrhythmias, renal dysfunction, and hepatic dysfunction (82).

## Continuous Venovenous Hemodialysis and Venovenous Slow Continuous Ultrafiltration

As renal function is often compromised with progressive heart failure or cardiogenic shock, replacement of renal function on a temporary basis becomes an important supportive therapy. However, standard hemodialysis or ultrafiltration is challenging in the setting of refractory heart failure or cardiogenic shock. In contrast, continuous venovenous hemodialysis and venovenous slow continuous ultrafiltration have relatively little effects on hemodynamic variables (83–85).

*Function.* In the standard techniques, a central venous dual-lumen catheter is needed to supply a blood flow of 100–300 mL/min for filtration or dialysis (84). Ultrafiltrate can be removed at a rate of up to 2 L/hr; however, usually ≤500 mL/hr is removed. The decision for the use of simple ultrafiltration vs. hemodialysis is made on the basis of the usual indications for hemodialysis, including uremia, the degree of azotemia that may lead to uremia, the acid–base condition and electrolyte balance.

*Indications.* The decision to use ultrafiltration in the treatment of an oliguric patient with refractory heart failure is made on the basis of the severity of the volume overload and the failure of medical measures to resolve this state.

*Clinical Results.* One series of 52 patients describes a mean daily weight loss of 1.2 kg using slow continuous ultrafiltration. Blood pressure was low before initiation of slow continuous ultrafiltration, with a mean blood pressure of 60 ± 22 mm Hg, and changed only slightly postultrafiltration, to 57 ± 25 mm Hg (84). Another report recently described slow continuous ultrafiltration using a smaller filter with lower blood flow rates to the filter. Blood flow rates were ≤40 mL/min, allowing the use of peripheral catheters (16–18 gauge) and removing up to 500 mL/hr (85). However, because of the low flow in the filter, heparin was recommended for anticoagulation during ultrafiltration. The group of patients were not as ill and had a substantially higher blood pressure, beginning at 116 ± 18/62 ± 10 and ending at 111 ± 16/58 ± 11 at the end of the ultrafiltration period, which lasted only up to 8 hrs. There were no significant changes in potassium, blood urea nitrogen, or creatinine.

Using these techniques, ultrafiltrate can be removed from the volume-overloaded patient who has compromised organ function with high venous pressures despite low systemic blood pressures. Although these techniques have been successfully employed in patients with refractory heart failure with apparently little hemodynamic compromise, there have been no sizeable randomized comparisons of intervention with either to guide the aggressive use, particularly when hemodialysis is not yet necessary.

*Complications.* Potential complications of ultrafiltration include all the potential complications of central venous access and problems associated with rapid volume removal and subsequent intravascular hypovolemia. Although electrolytes are generally preserved with either ultrafiltration or hemodialysis, frequent monitoring is warranted.

## Summary

Mechanical circulatory support is useful in the treatment of severe acute heart failure and cardiogenic shock. Mechanical ventilation, intraaortic balloon counterpulsation, and continuous venovenous hemodialysis have long been used in the critical care arena. We now have nearly three decades of long-term LVADs in the care of patients with heart failure, predominantly in the postcardiotomy shock and bridge to heart transplantation applications. Newer, smaller devices that can be placed without the need for extensive cardiac surgery and cardiopulmonary bypass may bring mechanical blood pumps into more common use in the intensive care setting.

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