An Overview of
Mechanical Circulatory Devices at Columbia:
Heartmate II, CentriMag and VA ECMO

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I. Types of ventricular assist devices

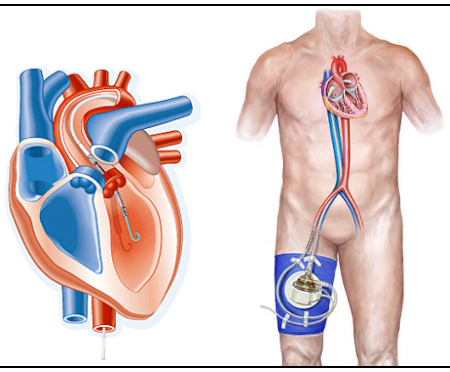
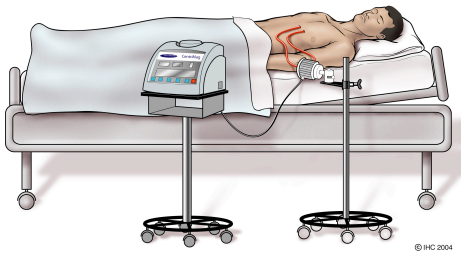
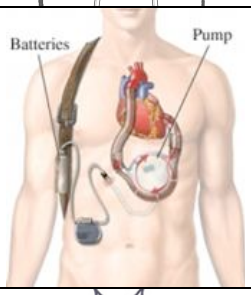
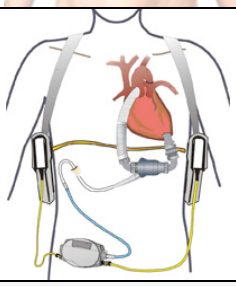
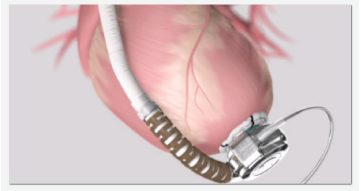
- Percutaneous
    - Placed via groin vessels
        - *Impella*: femoral arterial cannulation; the catheter is placed through the aortic valve into the L ventricle and a small pump at the tip of catheter pumps blood into the aortic root
        - *TandemHeart*: femoral arterial and venous cannulation; the catheter is placed through the femoral vein into the L atrium by crossing the interatrial septum and blood is then pumped into the femoral artery
- Extracorporeal:*
    - Pump located outside the body
- Paracorporeal:
    - Pump located next the body
- Intracorporeal:
    - Pump located inside the body
    - 1st generation: pulsatile-flow
    - 2nd generation: continuous-flow (axial)*
        - Impeller rotates like a screw propelling blood forward in a linear fashion
    - 3rd generation: continuous-flow (centrifugal)
        - Impeller rotates like a top spinning blood forward

- Support can be provided to:
    - Left ventricle (LVAD)
    - Right ventricle (RVAD)
    - Biventricular (BiVAD)

II. Other mechanical circulatory support devices

- Total Artificial Heart
    - The native ventricles are excised and replaced with two pneumatic pumping chambers
- Venous-Arterial Extracorporeal Membrane Oxygenation (VA ECMO)*
    - Venous and arterial cannulae and a membrane oxygenator oxygenate/ventilate and circulate blood for a failing heart/lungs

** Extracorporeal VADs (CentriMags), intracorporeal 2nd generation continuous flow VADs (Heartmate II), and VA ECMO support are routinely encountered in the CTICU at Columbia.*

VAD Type	Illustration	Name
Percutaneous		Impella (left) TandemHeart (right)
Extracorporeal		CentriMag
Paracorporeal		PVAD
Intracorporeal 1st generation: pulsatile-flow		Heartmate I
2nd generation: continuous-flow (axial)		Heartmate II
3rd generation: continuous-flow (centrifugal)		HeartWare

III. Goals of ventricular assist device therapy

- Bridge to transplant
  - Temporary circulatory support until a donor heart becomes available
- Bridge to recovery
  - Temporary circulatory support provided until myocardial recovery is achieved
  - e.g. myocarditis, post-cardiotomy shock
- Bridge to a decision
  - Temporary circulatory support to maintain organ perfusion until further information can be gathered
  - e.g. cardiac arrest with uncertain neurologic status - circulation is supported until brain function can be fully assessed, after which a plan of action can be made
- Bridge to a bridge
  - e.g. myocardial infarction with CentriMag support, subsequent lack of adequate recovery sufficient for full explantation, but enough myocardial function that a HM II as bridge to transplant can be implanted
- Destination therapy
  - Permanent implantation of HM II for non-transplant candidates

	VA ECMO	**CentriMag**	**Heartmate II**
Goals	-- -- Bridge to Decision Bridge to Recovery Bridge to a Bridge	-- Bridge to Transplant Bridge to Decision Bridge to Recovery Bridge to a Bridge	Destination Therapy Bridge to Transplant
Duration	≤ 7 days	Short term (wks-mo)	Longer term (mo-yrs)
Acuity	V. acute decompensation	Acute decompensation	Chronic heart failure
Placement	**Peripheral**: Groin cannulation is very easy and can be done at bedside **Central**: see text	Easy, but requires sternotomy	Involved – requires dissection of pocket for pump
Anticoagulation	Stringent – heparin gtt as soon as post-operative bleeding controlled	Less stringent, especially with flows > 4 L/min; heparin gtt	Aspirin POD 1-3 Warfarin POD >3 +/- dipyridamole
Support to:	Increases afterload; Primary goal of VA ECMO therapy is to support organ perfusion	LV RV Biventricular	LV

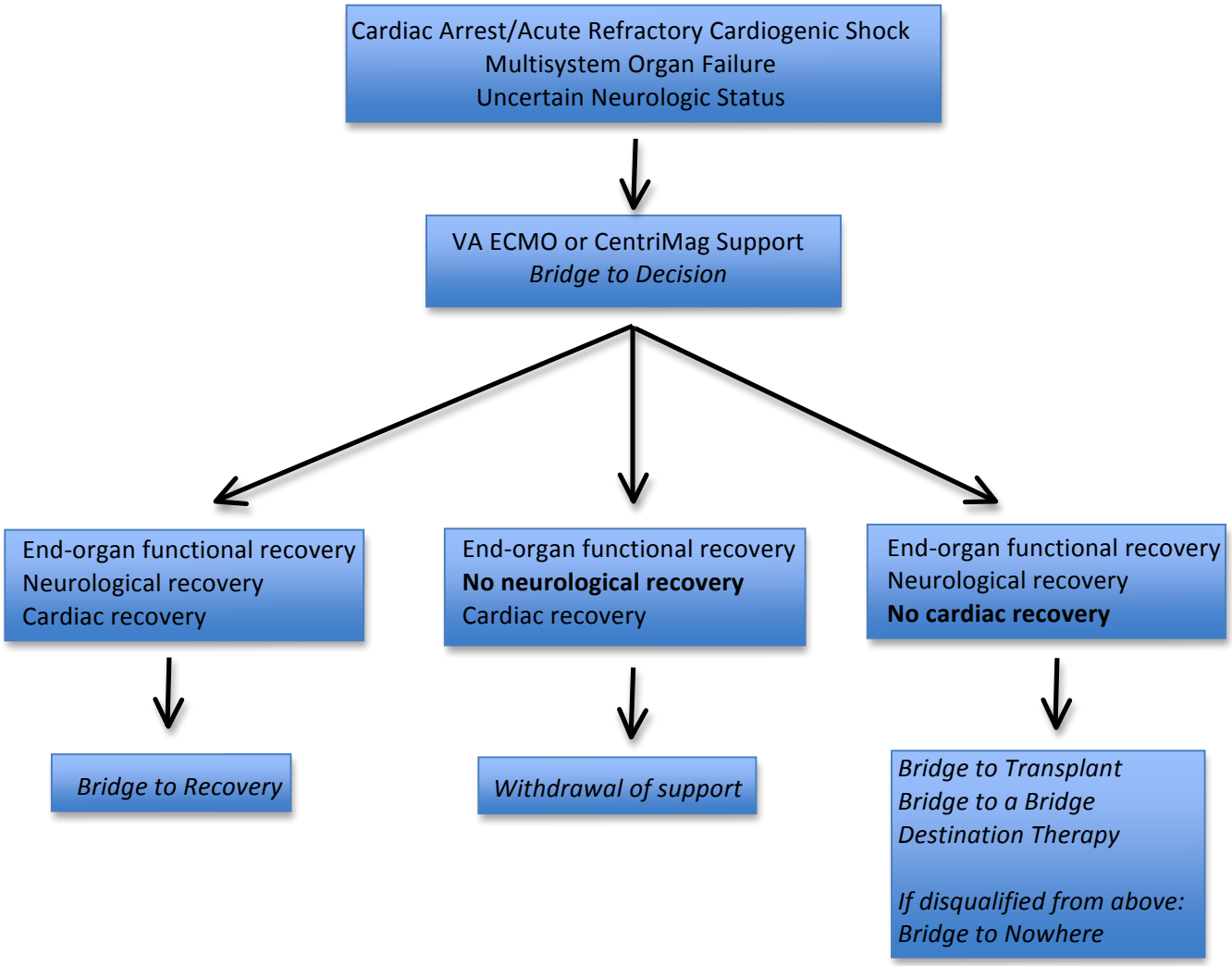
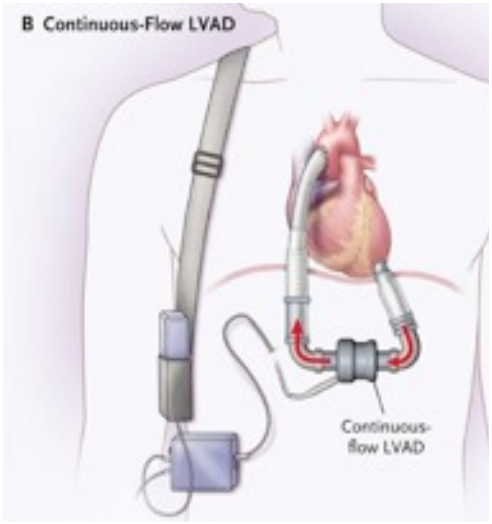
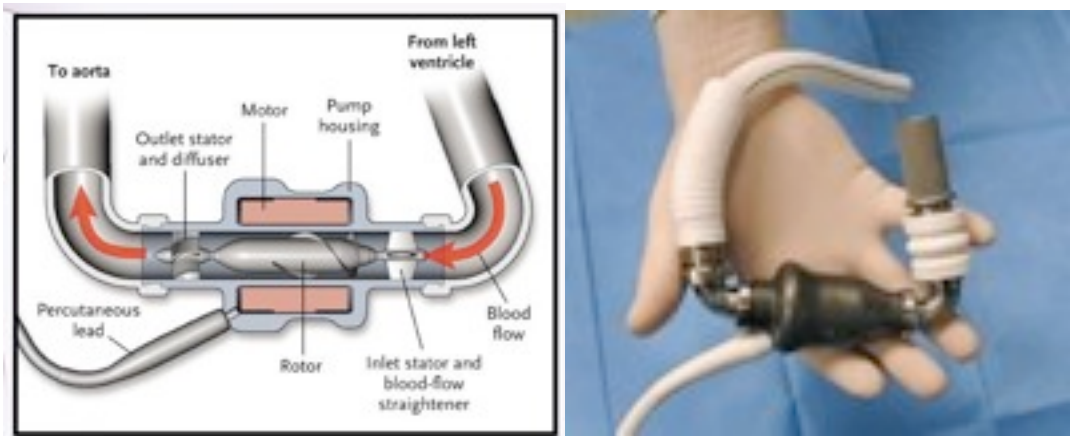


Figure adapted from John et al. "Experience with the Levitronix CentriMag circulatory support system as a bridge to decision in patients with refractory acute cardiogenic shock and multisystem organ failure." J Thorac Cardiovasc Surg. 2007. 134:351-8.

IV. Heartmate II Continuous-flow (axial) LVAD



The Heartmate II is a second-generation continuous-flow ventricular assist device (axial) with a linear conduit[1]. It has an inflow cannula anastamosed to the left ventricular apex and an outflow cannula anastamosed to the ascending aorta above the aortic valve. It receives blood from the left ventricle and pumps it into the ascending aorta, thereby reducing the work of a failing left ventricle. A percutaneous lead exits the skin; the lead connects to an external system controller, which itself connects to external battery packs. The pump can provide up to about 10 L/min of support.



The continuous-flow HM II LVAD contains a bladed rotor that, by rotating, draws blood from the heart and continuously propels it toward the ascending aorta. The rotor is the only moving part. The HM II does not contain any valves. It is smaller/lighter (0.8 lbs), much quieter, and more durable than its predecessor, the pulsatile-flow HM I. Cardiac lesions that must be corrected prior to/during LVAD implantation include: mitral stenosis (which limits flow to the LV), PFO (to prevent R-> L shunt and

[1] Slaughter MS et al. "Advanced Heart Failure Treated with Continuous-Flow Left Ventricular Assist Device." N Engl J Med 2009;361:2241-2251.

resultant hypoxemia), and aortic insufficiency (to prevent the futile circulation of blood). Mitral regurgitation may need to be repaired, as well.

Thrombosis and anticoagulation: Patients with the HM II are at risk of thromboembolic phenomenon and require anticoagulation. HM II patients require both warfarin and aspirin for anticoagulation; in select younger patients, dipyridamole is added to further decrease the risk. Unfortunately, the attempt to decrease the risk of thromboembolic phenomenon increases the risk of bleeding in this population.

A recent retrospective study published by Uriel, et al.[2] found that in 79 HM II patients, that 44% had major bleeding episodes within ~4 months after implantation requiring on average almost 6 units of PRBCs. The most common bleeding event was a GI bleed and the average INR at the time of the event was 1.67. This study looked at other potential mechanisms for why HM II patients have such a propensity to bleed; the study tested patients for von Willebrand factor levels and found that 31 out of 31 patients tested had decreased or absent von Willebrand multimers.

Von Willebrand factor is a very large multimeric glycoprotein that allows binding of platelets to damaged endothelium. It is postulated that the large von Willebrand multimers break apart from shear stress from the rotor causing a decrease in quantity. The reduced ability to form a platelet plug due to an acquired von Willebrand disease may be implicated in why bleeding is such a problem postoperatively in HM II patients.

V. The Heartmate II continuous-flow LVAD – how does it work?

A. Determinants of flow

The two factors that affect the flow of blood through a HM II continuous-flow LVAD are 1) pump speed and 2) the pressure gradient across the pump.[3][4] Increasing the pump speed results in a higher flow because an increase in RPM will cause more blood to get propelled through the device. Flow is also affected by the pressure gradient across the pump. The pressure gradient across the pump is the difference between the outlet and inlet pressures, which means the difference between the aortic root pressure and the left ventricular pressure.

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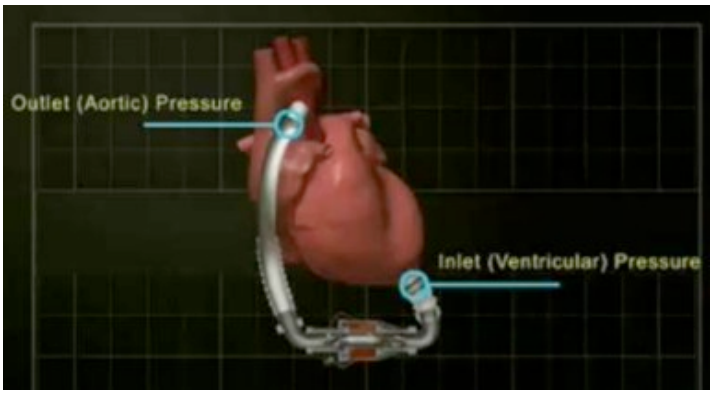
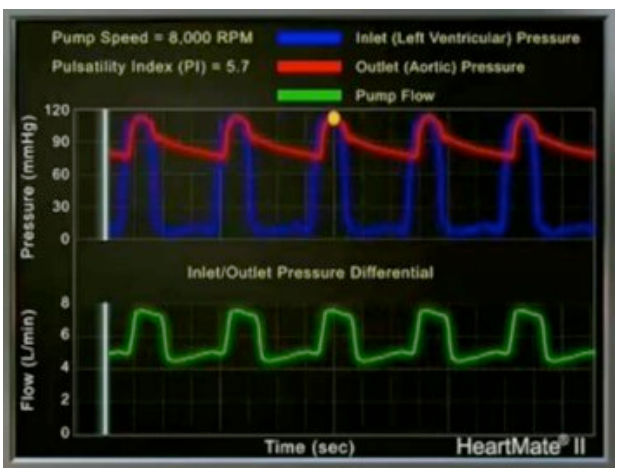
[2] Uriel et al. "Acquired von Willebrand Syndrome After Continuous-Flow Mechanical Device Support Contributes to a High Prevalence of Bleeding During Long-Term Support and at the Time of Transplantation." JACC. 2010. 56(15):1207-1a3.

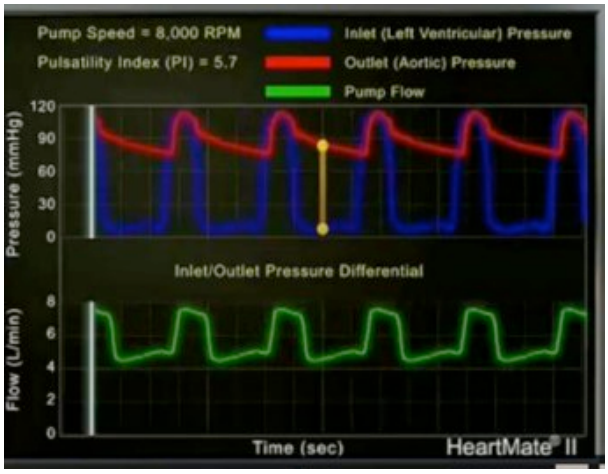
[3] http://www.thoratec.com/videos/mp-mcs.aspx?id=mp_hmII_IvasProfEduProGoGear; Chapter 2: System Overview

[4] Slaughter et al. "Clinical management of continuous-flow left ventricular assist devices in advanced heart failure." The Journal of Heart and Lung Transplantation. 2010. 29(4S):S1-37.



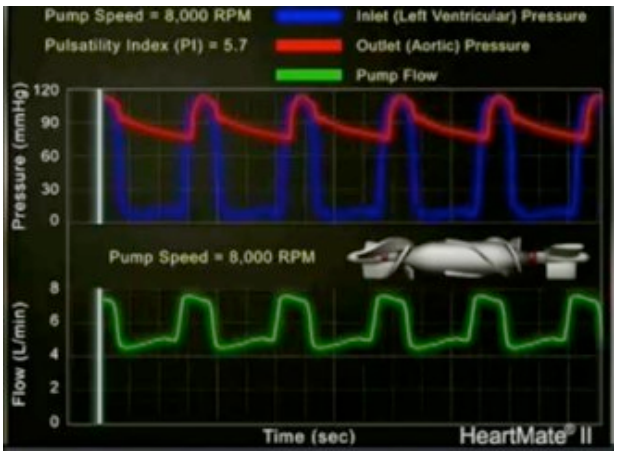
A large difference in pressure results in decreased flow through the pump; a small difference in pressure results in increased flow. Here is a simulated monitor with aortic and left ventricular pressures:

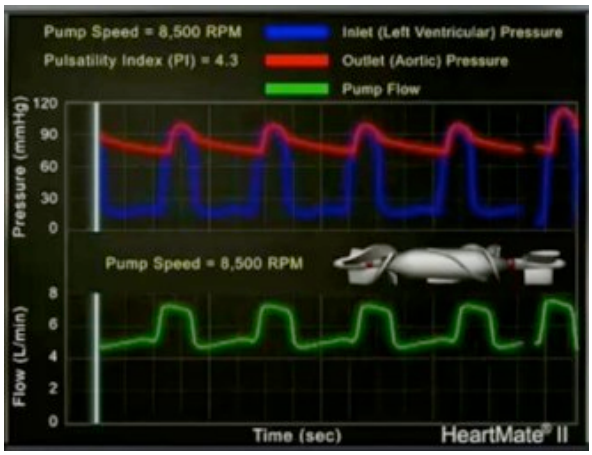


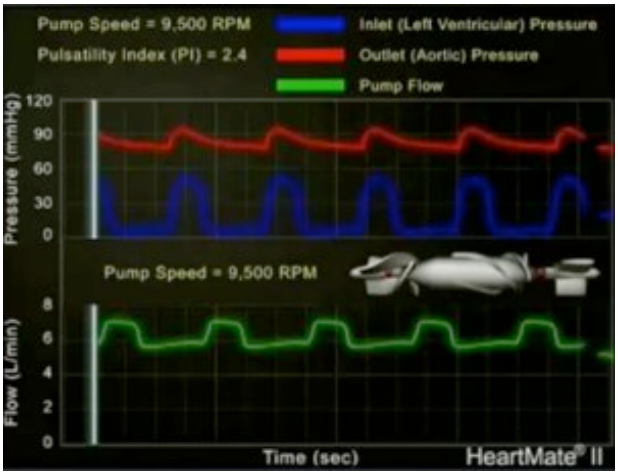


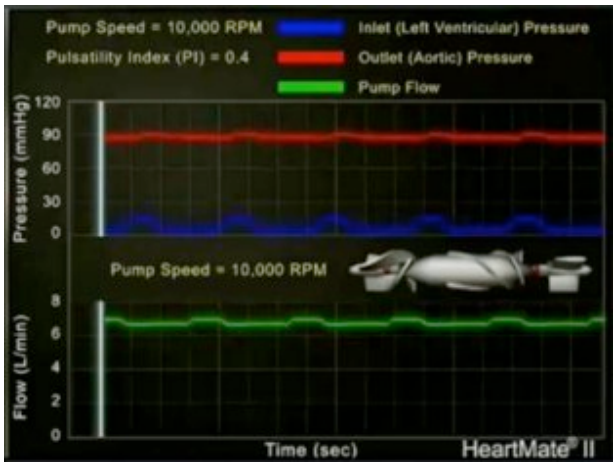
The red waveform represents the pressure at the pump outlet, which is the aortic pressure. The blue waveform represents the pressure at the pump inlet, which is the LV pressure. It is the difference between these two pressures that determines how much flow there will be through the pump, represented by the green waveform. During systole, denoted on the left panel by the yellow dot, the LV pressure rises and becomes identical to the aortic pressure. Because these two pressures are equal, there is no pressure gradient. As the pump inlet and outlet pressures become equalized, it becomes easier for the pump to propel blood forward, and therefore pump flow increases. This is shown by the increase in flow indicated by the green waveform.

The reverse is true during diastole. During diastole, the aortic and LV waveforms separate as the pressure in the aorta becomes much higher than the pressure in the left ventricle. This idea is illustrated on the right panel by the yellow line. Diastole is when the difference between the outlet aortic root and the inlet LV pressures is at its greatest. The pump must work against this increased pressure differential in order to propel blood forward, and as a result, flow decreases.









Here are four panels depicting the waveforms at 8000, 8500, 9500, and 10,000 RPM (left upper, right upper, left lower, right lower panels). The blue waveform denotes LV pressure. As pump speed increases, the ventricle becomes more unloaded resulting in less ventricular filling and decreased LV systolic pressure. As the speed increases and the pump takes over more of the work of the left ventricle, the blue waveform decreases in height.

The red waveform denotes aortic pressure. As the speed increases, the pulsatility decreases. As the speed increases, the diastolic pressure rises resulting in a decrease in the pulse pressure. The reduced pulse pressure with HM II continuous-flow LVAD support can make it difficult to palpate a pulse. This trend is mirrored by the green waveform, as flow through the pump also loses pulsatility.

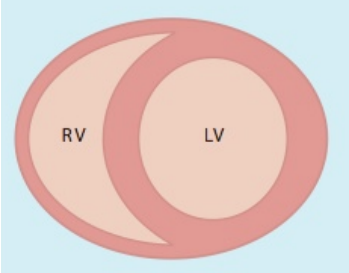
Now a key point is demonstrated with the panels at 8500 and 9500 RPM. At 8500 RPM, the red and blue waveforms overlap with each other. What this indicates is that at some point in the cycle, the pressure in the left ventricle exceeds the pressure in the aortic root, causing the aortic valve to open. In this scenario, blood flow across both the aortic valve and the pump. At 9500 RPM, the red waveform and the blue waveforms no longer overlap and have completely separated from one another. In this situation, the pump is doing so much of the work that the LV no longer generates a systolic pressure that exceeds the aortic systolic pressure. At this point, the aortic valve does not open; the flow of blood completely bypasses the aortic valve and goes solely through the pump.

While it might seem desirable to set the speed of the pump as high as possible to give the left ventricle maximal unloading, in reality, it is undesirable to have a situation where the aortic valve never opens. When the aortic valve does not open, it can create an area of stasis that can promote thrombus development, which can embolize and cause a stroke or occlude the coronary os; in addition, there have also been reports of fusion of the aortic valve leaflets and of the development of aortic insufficiency[5]. The pump speed is adjusted so that the aortic valve opening frequency is every beat or at least every 3rd beat.

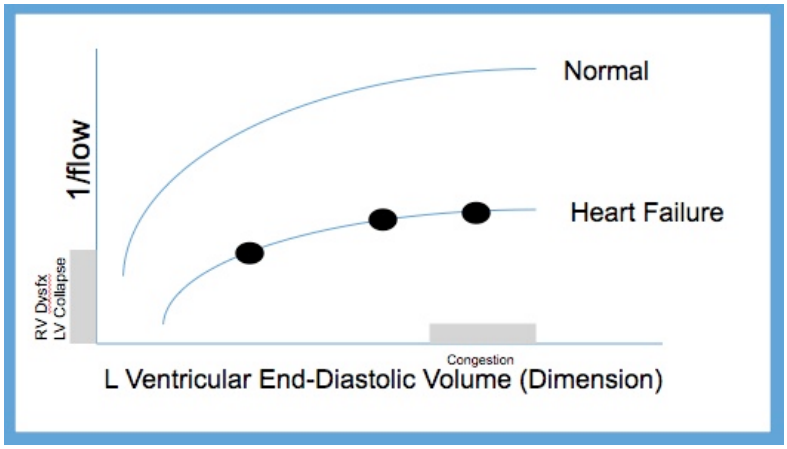
The important thing to remember is that a ventricular assist device is a ventricular assist device – in most circumstances, the device is not meant to completely take over the cardiac output of the patient; if possible, the patient's heart should take responsibility for some of its own cardiac output and promote flow across the aortic valve.

B. Determinants of optimum speed

After a patient has an LVAD placed, the optimum speed is determined and set before they go home. This determination is done with echocardiographic guidance. When the **speed is too low**, there will not be enough unloading of the left ventricle; LV size as assessed by the end-diastolic dimension (LVEDD) will remain increased and the filling pressures will remain high.

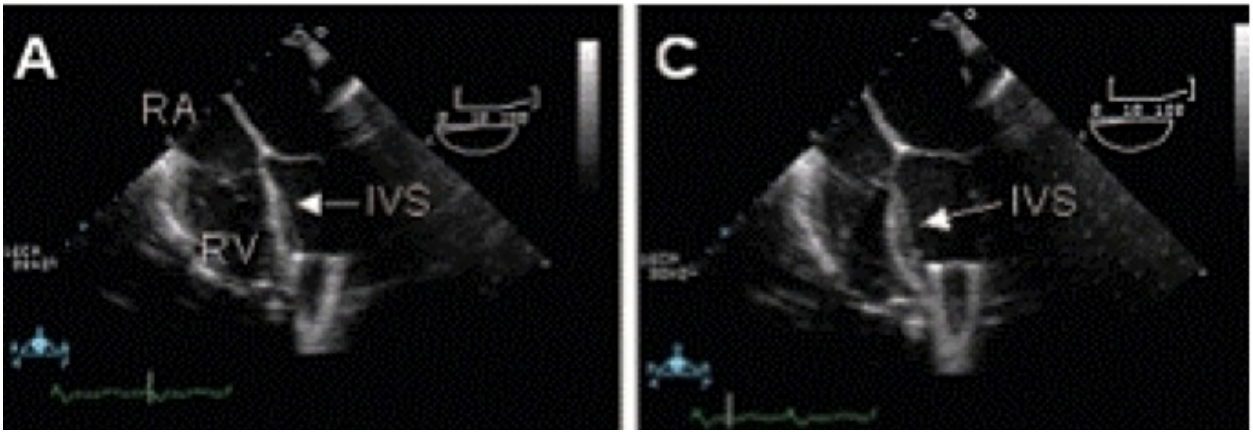


When the **speed is too high**, the LV decreases in size and the septum shifts to the left toward the inflow cannula. A) A leftward shift of the septum alters RV geometry and can cause an adverse effect on RV function. B) Continuous-flow LVADs have the ability to generate large negative pressures at the pump inlet; it is possible to create a flow rate that is higher than the preload going into the LV, which can result in a suction event where the walls of the LV collapse toward the inflow conduit; suction events can precipitate ventricular arrhythmias. C) Excessive pump speeds will decrease LV pressure and may prevent the aortic valve from opening.



Finding the right speed is just a reflection of finding the right place on the LVAD Frank-Starling-like curve. A speed that is too low, resulting in a large LVEDD, will not give enough help to the L ventricle and will result in congestion. A speed that is too high will reduce the LVEDD to the point where it may cause RV dysfunction and LV collapse. The right speed will avoid these two extremes. The **speed is just right** when LV size, filling pressures, and septal position have been optimized; in addition, the patient should demonstrate some arterial pressure pulsatility with intermittent aortic valve opening.

[5] Pak et al. "Prevalence of de novo aortic insufficiency during long-term support with left ventricular assist devices. " The Journal of Heart and Lung Transplantation. 2010. 29(10):1172-6.



Panel A[6] shows a TEE 4 chamber view showing the inflow cannula in the left ventricle with the arrow pointing to the interventricular septum. The speed is too high and the septum is being drawn in toward the left side causing RV dysfunction. As the speed is decreased, as shown in panel C, the position of the septum relaxes and moves more toward the right with improvement in RV function.

C. Optimal afterload

As mentioned above, flow through the pump is affected by **pump speed** and the **pressure gradient across the pump**. The pressure gradient is affected by the **afterload**. An increased afterload will increase the pressure differential across the pump, making it more difficult for the pump to propel blood forward. A decrease in afterload will decrease the pressure gradient across the pump and make it easier for blood to be propelled forward. Aiming for a MAP in the 70-80's should provide a good balance between perfusion pressure and forward flow.

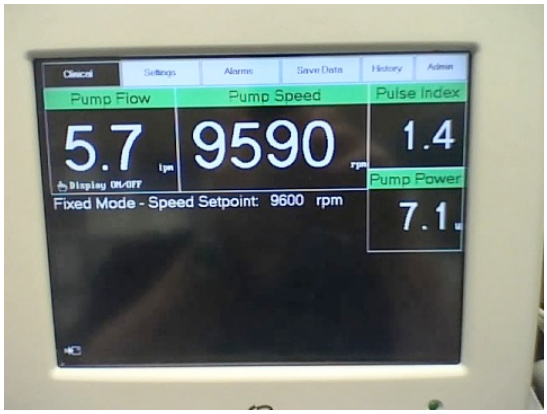
D. Optimal preload

Optimizing LVAD flow involves not only finding the right RPM and maintaining a reasonable afterload, but also in maintaining an adequate preload. An LVAD can only pump what is available in the left ventricle. Even if the patient leaves the hospital totally optimized, the preload can change dramatically due to factors like overdiuresis; if the LVAD flow exceeds the available preload, the patient may experience a suction event.

Low preload can occur not just from over-diuresis of a patient but also with reverse Trendelenberg and standing, hemorrhage, and **RV failure**. Right heart function is a crucial component to the function of an LVAD because after LVAD implantation, maintenance of an adequate cardiac output now depends on the ability of the unassisted right ventricle to deliver volume to the left side of the heart.

[6] Chumnanvej et al. "Perioperative Echocardiographic Examination for Ventricular Assist Device Implantation." Anesthesia Analgesia. 2007. 105(3):583-601.

E. The monitor

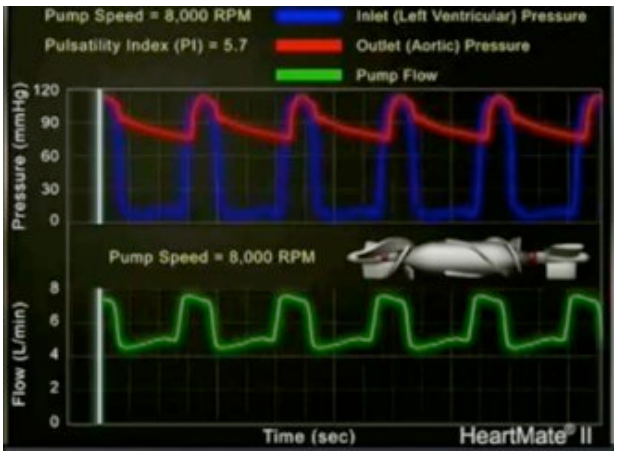


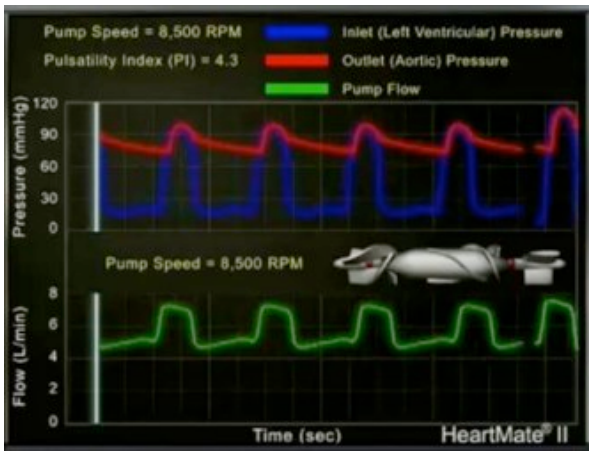
There are several components to a HM II monitor. The **speed** is displayed in RPMs. The **power** utilized by the pump is a directly measured function that normally ranges from 6-8 W. The power usage goes up as the RPMs are increased. In addition, if a thrombus develops on the rotor, this can increase the drag and hence the power needed to maintain a particular RPM. **Pump flow** is a **calculated entity**. It is estimated from the RPM and the power usage. It should not be used as an absolute estimate of cardiac output for two reasons: 1) at usual flows between 4-6 L/min, there can be up to a 20% difference between the flow estimate on the display and actual flows going through outflow graft. The device flow outputs may be inaccurate by nearly 1 L/min. 2) In addition, remember that total cardiac output comes not only from device flow but also from ejection by the ventricle through the aortic valve. The last part of the monitor is the **pulsatility index**.

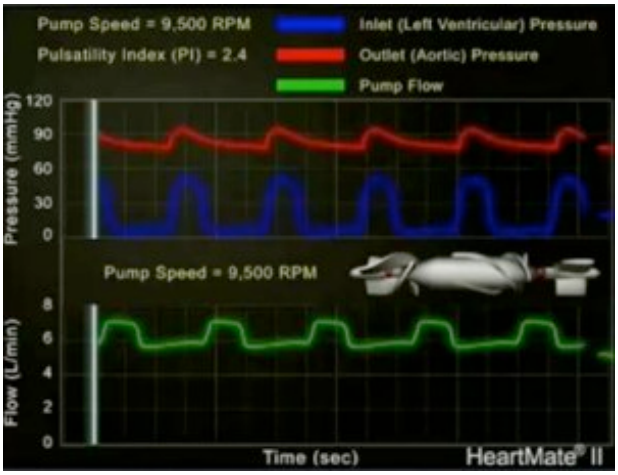
F. Pulsatility Index

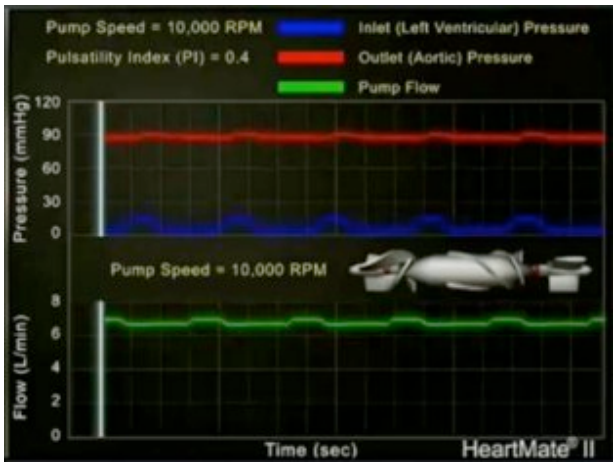
Pulsatility index is calculated by the formula: (Qmax-Qmin)/Qavg, i.e. maximum flow minus minimum flow divided by the average flow[7]. The pulsatility index is a dimensionless number that normally ranges between 4-6. As the LV contracts and relaxes, the LV pressures increases and decreases and subsequently causes a corresponding increase and decrease in pump flow; it is the magnitude of this change that is captured by the pulsatility index.

[7] Because flow is calculated, this calculation is actually done by pump detection of power pulsations.









Here are the images shown from before of the pressure waveforms at different speeds. Note the pulsatility index below the RPM designation. Observe the green tracing as the speed increases. The pulsatility of flow through the device decreases as the speed is increased. The difference between the maximum and minimum flow becomes attenuated, and this corresponds with a decrease in pulsatility index (i.e. 8000 RPM, PI 5.7; 8500 RPM, PI 4.3, 9500 RPM, PI 2.4, 10,000 RPM, PI 0.4).

A decreased pulsatility index can be caused by factors that result in a more decompressed ventricle. A) Increasing the RPMs of the pump will decompress the left ventricle more and hence cause less flow variability. B) For the same speed, a more decompressed ventricle can be created by decreasing the preload. A hypovolemic patient or a patient in RV failure, tamponade or with an arrhythmia will have decreased left ventricular preload, a more decompressed ventricle, and hence, decreased flow variability and a decreased pulsatility index. C) Finally, for the same speed, a more decompressed ventricle can be created by making it easier for the pump to work by decreasing afterload. Decreased afterload or a hypotensive or vasodilated state allows for greater flow of blood out of the left ventricle and will also cause a decreased pulsatility index.

Low Pulsatility Index (normal 4-6)	
Low power (Low Flows)	**High power (High flows)**
Low preload to left ventricle • RV failure • Hypovolemia • Arrhythmia • Tamponade	Increased RPMs Hypotension i.e. decreased afterload

If a decreased PI is due to low preload to the LV, either from hypovolemia, RV failure, arrhythmia, or tamponade, this is associated with a decreased power reading and estimated flow. The pump interacts with less blood volume and, hence, needs less power to function. In contrast, both increased speed and hypotension will cause a decreased PI that is associated with an increase in the power reading. An increase in RPM causes an increase in power usage. Hypotension/decreased afterload also causes an increase in power; because it is easier for the pump to propel blood forward, the rotor interacts with a greater volume of blood and, hence, requires more power.

Inflow/outflow obstructions (whether due to kinking of the inflow/outflow graft or due to clot) and thrombus on the rotor present totally different causes of low PI that are **not** the result of a decompressed left ventricle. In these situations, the left ventricle is full because of impeded flow through the pump; the PI is low because an obstruction/thrombus attenuates the difference between the Qmax and Qmin (remember the equation for calculating PI: (Qmax-Qmin)/Qavg). **Both** inflow and outflow obstructions present with also present with **low power** because the volume of blood in contact with the rotor is decreased. This observation makes intuitive sense for an inflow obstruction; with an outflow obstruction, the pump does not compensate by working harder or spinning faster; because an outflow obstruction diminishes the volume of blood that is able to go through the pump, this results in decreased power utilization by the rotor.

Thrombus on the rotor causes a decreased PI in the setting of increased power; as mentioned earlier, thrombus on the rotor increases the drag and causes the pump to utilize more power to maintain the same RPMs. In the case of both inflow/outflow obstructions and thrombus on the rotor, decreased flow through the pump results in increased filling of the left ventricle; if the left ventricle has sufficient function, this will result in a higher pulse pressure and more ejection through the aortic valve.

Low Pulsatility Index (normal 4-6)	
Low power (Low flows)	**High power (High flows)**
Left ventricle decompressed	
Low preload to left ventricle • RV failure • Hypovolemia • Arrhythmia • Tamponade	Increased RPMs Hypotension i.e. decreased afterload
Left ventricle well-filled (look for increased pulse pressure)	
Inflow obstruction (kink or clot)	Thrombus on rotor
Outflow obstruction (kink or clot)	

Outflow obstructions have been relatively more frequent as of late due to a problem in the bend relief of the HM II leading to a loosening of the connection between the outflow graft and the metal pump, allowing for kinking. This can be assessed by obtaining an AXR to look at the connection. If a patient is suspected of having a pump obstruction, a CT chest with contrast or ramp study can be useful in figuring out the diagnosis. A ramp study is accomplished via TTE; the RPMs are increased and the LVEDD is noted. A normal study should result in a decrease in the LVEDD as the RPMs are increased. If the LVEDD does not decrease despite an increase in RPM, this suggests that the flow through the pump cannot be augmented and suggests the presence of an obstruction. If the cause of the obstruction is suspected to be a clot, send off an LDH; if it returns > 1500, the result is suggestive of clot as the cause. Clot source in the ventricle can be assessed with a Definity contrast TTE.

There are very few causes of high pulsatility index.

High Pulsatility Index (normal 4-6)	
Low power	
Hypertension i.e. increased afterload	Increased contractility

Hypertension, or increased afterload, increases the pressure differential across the pump and makes it more difficult for the pump to propel blood forward. This causes the ventricle to be relatively more filled and, if there is enough left ventricular function, may stimulate stronger contractions and cause greater difference between Qmax and Qmin (i.e. greater pulsatility). The power is low because the volume of blood interacting with the rotor is decreased. Myocardial recovery and increased contractility will also increase the PI.

G. Management of low PI

The development of a low PI is a frequent occurrence in a fresh postoperative HM II LVAD patient. The two most common causes of a low PI in this situation are RV failure and hypovolemia. **RV failure** is common for many reasons; patients may have had a global myocardial pathophysiologic process. After the LV is assisted with a HM II, the cardiac output now depends solely on the function of the RV. In addition, the myocardium as a whole will be stunned/stiff due to having been cooled and arrested. **Hypovolemia** is also on the differential. HM II implantations are frequently reoperations, which tend to result in more bleediing, and which may be exacerbated by depletion of von Willebrand factor. Take care when deciding between RV failure and hypovolemia as a cause of low PI; the administration of fluid, even a seemingly innocuous amount as 500 cc crystalloid or colloid, can have disasterous consequences on a right ventricle on the verge of failing. When faced with a decreased PI, first rule out right heart failure. The diagnosis of right heart failure can be made several ways[89]:

A high CVP with the sequelae of cardiogenic shock. CVP: While it is not a good indicator of volume responsiveness, it is an excellent indicator of right heart failure and tamponade. Get a flat CVP from the nurse. Be wary of CVPs in the double digits; a patient can be in RV failure with a CVP of 14. **Shock**: Look for increasing lactate, a low mixed venous, rising pressor requirements, diminishing urine output.

[8] Kavarana et al. "Right ventricular dysfunction and organ failure in left ventricular assist device recipients: a continuing problem." Ann Thorac Surg. 2002;73:745-750.

[9] Matthews et al. "The Right Ventricular Failure Risk Score: A Pre-Operative Tool for Assessing the Risk of Right Ventricular Failure in Left Ventricular Assist Device Candidates." J Am Coll Cardiol. 2008;51;2163-2172.

Echocardiography: The Imacor is a disposable TEE that can be used for up to 72 hours. When a fresh LVAD is admitted to the CTICU, be sure to place one and get baseline pictures. Re-imaging the patient when the patient has a decreased PI can help tremendously with the diagnosis, especially if a dilated, hypocontractile ventricle is found.

Right ventricular stroke work index (RVSWI): One equation is (Mean PAP – CVP) x SVI where stroke volume index = Cardiac Index / HR; RVSWI < 6 gm x m2/beat is considered to be indicative of RV dysfunction. Another equation is [(Mean PAP – mean CVP) x SV]/BSA where RVSWI < 600 mm Hg x mL/m2 is indicative of RV dysfunction. These formulas create a relationship between mean PAP and CVP. Rather than calculating the formulatin, the formula can be considered conceptually: A high CVP in the setting of a relatively low mean PAP is indicative of a failing RV that cannot generate pressure and generates a high CVP because of lack of forward flow. A low CVP with a relatively higher mean PAP suggests a healthier RV that can generate pressure and promote forward flow.

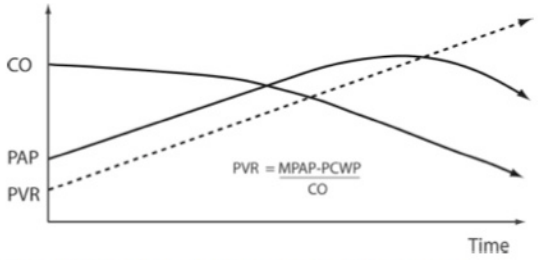


Figure 4. Hemodynamics in progressive pulmonary vascular disease. A decrease in pulmonary arterial pressure (PAP) in patients with PH may be a sign of low cardiac output (CO) and severe RV failure. PVR indicates pulmonary vascular resistance; PCWP, pulmonary artery capillary wedge pressure; and MPAP, mean PAP.

- **Contact the surgical attending and the ICU attending prior to any interventions.**

If the diagnosis is agreed to be RV failure, there are multiple maneuvers that can be done.[10][11]

- Decrease the pressure work of the right ventricle, i.e. PVR
  - Start inhaled NO (can also add iloprost, especially if the patient is not intubated)
- Decrease the volume work of the right ventricle
  - Initiate diuresis. Off-loading the right ventricle with diuretics can make pressor requirements go down.
- Increase contractility
  - Start or increase inotropes
    - Milrinone
    - Dobutamine
    - Epinephrine
- Increase the HR to at least 100 by pacing to increase the cardiac output.
  - The RV is stiff immediately post-op and as a result may have a limited SV; thus, increasing HR may result in an augmented CO (CO = HR x SV).

[10] Haddad F et al. "Right Ventricular Function in Cardiovascular Disease, Part II. Pathophysiology, Clinical Importance, and Management of Right Ventricular Failure." Circulation. 2008;117:1717-1731.
[11] Romano MA et al. "Diagnosis and Management of Right-Sided Heart Failure in Subjects Supported With Left Ventricular Assist Devices." Current Treatment Options in Cardiovascular Medicine. 2010. 12:420-430.

- Promotion of forward flow will help keep the RV decompressed. This will a) optimize RV geometry and help its function b) minimize dilation of the tricuspid annulus and minimize tricuspid regurgitation, which increases the volume work of the RV

- Ensure adequate perfusion pressure
  - To avoid RV ischemia with a MAP >70
- Decrease RPMs
  - RV dysfunction may be exacerbated by suboptimal geometry if there is distortion of the interventricular septum from too much suction from the in-flow cannula. This situation can be remediated by decreasing the RPMs.

If the diagnosis is agreed to be hypovolemia, then administered fluids cautiously in small amounts. Keep a very close eye on the CVP and pressor levels after each small bolus.

H. Arrhythmias

The differential diagnosis of arrhythmia in a patient with a mechanical circulatory support device should include: electrolytes, ischemia, high levels of catecholamines/inotropes (often from dobutamine, milrinone or epinephrine), irritation from scar, and in the case of ventricular arrhythmias, a preceding suction event.

I. Patient Monitors

Pulse oximetry: Pulse oximetry requires a pulse, and may or may not work depending on the magnitude of the pulse pressure in the patient.

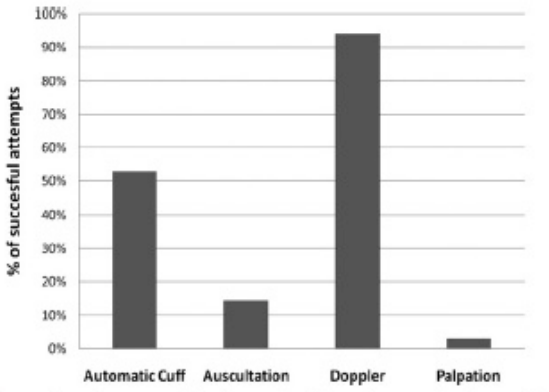


Figure 1 Success rate is shown for the various methods used to measure blood pressure in patients with a continuous-flow left ventricular assist device.

Noninvasive blood pressure cuffs: One study from Hopkins[12] from 2010 studied 17 patients with HM II devices to find out the optimal way to measure blood pressure, using arterial catheters as the gold standard. They found NIBP, auscultation, and doppler correlated closely to arterial catheter mean arterial pressure (r=0.7-0.8). In terms of reliability, however, they found that doppler was the most reliable way of obtaining a blood pressure noninvasively. In this study, the automatic cuff was successfully only about 50% of the time. Depending on the pulse pressure of the patient, the NIBP may be more or less successful, as the study did not address the rate of success with regards to pulse pressure. If the non-invasive blood pressure cuff gives a number, it is likely to be a real MAP, but remember that it is not the most reliable modality.

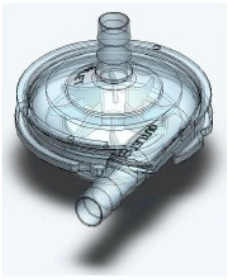
Arterial line: If a pulse is not palpable, use ultrasound or doppler.

[12] Russell et al. "Ideal methodology to assess systemic blood pressure in patients with continuous-flow left ventricular assist devices." The Journal of Heart and Lung Transplantation. 2010:29(5):593-4.

VI. The CentriMag Ventricular Assist Device

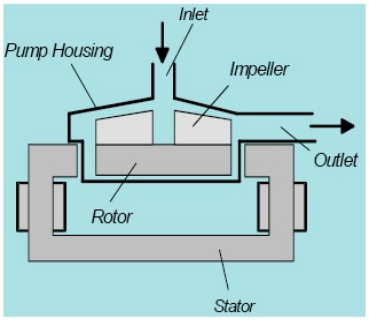
A. Components

The CentriMag is an extracorporeal ventricular assist device that can be used for weeks-months for temporary support (see introduction). It is a continuous-flow (centrifugal) device, meaning that the impeller turns like a top and spins the blood forward. The impeller is inside the pump, which itself nestles inside a motor; the motor spins the impeller magnetically.[13]



Pump

Motor



Long cannulae extend to and from the pump to support the LV, RV, or both ventricles. In sharp contrast to the HM II, a flow probe that clips onto the cannulae provides a **direct measurement of flow** (remember that in the HM II, flows are calculated based on RPM and power). The CentriMag can provide up to 10 L/min of flow. The only parameters listed on the monitor are RPM and flow.

B. Cannulation sites

CentriMag support to the right ventricle is almost always **RA to PA**. Left ventricular support can be accomplished one of two ways: LA to aorta or LV to aorta. **LA to aorta**: Cannulation from the LA is easier (anatomically more accessible), and can be done without bypass. However, blood may course through the LA to the LV and may become stagnant and form clots. LA cannulation is chosen when myocardial

[13] Levitronix. "CentriMag VAS Patient & Device Management Guidelines." November 2009.

recovery is either good (allowing accumulated blood to get ejected to decrease the risk of clot) or extremely poor. **LV to aorta**: Cannulation of the LV provides true rest and unloading of the left ventricle, but is technically more difficult as heart must be lifted up to access the apex.

C. Adjusting flows in a CentriMag BiVAD

Full flow is calculated by: BSA x 2.5 L/min. Remember to maintain LVAD flow > RVAD flow to prevent pulmonary edema. Exceptions:

- LVAD flow spuriously low due to output through aortic valve (will see pulsatility)
- RVAD flow spuriously high due to recirculation on RVAD due to PR/TR.

To increase flows, Increase then LVAD then the RVAD; to decrease flows, decrease then RVAD and then the LVAD.

D. Chattering

What is chattering? Chattering is a low-frequency jerking movement of the long cannulae. It is caused by two main groups of phenomena:

A physical interaction between inflow cannula and wall of heart due to decreased space:

- Decreased filling
- Increased contractility

(Less commonly) A problem with the cannula itself:

- Malpositioned cannula
- Obstructed cannula (kink, clot)

The most common causes of chatter are decreased filling (hypovolemia, excessive RPMs) and increased contractility.

RVAD chatter:

- Hypovolemia → insufficient RV preload
- RVAD RPM too high → excessive suction
- Increased RV contractility

Ways to address RVAD chatter are to give volume or decrease the RPMs. Rhythmic chatter due to increased RV contractility (verify by seeing if chatter corresponds to HR and if there is pulsatility on the PA catheter tracing) may indicate that it is time for the RVAD to be explanted, in which case RVAD chatter should merely be observed.

LVAD chatter

- If single LVAD CentriMag:
    - RV failure, hypovolemia
    - LVAD RPM too high → excessive suction
    - Increased LV contractility

- If BiVAD CentriMag:
    - RVAD RPM too low → insufficient LV preload
    - LVAD RPM too high → excessive suction
    - Increased LV contractility

When the patient has BiVAD CentriMags, giving volume only indirectly addresses LVAD chattering; do not give fluid routinely for LVAD chatter in this situation. Consider decreasing the RPMs, although remember that this may cause/exacerbate pulmonary edema. Rhythmic chattering due to increased LV contractility (look for pulsatility on the arterial catheter tracing) may signal that the patient should be explanted, in which case LVAD chatter should merely be observed.

E. Weaning CentriMags

Weaning trials are done in the cath lab with echocardiography as a test prior to a trip to the OR for explantation. Parameters to watch in the follow situations are as follows:

RVAD weaning
CVP: If the CVP remains stable during weaning, this indicates good RV function; a rising CVP suggests poor function. **PA catheter tracing:** Pulsatility of the tracing suggests good contractility of the RV. **LVAD flows and BP:** Stable LVAD flows and MAPs also suggest good RV function. If flows drop due to decreased preload to the LV and CO drops causing a decrease in MAP, the RV is likely to be dependent on CentriMag support.

LVAD weaning
Arterial catheter tracing: Pulsatility suggests good contractility of the LV. **MAP:** Stable blood pressures suggest good LV function.

Both
Echocardiography: Look for good function versus ventricular dilation and poor contractility. **Mixed venous:** A precipitous drop indicates an inability of the ventricle being interrogated to mount an appropriate cardiac output.

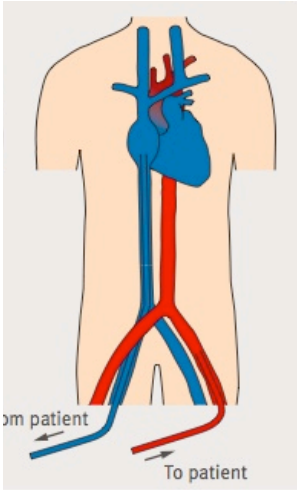
F. Monitors

Cardiac output calculation by thermodilution via the PA catheter: In the presence of an RVAD, the cardiac output calculation will be meaningless. Some fraction of the injected saline will get suctioned into the in-flow cannula and deposited into the pulmonary artery, rendering the results inaccurate.

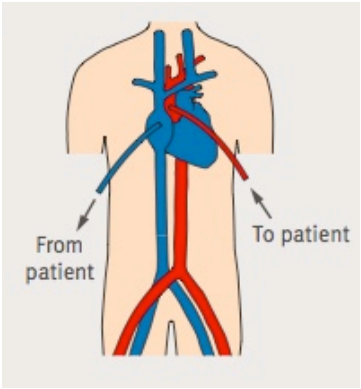
CVP: Controversial between cardiologists, intensivists, and surgeons. The CVP is meaningful when weaning an RVAD, as it is monitored for whether it is stable or starts to rise. The problem is that in the CTICU, the CVP can be easily manipulated by changing the RPMs, which occurs frequently. Hence, it can be difficult to make a meaningful comparison of two different CVP measurements on two different days when they occurred at different speeds.

VII. VA ECMO

The purpose of VA ECMO[14][15] is to support end-organ perfusion until a decision can be made or recovery can be attained; this modality takes over much of the oxygenation, ventilation, and circulatory functions of the body. VA ECMO actually increases afterload; it is, thus, important to make sure that the arterial line tracing in the patient is pulsatile. This suggests that the ventricle has enough contractile function despite the increased afterload to eject blood through the aortic valve and prevent stasis/clot formation. If there is no pulsatility due to extremely poor ventricular function, placement of an LV vent (which drains blood from the LV to the ECMO circuit) may be necessary.



With **peripheral cannulation** (femoral vein-femoral artery), oxygenated blood from the ECMO machine perfuses the body; however, blood to the head vessels (R innominate/L carotid) comes from blood ejected from the heart. The actual location of where the blood mixes depends on the location of the tip of the arterial cannula, the ECMO flow rate, and function of the heart. If there is significant injury to the lungs, the blood going to the coronaries/head/right arm may be quite desaturated. Adequate oxygenation of the brain can be ensured by following oxygenation via a pulse oximeter or ABGs from an A-line on the R arm. As with any device involving groin cannulation (e.g. IABP), perfusion of the distal leg should be monitored via pulses.



Central cannulation typically occurs during surgery when the patient is unable to come off bypass; the inflow is from the right atrium and the outflow from the ECMO goes into the aorta. This situation ensures oxygenation of the coronaries and head due to the central location of the outflow cannula.

Both: Follow SvO2 and lactates to assess total body oxygenation. Anticoagulation must be balanced with the risk of bleeding, but should be instituted as soon as possible given the risk of clotting of the membrane oxygenator. The sweep gas, the gas that interacts with the membrane oxygenator, is generally set to an FiO2 100% (this can be adjusted in VV ECMO to a lesser FiO2); the faster the rate of delivery, the more CO2 that is eliminated from the system.[*]

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[14] Gaffney AM et al. "Extracorporeal Life Support." 2010. 341: 982-6.

[15] Strickland R et al. "Royal Adelaide Hospital. General ICU ECMO Guidelines." October 2009.

[*] Acknowledgements: Desmond Jordan, Steve Miller, Hiroo Takayama, Yoshi Naka, Ulrich Jorde, Nir Uriel, Julia Sobol