Vasodilatory Shock After Ventricular Assist Device Placement: A Bench to Bedside Review

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With more than 2000 ventricular assist devices (VAD) placed annually in the United States, understanding postoperative management is important. One of the most common postoperative morbidities encountered with VAD implantation is vasodilatory shock. The mechanisms for this phenomenon are numerous and include cellular and hormonal aberrancies unique to the VAD recipient. Management of vasodilatory shock in VAD patients needs to be undertaken with an understanding of the side effects associated with each treatment, especially the effects on the right ventricle and pulmonary vasculature. This article focuses on the incidence, the pathogenesis, the consequences, and the management of vasodilatory shock in the post-operative VAD patient.

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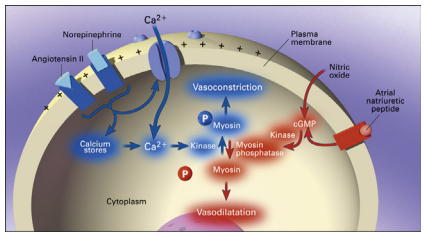

Understanding vasodilation at the cellular level is critical in understanding proper postoperative management.

Central Message

Vasodilatory shock following VAD placement is common and multifactorial. Treatment of this problem varies depending on etiology.

INTRODUCTION

The sixth report of the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) states that since 2006, over 12,000 ventricular assist devices (VAD) have been placed in the United States.[1] More than 2000 VADs are placed yearly with most being continuous flow, left sided devices (LVAD). Presently, destination therapy VAD placement occurs in 41%, which is a large increase from 2006 when destination therapy only occurred in 14% of patients.[1,2] As studies continue to corroborate that not only longevity of life is improved, but also quality of life, VAD placement has become a commonly accepted treatment for heart failure.

Although actuarial survival at 1 and 2 years is quite favorable (80% and 70%, respectively),[1] there are numerous adverse events that must be overcome following placement. Arguably, hypotension due to vasodilatory shock following VAD placement is the most common postoperative complication. Other causes of hypotension following VAD placement that need to be considered are: right ventricular failure, hypovolemia, bleeding, and sepsis. Elucidating the etiology of the shock is imperative as the treatment of 1 cause may counteract the treatment of another cause. This article focuses on hypotension due to vasodilatory shock following VAD placement.

INCIDENCE OF VASODILATORY SHOCK FOLLOWING VAD

Vasodilatory shock and the associated hypotension is a nearly universally anticipated postoperative complication following VAD implantation.[2] Vasodilatory shock after VAD placement has been defined as a mean arterial blood pressure (MAP) < 70 mm Hg, a cardiac index > 2.5 L/min/m^2 and dependence upon a pressor.[3] The incidence of vasodilatory shock after LVAD was found to be 42% in an early study[3]; however, most clinicians believe this estimate to be low as most VAD patients require pressor support for hours or days postimplantation.

CONSEQUENCES OF VASODILATORY SHOCK IN A VAD PATIENT

In all patients, vasodilatory shock can lead to hypoperfusion, lactic acidosis, and profound hypotension that is often unresponsive to catecholamine vasopressors.[4] Any impairment in perfusion in a VAD patient is especially deleterious as the reason for VAD implantation is to supply adequate perfusion to end-organs when the native heart is unable. Additionally, acidosis due to any cause increases pulmonary vascular resistance (PVR), which would add afterload to the right ventricle (RV). Even small increased in PVR can lead to RV

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Vasodilatory shock following ventricular assist device implantation is a common postoperative morbidity requiring understanding of the pathogenesis and treatment options.

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Table 1. Various VADs and Associated Type of Pump

VAD	Type of Pump
Heartmate II	Axial flow
HeartWare HVAD	Centrifugal (hydromagnetically suspended)
Centrimag	Centrifugal (magnetically levitated)
Heartmate III	Axial flow rotor (magnetically suspended)
Jarvik 2000	Axial flow

failure in the setting of newly implanted LVAD. Acidosis also creates a milieu prime for dysrhythmias, which can be detrimental to VAD flows and decrease blood pressure further.

Table 1 lists the most common VAD types placed in the United States with respect to type of pump: centrifugal or axial flow devices. Centrifugal pumps, such as the HeartWare HVAD (HeartWare HVAD Inc, Miramar, FL) have a higher afterload sensitivity compared with axial pumps, such as the HeartMate II (Thoratec Corp, Pleasanton, CA).[5,6] This may be in part because of the functionality of these devices. Axial pumps "push" or "propel" blood to generate flow, whereas centrifugal pumps "throw" blood to generate flow.[6] Thus, patients with centrifugal pumps necessitate a more rigorous control of systemic vascular resistance (SVR) than patients with axial flow VADs. However, maintenance of appropriate SVR with MAP in the 60-80 mm Hg range, is beneficial in all VAD patients.

PATHOPHYSIOLOGY OF VASODILATORY SHOCK

In all forms of vasodilatory shock, plasma catecholamine levels are markedly increased and the renin-angiotensin system is activated. Thus, the underlying problem is failure of the vascular smooth muscle to constrict, not lack of vasoconstrictors.[7] In order for vasoconstriction to occur, hormonal and neuronal ligands such as angiotensin II, endothelin, and norepinephrine must bind to receptors on smooth muscle cells. After which, a cascade of second messengers would lead to an increase in calcium concentration in the cytosol (Fig. 1). Calcium binds with calmodulin leading to phosphorylation of the light chain of myosin. This allows for the activation of myosin ATPase by actin and cycling of myosin cross bridges, leading in muscle contraction. Conversely, vasodilators such as atrial natriuretic peptide (ANP) and nitric oxide activate a kinase that dephosphorylates myosin and prevents muscle contraction.[7] This highly regulated systemic cascade fails after VAD implantation for a variety of reasons.

Firstly, prolonged hypotension and low flow can lead to death of vascular cells. The contribution of nonpulsatile flow to vascular cell death is yet to be determined. Secondly, mitochondrial dysfunction can lead to inadequate oxygen extraction at the cellular level. Thirdly, endothelial injury and the release of vasodilatory substances in the blood, such as prostaglandins, which are released with both critical illness and extracorporeal circulation, can produce vasodilation and shock following CPB and VAD placement.

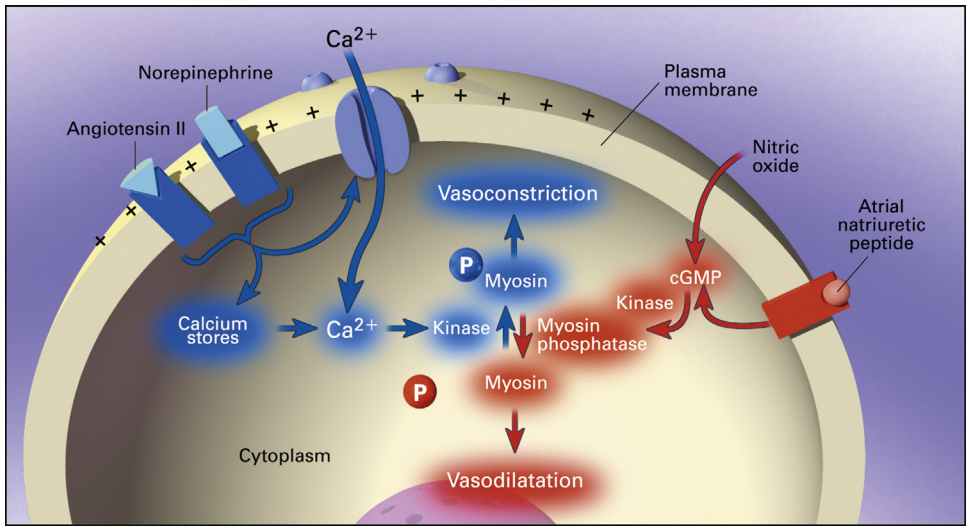

Figure 1. Regulation of Vascular Smooth Muscle Tone. The steps in vasoconstriction are shown in blue. The steps involved in vasodilation are shown in red. (Ca^{2+}=calcium; cGMP=cyclic guanosine monophosphate.) Reprinted with permission from Massachusetts Medical Society. N Engl J Med. Landry, D., Oliver J. Pathogenesis of Vasodilatory Shock. 345;588-595. Copyright © 2001, Massachusetts Medical Society. (Color version of figure is available online at http://www.semthorcardiovascsurg.com.)