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EDITORIAL COMMENTARY

Mock loop revelations and the calculus for recovery

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See related article on pages 343-8.

The clinical application of continuous flow (CF) left ventricular assistance devices (LVAD) evolved from decades of biomedical research to produce mechanical solutions for the chronically failing heart. Durable forms of CF pumps were designed for 5, 10, or more years of support (so-called destination therapy). With the ongoing shortage of available donor hearts, these devices have increasingly been used to support the hearts of patients awaiting heart transplantation (bridge-to-transplant strategy) when rapid cardiac decompensation portends imminent death. The concept of cardiac recovery did not really figure into the original paradigm. Although the possibility that a chronically dilated,

cardiomyopathic heart could recover after LVAD support has been recognized for several decades, actual reports of successful recovery and pump explantation were rare before about 2000.¹

Early in the new millennium, recovery gained traction as a viable clinical goal. The seminal publication by Birks and colleagues² in 2006, for example, reported a remarkable rate of recovery in patients with nonischemic cardiomyopathy with a program of durable LVAD support and administration of clenbuterol, a selective beta 2-adrenergic agonist. As CF technology in North America and Europe developed, additional successful experiences emerged.³⁻⁵

During the past decade, a flurry of basic science activity has targeted genetic upregulatory responses as they relate to unloading of a maladapted failing left ventricle, which is a requisite for myocardial recovery. Chronic LVAD support in the failing heart has been shown to decrease neurohormone levels, reduce cytokine release, decrease apoptosis, reduce myocyte size, normalize beta adrenergic pathways, and improve calcium handling at the cellular level⁶⁻¹¹; all of which could contribute to favorable reverse remodeling of the failing ventricle. The fact that chronic heart failure

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reverse remodeling hypotheses did not translate routinely into successful ventricular recovery following LVAD implantation added to the challenge and intrigue of deciphering the critical events at the myocyte level that could unlock the recipe for recovery.¹²

Unfortunately, most centers have experienced only limited success in achieving recovery of ventricular function to a level that would allow successful removal (explantation) of the LVAD. Although essentially all programs look for signs of recovery by periodically performing echocardiograms to examine left ventricular ejection fraction during LVAD support, there is no consensus on the best protocol to encourage recovery of ventricular function. Such protocols generally describe algorithms for gradual reduction of pump speed and verification of ventricular loading by observing intermittent aortic valve opening. An analysis of the US experience indicates that only approximately 5% of pump implants result in explant for recovery.¹³

Heart failure remission is determined by the achievement of effective cardiac function without mechanical assistance. The final stages of the recovery process include further reduction of pump speed, usually in the cardiac catheterization lab, while monitoring a patient's hemodynamic parameters via a pulmonary artery catheter and assessing ventricular geometry and wall motion with transesophageal echocardiography.

Sunagawa and colleagues¹⁴ in this issue present an elegant mock loop study that provides quantification of the competing contributions of regurgitant (backward) graft flow and forward pump flow. Knowledge of the overall sum of these competing flows at low pump speeds is an important part of deciding when pump removal can be safely performed. The authors observed that the large diastolic regurgitant flow plus forward systolic pump flow at 6000 rpm with the HeartMate II (Thoratec Corporation, Pleasanton, Calif) LVAD yielded a net flow of 0 in the normal heart state. But in heart failure, with less regurgitant flow, LVAD forward flow contributed significantly to output. Accurate predictions of non-LVAD-supported cardiac performance becomes more challenging as a recovering heart moves along the spectrum from 0 net flow in a normal heart to progressively greater net flow (ie, less regurgitant flow) in a partially recovered heart. Therefore, depending on where a patient's heart lies on the spectrum of recovery, the estimate of total cardiac output from the native heart at 6000 rpm may lead to overoptimistic conclusions about the myocardial state, particularly in borderline situations.

The authors suggest that reliable assessment of recovery likely requires total cessation of pump flow with balloon occlusion of the outflow graft. Whether this would present a risk for thrombus formation despite full heparinization during the period of balloon occlusion and pump cessation remains speculative.

Greater confidence in predicting recovery could generate increased interest in routinely pursuing protocols for weaning patients from LVAD support. The findings of Sunagawa and colleagues¹⁴ have particular relevance to enhancing the accuracy of predicting cardiac recovery during the weaning process.

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