

EDITORIAL COMMENT

The Right Prediction*

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Despite much improvement in the outcomes of patients undergoing implantation with left ventricular assist devices (LVADs), right ventricle (RV) failure remains a challenging clinical dilemma, particularly perioperatively as a significant cause of morbidity and mortality (1). In the patient freshly implanted with an LVAD, the RV becomes the limiting step to increase forward cardiac output. With increasing right atrial pressures in a failing RV, along with poor forward flow and, thereby, blood pressure, organ perfusion is compromised, leading to multiple organ dysfunction. Accordingly, many clinical investigators who work in this area have attempted to assess RV function pre-operatively, so as to either better select appropriate candidates for LVAD support, mitigate the impact of poor RV function post-operatively, or allow planning for interventions such as RV assist devices.

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However, assessing RV function is challenging. The RV has an unusual shape, making quantitation of RV volumes challenging. In patients with normal physiology, low pulmonary arterial pressures require relatively low stroke work in the RV. Chronic heart failure typically increases loading conditions on the RV, and the RV becomes conditioned to increase stroke work. Unfortunately, a number of factors then combine to cause RV dysfunction post-operatively. Even under optimal conditions, cardiopulmonary bypass may cause transient depression of RV function. Furthermore, the RV is uniquely susceptible to air embolism, with much of its blood supply typically coming from the right coronary artery, which is anatomically the more superior artery as the heart is being de-aired. Cardiopulmonary bypass often leads to transient increases in pulmonary vascular resistance, causing further increases in afterload on the RV. Blood products along with colloid and crystalloid solutions add to the loading conditions on the RV. Finally,

an LVAD decompresses the left ventricle and may cause a shift of the interventricular septum toward the left. The LV–RV interactions cause unusual loading conditions on the septum and may lead to further impairment of global RV function.

In this issue of the *Journal*, Grant et al. (2) describe the use of velocity vector imaging of RV free wall longitudinal strain to predict post-operative RV failure after LVAD implantation. The authors are an experienced group with a fairly high volume of implantations. They use a reasonably complete set of variables in this work that have been reported to be useful in the prediction of RV failure. Therefore, they provide a fair comparison with what has been done previously. A number of variables were associated with an increased risk for RV failure. These included hemodynamic parameters (right atrial pressure and pulmonary vascular resistance), clinical parameters (inotrope use), and biochemical measurements (total bilirubin). The authors also use a previously reported composite to predict RV failure, the Michigan RV risk score (3). The Michigan RV risk score was derived from a multivariable analysis of their single-center population. The variables included in the risk score are clinical (vasopressor use) and biochemical (elevated aspartate aminotransferase, total bilirubin, and creatinine). The echocardiographic parameters correlated with RV failure were the subjective assessment of RV systolic function and RV free wall longitudinal strain. When added to the Michigan RV risk score, RV longitudinal strain had an incremental benefit.

The authors do a commendable job of describing the limitations of their work. They deal in detail with the percentage of patients who did not have acceptable images, which will obviously affect the wider applicability of the method. Additionally, this was a single-center study, and the reproducibility of their findings in other centers has not been assessed. However, because the study was retrospective, the images obtained during routine clinical practice may be improved with further work.

Another issue is that local practices for the selection of LVAD candidates and their management before and after surgery are not uniform. For example, the right atrial pressures of the population in this study ranged only from 6 to 15 mm Hg, suggesting that patients were either aggressively treated or screened pre-operatively to create this unusual finding for an advanced heart failure population. Furthermore, the authors report an incidence of RV failure that is at the upper end of what has been reported in the literature. It may be that their population was unusually ill or had poor RV function pre-operatively. However, it would appear more likely that this particular group of clinicians monitored RV function closely and treated RV failure aggressively. Therefore, the definition of RV failure used, while an accepted definition used in prior similar studies, may be flawed in that the definition relies heavily on a physician's reaction to a patient's condition more so than

*Editorials published in the *Journal of the American College of Cardiology* reflect the views of the authors and do not necessarily represent the views of *JACC* or the American College of Cardiology.

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simply measuring the patient's condition. The reason to use such a definition falls immediately back to the original problem—that RV function is difficult to measure. Because each program is unique, assessing the utility of these parameters in multiple centers is necessary to make certain that the findings apply to various practices.

A number of other echocardiographic parameters have been evaluated for this purpose. Matthews et al. (3) reported 8 echocardiographic parameters included in the development of the Michigan RV risk score. However, the data were collected retrospectively, and sophisticated measures of RV mechanics were not routine. Another recent publication, by Topilsky et al. (4), found that a decrease in the timing interval between the onset and the cessation of tricuspid regurgitation flow corrected for heart rate predicted adverse outcomes following LVAD implantation. A number of other studies using fewer variables in their analyses have found clinical, biochemical, hemodynamic, and echocardiographic parameters to be useful in their populations. However, there has been no report from larger, multicenter cohorts to confirm the utility of many of these metrics.

The authors should be commended for their work to increase our knowledge on the prediction of RV failure in the LVAD recipient. More work is needed to determine whether these findings can be applied to other centers with different imaging and clinical practices. Additionally, more work needs to be done to improve outcomes in high-risk patients once they are identified. A number of studies have now reported improving perioperative survival with our current methods (5,6). Further improvements may involve more aggressive use of mechanical support of the RV, novel

mechanic support devices, and/or novel pharmacologic therapy, or patient selection should simply be refined. However, as progress is made in the perioperative management of the LVAD recipient, better prediction of RV failure will become more important in selection and management decisions. This issue is vital because getting the prediction right is critically important to outcomes in LVAD candidates.

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Key Words: assist device ■ heart failure ■ right ventricle ■ strain.