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Reviews:

[Proximal thoracic aorta dimensions after continuous-flow left ventricular assist device implantation: longitudinal changes and relation to aortic valve insufficiency.](#) Fine NM, Park SJ, Stulak JM, Topilsky Y, Daly RC, Joyce LD, Pereira NL, Schirger JA, Edwards BS, Lin G5, Kushwaha SS. J Heart Lung Transplant 35(4): 423-432.

It is well-known that aortic valve (AV) regurgitation (AR) occurs de novo and/or worsens following left ventricular assist device (LVAD) support, particularly using continuous-flow VADs. However, the physiological and molecular mechanisms that underlie this process are poorly understood. In patients with well-functioning LVADs, the trans-aortic valve regurgitant flow rate is determined by the difference between the aortic pressure and intracavitary LV pressure, and impedance of the AV to regurgitant flow. In turn, the aorta-LV pressure difference is causally dependent upon the LVAD pump speed and the impedance of the systemic arterial circulation (the primary component of systemic afterload) for any given level of LV preload. Thus, the regurgitant flow rate and AR "severity" are proportional to left-sided output (principally through the LVAD), although the nature of the relationship is influenced by left-sided afterload. Another way to state this is that the absolute regurgitant flow rate and the regurgitant fraction are distinct entities; the former is what the systemic tissues/organs "see" in terms of the deleterious effects of AR, while the latter quantifies the circulatory inefficiency that AR causes. In summary, both de novo and worsening AR need to be assessed in the context of native LV and LVAD function, and left-sided loading conditions.

Moreover, the physiological and clinical significance of post-LVAD implantation AR are unclear. While a sizable percentage of patients develop a minimum of "mild-to-moderate" AR, comparatively few develop severe AR or AR requiring procedural treatment. Previous studies from the University of Michigan and Duke have shown a small clinical impact of post-LVAD implantation AR, in terms of patients requiring procedures to treat AR. In aggregate, these findings are consistent with the concept articulated above, i.e., that much of worsening and even de novo AR (although "de novo" may represent an increase relative to trace AR not well-appreciated pre-LVAD implantation) is due to an assessment of worsening overall regurgitant flow rates in the setting of increased left-sided output in the post-LVAD state compared to the pre-LVAD state, rather than a meaningful increase in the regurgitant fraction of left-sided output. That stated, patients with moderate or worse AR at the time of LVAD implantation typically undergo AV repair or replacement, and thus, these patients who might otherwise progress to severe AR post-LVAD implantation are effectively censored due to their up-front treatment.

It is in this complex background and context that Fine et al. from the Mayo Clinic publish a novel study in this month's Journal. This group has previously contributed greatly to the understanding of the effects of LVAD dynamics on AV function. **The effects of LVAD support on the housing structure of the AV – the aortic root, and the ascending aorta as well, are largely unknown. Moreover, relationships between AV function and aortic geometry in the context of LVAD support are also unknown.**

The authors reviewed a series of 162 patients implanted with HeartMate II LVADs. 9 of these patients experienced what the authors defined as accelerated proximal (aortic root or ascending thoracic aorta) aortic expansion post-LVAD support, as defined by a well-accepted criterion of diameter increase of >0.5 cm/year. 8 of the 9 patients had pre-existing aortic root/ascending thoracic aortic dilatation, in comparison to 10 of 153 patients who did not exhibit rapid aortic expansion. Patients with accelerated aortic dilatation also had a greater incidence of hypertension (although blood pressure while on LVAD support was not significantly different), and were older. Finally, with respect to the kinetics of aortic dilatation, changes were not evident at 1 month, but were evident at 6 months and 1 year; moreover, further aortic expansion beyond 1 year was not observed.

Next, the authors examined AV function in their patient group, and identified factors associated with >mild AR post-LVAD implantation. 45/162 patients developed >mild AR, but only 1 patient required procedural treatment. Multivariable data analysis identified two factors associated with the development of >mild AR. First, although the majority of patients in the study had LVAD speed and loading conditions titrated to persistent AV closure, 80% of patients in the >mild AR group had persistent AV closure, in contrast to 53% of patients in the non-progression group. Second, a greater percentage of patients in the >mild AR group had rapid aortic dilatation (13% versus 0%).

In summary, this comprehensive single-center analysis identified that: (1) a small subgroup of LVAD recipients exhibit rapidly progressive proximal aortic aneurysmal dilatation, but that this generally occurs in patients with pre-existing proximal aortic aneurysm/dilatation and stabilizes after 1 year post-implant, and (2) >mild AR occurs in a large subset of LVAD recipients, but rarely is severe or warrants intervention on the AV, with >mild AR being associated with persistent AV closure and rapidly progressive aortic dilatation.

The study prompts several questions. First, with respect to proximal aortic dilatation, although all patients with progressive dilatation had pre-existing dilatation, 10/18 patients who had pre-LVAD implant aortic dilatation did not experience rapidly progressive aortic expansion. LVAD support appears to cause many patients with pre-existing aneurysmal disease to develop progression, but many patients do not develop progressive expansion. What are the determinants of who does and does not develop expansion? Second, with respect to post-LVAD implantation AR, the study identified a high incidence of >mild AR, but only 1 patient underwent an intervention to treat AR (presumably severe). This underscores the previously outlined concepts that much observed AR may be an "artifactual" increase (and de novo AR may really be a change from "trace") due to higher overall left-sided output post-LVAD implantation relative to the low output state of the failing LV. In addition, the majority of patients in both those who did and did not develop >mild AR had persistent AV closure (98/162 patients). Although persistent AV closure was even more common in the >mild AR subgroup, it is important to note that, viewed using AV closure rather than >mild AR as the denominator, 62/98 patients with persistent AV closure did not develop >mild AR. Similarly, although progressive aortic dilatation was more common in the >mild AR subgroup, this amounted to only 13% of this subgroup; 87% of patients with >mild AR did not have progressive aortic dilatation. Consequently, while the study by Fine et al. is an excellent descriptive study, many questions regarding how LVAD support influences the physiology of AV function and aortic wall mechanics remain.

[Effect of body mass index on outcomes in left ventricular assist device recipients.](#) Go PH, Nemeh HW, Borgi J, Paone G, Morgan JA. J Card Surg 31(4): 242-247.

LVAD implantation is undertaken, broadly, in two patient populations: bridge-to-transplant (BTT), and destination therapy. With a potential heart transplant recipient population in vast excess of donor organ supply, organs are carefully allocated to "suitable" recipients. As a result, patients with conditions that render them poor heart transplant recipients are typically implanted with LVADs as destination therapy (DT).

Obesity, and most dramatically morbid obesity, are increasing in incidence, and are frequently reasons that patients are declared ineligible for heart transplantation, and thus rendered candidates for DT LVAD implantation. The impact of BMI and obesity upon post-LVAD implantation outcomes is unclear.

The authors, from Henry Ford Hospital, undertook an institutional review of 200 patients undergoing Heartmate II or Heartware HVAD LVAD implantation from 2006-2014. Mean BMI was within the overweight range: 28.3. 36.5% of patients were frankly obese (BMI >30), while an additional 36% were overweight (BMI 25-30). Patients who were obese were younger, with a median age of 51.9. This may reflect a selection bias insofar as older obese patients were not deemed suitable for LVAD implantation, or alternatively, few older obese patients were evaluated because of the adverse impact of obesity upon life expectancy. Obese patients more commonly had lower extremity arterial occlusive disease, and consistent with this, had a higher incidence of diabetes mellitus. Surprisingly, normal BMI patients were more commonly declared DT (67%), in contrast to overweight (39%) and obese (51%) patients. Moreover, postoperative stroke was more common in normal BMI patients (22%) than in overweight (6.9%) and obese (12.3%) of patients. Postoperative bleeding was also more common in normal BMI patients (27.8%) than in overweight (12.5%) or obese (9.6%) patients. Consistent with a higher complication rate in normal BMI patients, BMI was not found to be associated with an adverse impact upon post-LVAD implantation survival.

Although this study does suggest that in selected patients, BMI does not adversely impact outcomes post-LVAD implantation, the results are somewhat counterintuitive. It is likely that patient selection biases are inherent and explain much of the findings. Obesity is generally a reason to declare a patient DT as opposed to BTT; yet, many obese patients were BTT (presumably they were not morbidly obese). Moreover, the fact that a clear majority of normal BMI patients were declared DT suggests that other characteristics of these patients rendered them what we might call "poor protoplasm." This interpretation of the data is consistent with the higher incidence of postoperative complications such as bleeding and stroke. Nonetheless, the authors have shown that at least in selected patients, higher BMI does not substantially adversely affect outcomes.

Circulation

None

European Heart Journal

None

JACC: Heart Failure

[Development of a transplantation risk index in patients with mechanical circulatory support](#). Johnston LE, Grimm JC, Magruder JT, Shah AS. JCHF 4(4): 277-286.

The authors have developed a multi-factorial index that is geared towards ascertaining the risk of 1-year post-heart transplant mortality in patients with pre-transplant mechanical circulatory support. This may aid in determining the best candidates for carrying out bridging to heart transplantation. A potentially interesting subsequent question is whether some patients identified as "destination therapy" might be re-classified as being reasonable for bridging to transplantation based upon this study, just as the study has identified patients who underwent bridging with poor outcomes.

Annals of Thoracic Surgery

[National trends in utilization, mortality, complications, and cost of care after left ventricular assist device implantation from 2005 to 2011](#). Shah N, Agarwal V, Patel N, Deshmukh A, Chothani A, Garg J, Badheka A, Martinez M, Islam N, Freudenberger R. Ann Thor Surg 101(4): 1477-1484.