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Commentary: Measuring the impact of thoracic endovascular aortic repair on the heart

Jennifer Worsham, MD,[a] and Abe DeAnda, Jr, MD[b]

Abe DeAnda, Jr, MD, and Jennifer Worsham, MD

CENTRAL MESSAGE

TEVAR may have a detrimental impact on the left ventricle. The technique described herein provides a novel approach to assessing this impact.

Although the aorta is frequently regarded as simply a conduit, it can be appreciated as also having (in an electrical circuit analogy) resistive and capacitance properties, with a direct impact on downstream properties and function. Because of its capacitance qualities, the aortic wall may also have an impact on proximal (ie, left ventricular) function. In a stiffer, diseased aorta, cardiac output encounters a less compliant (ie, lower capacitance) conduit, resulting in increased afterload on the ventricle.

In the study reported in this issue of the *Journal*, Orihashi[1] has attempted to assess the impact of thoracic endovascular aortic repair (TEVAR) on left ventricular afterload and workload. The premise is simple; TEVAR by its nature changes the mechanical properties of the aortic wall by altering the stiffness of the wall. In the case report presented, Orihashi uses a variant of the familiar pressure–volume relationship usually associated with ventricular function studies and demonstrates increases in afterload and workload, both of which are detrimental to a failing heart. The traditional pressure–volume loops were not generated for multiple reasons, but nonetheless the resultant measures of afterload and workload appear to be appropriate.

As a clinical tool, this method might not be useful other than to alert the clinician of the potential need to modify cardiac therapy for the post-TEVAR patient, because the calculation needed to determine afterload and workload is post hoc. An increase in central pulse pressure alone, adjusted for age and risk factors, has been shown to have a relative risk of total cardiovascular events of 1.137 (95% confidence interval, 1.063–1.215).[2] The impact of increased stiffness is not limited just to changes in afterload and workload; Moulakakis and colleagues[3] demonstrated statistically significant increases in N-terminal pro b-type natriuretic peptide (from baseline) at 24 hours, 48 hours, and 6 months postoperatively. The method described by Orihashi has the benefit of near real-time assessment. Predictive models would be difficult although not impossible, as each patient would have a different ''circuit'' based on the material of the graft, the length and diameter used,[4] and the downstream aortic properties. On the other hand, as an experimental study, the approach described by Orihashi adds to our understanding of ventricular–aortic interaction and coupling. We believe that this latter result will having more staying power.

From the [a]Division of Vascular and Endovascular Surgery and [b]Division of Cardiovascular and Thoracic Surgery, UTMB-Galveston, Galveston, Tex.

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Address for reprints: Abe DeAnda, Jr, MD, Division of Cardiothoracic Surgery, UTMB-Galveston, 301 University Blvd, Galveston, TX 77551 (E-mail: abdeanda@utmb.edu).

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