Commentary Coselli

See Article page 498.



## Commentary: The hinge point doubles down—What's next for descending thoracic and thoracoabdominal aortic repair

Joseph S. Coselli, MD

Despite decades of study, there remains much to be learned about the natural history of descending thoracic and thoracoabdominal aortic pathology, both aneurysmal and dissection. In 1986, with limited information available on the natural history of distal aortic aneurysms, Crawford and DeNatale reported the long-term results of a small number of patients (n = 94) with thoracoabdominal aortic aneurysm who qualified for surgery after evaluation; ultimately, these patients were not operated on and were followed without surgical intervention over a 25-year period. Crawford and DeNatale observed that the natural history of this disease was dismal—only 24% of patients were alive at 2 years after evaluation, with one-half of all deaths due to rupture. In 1999, Randall Griepp and associates provided additional insight into the natural history of descending thoracic and thoracoabdominal aortic disease, studying 165 patients with degenerative aneurysm or chronic aortic dissection who were initially managed nonoperatively.2 Like Crawford and DeNatale, Griepp's group found a substantial rate of rupture (20%) during long-term observation, as well as high mortality in patients experiencing rupture, ranging from 75% in patients with degenerative aneurysm to 90% in patients with chronic dissection. They found that aneurysms related to chronic dissection were at an elevated risk of rupture at smaller aortic dimensions compared with those who had degenerative aneurysms (5.4 cm vs 5.8 cm). In

From the Division of Cardiothoracic Surgery, Michael E. DeBakey Department of Surgery, Baylor College of Medicine, Department of Cardiovascular Surgery, Texas Heart Institute, and CHI St Luke's Health–Baylor St. Luke's Medical Center, Houston, Tex.

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Joseph S. Coselli, MD

## **CENTRAL MESSAGE**

The descending thoracic and thoracoabdominal aorta appear to dilate more quickly than previously thought; likewise, the risk of an acute aortic event may occur at a smaller diameter. Should the diameter-based threshold of repair be shifted lower?

addition, they developed a formula to weigh the risk of aortic rupture versus the risk of surgery to better manage patients.

Over the years, John Elefteriades and others at Yale<sup>3,4</sup> have provided us with crucial information regarding the natural history of thoracic aortic disease by analyzing the robust database at the Aortic Institute at Yale-New Haven Hospital, which now includes data from nearly 4000 patients with thoracic aortic disease. In this issue of the Journal, Mohammad Zafar and coauthors at Yale<sup>5</sup> review a subset of patients with serial radiographic studies (n = 2384) after excluding patients (n = 1477) with congenital aortic malformation, traumatic aortic lesion, intramural hematoma, penetrating atherosclerotic aortic ulcer, iatrogenic aortic dissection, chronic aortic dissection (either Stanford type A or B aortic dissection) at the time of presentation to their center, and syndromic patients (ie, Marfan, Ehlers–Danlos, and Loeys–Dietz syndromes), as well as those patients with a descending thoracic aortic ≤3 cm in diameter. Longitudinal data from the resulting 907 patients were analyzed—a much larger study than prior natural history studies from our colleagues at Yale<sup>3,4</sup> or the studies by Crawford and colleagues<sup>1</sup> and Griepp and colleagues.<sup>2</sup>

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Address for reprints: Joseph S. Coselli, MD, One Baylor Plaza, BCM 390, Houston, TX 77030 (E-mail: jcoselli@bcm.edu).

Coselli Commentary

For patients with clear-cut symptoms (such as pain in patients with a contained rupture), there is general agreement that operative intervention should be performed on an urgent or emergent basis in nearly all patients. The authors point out that the current international guidelines recommend preemptive surgical intervention asymptomatic patients once an aortic diameter of 5.5 to 6.0 cm is reached.<sup>6,7</sup> However, it is well known that adverse aortic events-aortic dissection, rupture, and death-may occur in patients whose aortic size has not yet reached those dimensions. The authors report an important finding, that it may be impossible to use a diameter-based threshold of repair to prevent type B aortic dissection; the median diameter before dissection was only 4.1 cm, which is far below current guidelines. Fortunately, patients who experience type B aortic dissection typically survive, and there is little incentive to push for a lower threshold.

In addition, Zafar and coauthors determined the rate of distal aortic growth, finding an average growth rate of 0.19 cm per year that increased as the aorta grew larger. This rate was nearly double that previously determined by the Yale group. Likewise, the hinge point—the diameter at which the risk of a catastrophic aortic event is strikingly apparent—changed from that previously identified by this group. In a 1999 report, Coady and colleagues noted that the risk of a serious distal aortic complication increased dramatically at 7.0 cm (the hinge point); thus, the target for repair was set at 6.5 cm to avoid complication. In the study of Zafar and colleagues, the sample size was sufficiently large to allow analysis of the risk of aortic rupture and aortic death separately. Here the hinge points of these catastrophic events have shifted to lower diameters:

6.0 cm for aortic death and 6.6 cm for aortic rupture. As a result, the authors suggest that the targets for aortic repair shift lower, to a range of 5.0 to 5.5 cm, that can then be fine-tuned by patient-specific factors. Although it may be time to double down on the threshold of repair, only experienced aortic centers with outstanding results should manage patients toward elective repair at the lower end of this range (5.0 cm).

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