A step forward or running from risk?

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Type A acute aortic dissection is a common aortic emergency that has an exceedingly high mortality when left untreated. Even with surgical treatment, the early International Registry of Acute Aortic Dissections (IRAD) survival data revealed a hospital mortality of 349 of 1160 (29.5%) for all type A dissections.¹ Visceral malperfusion has long been recognized as an independent predictor of in-hospital mortality, despite central repair of type A dissection.² Fortunately, IRAD has demonstrated that only about 3% of patients presenting with Type A aortic dissections have concomitant mesenteric ischemia.³ Similarly, an Italian registry described inferior results for patients with mesenteric ischemia despite timely repair of the central aortic dissection.⁴ This is unequivocal, and has been reported numerous times. It is therefore evident that a change in approach should be considered, addressing the malperfusion at the outset if possible. This has been well described by numerous authors, but probably best by the Michigan group. As early as 1997, Deeb and associates⁵ proposed an alternative pathway, performing fenestrations to restore visceral flow before central repair. Addressing the ischemia up front promoted improved survival in 88% of patients. In light of this evidence and further clinical experience, Patel and colleagues⁶ proposed an alternative algorithm that addressed the malperfusion syndrome first. In a further study by the Michigan group,⁷ a mortality of 17% from aortic rupture was observed while patients awaited resolution of their malperfusion syndrome under a protocol that specified distal repair first.

In this issue of the *Journal*, Lawton and coauthors⁸ have looked at an almost 20-year experience with type A dissection at an IRAD aortic center. The purpose of the study was to look at malperfusion, acidosis, and the combination of these as markers for mortality risk. There were a total of 282 type A dissections seen, with an operative mortality of 18%. There were 80 cases of malperfusion, with a mortality of 80 of 282 (28%), versus 22 of 202 (11%) for those without malperfusion. There were 66 patients who had a base deficit of 5 or greater, with 24 of these having a base

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Central Message

Type A dissection with acidosis and malperfusion needs a new treatment strategy.

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deficit of 10 or greater. For the 44 patients with a base deficit of 5 to 10, the mortality was 11 of 42 (26%); for those with a base deficit greater than 10, the mortality was 11 of 24 (46%). Finally, they looked at the combination of acidosis and malperfusion, of which there were 21 cases. For malperfusion with a base deficit of 5 to 10, the mortality was 10 of 21 (48%); for malperfusion with a base deficit of greater than 10, the mortality was 11 of 12 (92%). Aortic surgeons already know most of this in broad terms, with Lawton and colleagues⁸ adding a more exact measure of the risks. This article points out the continued high mortality associated with type A dissection and better defines groups with an exceedingly high mortality.

For type A dissection with malperfusion or acidosis, the fundamental concept of tackling the malperfusion syndrome first by a less invasive and more prompt endovascular route raises a question: Why not extend that repair to the ascending aorta? The potential benefits of delaying the repair of a type A dissection to let the inflammatory cascade of mesenteric ischemia abate is contrasted by a persistent notable risk of rupture for those whose definitive aortic operation is delayed. In describing poor outcomes for patients with mesenteric ischemia in the setting of a type A dissection, Lawton and coauthors⁸ have better stressed something we already know. They conclude that an alternate algorithm should be entertained. In this age of high scrutiny for cardiac surgery outcomes, in which every death may be held against the surgeon, the question is what we do with this information. In the early days of repair of post-myocardial infarction ventricular septal defect it was found that waiting 6 weeks for the tissues to heal greatly improved the surgical mortality. Further study, however, showed that

this was at the cost of losing 90% of the patients during this interval of healing.⁹ So in essence, what is the risk of avoiding risk? Is the solution to avoid surgery in these patients, who only account for about 3% to 4% of all patients with type A dissection, or should we simply restore perfusion to the gut and accept the ruptures that may occur during the interval between mesenteric recovery and definitive aortic repair? We thank Lawton and coworkers⁸ for bringing this information forward to the aortic community. Now the choice is ours to make: Do we take a step forward, or run from risk? We propose a step forward, with minimally invasive endovascular approaches being extended not only to restore the mesenteric insufficiency but also to repair the ascending aorta definitively by thoracic endovascular aortic repair.

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