Peas and carrots, apples and oranges: Not all malperfusion is the same

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Central Message

The jury is still out on the best management strategy for acute type A aortic dissection, delayed operation after endovascular reperfusion versus immediate operation.

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In a provocative early study, Deeb and colleagues¹ proposed the notion of an endovascular reperfusion first approach to treat acute type A aortic dissection (ATAD) complicated by severe end-organ dysfunction. This strategy was based on a seminal work by Williams and colleagues² that described the pathophysiology contributing to malperfusion syndrome with aortic dissection. With this background, the Michigan group (of which I am part) has for almost 25 years espoused the notion of delaying operative management for those presenting with acute type A aortic dissection and severe malperfusion syndrome until after resolution of the ischemic insult. The late results of this approach were described in the *Journal* 10 years ago and suggested that when revascularization was undertaken first, late survival for patients with malperfusion was similar to that of those who presented without malperfusion.³ In that study, of the 70 patients with malperfusion, 23 (32.8%) died before open repair; these included 12 deaths (17.4%) from aortic rupture and 11 deaths (15.7%) from complications associated with the malperfusion syndrome.

In the 2 decades since the initial descriptive study of Deeb and colleagues,¹ a number of investigators have described results of both immediate and delayed strategies for acute type A aortic dissection with malperfusion, with varying results. The study by the Stanford group reported in this issue of the *Journal* by Chiu and colleagues⁴ extends this work and espouses an immediate surgical strategy, with acceptable results. In all these evaluations, however, a direct comparison between strategies is lacking.⁴⁻⁸

The primary issue that likely contributes to the confusion remains the definition of what constitutes malperfusion. Is it a radiographic finding of reduced enhancement of a vascular bed, which could be confounded simply by poor timing of image acquisition relative to contrast bolus administration? Is it simply the loss of a pulse in an extremity, as described in the current work by Chiu and colleagues⁴? Or is it as Deeb and colleagues¹ described it,

involving significant end-organ dysfunction from prolonged ischemia resulting in hematochezia or limb sensorimotor changes?

The second but equally important issue that contributes to confusion revolves around the vascular beds affected by ischemia. In the experience of my group, malperfusion is assessed not only by clinical means but also by manometric assessment of aortic root to branch vessel gradients. My group's previous study,³ which used this approach, found multiple territories to be affected in 40 of 70 patients (57.1%); this finding contrasts with the 27% identified in the report of Chiu and colleagues.⁴

The final contributor to confusion lies in the relative distributions of visceral, renal, or limb malperfusions. For example, the study of Chiu and colleagues⁴ describes 7 patients (8.5%) with visceral malperfusion, whereas the previous report of my Michigan group³ describes 37 (52.8%).

The Michigan message has been clear. Operate on all patients with acute type A dissection immediately, unless there is evidence of significant end-organ dysfunction from ischemia. In that case, restore perfusion with an endovascular approach, wait until the ischemia-reperfusion injury has resolved, and then take the patient to the operating room. As far as deciding which strategy should be the criterion standard, as suggested by the Stanford group, until the issues described above are consistently clarified and categorized, we will continue talking about peas and carrots, apples and oranges. They are all fruits and

vegetables—no real scientific evidence exists to say which is better, and it is all a subjective, opinionated matter of taste.

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