

Commentary: Myocardial protection is a process, not an event



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Nathaniel B. Langer, MD, MSc (left), and Gus J. Vlahakes, MD (right)

Central Message

Myocardial protection is a process, not an event.

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The study by Timek and colleagues¹ in this issue of the *Journal* is the most recent addition to the literature on myocardial protection in adult patients. Dr Pedro del Nido's cardioplegia formulation has made a major contribution to pediatric cardiac surgery.² Because of this success, it continues to gain a greater and greater foothold in adult cardiac surgery; however, its broad adoption remains controversial. The current publication of Timek and colleagues¹ is another contribution supporting its application, but it must be interpreted with certain caveats.

The advent of myocardial protection, heralded by the landmark contribution of Gay and Ebert,³ revolutionized cardiac surgery, making significant improvement in outcomes, allowing more complex surgeries to be done, and allowing surgeons to take the needed time to take trainees through operations.⁴ Irrespective of the formulation used, the basic principles of myocardial protection remain constant: delivery to the entire heart; complete, sustained electromechanical arrest; cooling; and buffering capacity. These principles have not changed and continue to be the underpinning of any myocardial protection method. Traditional cold blood cardioplegia achieves this, but repeated dosing is needed, especially for crossclamp times that exceed an hour, potentially interrupting the flow of the surgical steps in progress.

Multiple studies, including single-institution series, meta-analyses, and a prospective, randomized trial, have shown the relative safety and efficacy of del Nido cardioplegia in adult patients with both valve and coronary disease.⁵⁻⁷ Proposed advantages include shorter crossclamp times, smaller volume administration, and even superior myocardial protection. Timek and colleagues¹ do not report significant advantages with del Nido cardioplegia, but they did find noninferior myocardial protection and overall postoperative outcomes.

The study of Timek and colleagues¹ must be interpreted keeping in mind that the crossclamp times were not inordinately long. The issue of more complex cases requiring

extended crossclamp times, however, remains in question vis-à-vis the applicability of del Nido cardioplegia with long dosing intervals.

One advantage of del Nido cardioplegia may be its superior ability to produce uninterrupted, dense electromechanical arrest, including suppression of micromotion not evident on simple electrocardiographic leads. Thus, extended crossclamp times well beyond those used in this study still require enough repetitive dosing to maintain adequate protection, particularly of the abnormal ventricle. Evidenced-based dosing intervals do not yet exist.

If the literature supports its use, and del Nido cardioplegia streamlines the flow of procedures, then why has it not been more widely adopted? We believe that this is likely due to individual surgeon comfort and to concern regarding how del Nido cardioplegia might perform during extended crossclamp periods. The available studies, including this study, do not include enough patients with long crossclamp times. Caution must therefore be taken in adopting its conclusions for cases requiring extended crossclamp times.

The study reported by Timek and colleagues¹ is further evidence that single-dose del Nido cardioplegia is safe for revascularization operations conducted with the crossclamp times in this study. Timek and colleagues¹ acknowledge that they do not address the major remaining question, however, which is how best to protect the heart for extended periods. Until that question has been answered, the debate will continue.

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