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Commentary: Potential advantages of terminal warm blood cardioplegia: Will the “hot shot” approach become hot stuff?

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The search for optimal strategies to protect the arrested heart during cardiac surgery continues unabated. Current anesthesia and perioperative management protocols have made reperfusion-mediated injury upon declamping seem less of a problem.^{1,2} Yet cardiac arrest intrinsically carries with it deleterious effects on myocardial metabolism, particularly when prolonged beyond 60 minutes.

Many different approaches have been proposed to minimize this problem; however, research has been hampered by limitations when it comes to assess results. Differences in “hard” endpoints (eg, mortality) are difficult to explore in clinical trials owing to the huge sample size required and virtually impossible in experimental studies; thus, investigators have typically resorted to much “softer” endpoints, such as troponin release or incidence of arrhythmias following declamping. Clearly, this is unlikely to provide solid, satisfactory answers.

The study by Nakao and colleagues³ reported in this issue of the *Journal* goes a step forward. Using a left ventricular (LV) conductance catheter, they were able to precisely assess, in a load-independent fashion, LV contractile function through measurements of end-systolic elastance (% Ees) and compliance via the end-diastolic pressure–volume relationship (%EDPVR), to investigate in piglets the effects of warm-blood cardioplegia (TWBCP) administered at the end of a prolonged (120 minutes) single-dose

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CENTRAL MESSAGE

A shot of warm-blood cardioplegia before declamping achieves prompt metabolic and contractile recovery of arrested hearts in an experimental model, as demonstrated by an objective assessment of function.

cardioplegic arrest achieved with del Nido solution compared with del Nido cardioplegia alone. After a 150-minute recovery, Ees was largely depressed in the del Nido group but substantially better in the TWBCP group ($57.9 \pm 17.8\%$ vs $94.7 \pm 13.1\%$; $P < .028$); EDPVR was also better in the TWBCP group ($88.5 \pm 24.0\%$ vs $101.4 \pm 16.8\%$; $P = .050$). These findings objectively support the authors' conclusions that a “hot shot” of warm cardioplegia before declamping on top of del Nido cardioplegia may allow safe prolongation of the arrest time up to 120 minutes.

On the mechanistic side, the authors complemented the study with data from cardiac biopsies, showing higher myocardial glycogen content in the TWBCP group, whereas mitochondria ultrastructure was altered only modestly and was similar in the 2 groups. The glycogen data hint at preserved glycolysis as a possible mechanism underpinning those results,⁴⁻⁶ whereas in the authors' model, preserved mitochondrial morphology might be indicative of myocardial “stunning”, not of irreversible injury.⁷

Although interesting, these findings might not provide the ultimate answer as to the best cardiac protection strategy for prolonged arrest. Important issues are left unaddressed and/or require further research. Piglets are not human babies, let alone adult or elderly people—those in whom cardiac surgery is performed. In addition, these results apply

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only to a strategy of single-dosing del Nido cardioplegia; it is unknown whether a terminal “hot shot” maintains an edge when redosing and/or different cardioplegic solutions are used. Finally, as differences in LV function were documented just a few hours after declamping, it is possible that we might be looking at a speedier recovery rather than a persistent difference in cardiac function, as the finding of substantially preserved ultrastructure (consistent with stunning) might suggest.

In conclusion, Nakao and colleagues are to be commended for addressing an important issue through appropriate methodology. Specifically tailored studies in patients will demonstrate whether the hot shot approach will live up to its promise.

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Commentary: Del Nido cardioplegia with a “hot shot”

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In this issue of the *Journal*, Nakao and colleagues¹ use a piglet model of cardiopulmonary bypass with Del Nido cardioplegic arrest to demonstrate enhanced early recovery of left ventricular function with administration of a dose of terminal warm blood cardioplegia (TWBCP, aka a “hot shot”). The physiologic effects of the combination of prolonged Del Nido cardioplegia followed by TWBCP have not been characterized in an experimental model previously. Designed to facilitate pediatric surgeries, Del Nido cardioplegia use in adult cardiac surgery is not commonly used.

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CENTRAL MESSAGE

Use of terminal normothermic whole blood cardioplegia promotes recovery of left ventricular contractility and compliance after prolonged single-dose Del Nido cardioplegic arrest.

The use of Del Nido cardioplegia in adult patients has been limited by concerns about increased arrhythmias, decreased contractility, and decreased glycogen stores outweighing the potential benefits of uninterrupted flow of the surgical procedure.² However, if capable of comparable outcomes in the adult population, single-dose del Nido

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