the functional outcome in addition to mere histologic injuries.

Jung and colleagues³ show preliminary observational data that indicate that myocardial ischemic injury progresses gradually, and that irreversible ischemic injury of the heart with severe reduced cardiac function begins to occur 90 minutes after initial DNC infusion.

See Article page 528.

Commentary: How long can you go with del Nido?

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“Another 5 minutes...” Most cardiac surgeons have uttered something to this effect while our perfusionist is hinting that we should redose del Nido cardioplegia. Although clinical experience tends to dictate much of what we do, this holds especially true with our use of del Nido. A multitude of studies have demonstrated equivalent myocardial protection compared with conventional cold blood cardioplegia in adult cardiac surgery, with the advantage of a single dosing regimen. Expert opinion suggests redosing if the ischemic time is likely to extend much beyond 90 minutes.^{1,2} Are we just settling, or are we truly providing optimal myocardial protection? Answering this question in a clinical study is impractical and would require a prohibitive number of patients.

In an attempt to better understand the progression of myocardial ischemic injury and a safe ischemic time after single dose del Nido, Jung and colleagues conducted an ingenious experiment, reported in this issue of the

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CENTRAL MESSAGE

Pathoanatomic data suggest that a single dose of del Nido cardioplegia with myocardial hypothermia provides 90 minutes of safe ischemia.

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Journal.³ The authors obtained myocardial tissue from the explanted hearts of transplant recipients, which were arrested before explantation with 1000 mL of del Nido cardioplegia, and then placed in a cold saline bath with the myocardial temperature maintained at 18°C. Using electron microscopy, they evaluated mitochondrial and nuclear changes that can distinguish between reversible and lethal myocyte damage over the first several hours after the cardioplegia dose. The mitochondrial data are most compelling. Injury scores worsened with longer ischemic time, but even at 90 minutes, only 1 in 100 mitochondria had a lethal injury. The proportion increased 5-fold over the next 90 minutes.

These data provide compelling pathoanatomic support for a single-dose strategy providing up to 90 minutes of safe ischemia. Whether redosing at the 90-minute mark attenuates further lethal injury remains to be established. Of

course, the conditions of this study might not be applicable to the operating theatre, in which heat sources such as the viscera and overhead lights make it impossible to maintain a constant and uniform myocardial temperature of 18°C. In addition, the hearts in this study suffered from chronic failure and thus might have tolerated longer ischemic times.

Del Nido cardioplegia was developed with attention to sophisticated biochemical measurements of high energy phosphates, intracellular calcium, and acid-base buffering levels within myocytes.⁴ It is fascinating to see the application of more traditional anatomic phenotyping to correlate with these. This study should provide some reassurance to those skeptical of single-dose cardioplegia, at least as far as the first 90 minutes go, and with more

aggressive myocardial cooling, perhaps even up to 120 minutes.

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