New model for an old problem: Myocardial edema and dysfunction in neonates

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The neonatal myocardium is intrinsically different at many levels and more susceptible to ischemia when compared with adults, even more evident after surgical corrections for congenital heart disease. One way the myocardium of the neonate responds to the ischemic condition is by increasing myocardial edema. This response often results in delayed sternal closure for the neonate, which may cause increased postoperative morbidity and mortality. Developing a methodology in an animal model to measure myocardial edema is critical to better elucidate the biology of this condition and to develop novel therapeutic interventions.

In this issue of the *Journal*, Lueck and colleagues¹ present for the first time a comparison of the effect of ischemia on neonatal versus adult myocardial edema using bioelectrical impedance and electron microscopy. The authors demonstrated an earlier and more pronounced effect of ischemia in gap junction uncoupling and alterations of myocardial structure in neonatal swine myocardium compared with adult swine. The vulnerability of immature relative to mature myocardium to ischemia and subsequent acidosis has been established and is related to a diminished buffering capacity and increased lactate production in neonatal myocardium. This in turn leads to an increase in the intracellular sodium concentration and resultant flow of water from the extracellular to intracellular compartment, leading to the clinical sequelae of myocardial edema and dysfunction. In the setting of postoperative congenital cardiac surgery, this response of the myocardium can translate to delayed sternal closure in the neonates. Factors that cause delayed sternal closure in neonates after cardiac surgery have been attributed to duration of cardiopulmonary bypass and crossclamp time, type of congenital heart defect, age, and other variables.² However, the exact cause and timing of pathologic myocardial edema and gap junction uncoupling are not clearly delineated.

With the current study, however, Lueck and colleagues¹ have demonstrated a successful and reproducible

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Central Message

The potential clinical implications of the model described in this issue of the *Journal* as it applies to neonatal congenital cardiac surgery are discussed.

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methodology for determining the timing and severity of myocardial intracellular edema and alterations of myocardial structure. The potential clinical implications of determining myocardial edema are encouraging. One can imagine similar experiments performed in neonatal swine on cardiopulmonary bypass or circulatory arrest for varying durations, evaluation of new cardioplegia solutions, and the effect of targeted interventions. In addition, one can assess how current practices, such as the use of modified ultrafiltration, for example, affect the parameters of bioelectrical impedance and the number of light gray areas seen on electron microscopy. Other applications of this methodology would include how nonischemic conditions of pressure and volume overload affect myocardial edema. When applied to clinical conditions relevant to congenital cardiac surgery, the readouts from this model could influence clinical decision making for these challenging neonates.

References

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