Is Too Much Oxygen Bad for the Heart?

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In this addition of the Seminars, Peng et al report the effects of normoxic (21% O_2) vs hyperoxic (100% O_2) reperfusion following cardioplegic arrest.[1] In an in-vitro model of neonatal cardiomyocytes, they demonstrated that hyperoxic reoxygenation increased the inflammatory response and apoptosis compared to normoxic reperfusion. In an intact rabbit model with 60 minutes of crystalloid cardioplegic arrest followed by 120 minutes of reperfusion (60 minutes on partial cardiopulmonary bypass (CPB) and 60 minutes off CPB), rabbits perfused with 100% O_2 had a significant decrease in +dP/dt, LVDP, and an increased production of reactive oxygen species, IL-6, and apoptosis. The authors concluded that hyperoxia should be avoided during reperfusion following cardioplegic arrest on CPB.

How can we relate these experimental findings to the current practice of cardiac surgery involving cardioplegic arrest and reperfusion on CPB? In order to do so, we need to determine whether the experimental model used by the authors is analogous to current techniques of myocardial protection and CPB.

The authors chose to examine 2 extreme groups of oxygenation during reperfusion—hyperoxic (100% O_2) and normoxic (21% O_2). Many centers may start reperfusion with 100% O_2 for a short period followed by 40–60% oxygenation for the remainder of the reperfusion period which is usually within 30 minutes following aortic unclamping. The authors not only used an increased level of oxygenation but also a prolonged period of partial CPB which, by itself, could have contributed to altered myocardial contractility and compliance, and increased levels of inflammatory factors such as IL6. Furthermore, the hemodilution associated with the prime used this rabbit model on CPB, undoubtedly contributed to changes in myocardial edema which alters contractility and compliance. Although both normoxic and hyperoxic groups received a crystalloid prime, a blood prime would have resulted in less hemodilution. Similarly, a blood cardioplegic solution has been associated with superior myocardial protection, and should have been used. Although the authors claim that they use a crystalloid solution in their own practice, the solution used in these experiments resulted in only a 70% recovery of +dP/dt and an 80% recovery of LVDP in normoxic hearts—far less than one would expect following 60 minutes of aortic clamping.

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Central Message

Reperfusion under hyperoxemia (100% O_2) in a rabbit model of cardioplegic arrest and reperfusion on cardiopulmonary bypass resulted in decreased myocardial contractility and an increased inflammatory response compared to reperfusion under normoxic (21% O_2) conditions. These findings will need to be confirmed in clinical studies utilizing standard protocols of cardiopulmonary bypass and myocardial protection commonly used in clinical practice.

The authors based the recovery of myocardial function on data derived from a Millar catheter to determine +dP/dt and left ventricular diastolic pressure (LVDP). A more accurate estimation of myocardial performance could have been obtained from intraoperative two-dimensional echocardiography which provides an assessment of not only contractility but also compliance and wall motion changes. Finally, the authors focused only on the recovery of myocardial function following normoxic vs hyperoxic reperfusion. However, it is not known whether hyperoxic reperfusion may actually be beneficial to the brain, kidney, or other visceral organs following periods of CPB.

The authors plan to assess the implications of normoxic vs hyperoxic reperfusion from their experimental studies on

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clinical outcomes following congenital heart surgery using their established institution protocols for CPB and myocardial protection. I would encourage them to also include a group of patients that will undergo reperfusion with oxygenation between 40% and 60%. In addition, the effects of oxygenation should also be determined not only on the myocardium, but also on other organs such as the brain and kidney. Such a study will help to determine whether too much oxygen is really bad for the heart.

REFERENCE

1. Peng Y-W, Mohammed A, Deatrick KB, et al: Differential effects of normoxic and hyeroxic reperfusion on global myocardial ischemia-reperfusion injury. Semin Thorac Cardiovasc Surg 31:188–198, 2019