Myocardial insult during cyanotic heart disease surgery

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Although myocardial ischemic damage has been reported to be a common cause of early death in children, in the current era, myocardial failure is a rare primary cause of death after pediatric cardiac surgery[1,2].Nonetheless, a fall in cardiac output is seen in most patients between 6 and 12 hours postoperatively[3]. The most important cause of an excessive fall in cardiac output postoperatively is a residual volume load or pressure load, I.e., an imperfect repair[2]. Other causes include

- Ventricular distention - failure to vent the heart adequately
- Retraction / stretch injury to the myocardium
- Coronary artery injury
- Ventriculotomy
- Edema - inappropriate degree of hemodilution of red cells or colloid oncotic pressure
- Perfusion factors
- Reperfusion condition, e.g., pressure, calcium, oxygen, additives such as adenosine and free radical scavengers

To prevent or reduce a myocardial insult during cyanotic heart surgery, we should focus on patients with cyanotic congenital heart disease (CHD), which are divided into two groups, such as neonates or young infants with cyanotic CHD and older children or adults with untreated or palliated cyanotic CHD. The presence of severe hypoxia has been identified to be deleterious to the myocardial metabolic profile and ventricular function in animals[4] and clinical settings[5]. Najm et al[5] investigated the effect of cyanosis on myocardial adenosine triphosphate (ATP) levels and function in children with tetralogy of Fallot undergoing surgical procedures, demonstrating the deleterious effects of cyanosis on preoperative ventricular function and base-line ATP levels and its adverse effects on ATP levels during early ischemia and reperfusion. In addition, they showed an association of cyanosis with worse postoperative ventricular function and a more complex postoperative hospital course.

With regard to myocardial insult in cyanotic patients, several studies have documented the occurrence of an oxygen-derived free radical injury with reoxygenation on cardiopulmonary bypass (CPB)[6-9].

Despite experimental and clinical findings verifying the existence of reoxygenation injury, many surgeons may still believe that this is an unimportant injury, and of little clinical significance,

because of lack of data verifying impaired cardiac function and of biochemical parameters closely correlating with poor surgical outcome attributable to reoxygenation injury[10].
A well-organized randomized clinical trial with larger volume, appropriate stratification by age and degree of preoperative cyanosis and with more sensitive methodology to assess ventricular performance will be essential to clarify the true role of this surgical injury and the validity of therapeutic modalities designed to reduce reoxygenation injury.

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