OBJECTIVES:
1- Review the risk of myocardial injury in pediatric cardiac surgery.
2- Discuss the various cardioplegia preparations for children with congenital heart disease.
3- Present the various methods of delivery of myocardial protection in children.

MYOCARDIAL PRESERVATION:
The surgical management of children with congenital heart disease has significantly advanced in the past decade. Patients with complex defects are treated at a younger age, and definitive repairs are pursued rather than palliations. Advances in the understanding of the mechanisms of myocardial injury, and the composition, delivery and conduct of myocardial preservation are an integral part of improved outcome. However, the surgical mortality secondary to myocardial failure remains a major obstacle despite these advances. Scattered areas of myocardial necrosis due to inadequate preservation were documented in 30% of the left ventricular myocardium. The concept of reperfusion injury, causing a “stone heart” secondary to a massive myocardial infarction remains a challenge despite being reported by Cooley and colleagues over thirty years ago.

Deteriorating cardiac performance was the major factor contributing to postoperative mortality in 16/27 infants following cardiac surgery in a 1975 report. Myocardial dysfunction correlated with postoperative mixed venous oxygen partial pressure and systemic vascular resistance. With intermittent ischemia and reperfusion despite cardioplegic protection, the mortality increases sharply with a total cross-clamp time more than 85 minutes. Myocardial failure, documented by deteriorating cytochemical and biophysical parameters, is the cause of 50% of postoperative hospital deaths in children following cardiac surgery. Using conductance and Mikro-Tip pressure catheters to create real-time pressure-volume loops reflecting load-independent indices of left ventricular function, a recent report showed 40.7% decrease in end-systolic elastance, even in infants undergoing repair of simple congenital heart defects with cardioplegic myocardial protection and short cross-clamp durations. There is a clear correlation between age at operation, ischemic time and degree of myocardial injury as documented by postoperative ventricular compliance or serum markers. Although the immature myocardium is more tolerant to ischemia than the adult myocardium, myocardial injury and mortality are higher in the younger age group following cardiac surgery with ischemic arrest.

INTEGRATED APPROACH TO MYOCARDIAL PRESERVATION:
Myocardial protection in children undergoing repair of congenital heart disease requires an understanding of the mechanisms of injury, and an integration of the various methods of protection into a planned approach that begins preoperatively and continues through the surgical procedure. Preoperative interventions are aimed at resuscitating the myocardium and restoring metabolic state, especially in the neonate with a critical lesion. Ductal dependent lesions require rapid institution of prostaglandin E1 infusion to maintain coronary and systemic perfusion (as in hypoplastic left heart syndrome) or
pulmonary blood flow (as in pulmonary atresia). Adjusting the pH, fluid status, inotropic support and parenteral nutrition will help recover myocardial, renal, and hepatic function. Institution of mechanical ventilation must avoid run-off into the pulmonary circulation with hyperventilation, and ventricular distension from increased left-to-right shunting. In the operating room, rapid cooling and preischemic hypothermia may cause myocardial contracture especially in the presence of normocalcemia. The use of steroids (methylprednisolone or dexamethasone) pre-bypass can attenuate the inflammatory response and ischemia-reperfusion injury in neonates and children. Cardioplegia induction in the non-hypoxic neonatal heart can be done using cold crystalloid (4°C) or blood cardioplegia, with complete recovery of ventricular function. However, in the child with significant preoperative stress (hypoxia, pressure or volume overload), post-ischemic recovery of systolic and diastolic function is only achieved with a 5-minute infusion of warm, substrate-enriched (aspartate/glutamate) induction. The mechanism of warm, enriched cardioplegia protection in adults with ischemic heart disease is through improved cellular repair, re-establishment of ion gradients and a 5-fold increase in the capacity for oxygen utilization. In the hypoxic, stressed neonatal myocardium, warm induction improves recovery through amino acid enrichment causing increased endothelial NO production and antioxidant properties. Magnesium is added to the induction dose to counteract normocalcemia, and limit the amount of K+ needed for electromechanical arrest.

Non coronary collateral flow causes wash-out of cardioplegia and rapid rewarming of the myocardium from systemic perfusate, especially in the presence of large aorto-pulmonary collaterals. Myocardial hypothermia can be maintained by decreasing systemic flow rate (low-flow bypass), or using profound systemic hypothermia (<20°C). Alternatively, cardioplegia can be replenished by periodical dosing every 15-20 minutes. Intermittent cold cardioplegia will maintain myocardial hypothermia, wash out metabolites, buffer acidosis, and replenish high energy phosphates and depleted substrate. Continuous infusion of a cold, modified (no K+) integrated cardioplegia solution, antegrade or retrograde, at low pressure (30-50 mmHg) significantly improves recovery of ventricular function and coronary flow more than intermittent cardioplegia in neonates with significant hypoxic-ischemic injury. Removal of the cross-clamp exposes the neonatal myocardium to significant reperfusion injury with metabolic, structural and functional alterations. The brief antegrade infusion of warm, substrate enriched reperfusate (hot-shot) immediately before clamp removal can significantly improve systolic and diastolic recovery of the hypoxic myocardium following prolonged ischemic arrest.