Arginine-Vasopressin is Beneficial In Weaning Children From Cardiopulmonary Bypass Following Open Heart Surgery


Background: A relative arginine-vasopressin (AVP) deficiency state has been described as a potential cause of vasodilatory shock following cardiopulmonary bypass (CPB) in adults and children. Although there is considerable experience using low dose AVP in adults with vasodilatory shock coming off CPB, data on the use of AVP in children is limited. The purpose of this report is to describe the intraoperative use of low dose AVP during attempts to wean children from CPB after open-heart surgery.

Methods: We reviewed the records of 10 children (median age 2 months, range 3 days to 15 years) who received AVP for blood pressure (BP) support during weaning from CPB, following repair of congenital heart disease (n=9) or cardiac transplantation (n=1).

Before treatment with AVP, children received multiple pressors and inotropes including dopamine (n=1), dobutamine (n=8), epinephrine (n=7) and milrinone (n=6). AVP was used during the initial attempt to wean from CPB in 4, after an unsuccessful attempt in 5 and after two unsuccessful attempts in 1. Median dose of AVP was 0.004 units/kg/min and ranged from 0.0006 to 0.01 units/kg/min.

Results: After 15-30 minutes of AVP, mean arterial BP increased from 30 ± 10 to 57 ± 12mmHg (p<0.0001; paired t test; n=10). Systolic BP increased from 50 ± 14 to 82 ± 17mmHg (p<0.001; n=6) and diastolic BP increased from 29 ± 10 to 43 ± 14mmHg (p<0.05; n=6). Eight children were successfully weaned off CPB with concomitant rise in systolic BP. Two children went on to extra corporeal membrane oxygenation despite an initial rise in systolic BP with AVP. Eight children were successfully weaned off AVP and survived the perioperative period. Seven of these children were discharged from the hospital and one died 5 months following surgery. Two patients died 5 hours after CPB secondary to cardiogenic shock, despite treatment with AVP.

Conclusion: Low dose AVP is associated with BP in patients following CPB. Our data suggest that AVP may be beneficial in infants and children with hypotension secondary to vasodilatory shock who are unable to be weaned from CPB with conventional therapy. Further studies to identify ideal candidates for AVP therapy following CPB are warranted.

Glucocorticoids Reduce Troponin I Degradation in a Neonatal Piglet Model of Cardiopulmonary Bypass

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Degradation of troponin I (Tnl) by the calcium activated protease calpain is a cause of myocardial stunning due to ischemia/reperfusion injury. Glucocorticoids (GC) attenuate myocardial ischemia/reperfusion injury but their effect on Tnl degradation is unknown. We used a neonatal piglet model of cardiopulmonary bypass (CPB) to test the hypotheses that 1. CPB is associated with Tnl degradation in the neonatal heart and 2. Preoperative GC are associated with a decrease in CPB-mediated Tnl degradation and improved left ventricular (LV) function. METHODS: Neonatal piglets were cooled to 18°C C on CPB and subjected to 2 hours of circulatory arrest. Animals were rewarmed to 37°C and allowed to recover for 2 hours. Myophosphonol was administered 6 hours before surgery (30mg/kg) and at initiation of CPB (30 mg/kg). The control group received saline. LV tissue was collected at the end of recovery and tissue analyzed by chemiluminescent western blot for Tnl, calpain and the peptide inhibitor of calpain, calpastatin. RESULTS: Tnl degradation products were detected in LV myocardium after CPB, cold ischemia and reperfusion. In addition to intact Tnl at 29 kDa, selective degradation products of 22, 18, and 15 kDA were identified. Administration of GC was associated with alteration in the pattern of Tnl degradation products with increased preservation of intact Tnl and a decrease in the 22 kDa degradation product. Although the 80 kDa catalytic subunit of calpain 1 was increased to 111%±16 of controls (p<0.05), calpastatin levels were increased to 128%±7 of control levels (p<0.05). Mean LV +dp/dt decreased to 68%±15 of baseline in control animals by 2 hours of recovery, but was maintained at 92%±12 (p<0.05) of baseline in the GC group. CONCLUSIONS: Tnl degradation may be an important cause of myocardial dysfunction following CPB in neonates. Reduction in reperfusion injury by GC may be partly dependent on increases in calpastatin activity and/or subsequent preservation of intact Tnl.