Mode of Death After Congenital Heart Surgery

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Background. Two thirds of deaths after adult coronary artery bypass surgery are due to heart failure (O'Connor, 1998).

Method. To determine whether mode of death is the same or differs after congenital heart surgery, we evaluated the mode of death for the most recent 100 deaths at our institution.

Results. The mode of death was determined based on retrospective chart review including autopsy reports when available, and was assigned as the best understanding of the treating physicians at the time of death. Patients who died of a low output state were further categorized into pump failure (ventricular dysfunction); failed physiology (technically adequate surgery and ventricular function, but persistent low output state); and pulmonary hypertension (PAH). Additional variables indicative of pre- and post-operative status were also evaluated.

Conclusion. There was considerable anatomic diversity among patients who died; 44 cases had single ventricle physiology. Most deaths occurred in infants; median age at surgery 15 days; median age at death 47 days. The vast majority of patients (79) were in the ICU prior to the surgical procedure; 62 intubated, 52 intravenous inotropy, 49 prostaglandin, 6 nitric oxide, 12 hypoventilated, and 6 extracorporeal membrane oxygenation. Surgical reentry was noted in 23 cases; 7 patients died in the operating room.

Of 100 deaths, 56 were due to low output: 29 failed physiology, 18 pump failure, 6 PAH, and 1 other. Other causes of death included sudden cardiac arrest (11), sepsis (10), procedural complication (7), neurological injury (4), chronic lung disease (2), and other (10).

Among the 95 patients who survived to ICU, 48 had no known residual anatomical defect, 44 had residual defects, and 1 unknown. Of these, 38 underwent additional surgical (28) or catheter-based (10) procedures, although in some cases these procedures were performed to treat mild residual defects in an attempt to prevent the patient from death.

Conclusion. Mode of death after congenital heart surgery differs from adult cardiac surgery. Over half of the deaths were due to low output state, but not to pump failure.

Importance of Systemic-to-Pulmonary Artery Collaterals in Pediatric Heart Transplant Recipients

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The presence of systemic-to-pulmonary collaterals (SPC) after the Fontan operation can result in hemodynamic compromise, increased morbidity and mortality. METHODS: We retrospectively collected data on the occurrence of SPC and their relationship to morbidity and mortality after heart transplantation (HT). Between July 1988 and August 2003, we performed 152 transplant procedures. 73 transplants (48%) were performed for congenital heart disease and 20 (13%) were retransplant patients. Of the 73 patients with CHD, we performed 152 transplant procedures. 73 transplants (48%) were performed for congenital heart disease and 20 (13%) were retransplant patients. Among the 93 patients who survived to ICU: 48 had no known residual anatomical defect, 44 had residual defects, and 1 unknown. Of these, 38 underwent additional surgical (28) or catheter-based (10) procedures, although in some cases these procedures were performed to treat mild residual defects in an attempt to prevent the patient from death.

Conclusion. Of the 93 patients who survived to ICU, 48 had no known residual anatomical defect, 44 had residual defects, and 1 unknown. Of these, 38 underwent additional surgical (28) or catheter-based (10) procedures, although in some cases these procedures were performed to treat mild residual defects in an attempt to prevent the patient from death.

Role of the Lipoygenase-1 Receptor in Transducing the Apoptotic Signals of Circulating Atherogenic Low-Density Lipoprotein

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Background: LOX-1, a lectin-like receptor for oxidized LDL (oxLDL), is known to mediate various effects of oxLDL produced in vitro. A highly electronegative subfraction of human plasma LDL is oxidatively modified LDL (oxLDL), can inhibit thromboxane growth factor 2 (FGF2) transcription and induce profound apoptosis in vascular endothelial cells (EC) in the absence of oxLDL. To confirm the physiological significance of observations made with experimental oxLDL, we examined the role of LOX-1 in the transduction of LDL's signals.

Methods: LDL from hypercholesterolemic (HC; LDL-C > 160 mg/dL) human plasma analyzed by ion exchange chromatography yielded five subfractions, L1-L5. Apoptotic signaling was examined in bovine aortic EC (BAEC) exposed to L1-L5 in the presence or absence of JTX20, a specific LOX-1 neutralizing antibody.

Results: In cultured BAEC, L5, but not L1–L4, induced apoptosis in a time- and concentration-dependent manner. At 50 µg/mL, more than 50% of the cells exhibited DNA fragmentation and increased capase 3 activity at 24 hours. In the Bcl-2 family, Bcl-2 is antiapoptotic while BAX is proapoptotic. In L5-treated BAEC, the mRNA and protein levels of Bcl-2 were reduced, whereas those of BAX were unchanged, resulting in a reduced Bcl-2/BAX ratio. Supplementation of FGF2 prevented Bcl-2 downregulation and apoptosis. In contrast, vascular endothelial growth factor (VEGF), despite its mitogenic and antiapoptotic properties resembling those of FGF2, was ineffective. As expected, FGF2 transcription was inhibited in L5-treated cells. Interestingly, pretreatment of BAEC with JTX-20 attenuated FGF2 and Bcl-2 downregulation in a concentration-dependent manner. At 30 µg/mL, JTX-20 completely prevented FGF2 and Bcl-2 downregulation and apoptosis in L5-exposed BAEC.

Conclusion: L5, a novel proapoptotic LDL subfraction, acts in part by downregulating FGF2 and Bcl-2. These effects of L5 can be prevented by blockade of the LOX-1 receptor. Because EC apoptosis contributes to the initiation and progression of atherosclerosis, our results strongly indicate that the atherogenic effects of circulating electronegative LDL depends on signaling through the LOX-1 receptor.