ANESTHETIC CONSIDERATIONS IN CONGENITAL HEART DISEASE

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The approach to anesthesia in the child with congenital heart disease (CHD) is the same whether the heart has been reconstructed or the congenital heart disease has not been surgically treated. In both cases, the current clinical status of the child's cardiac pathophysiology must be understood. When anesthesia is being given for noncardiac surgery, cardiopulmonary bypass is not available for support if surgical and anesthetic intrusions overwhelm circulatory homeostasis. During noncardiac surgical procedures, surgeons unfamiliar with the physiologic limitations imposed by congenital heart disease may place substantial burdens on the circulation in the course of their procedures. Since the anesthesiologist must maintain the often fragile circulatory balance in spite of surgical trespass, destabilizing surgical insults must be anticipated in the anesthetic plan. Familiarity with the child's pathophysiology, the details of any reparative cardiac surgery, and the planned noncardiac operative procedure should avoid major problems in anesthetic management. Evaluation, preoperative preparation, choice of monitoring, induction, maintenance, emergence, and plans for postoperative care all are predicated on this familiarity.

STATUS OF REPAIR: THE RECONSTRUCTED HEART VS. THE UNRECONSTRUCTED HEART

Children with heart disease may present before cardiac surgical treatment, after palliation, or after "repair." Palliated patients still have a distinctly abnormal circulation but, hopefully, severe consequences of pediatric heart disease (severe congestive heart failure, severe hypoxemia, polycythemia, and pulmonary vascular disease) will not be a problem during anesthesia. It is important to note that, even with a reconstructed heart in patients whose heart disease has been surgically "corrected," the circulation must still be considered abnormal in the majority of cases. Arrhythmias, ventricular dysfunction, residual shunts, residual valvular
stenosis or regurgitation, and residual pulmonary hypertension all may remain or develop after surgical "repair" of pediatric heart disease. The most important aspects of the pathophysiology and the common circulatory abnormalities in the unreconstructed heart, or in the reconstructed heart after repair or palliation of common lesions, are outlined below.

**EFFECTS OF ANESTHESIA AND SURGICAL PROCEDURES ON PATHOPHYSIOLOGY**

Knowledge of the effects of anesthetic and operative manipulations on the pathophysiology of pediatric heart disease is necessary for optimal anesthetic management. Such manipulations may have a potent influence on cardiac function, venous return, and the ratio of pulmonary-to-systemic vascular resistance (PVR/SVR), which is particularly important in the pathophysiology of pediatric heart disease. Although anesthesiologists are familiar with manipulations of SVR, manipulations of PVR are less well understood. In lesions with inadequate pulmonary blood flow and hypoxemia, such as tetralogy of Fallot or a long-standing VSD-producing pulmonary vascular disease and high PVR with R-L shunting, anesthetic and surgical manipulations should be directed towards increasing pulmonary blood flow by decreasing PVR. At the same time, systemic vascular resistance (SVR) ideally is maintained or even increased to further favorably alter PVR/SVR. Although hyperventilation is the most reliable and efficacious way of decreasing PVR in children, it is pH and not pCO₂ that controls pulmonary vasoconstriction. Additionally, use of 100% O₂ is effective in decreasing PVR, as is optimizing FRC by avoiding PEEP and adjusting ventilation patterns to avoid hyperinflation of the lungs. Unfortunately, although many drugs are still touted as selective pulmonary vasodilators, none of these drugs reduces elevated PVR without also decreasing systemic vascular resistance. Nitric oxide (an investigational inhalation agent) administered to the lungs in a range of 10-80 ppm has specific pulmonary vascular dilating properties but has not been effective in all patients with increased PVR in early studies. Equipment for its administration is not yet commercially available, so its therapeutic usefulness in patients with congenital heart disease has yet to be defined.

Distention of the abdomen and collections of blood or fluid in pleural spaces also can substantially increase PVR, so drainage of these collections may be necessary. These maneuvers can have substantial