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Hypoplastic Left Heart Syndrome: Anesthesia for Elective Noncardiac Surgery

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Until this decade, hypoplastic left heart syndrome (HLHS, fig. 1*A*) was uniformly fatal. However, advances in surgical management of this lesion have improved the outlook for these infants. Currently, a two-stage procedure is the most common form of surgical intervention.[1,2] In the neonatal period, the palliative first stage is performed to ensure unobstructed pulmonary venous return and systemic outflow, and includes a systemic to pulmonary artery shunt to regulate pulmonary blood flow (Norwood Stage 1, fig. 1*B*). It is followed by a Fontan operation at 1–3 yr of age when pulmonary vascular resistance is low.[2,3] Survival after Stage 1 ranges between 60% and 70%,[1] and the oldest survivors of both stages of repair are now 10 yr of age. Although most of these infants are free from other congenital anomalies,[2,3] they can be expected to present for procedures commonly considered routine for pediatric patients.

Reviews of the anesthetic management of patients with HLHS[3–6] focus on that for repair of the cardiac anomaly; there have been no reports of anesthesia for noncardiac procedures after first-stage palliation. Many of the perioperative concerns in these patients are no different from those in other patients with complex congenital heart disease and systemic to pulmonary artery shunts. Maintenance of stable shunt flow is central to the anesthetic management of all these patients.

CASE REPORT

An 8-month-old 6.5-kg female infant presented for elective bilateral medial rectus muscle recessions with left lateral rectus resection. She was 4 kg at birth, the product of an uncomplicated term pregnancy. In the neonatal period she was noted to have a heart murmur, echocardiographic findings of HLHS, and significant strabismus. On day 9 of life, she underwent Norwood Stage 1 palliation of her cardiac anomaly. Postoperative echocardiograms showed good ventricular function, but her course was complicated by pneumonia and bronchospasm. She was discharged home at 7 weeks. Her only medication was digoxin.

After discharge she developed mild tachypnea (50–60/min), diaphoresis with feeding, acrocyanosis with vigorous crying, and moderate hepatomegaly. She was hospitalized once for viral pneumonitis. Addition of diuril and captopril to her cardiac medications decreased her congestive heart failure, and she grew steadily along a growth curve just below the 5th percentile for age. By the time of her preoperative evaluation for correction of severe esotropia (70 prism diopters), she was active, vigorous, and eating well. Medications included digoxin 30 μg po bid, diuril 60 mg po bid, and captopril 1 mg po bid. Her blood pressure was 120/70 mmHg, with a regular heart rate of 115–150 beats/min. She was not tachypneic and had minimal central and peripheral cyanosis at rest (peripheral oxygen saturation, Sp_{O_2} 84–85%). On auscultation, her second heart sound was single, with a grade 2–3/6 continuous shunt murmur over the anterior precordium. Her lungs were clear. The liver edge was palpable just below the costal margin. Her hematocrit was 47% and serum electrolytes were within normal limits. The preoperative chest x-ray showed stable borderline cardiomegaly with near-normal pulmonary vascularity, and her ECG demonstrated right ventricular hypertrophy unchanged since discharge. An echocardiogram performed 3 months preoperatively and just prior to instituting diuril and captopril showed normal ventricular function, trivial tricuspid valve insufficiency, mild pulmonic insufficiency, a widely patent interatrial communication, and well-functioning systemic to pulmonary artery shunt.

On the morning of surgery, she received her usual cardiac medications and atropine 0.13 mg im on call to the operating room. She was monitored with an automatic blood pressure monitor (Dinamapp®), a pulse oximeter (Nellcor®), ECG, precordial stethoscope, and esophageal temperature probe. Her vital signs were unchanged from the preoperative examination. A 22-G catheter was inserted in a peripheral vein and oxygen was administered by mask prior to induction. Gentamicin (12 mg) and ampicillin (325 mg) were given for endocarditis prophylaxis. An initial dose of 25 mg of thiopental (3.8 mg/kg) did not significantly change her systolic blood pressure or oxygen saturation. An additional 25 mg of thiopental in small divided doses was given during induction as required. She received 0.1 mg/kg of pancuronium and the trachea was intubated with a 3.5-mm ID uncuffed endotracheal tube. Anesthesia was maintained with air, oxygen, and halothane. $F_{I_{O_2}}$ was decreased to keep Sp_{O_2} between 80% and 85%; the inspired halothane concentration was titrated to maintain systolic

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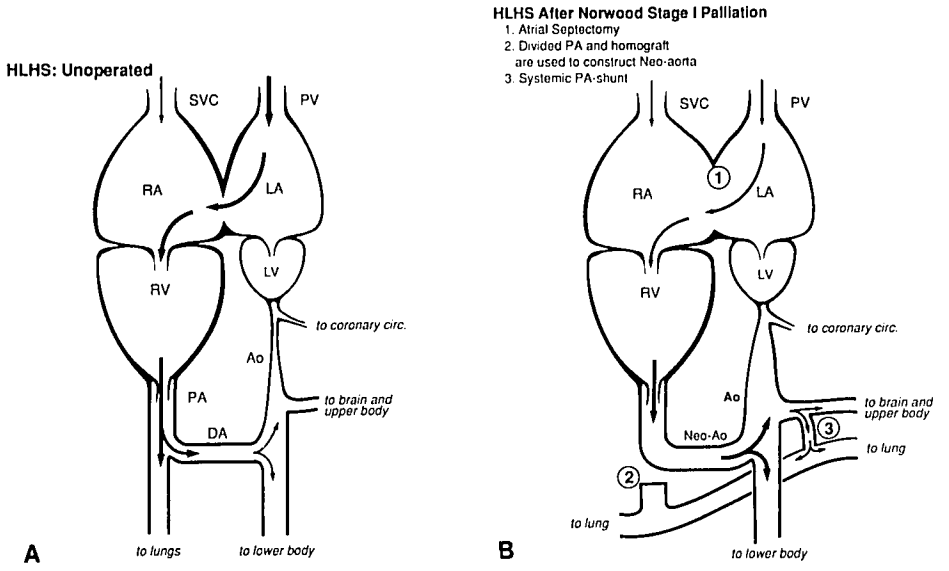

FIG. 1. Physiologic schematic of the heart and central circulation in HLHS. *A.* Unoperated: Branched arrows, their thickness approximately proportional to the volume of blood flow, indicate division of flow into parallel circulations in which perfusion depends on relative resistances. Pulmonary veins (PV), left atrium (LA), left ventricle (LV), superior vena cava (SVC), right atrium (RA), right ventricle (RV), pulmonary artery (PA), ductus arteriosus (DA), and aorta (Ao) are depicted schematically as labeled. *B.* After Norwood Stage 1 palliation: As in *A*, arrow thickness is approximately proportional to the usual blood flow; PA and DA have been replaced by neoaorta (Neo-Ao). The three components of the procedure are as follows: 1) an atrial septectomy, which assures unrestricted PV return, thereby protecting the pulmonary vasculature from high venous pressure, which would prevent its normal development; 2) division of the main PA with anastomosis of proximal PA to Ao (Addition of pulmonary homograft material augments the native tissue; the appropriately sized neoaortic arch provides the unobstructed ventricular outflow necessary for good ventricular function and systemic perfusion.); and 3) construction of a (modified Blalock-Taussig) systemic to PA shunt, which establishes the controlled pulmonary blood flow essential for adequate oxygenation and normal maturation of the pulmonary vasculature.[3]

blood pressure at 100 mmHg (0.6–1.5%). End-tidal carbon dioxide of 35–40 mmHg was maintained. During the 2-h procedure she received 15 ml/kg of iv fluids. At the end of the case, muscle relaxation was reversed with atropine (0.02 mg/kg) and neostigmine (0.05 mg/kg); there was no significant change in heart rate. The trachea was extubated without incident, and oxygen was administered *via* mask and monitored with a pulse oximeter during transport to the recovery room.

In the recovery room Sp_{O_2} measured while breathing oxygen *via* face mask and then room air was stable at 85%. Her systolic blood pressure was unchanged. The only apparent abnormality was intermittent episodes of sinus slowing with a junctional escape rhythm noted on the ECG. During these episodes her heart rate would decrease to 80–90 beats/min without a change in blood pressure or oxygen saturation. After 3 h of close observation she was transported to an intermediate care unit where she exhibited several more episodes of sinus slowing with junctional escape rhythm. By the following morning her heart rate was within its usual range. The ECG obtained just prior to discharge was unchanged from the preoperative ECG, and the serum digoxin level (0.5 ng/ml) was within the therapeutic range (0.5–2 ng/ml).

DISCUSSION

Based on currently available data on survival after palliation and repair of HLHS, this infant has the potential for at least a 10-yr life span. Eye muscle surgery would provide significant enhancement of the quality of her life. Correction of ocular alignment was necessary to allow her binocular vision. In addition, the parent–child relationship is known to suffer when there is a dichotomy between parents' expectations of a perfect infant and the reality of one with congenital anomalies. Bonding to a child with esotropia may be particularly difficult for parents, and normal ocular alignment might improve the parent–child relationship.[7,8] Thus, although the risks of perioperative morbidity and mortality were substantially greater in this infant than in healthy children, this elective procedure could be anticipated to have substantial physical and psychologic benefits.

HLHS encompasses a constellation of congenital cardiac lesions affecting left heart structures, ascending aorta, and aortic arch. The most common form includes atresia of the aortic valve and ascending aorta (fig. 1*A*). Mitral valve hypoplasia or atresia, varying degrees of hypoplasia of the left atrium and ventricle, juxtaductal coarctation of the aorta, or a coarctation shelf are frequently associated findings.[2,3,9] In patients with severe left ventricular outflow obstruction, pulmonary venous return crosses to the right heart *via* a patent foramen ovale or atrial septal defect, mixes with systemic venous blood, and is pumped by the right ventricle through the pulmonary valve. Blood reaches the systemic circulation *via* the ductus arteriosus. The cerebral and coronary vessels are perfused retrograde through the hypoplastic aortic segment, and the abdominal organs and lower extremities receive antegrade flow through the normal descending aorta. The lungs are supplied in parallel *via* the pulmonary artery. The amount of blood flow that the systemic circuit and pulmonary circuit receive depends on the ratio of vascular resistances in each circuit. When pulmonary vascular resistance decreases soon after birth, progressive pulmonary overcir-

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culation develops. Ductal closure would result in decreased systemic perfusion and death; infusion of prostaglandin E_1 prevents closure and allows medical resuscitation of the neonate prior to surgical intervention.[2–4]

The survival of infants with other forms of congenital heart disease can also depend on ductal patency and maintenance of parallel circulations. Like our patient with HLHS, infants with severe aortic coarctation and a ventricular septal defect (VSD) have both severe left heart obstruction and a significant left-to-right shunt through the VSD. These patients require right-to-left flow through the ductus arteriosus to provide adequate systemic perfusion. Obstruction to right heart outflow, such as tricuspid atresia, severe pulmonary valve stenosis, or pulmonary atresia, occurs more commonly. These neonates require left-to-right ductal flow to perfuse the pulmonary circulation; however, the pathophysiology and presentation of neonates with inadequate pulmonary blood flow are different from those who have pulmonary overcirculation.[6]

"Physiologic" repair of congenital heart disease characterized by persistent parallel circulations involves isolation of the systemic and pulmonary circulations in series. Correction of HLHS and other anomalies in which the patient has a single functional ventricle is based on principles described by Fontan in his surgical treatment of tricuspid atresia.[3,6] He showed that when pulmonary vascular resistance is at normal low levels, adequate blood flow can be achieved without a pulmonary ventricle; however, the high pulmonary vascular resistance present in the neonatal period precludes a Fontan operation and makes a staged surgical approach necessary. Initial palliation is accomplished in HLHS by performance of the Norwood Stage 1 procedure (fig. 1*B*). When Stage 1 palliation is complete as in the patient described herein, the overall physiology is similar to other forms of congenital heart disease in which pulmonary blood flow is shunt-dependent: the systemic and pulmonary circulations are still perfused in parallel, but ductal patency is no longer required for survival.

As with all children with congenital heart disease, preoperative evaluation of patients after Stage 1 palliation requires particular attention to the expected areas of physiologic compromise. Poor ventricular function is not uncommon in these patients. Maladaption of the morphologic right ventricle for contraction against systemic afterload, and volume overload of a single ventricle expected to supply both the systemic and pulmonary circuits are the primary mechanisms for decreased ventricular function. Some patients, like ours, do well with diuretics, digitalis, and/or afterload-reducing agents; others have severely depressed ventricular function despite maximal medical therapy. In infants poor ventricular function from any cause results in signs of excess catecholamine production and pulmonary and systemic venous congestion.[3,10] These include increased heart and respiratory rates, prolonged capillary refill, poor feeding and growth retardation, recurrent pneumonia, rales or wheezing, and hepatomegaly. The relative cardiac diameter and degree of pulmonary vascularity on chest radiograph would be increased. The degree of pulmonary vascularity also may indicate inadequate or excessive shunt flow. Specifically, in patients with HLHS circulation may be further compromised by tricuspid valve regurgitation,[1,11] restrictive interatrial communication,[4] or aortic coarctation,[9] all of which may be seen with echocardiography. The presence of unequal peripheral pulses strongly suggests the presence of aortic coarctation.

A pulmonary to systemic flow ratio of approximately 1.3:1, Sp_{O_2} between 80% and 85% due to the mixing of systemic and pulmonary venous return, and hematocrit of 45–50% are considered optimal in this group of patients.[12] Worsening cyanosis and increasing hematocrit indicate probable relative stenosis of the systemic to pulmonary artery shunt due to growth of the child. Angiograms have shown some growth of the aortic root and coronary arteries. There have been no reported cases of coronary insufficiency, but the persistently small diameter of the native proximal aortic root (fig. 2) makes this possibility one of long-term concern.[13]

In the absence of clinical signs of diminished systemic

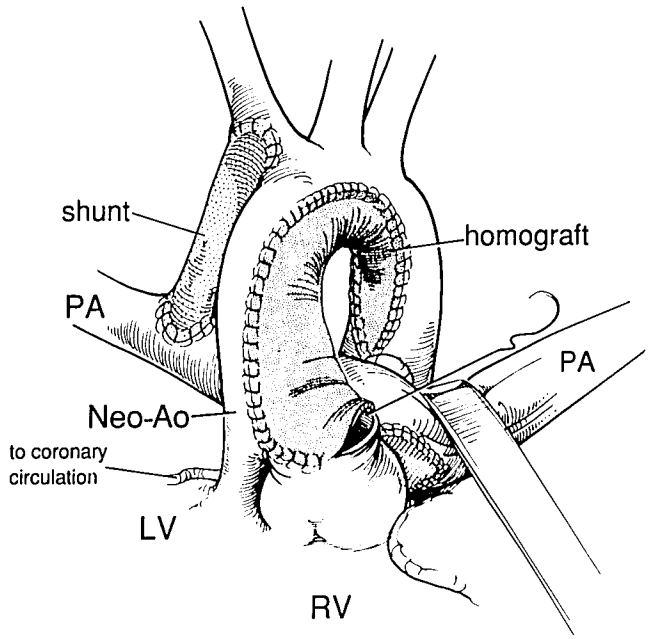

FIG. 2. Surgical drawing after completion of Norwood Stage 1 repair. Anterior view of the heart with hypoplastic aortic root, divided main PA, Neo-ao, and shunt between the brachiocephalic and right pulmonary arteries.

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or pulmonary circulation, a thorough history and physical examination supplemented by hematocrit, serum electrolyte determination, ECG, and chest x-ray constitute adequate preoperative evaluation for patients undergoing elective surgery. Echocardiographic evaluation should be tailored to the patient's clinical presentation. Doses of cardiac medications may require adjustment to ensure that moderate degrees of congestive heart failure are optimally controlled prior to elective surgery. Infants with clinical signs of severe congestive heart failure are poor candidates for elective surgery and require more extensive studies, such as cardiac catheterization.

Anesthetic management after Norwood Stage 1 palliation is tailored to the physiology of parallel circulations.[4–6] As in any patient with a nonrestrictive shunt and/or a common cardiac chamber, the ratio of resistances of the systemic and pulmonary circuits controls the amount of systemic and pulmonary blood flow.[6] If systemic to pulmonary artery shunt flow is reduced by elevations in pulmonary vascular resistance and/or decreases in systemic vascular resistance, hypoxemia will ensue. Conversely, injudicious use of pulmonary vasodilating agents, of which oxygen and hyperventilation are the most potent, results in excessive pulmonary blood flow with the possibility of decreased systemic perfusion, hypotension, and acidosis. A smooth course, maintaining normal systemic pressure (measured on the arm not involved with a Blalock-Taussig shunt), Sp_{O_2} between 80% and 85%, and normocapnia, is central to the safe performance of elective surgery. As with all patients with intracardiac communications or shunts, careful removal of air bubbles from iv fluids is necessary to prevent systemic air embolization.

The effects of specific anesthetic agents on vascular resistance and ventricular function depend on the doses administered and on the patient's preanesthetic vascular tone and cardiac reserve. Patients with HLHS are no exception in this regard. Patients with good ventricular function and no residual anatomic obstruction can be monitored noninvasively. Administration of commonly used anesthetics titrated to the patient's systemic blood pressure is appropriate.[4,6] This technique allows rapid recovery from anesthesia and avoids the potential complications associated with prolonged intubation. In contrast, infants with impaired ventricular function or inadequate shunt blood flow and those requiring more extensive intraabdominal or intrathoracic procedure with fluid shifts, ventilatory compromise, and temperature fluctuations may require more invasive monitoring and high-dose opioid anesthetics.[4–6] In that case, the potential benefits of the elective procedure must be balanced against the additional risks associated with invasive monitoring and prolonged intubation.

In the setting of parallel circulations, such as after palliation of HLHS, bradycardia decreases systemic cardiac output and flow through the systemic to pulmonary artery shunt. Accordingly, both systemic hypotension and hypoxemia may result. Patients with HLHS have not been reported to have abnormal increases in vagal tone and are thus not necessarily prone to bradycardia. Many of them, however, are maintained on digoxin for inotropic augmentation. Digoxin has well-known vagotonic effects that can lead to slowing of the sinus rate.[14] Extraocular muscle surgery might further enhance vagal tone and thus increase the degree of sinus slowing. The sinus bradycardia with a junctional escape rhythm exhibited by this patient is characteristic of the rhythms seen with elevations in vagal tone. This dysrhythmia in patients with normal cardiac anatomy is essentially benign; however, because of the possibility of diminished shunt flow, careful postoperative monitoring of HLHS patients is necessary.

In summary, we have presented a case of an infant following Norwood Stage 1 palliation of HLHS who underwent eye muscle surgery successfully with noninvasive monitoring, iv induction, and inhalation anesthesia. Preoperative assessment should include evaluation of ventricular function and systemic to pulmonary artery shunt flow. Like other stable patients with complex congenital heart disease, when infants with HLHS and congestive heart failure are medically well controlled after good surgical palliation and the effects of anesthetic techniques on the physiology of parallel circulations are well understood, elective surgery can be considered.

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Hemodynamic Effects of Epinephrine, Dopamine, Nitroglycerin, and Nitroprusside in a Patient with a Total Artificial Heart (TAH)

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Many drugs with vascular effects also have a direct cardiac effect. The cardiac responses to these drugs are naturally absent in patients with a total artificial heart (TAH). Vascular effects of cardiovascular drugs have been described in animals subjected to TAH implantation,[1–5] but to our knowledge investigations in patients have not been reported. The purpose of this study was to evaluate the circulatory effects of epinephrine, dopamine, nitroglycerin, and nitroprusside in a hemodynamically stable patient after a TAH implantation.

CASE REPORT

A 55-yr-old man with a 5-yr history of angina symptoms and four previous myocardial infarctions was admitted for coronary artery bypass surgery. Preoperative angiography demonstrated obstruction of four large coronary artery branches and depressed left ventricular function. Four bypass grafts were implanted. Two months later the patient had an additional acute myocardial infarction complicated by cardigenic shock, including hypotension and oliguria. Catheterization revealed a cardiac index of 1.3 $l \cdot min^{-1} \cdot m^{-2}$ and an arteriovenous oxygen difference (AVO_2 difference) of 90 ml/l. Repeated angiography showed occlusion of three grafts and poor left ventricular function with an ejection fraction of less than 10%. As the patient deteriorated rapidly, a Jarvik-7/70 ml cardiac prosthesis was implanted. The surgical technique and TAH design has been reported elsewhere.[6] The postoperative course was uncomplicated and the trachea was extubated on the second postoperative day. On the tenth postoperative day the patient underwent a biological orthotopic heart transplantation with an uncomplicated postoperative course.

METHODS

On the seventh postoperative day after the TAH implantation, having been hemodynamically stable for several days and without vasoactive drug therapy, the patient's response to cardiovacular drugs was evaluated. The study was approved by the Ethical Committee of the Karolinska Hospital and the patient's informed consent was obtained. The drugs were administered intravenously as a continuous infusion through a catheter positioned in the left internal jugular vein. The dosage for each drug was epinephrine (Adrenalin®, ACO): 25, 50, and 75 $ng \cdot kg^{-1} \cdot min^{-1}$; dopamine (Intropin®, American Critical Care): 2.5, 5, and 7.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$; nitroglycerin (5 mg/ml clinical solution from the pharmacy of the Karolinska Hospital): 0.5, 1, and 2 $\mu g \cdot kg^{-1} \cdot min^{-1}$; nitroprusside (Nipride®, Roche); 0.5, 1, and 2 $\mu g \cdot kg^{-1} \cdot min^{-1}$. The infusion was increased in a stepwise fashion for 15 min periods, at each infusion rate. Each drug was tested with at least a 1-h interval between different drugs. The hemodynamic condition of the patient prior to each drug tested served as a control. The TAH driver (Utah drive™ System II) was adjusted to the following drive settings: pump rate, 100 beats per min; systolic duration, 50%; left drive and right drive pressures, 170 mmHg and 35 mmHg, respectively. The following pressures, central venous pressure (CVP), mean pulmonary artery pressure (MPAP), left atrial pressure (LAP), and mean arterial pressure (MAP), were obtained by positioning catheters in the right internal jugular vein, pulmonary artery, left

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