Neonatal Cardiac Care, a Perspective
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Every year in the United States approximately 40,000 infants are born with congenital heart disease. Several of these infants require corrective or palliative surgery in the neonatal period. Mortality rates after cardiac surgery are highest amongst neonates, particularly those born prematurely. There are several reasons for the increased surgical mortality risk in neonates. This review outlines these risks, with particular emphasis on the relative immaturity of the organ systems in the term and preterm neonate.

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Introduction

Congenital heart disease is the most common birth defect.1 Recent prevalence estimates range from 6 to 10 per 1,000 live births.2-4 Nearly 40,000 infants are born with a congenital heart defect each year in the United States; worldwide, over 1 million such babies are born every year.1,5 Many of these infants require surgery to correct or palliate their heart defect during their lifetime; several require surgery in the newborn period.

Between 2007 and 2010, approximately 80,000 patients underwent cardiac surgery for congenital heart disease across 96 North American centers.6 Figure 1 depicts the age distribution of these patients. Although neonates comprised only 25% of the total surgical volume, they accounted for more than 50% of all deaths that occurred during this time period (Fig. 2). Tremendous strides in congenital heart surgery, advances in cardiopulmonary bypass techniques, and improved preoperative and postoperative management skills have resulted in a general decline in operative mortality across all age groups.7 However, mortality rates after neonatal cardiac surgery continue to be high.6,7

One in 10 neonates does not survive to discharge after cardiac surgery.6 Multiple factors contribute. Premature birth and low birth weight add substantial risk.8,9 Lesions requiring surgery in the neonatal period are often quite complex. Performance of intricate surgical procedures in tiny hearts requires superior technical skills and several years of experience for mastery. Neonates pose technically challenging issues related to structure, cannulation, and cardiopulmonary bypass. Abnormal preoperative circulation and effects of cardiopulmonary bypass on immature organ systems are additional factors that place neonates at greater risk for death after surgery.

Neonates born prematurely, ie, before 37 completed weeks of gestation, are at greater risk of death after cardiac surgery than those born after 37 weeks.8,10 This dichotomous distinction, although not untrue, makes an erroneous assumption that the risk of death after 37 weeks is uniformly equivalent. Population and single-center studies have disproven this theory in babies born with congenital heart disease and in those born without birth defects.11,12 There is an incremental decline in death rate from 37 to 40 weeks, with the nadir at 39 to 40 weeks.8,10 Death rates increase again if delivery is delayed beyond 41 weeks. Extension of pregnancy from 37-38 weeks to 39-40 weeks provides a significant survival benefit and reduces the risk of complications.8,10

The majority of babies with congenital heart disease are born before 39-40 weeks of gestation.8 Many babies are electively delivered before the due date, for better coordination of delivery, catheter intervention if necessary, and to avoid intrauterine demise. The recent spate of single-center and population studies have shown significant risk of mortality and morbidity among near-term babies and should caution against this practice.8,10-12 Elective delivery of babies before 39 completed weeks of gestation should be discouraged, absent any obstetrical or fetal risk. Local, state, and regional initiatives to eliminate non-medically indicated elective deliveries before 39 weeks are critical endeavors that may help reduce neonatal death rate, including those after cardiac surgery.13

Why should birth that occurs only 2 to 3 weeks before the due date confer such a disadvantage? The answer is un-
known. Human gestation is called “full term” when it lasts 280 days (or 40 completed weeks). Term gestation, delineated for statistical probability, ranges from 37 weeks to 42 weeks. Hence, 37 weeks is an entirely arbitrary beginning for term gestation and the period between the two limits represents a continuum where organ maturity continues. Therefore, babies born in the “early term” period are physiologically less mature than babies born in “late term.” The exact physiological immaturity that places early term neonates at greater risk of mortality is not known, but likely represents incomplete development of several organ systems. The authors extend the argument that organ maturity is not conferred, even at late term or “due date.” Maturation is a gradual process that continues for several months and years after birth. Neonates are disadvantaged in comparison to infants and older children because organ systems are comparatively less mature.

**Fetal Circulation**

Neonates are recent occupants of a fetal environment where demands are few and there is dependence on the utero-placental unit for survival. At birth, the fetus transitions from this sheltered milieu to a place of high metabolic rate and self-dependence for gas exchange.

Fetal lungs do not serve a respiratory function; they are solid, fluid filled, and collapsed. Placenta assumes the role of organ for gas exchange in the fetus. Oxygenated blood is conveyed from the placenta to the fetal heart and systemic blood is routed through the ductus arteriosus and descending aorta to the placenta.

Preferential streaming of oxygen-rich blood in the inferior vena cava and across the foramen ovale to the left side of the heart facilitates delivery of blood with relatively higher oxygen content to the fetal myocardium and brain. Because the resistance in the fetal pulmonary vasculature is high, less than 15% of right ventricular output is delivered to the lungs. The majority of right ventricular output flows through the ductus arteriosus into the descending aorta to perfuse the lower body and the low-resistance placental bed. The parallel fetal circulatory system promotes efficient oxygen redistribution in a relatively hypoxic environment (Fig. 3).

Oxygen delivery to tissues depends on regional blood flow and oxygen content in the blood. Hemoglobin concentration and oxygen saturation of hemoglobin are the major determinants of oxygen content. Despite the low fetal blood oxygen

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**Figure 1** Age distribution of patients who underwent cardiac surgery for a congenital heart defect between 2007 and 2010 in 96 centers in North America. Y axis represents the number of cardiac surgical patients. (Adapted from the Society of Thoracic Surgeons Congenital Heart Surgery Database, 14th Harvest, with permission.6).

**Figure 2** Proportional distribution of cardiac surgical mortality by age group between 2007 and 2010 in 96 centers in North America. (Adapted from the Society of Thoracic Surgeons Congenital Heart Surgery Database, 14th Harvest, with permission.6).

**Figure 3** Mammalian fetal circulation. Ao, aorta; DA, ductus arteriosus; DV, ductus venosus; LA, left atrium; LV, left ventricle; PA, pulmonary artery; RA, right atrium; RV, right ventricle. (Reprinted with permission from Rudolph AM: Congenital Diseases of the Heart. Clinical-Physiological Considerations. Ed 3. Wiley-Blackwell, 2009).
tension, the cellular oxygen demands of the fetus are met by well-designed oxygen transport and delivery mechanisms. Greater oxygen affinity of fetal hemoglobin permits efficient oxygen extraction from the placenta. High concentration of hemoglobin, the presence of fetal hemoglobin, and a high combined ventricular output maintain tissue oxygen delivery under unstressed conditions.

The fetal circulation is forgiving to even the most severe forms of congenital heart disease. Intra- and extra-cardiac shunts allow fetal circulatory adaptations to abnormal heart anatomy.

Transitional Circulation

Most of the circulatory changes occur in the first few moments after birth. Additional circulatory adjustments occur over a period of several weeks. The primary event triggering the alteration in blood flow patterns is the establishment of alveolar respiration. As a consequence, there is a substantial decline in pulmonary vascular resistance and a several-fold increase in pulmonary blood flow. A rise in left atrial pressure caused by an increase in pulmonary venous return allows closure of the foramen ovale and abolishes the atrial level shunt. The higher oxygen tension in the blood initiates the postnatal closure of the ductus arteriosus, establishes complete separation of pulmonary and systemic blood flows, and leads to a circulation in series (Fig. 4).

Perinatal Cardiovascular System

Postnatal Increase in Left Ventricular Output

The “inappropriately” low for size fetal metabolic rate is an adaptation to low oxygen tension. Heat regulation is not needed and significant fetal physical or respiratory activity is absent. The metabolic rate in late-term fetus approximates the low level of its adult mother. At birth, metabolic rate/ oxygen consumption increases several-fold because of the additional demands imposed by heat conservation mechanisms and respiratory activity. Oxygen delivery increases in a similar proportion to maintain normal oxygen reserve capacity. Much of the increase in oxygen delivery is attributed to a substantial increase in left ventricular output after birth. Enhanced left ventricular output is caused by an increase in heart rate, an increase in left ventricular preload, and a greater inotropic state. The exact mechanisms causing postnatal increase in cardiac output are not known, but thyroid hormone is believed to play a role. Fetal lambs in which the thyroid gland was removed 2 weeks before delivery demonstrated low plasma levels of T3 and failed to demonstrate the expected postnatal increase in T3 levels and cardiac output. The same lambs had fewer β-adrenergic receptors on myocardial surface and exhibited a blunted response to β-adrenergic stimulation. Elevation in cortisol levels, catecholamine surge, and relief from ventricular constraintment at delivery also contribute to postnatal elevation in cardiac output.

The transition to extra-uterine life depletes much of the neonate’s circulatory reserves and exposes it’s vulnerability to additional challenges. In the weeks that follow, the newborn replenishes some of these reserves as evidenced by a decline in resting heart rate, a reduction in basal inotropic state, and improved response to exogenous catecholamines. As postnatal age advances, the cardiac output declines in relation to body weight (Fig. 5). This decrease parallels a decline in oxygen consumption in relation to body weight, an improved efficiency in oxygen unloading at the tissue level, and a transition to adult hemoglobin.
Developmental Differences in Myocardial Structure and Excitation-Contraction Coupling

Generation of myocardial contractile force increases with maturation. Developmental differences in contractility are, in large part, caused by age-related differences in myocardial structure (Fig. 6).

Immature myocytes possess fewer myofilaments, the fundamental units of cross-bridge formation. Increase in the number of myofilaments with age correlates with increase in myocardial force generation. Isoform switching of myofibrillar proteins with development contributes to improved contractile efficiency with age. The immature myocyte is smaller and reveals intracellular spatial disorganization. The myofibrils assume a random arrangement rather than the parallel arrangement seen in adult myocytes. A large proportion of the immature myocyte is inhabited by non-contractile organelles that do not contribute to force generation. Biophysical disadvantage to shortening is also imposed by the small, spherical structure of the immature myocyte and the central location of noncontractile elements.

The calcium handling mechanism in the neonate is underdeveloped and inefficient. T-tubules and sarcoplasmic reticulum are scarce, intracellular cardiac regulatory proteins exhibit functional immaturity. Therefore, cytosolic calcium concentration is primarily dependent on trans-sarcolemmal flux of calcium.

A densely arborized plexus of sympathetic nerves innervates the adult myocardium. Sympathetic innervation of the heart is incomplete at birth and continues postnatally. Cardiac norepinephrine stores, a reflection of sympathetic innervation, is the lowest in late-term fetuses and increases postnatally to approach adult levels by 4 weeks of age.

Maturation of adrenergic receptors predates myocardial sympathetic innervation. β-adrenergic receptors on myocardial cell surface increase in number during fetal development, with no significant quantitative difference between newborns and adults. However, functional uncoupling of β receptor-G protein-adenylate cyclase complex in the newborn limits the effectiveness of catecholamine-modulated contractility in this age group. Maturational changes in myocyte ultra structure, calcium handling, and sympathetic innervation contribute to improved myocardial performance with age.

Neonatal Ventricular Performance

Ventricular performance improves with age. Circulatory adaptation at birth is vital to meet the increased metabolic demands of extra-uterine life. An acute increase in heart rate, pulmonary venous return, and contractile state contributes to the postnatal enhancement in cardiac output. High resting inotropism limits contractile reserve in newborns. Immature hearts exhibit a blunted response to exogenous catecholamines compared with mature hearts. Improvement in contractile performance with maturation parallels several developmental changes in the myocardium, as described previously. Limitation in contractile reserve in neonates favors utilization of rate-dependent mechanisms to improve cardiac output.

Increase in preload augments stroke volume; the Frank-Starling relationship. Immature hearts demonstrate a Frank-Starling relationship, albeit a modest one (Fig. 7). The mature heart shows an extension of the Frank-Starling slope beyond that seen in immature hearts, ie, a more robust increase in stroke volume after volume loading. Therefore, the immature heart has a lesser recruitable preload reserve. Limited response to volume loading may in part be caused by...
decreased compliance of immature hearts. Maturational changes in cytoskeleton and extracellular matrix improve myocardial compliance with age.34,36,60-62

Decreased ventricular compliance exposes the deleterious effects of ventricular interdependence. Volume or pressure loading of one ventricle can impact filling of the contralateral ventricle to a greater extent in immature hearts than in more mature ones.63 This restrictive effect is particularly evident in neonates who have endured an unfavorable postnatal transition and exhibit persistent fetal circulation. The pressure-loaded right ventricle alters septal dynamics and limits left ventricular filling and left ventricular stroke volume.

Increase in afterload profoundly diminishes ventricular performance in the fetus and neonate (Fig. 8).17,36 When exposed to similar afterloads, the immature myocyte shortens to a lesser extent and more slowly than a mature myocyte.36 Developmental changes in myocyte architecture permits the adult heart to counteract afterload stressors more effectively.34,36

**Congenital Heart Disease and Postnatal Circulation**

Most babies with structural heart disease experience an unremarkable transition to ex-utero conditions. However, abnormal circulatory patterns in some forms of congenital heart disease may impose immediate hemodynamic challenges at birth, ie, babies with hypoplastic left heart syndrome with a restrictive atrial communication, d-transposed great vessels with intact ventricular septum, and restrictive foramen ovale. In others with circulations dependent on ductal patency, the pressure-loaded right ventricle alters septal dynamics and limits left ventricular filling and left ventricular stroke volume.

Inherent limitations in cardiac mechanics of the newborn heart are exposed in some forms of congenital heart disease. Neonates with severe aortic stenosis are limited in their ability to increase myocardial performance in the face of an increased afterload. Lesions with left-to-right shunts require an increase in left ventricular output to maintain adequate systemic flow. Recruiting the Frank-Starling mechanism and increasing inotropic state accomplish much of the increased stroke work. Large shunts may overwhelm the limited preload and contractile reserve of the newborn heart. Babies with hypoplastic left heart syndrome are particularly vulnerable. In these neonates, excessive pulmonary blood flow through a patent ductus arteriosus can limit systemic flow. Right ventricular output must increase several-fold to maintain systemic flow. Right ventricular functional reserves may be insufficient to accomplish the stroke work needed to maintain systemic flow.

### Other Immature Organ Systems

Other organ systems may also be incompletely developed, even in the infant born at term. An extensive description is beyond the scope of this review. A brief summation of critical systems or issues is described.

**Respiratory**

Chest wall structure and limited diaphragmatic apposition introduces mechanical inefficiencies in neonatal ventilation.64 Neonatal lungs and chest wall possess variable compliances.64,65 The lungs are less compliant, while the chest wall is extremely compliant. This uncoupling predisposes the chest wall to deformational forces and much of the respiratory energy is expended in counteracting these forces.65 The neonate compensates with a higher resting respiratory rate than is seen in older children and adults. Diminished respiratory reserves in the neonate are exposed with parenchymal lung disease, fluid, or air accumulation in pleural space. Chest wall structure and mechanics improve with age, making the older child or adult more equipped to face challenges requiring an increase in respiratory work.65

**Renal**

Nephrogenesis is completed at 35 weeks of gestation.66 Structural and functional growth of the kidney continues for several months after birth. The biggest limitation in renal function in the neonate is the rate of glomerular filtration, which, in the first few days of life, is one third that seen in adults.67 Tubular and medullary renal function limit the maximal urine concentrating ability of the newborn infant to half that of an adult.68 These functional limitations make the neonate more vulnerable to fluid overload or depletion.

**Temperature Regulation**

Newborn infants, particularly those born prematurely are susceptible to hypothermia.69,70 A large surface area in relation to body weight permits greater heat loss in neonates than in older children. Neonates are limited in their ability to conserve heat in the presence of cold stressors.69,70 Shivering thermogenesis is limited in the first few weeks to months of life.69,70 Non-shivering mechanisms, ie, brown fat metabolism, is recruited for heat production in neonates, but this increases oxygen consumption.69,70 Therefore, neonates benefit from care in a thermoneutral environment, which is the temperature at which normal core temperature is maintained with minimal energy expenditure.

**Immune System**

Neonates are susceptible to infections. Skin and mucosa serve as ineffective barriers. Immature cellular and humoral systems limit their ability to mount an effective immune response.71 Neonates, particularly premature infants with long-standing indwelling venous catheters, are particularly at risk.
Effects of Cardiopulmonary Bypass on Neonates

The damaging effects of cardiopulmonary bypass including hemodilution, systemic inflammation and bleeding are more pronounced in neonates than in older children and adults.72 The total blood volume in term neonates is approximately 80 mL/kg.73 The priming volume of the extracorporeal circuit may be as high as two or three times the circulating blood volume of the neonate. This disparity between the circulating blood volume and bypass circuit size results in marked hemodilution causing anemia, hypoproteinemia, and a reduction in coagulation factors. Significant hypoproteinemia can lead to greater transflux of fluid into the extracellular space from the intra vascular compartment.

Surgical trauma and extracorporeal circulation triggers an exuberant systemic inflammatory response.74,75 Neutrophil, contact and complement activation, cytokine release, platelet aggregation, and activation of coagulation cascade are components of systemic inflammation. Systemic inflammatory mediators can cause cellular and organ dysfunction. Release of C3a increases vascular permeability, TNF-α and IL-1β depress myocardial contractile function, TNF-α increases vascular permeability and lung water content and decreases glomerular filtration.75 The adverse effects of global inflammation are more pronounced on the immature organ systems of neonates.

Several strategies have been used to counteract the damaging effects of cardiopulmonary bypass.72 Reducing the extracorporeal circuitry will decrease the artificial surface area of exposure. Deploying tubes of smaller length and diameter, decreasing distance of circuit from surgical table, and eliminating arterial filters and other components can miniaturize circuits. Such small circuits have the additional advantage of requiring a smaller priming volume. Other strategies include maintenance of a higher oncotic pressure in the bypass circuit, use of anti-inflammatory agents like corticosteroids, and removal of inflammatory mediators by continuous or modified ultrafiltration.72

Implications for Postoperative Care

The immature organ systems and the deleterious effects of cardiopulmonary bypass have been described in previous paragraphs. Cardiac surgery exposes the limited reserves of the neonate.

Hemodynamic Optimization

Myocardial edema and ischemia–reperfusion injury after cardiac surgery decreases ventricular performance. The decline in contractility is usually transient, unless there is a significant residual surgical defect. Despite mounting a robust sympatho-adrenal response during surgery, the immature neonatal heart is limited in its ability to augment ventricular performance.76,77 Therefore, exogenous infusions of inotropes are required to enhance the contractile state after separation from cardiopulmonary bypass. The inotropic response with exogenous agents is lesser in neonates than in older children because of the high baseline adrenergic state.30

There are no clear advantages of one inotrope combination over another, and institutional choices determine practice. Factors that may influence response to inotropic agents in the neonate are discussed.

Dopamine exerts its cardiovascular, renal, and hormonal effects in a dose-dependent manner.78-81 Renal effects predominate at low doses in older children and adults. Moderate doses (5 to 10 mcg/kg/min) stimulate cardiac adrenergic receptors and increase cardiac output, while doses >10 mcg/kg/min stimulate vascular α-adrenergic receptors and increase systemic vascular resistance. Maturational differences in the expression and sensitivity of α- and β-adrenergic receptors in the neonate make the response to dopamine less predictable, particularly in the preterm neonate.78,81,82 Therefore, α-adrenergic stimulation and increase in systemic vascular resistance may become apparent at low doses.78,81,82 Metabolism and elimination of dopamine is a complex process and wide inter-individual variations in clearance are noted.78,81-83 Reduced dopamine clearances in premature neonates and patients with hepatic and/or renal failure may make the cardiovascular response to conventional dosing more difficult to predict.82 Finally, dopamine exerts its effects on the heart in part by releasing norepinephrine from nerve terminals. Hence, it may be less effective when myocardial norepinephrine stores are depleted or low, as seen in immature hearts.45

Effects of epinephrine on β- and α-adrenergic receptors make it a good choice for post cardiac surgery patients with diminished cardiac function and vascular tone. However, there is limited data on the cardiovascular effects of epinephrine in neonates. Metabolic effects of epinephrine, like hyperlactatemia and hyperglycemia, warrant cautious use.84 Neonates are more susceptible to myocardial damage and necrosis after prolonged high-dose infusions of epinephrine.85 Milrinone has several positive modulating effects on ventricular performance.81,86,87 Decreased milrinone clearance in the setting of renal insufficiency may cause systemic hypotension. Neonates are particularly vulnerable because they have a low baseline glomerular filtration rate.

It is not uncommon for neonates to develop catecholamine-resistant hypotension after cardiac surgery.88 Downregulation of adrenergic receptors and related insufficiency or resistance to corticosteroid action may contribute to catecholamine resistance.89 In these cases, hydrocortisone administration induces a dramatic improvement in hemodynamics through its genomic and non-genomic effects.90-92 Non-adrenergic agents like arginine–vasopressin may also be used for vasodilatory shock when ventricular function is preserved.93,94

Finally, exploiting the Frank-Starling relationship to improve stroke volume may be less effective in neonates because of decreased myocardial compliance and mismatch of afterload to contractile state.59

Adrenergic agents primarily exert their effects by increasing intracellular calcium levels.35 In neonates, intracellular calcium levels are maintained through a trans-sarcloemmal flux of calcium. Hence, maintaining normal serum levels of ionized calcium is critical to optimize contractile function. Trans-capillary fluid shifts, bleeding and osmotic diuresis can
deplete circulating volume and decrease venous return. Dynamic and static assessments of volume status and fluid responsiveness are often used in older children and adults. There are no perfect measures to assess intravascular volume status in neonates. Single-point measurements of right atrial pressure are uninformative of volume status. Despite this, most intensivists use right atrial pressure or central venous pressure to guide fluid therapy.

Hypovolemia and decreased ventricular end-diastolic volume is inferred from declining trends in right atrial pressure measures. In these cases, correction of hypovolemia will increase venous return and stroke volume.

In the absence of bleeding, choice of replacement fluid for correction of hypovolemia is not clear. There is no convincing data that 4% to 5% albumin secures sustained improvement in hemodynamics compared with 0.9% saline.

**Fluid Management**

Total body water is increased and there is expansion of the extra-cellular space after cardiopulmonary bypass. There fore, maintenance fluid and sodium requirements are lower than usual.

The postoperative course is more often than not accompanied by trans-capillary fluid shifts. Fluid shifts can be quite pronounced in neonates if there is a concurrent leakage of albumin into the interstitial space. Attempts to maintain intravascular volume by increasing circulating albumin levels in such a “leaky” circulation may precipitate pulmonary edema, particularly in premature infants with lung disease.

Administration of systemic corticosteroids in the operating room and/or in the postoperative period may mitigate “capillary leak” but have their risks. Leakage of fluid into the interstitial space of tissues and organs can affect function. For example, accumulation of fluid in the lung parenchyma, pleural space, or chest wall leads to diffusion abnormalities, atelectasis, and worsening chest wall compliance.

Spontaneous fluid mobilization from the extracellular space is accomplished by encouraging lymph flow. Lymph flow increases with muscle contraction and spontaneous breathing. It is our institution’s practice to allow early resumption of spontaneous respiratory and physical activity and avoidance of muscle relaxation when possible.

**Respiratory Care**

Neonatal ventilator modes (ie, pressure-limited, time-cycled) are used at our institution in the postoperative period. Transition to nasal continuous positive airway pressure (CPAP) maintains functional residual capacity, decreases the work of breathing, and has the advantage of permitting earlier extubation. In this institution, extubation to “bubble” CPAP is favored compared with constant pressure ventilator-derived CPAP. Bubble-CPAP enhances gas exchange, lung mechanics, gas mixing efficiency, and lung volume compared with constant-pressure CPAP.

**Prematurity and Congenital Heart Disease**

One in eight babies in the United States are born before 37 completed weeks of gestation and are considered premature. Mortality risk increases with declining gestational age. Prematurely born infants who survive are at risk for motor, cognitive, visual, and auditory disabilities.

Congenital heart disease is more common in premature infants than those born at full gestational term. Interestingly, approximately one in five neonates with congenital heart disease born prematurely. Gestational age is an important contributor to mortality. Crude hospital mortality rates for prematurely born infants with congenital heart disease range from 16.4% in late preterm infants (34 to 36 weeks) to twice that in infants born before 34 weeks. In very low birth weight infants (<1500 g), mortality rates are much higher.

Description of the developmental immaturities of the preterm newborn is beyond the scope of this review. A brief summary is provided below.

Premature lungs are immature in structure and function. Premature infants may be deficient in surfactant
Neonatal Cardiac Care, A Collaborative Model

The benefits of specialized units were realized in the 1960s, leading to the establishment of dedicated coronary care units. Quick proliferation of adult cardiac intensive/corony care units followed. A similar pattern is observed in critical care of children. Cardiac intensive care units dedicated to the care of children with heart disease have rapidly flourished. A recent analysis of outcomes based on care models in congenital heart surgery has not demonstrated superior outcomes in dedicated pediatric cardiac intensive care units. This result lends support to the thesis that although physical structural elements of a dedicated unit are important, human resources and processes involved in delivering care are more critical.

We believe that term and preterm neonates with congenital heart disease benefit from the expertise of personnel trained or experienced in the care of newborns. This should include pediatric intensivists, cardiologists, neonatologists and neonatal nurses, and other medical caregivers trained in the care of the newborn infant. A collaborative model with all caregivers exhibiting expertise in neonatal cardiac care would be ideal and would meet the specific needs of the newborn infant with congenital heart disease.

In summary, neonates are not just “little children.” They differ from older children not just in weight or height, but in the physiology of their maturing organ systems. Understanding their limitations is the first step in differentiating the care delivered to them.

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