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Nutrition Support After Neonatal Cardiac Surgery

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Congenital heart disease is the most common birth defect in the United States, with an estimated frequency of approximately 12–14 of 1000 live births per year. Neonates with congenital heart disease often need palliative or corrective surgery requiring cardiopulmonary bypass during the first weeks of life. The neonate undergoing cardiopulmonary bypass surgery experiences a more profound metabolic response to stress than that seen in older children and adults undergoing surgery. However, compared with older children and adults, the neonate has less metabolic reserves and is extremely vulnerable to the negative metabolic impact induced by stress, which can lead to suboptimal wound healing and growth failure. There are complications associated with the metabolic derangements of neonatal surgery requiring cardiopulmonary bypass, including but not limited to acute renal failure, chylothorax, and neurological dysfunction. This article discusses the importance of nutrition and metabolic support for the neonate undergoing cardiopulmonary bypass and the immediate postoperative nutrition needs of such a patient. Also, this article uses a case study to examine the feeding methodology used at one particular institution after neonatal cardiac surgery. The purpose of the case study is to provide an illustration of the many factors and obstacles that clinicians often face in the provision and timing of nutrition support. (*Nutr Clin Pract*. 2009;24:242-249)

Keywords: infant, newborn; cardiac surgical procedures; nutritional support; enteral nutrition; parenteral nutrition

The neonate undergoing surgery requiring cardiopulmonary bypass (CPB) experiences a more profound metabolic response to stress than that seen in older children and adults undergoing surgery.[1-3] However, compared with older children and adults, neonates have less metabolic reserves, leaving them extremely vulnerable to the negative metabolic impact induced by stress.[1] This can potentially lead to suboptimal wound healing and growth failure. In this article, the importance of nutrition and metabolic support for the neonate subjected to cardiac surgery requiring CPB and the immediate postoperative nutrition needs are discussed. Additional complications are associated with the metabolic derangements of neonatal surgery requiring CPB. Three of these complications—acute renal failure (ARF), chylothorax, and neurological dysfunction—are reviewed.[4-7] A case study reviewing a feeding methodology used at one institution after neonatal cardiac surgery is provided. The case study delineates obstacles and complications that affect the provision of nutrition support after cardiac surgery in the neonate.

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Congenital Heart Disease

Congenital heart disease (CHD) can be defined as an abnormality in the structure and function of the heart as a consequence of abnormal intrauterine development. CHD is the most common developmental anomaly in the United States. It is estimated that about 40,000 newborns per year are born with CHD, or approximately 12–14 of 1,000 live births per year.[8] There are more deaths due to CHD than all other congenital defects combined.[9] Many infants with CHD require surgery before the age of 1 year.[10] Surgery can be corrective or palliative, depending on the specific cardiac lesion.[11,12] CHD can be classified according to its clinical presentation of acyanotic or cyanotic, with the absence or presence of large intracardiac shunts.[13] Acyanotic lesions include ventricular septal defect, patent ductus arteriosus, and atrioventricular septal defect and are usually repaired within the first year of life.[13] Cyanotic lesions are evidenced by a bluish discoloration of the mucous membranes, skin, and nail beds (see Table 1 for classifications and examples). Cyanotic lesions can be further classified as ductal-dependent pulmonary blood flow, ductal-dependent systemic blood flow, and ductal-independent mixing lesions.[13] Some of these lesions may require surgical palliation or repair in the neonatal period. These surgical procedures often require the use of CPB, which places the neonate at risk for

Table 1. Classification and Examples of Congenital Heart Disease

Acyanotic Lesions		
Ventricular septal defect		
Patent ductus arteriosus		
Atrial septal defect		
Atrioventicular septal defect		
Cyanotic Lesions		
Ductal-Dependent Pulmonary Blood Flow	**Ductal-Dependent Systemic Flow**	**Ductal-Independent Mixing Lesions**
Tetralogy of Fallot	Hypoplastic left heart syndrome	Total anomalous pulmonary venous return
Critical pulmonary stenosis	Interrupted aortic arch	Truncus arteriosus
Pulmonary atresia with intact ventricular septum	Coartctation of the aorta	Transposition of the great arteries
Tricuspid atresia	Critical aortic stenosis	

Classification of congenital heart disease based on clinical presentation of acyanotic or cyanotic lesions. Cyanotic lesions can be further subdivided into ductual dependent or independent categories.

postoperative morbidity, including extracardiac organ dysfunction and possibly organ failure.[3,14] In recent years, there has been a reduction in mortality due to advancement in surgical techniques and an improved understanding of the pathophysiologic changes that occur in the postoperative period; however, complications remain a significant cause of prolonged length of stay in the pediatric intensive care unit (PICU).[14] The postoperative care of neonates after cardiac surgery is multifaceted and complex. Infancy is associated with a more rapid increase in body dimensions than at any other time in life. Nutrition forms an important aspect of care.[15] Many factors affect the timing and provision of nutrition after cardiac surgery, and these are discussed throughout this article.

Overview of the Metabolic Response to Stress

The metabolic response to stress is mediated by the body's release of cytokines or inflammatory mediators.[2,3,14] After the body has depleted its short-term energy stores of glucose through glycogenolysis, the mobilization of amino acids from skeletal muscle (gluconeogenesis) is used for energy (glucose) to support vital organ functions, tissue repair, and wound healing.[1] This process also enhances immune function through decreased synthesis of hepatic transport proteins and increased production of acute-phase reactants such as C-reactive protein, ceruloplasmin, fibrinogen, and haptoglobin.[1] This decline in the synthesis of transport proteins reflects the severity of illness rather than the patient's nutrition status.[16]

Metabolic Consequences After Cardiac Surgery in the Neonate

In the immediate postoperative period, most neonates experience a transient increase in resting energy expenditure (REE) that typically lasts 12–24 hours before returning to near REE for age.[2] This period of increased REE is accompanied by an increase in respiratory rate and heart rate.[1,2,17] However, the increase is less in newborns undergoing surgery during the first 24 hours of life than in newborns undergoing surgery more than 48 hours after birth.[2,3] Skeletal muscle breakdown to provide energy (glucose) plays a crucial role in the ability of neonates to survive a prolonged catabolic response to stress. Compared with older children or adults, infants have less body stores of protein and fat and a 25% higher rate of protein breakdown.[1] Because of limited metabolic reserves, the neonate is at greater risk for malnutrition and the detrimental consequences of a prolonged metabolic response to stress or injury than is an older child or adult.[2,3] In addition, neonates who have undergone CPB are at an increased risk for developing an exaggerated inflammatory response, which is often manifested by generalized edema, capillary leak syndrome, and multisystem organ failure.[7] After CPB, modified ultrafiltration (MUF), a specialized form of continuous renal replacement therapy (CRRT), may be used as a method to remove proinflammatory cytokines and attenuate the inflammatory response.[3,18-20] Also, corticosteroids are often administered to reduce the inflammatory response after CPB.[3,7] If a prolonged inflammatory response to stress occurs without nutrition intervention, malnutrition develops, causing a loss of lean body mass and deterioration of organ function. After one-third of lean body mass has been lost, respiratory compromise and

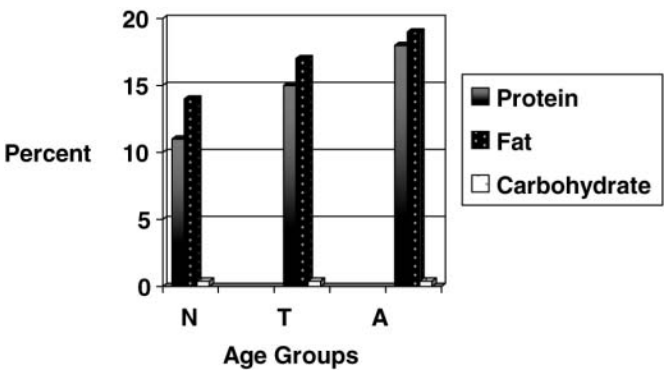

Figure 1. Body composition comparison as a percentage of total weight. A, adult; N, neonate; T, 10 years old. Adapted from Agus SD, Jaksic T. The body composition of neonates, children, and nonobese adults as a percentage of total body weight. *Curr Opin Pediatr*. 2002;14:470-481 with permission from Lippincott Williams & Wilkins.

cardiac arrhythmia may occur.[1,2] Comparing their body compositions as a percentage of total body weight for protein, fat, and carbohydrates, it is evident that neonates have markedly lower reserves of protein and fat than older children and adults (see Figure 1).

Pain is another influential factor in the metabolic response to stress and injury. Sufficient control of pain through analgesia and anesthesia is paramount in reducing the depth and duration of the catabolic response associated with injury and surgery in children.[1,21] Insufficient control of pain can prolong or exacerbate the metabolic response following surgery and can have detrimental consequences in the neonate recovering from cardiac surgery.[1,21]

Barriers to Nutrition Support After Cardiac Surgery in the Neonate

The provision of early nutrition support is crucial to the preservation of lean body mass. The initial goals of nutrition support are to minimize the loss of lean body mass and to support vital organ function. Nutrition support can minimize the loss of lean body mass but cannot completely prevent catabolism.[1,22] There are many barriers to optimizing nutrition support after cardiac surgery in the neonate. These barriers include hemodynamic instability, hypotension, hyperglycemia, and fluid limitations due to medication infusions, flushes, and the use of intravenous fluids. Other barriers include mechanical ventilation, electrolyte derangements, and impaired renal function. Electrolyte derangements and ARF often occur in patients who have been placed on CPB during surgery. After surgery, increased secretion of cytokines is accompanied by activation of the complement cascade.[2] This leads to an inflammatory response, which is associated with the development of myocardial injury, capillary leak syndrome, fluid retention, generalized edema, and potentially multisystem organ failure.[7] Aggressive diuresis is required, particularly if the postoperative management includes delayed sternal closure. Sternal closure is sometimes delayed after CPB to allow hemodynamic and pulmonary stability in patients whose cardiopulmonary interactions may have a significant impact on immediate postoperative recovery.[23,24] After cardiac surgery, total fluid intake is typically limited to avoid fluid retention and its associated morbidities.[7] Fluid administration is often regulated so that total fluid intake does not exceed a specific volume, if feasible, in a 24-hour period. Medication infusions, flushes, and other carrier fluids are included in this volume. The volume remaining after the inclusion of medication infusions, flushes, and carrier fluids is available for nutrition. This remaining allowable fluid volume is often severely restricted; thus, intravenous fluid (IVF) is most often used. If only a very small volume is available, ~15–17 kcal/kg/d can easily be given via IVF with a more concentrated dextrose solution at a 10th of the cost of parenteral nutrition (PN). Generally, the amount of fluid available for nutrition increases as continuous medication infusions are weaned and after sternal closure, if delayed sternal closure is an employed strategy. When CPB is used during cardiac surgery, postoperative fluid goals are often restricted to 50%–80% of maintenance fluid needs.[25]

Neonatal Nutrition Support After Cardiac Surgery in the Neonate

Initiation of Nutrition Support

Many factors influence the choice of which method of nutrition support to initiate after cardiac surgery. The medical team's clinical assessment of the neonate's cardiac output is crucial in deciding whether to initiate PN or enteral nutrition (EN). After cardiac surgery, decreased cardiac output raises the concern of hypoperfusion to the organs of the body. This concern increases in hemodynamically unstable patients requiring inotropic support. When low cardiac output is present, blood is shunted away from the splanchnic bed to the heart and brain, thereby making the splanchnic bed at risk for ischemia.[26] In patients with single ventricle physiology, who have undergone Norwood stage 1 palliation, the balance of circulation becomes critical because the single ventricle is pumping to both the systemic and pulmonary circulation. In patients with pulmonary overcirculation, poor systemic output may occur, thus further compromising

blood flow to the splanchnic bed. The Norwood procedure is the initial surgical palliation for hypoplastic left heart syndrome (HLHS) and includes complete relief of systemic outflow obstruction with arch reconstruction, creation of an unrestrictive atrial septal defect to prevent pulmonary venous hypertension, and formation of a systemic to pulmonary artery shunt for pulmonary blood flow. Following the Norwood operation, infants are left with inefficient parallel circulation and dependence on a functionally inferior systemic right ventricle.[27,28]

Another important factor is the actual amount of fluid available for nutrition. In severely fluid-restricted situations, PN offers the advantage of delivering a more concentrated form of nutrition compared with other methods. EN requires larger volumes of fluid to provide comparable nutrition but offers advantages that PN does not. These include promotion of gut integrity and gut motility as well as enhancement of immune function.[29] EN is sometimes given in the form of low-rate "trophic" feedings at the same time that PN is being administered to the patient.[26,29] Independent of fluid restrictions, the optimum time frame to initiate nutrition support is based on several factors, including the baseline nutrition status of the infant, disease acuity, and age.[30]

Neonatal Energy Expenditure After Cardiac Surgery

Neonates have a metabolic response to stress and injury similar to older children and adults but much more profound with the use of CPB.[3,7] The practice of using predictive equations that were developed for healthy, active participants to assess the energy requirements in hospitalized patients can lead to the overfeeding of critically ill patients.[17,22] Overfeeding is associated with increased carbon dioxide production, difficulties in weaning from ventilatory support, impaired immune function, and impaired organ function.[17,22] Underfeeding is associated with impaired immune function, poor wound healing, and difficulty in weaning from ventilatory support.[31] Although growth is crucial for neonates, the need to grow is not a critical priority during the immediate postoperative period. Growth cannot occur until the convalescent period of the stress response has begun.[1] A positive nitrogen balance is crucial for growth.[1,2] Critically ill neonates have considerably lower energy needs compared with healthy neonates due to absence of growth, decreased activity, and reduction in insensible losses during stress states.[32] One clinically useful method of determining the end of the stress response is by monitoring trends in serial serum C-reactive protein (CRP) levels. A CRP level <2.0 mg/dL is associated with an anabolic state.[32] Overfeeding and the potential consequences can be avoided by providing the measured resting energy expenditure (MEE)/REE or basal energy requirements until the serum CRP level is <2.0 mg/dL.[32]

One group of investigators used indirect calorimetry (IC) to measure REE before and after cardiac surgery in neonates and children with cyanotic and acyanotic CHD.[33] REE measurements were comparable between the 2 groups. The study measured REE before surgery at 57 ± 13 kcal/kg/d in the cyanotic group and 58 ± 9 kcal/kg/d in the acyanotic group. Five days after surgery, measured REE was again obtained. The cyanotic group's mean REE was 59 ± 10 kcal/kg/d, and the acyanotic group's mean REE was 62 ± 10 kcal/kg/d. The measured REE measurements obtained from both groups of patients before and after surgery were compared to the use of 2 REE predictive equations used for healthy infants and children based on age and weight.[22] The study concluded that predictive equations do not adequately predict energy expenditure in these children. Measured REE more accurately assessed energy needs in children before and after cardiac surgery.[33]

Another group of investigators calculated energy expenditure in 17 infants daily for 3 days after the Norwood procedure.[34] The investigators calculated energy expenditure using the modified Weir equation. Average energy expenditure in kcal/kg/d was 43 ± 11 (day 0), 39 ± 8 (day 1), 39 ± 8 (day 2), and 41 ± 6 (day 3).[34] Postoperatively, neonates are often mechanically ventilated and on medications such as sedatives and neuromuscular blocking agents that reduce resting energy needs. The neonate's postoperative REE after cardiac surgery peaks in the first 12–24 hours.[2] After the first postoperative day, energy needs in the neonate are similar to their basal metabolic rate in most situations.[2,17,33] Because metabolic rate varies between individuals, IC is recommended to assess individual REE. IC is a more accurate measurement of REE than standard predictive equations, but it is not always feasible. Equations have been developed to estimate REE for postoperative surgical and critically ill children.[17] However, these equations do not take into account patient variability. The neonate who has undergone cardiac surgery has higher protein needs than the normal healthy neonate due to the increased proteolysis that occurs in stressful states.[1,22] Based on the metabolic response to stress that the neonate experiences following cardiac surgery, nutrition support goals for the postoperative critically ill neonate have been suggested (see Table 2).[1,17,22]

Complications After Neonatal Cardiac Surgery With Nutrition Implications

Neonates who undergo CPB during cardiac surgery are at risk for inflammatory complications that can lead to

Table 2. Suggested Postoperative Nutrition Goals for Energy, Amino Acids, Carbohydrate, and Lipids for Neonates After Cardiac Surgery

Substrate	Amount
Energy	Acute: 55-60 kcal/kg/d (basal energy/REE) Goal: 90-100 kcal/kg/d
Amino acids	Term: 3-3.5 g/kg/d Low birth weight: 3-4 g/kg/d
Carbohydrate	40%-60% of total kcal Maximum glucose infusion rate: 13 mg/kg/min
Lipids	3-4 g/kg/d to a maximum of 0.13-0.17 g/kg/h

REE, resting energy expenditure.

multiorgan failure. It is unclear whether these patients have more complications because of their marked inflammatory response and multiorgan failure or whether it is due to the fact their nutrition goals cannot be met because of their complications. This dilemma is complex, and more research is needed in this area.

Three significant complications that occur after neonatal cardiac surgery are renal dysfunction or ARF, chylothorax, and neurological dysfunction.[4-7] These do not represent an exhaustive list of complications that can be incurred after neonatal surgery that employs CPB.

Acute Renal Failure

ARF is an abrupt decline in the glomerular filtration rate.[4] The kidneys are not able to regulate fluid or electrolytes or to maintain acid–base homeostasis. ARF in pediatric patients is defined as a 50% elevation in serum creatinine from baseline, a decline in urine output to < 0.5–1.0 mL/kg/h, and an increase in blood urea nitrogen (BUN).[4] The incidence of ARF in pediatric patients after cardiac surgery is 1%–9%. Dialysis treatment is required by 1%–17% of patients who required CPB.[35] Management of ARF involves preservation of fluid and electrolyte homeostasis and allows the provision of adequate nutrition support.[4]

The provision of nutrition support during ARF is greatly affected by fluid availability and treatment modality. After cardiac surgery, care and recovery have been reported to improve with the maintenance of fluid status stability.[4] In addition, ARF induces alterations in protein, carbohydrate, and lipid metabolism. ARF yields a proinflammatory response and a significant impact on the antioxidant system.[35] When dialysis is indicated, the trend over the past decade in pediatric nephrology has been toward CRRT.[4] (Examples of CRRT include continuous venovenous hemofiltration, continuous venovenous hemodialysis, and continuous venovenous hemodiafiltration.[4,36]) Compared with intermittent dialysis, the use of CRRT allows more fluid to be administered, leading to greater provision of adequate nutrition support. Obtaining and maintaining vascular access for hemofiltration is not always feasible, and peritoneal dialysis may be preferred, especially in the neonate. Management of ARF consists of prevention and/or treatment of hyperkalemia and volume overload.[4]

In ARF, protein catabolism is increased, resulting in a negative nitrogen balance.[35] Protein needs are significantly affected by the degree of stress and the presence of CRRT, oliguria, and other medical conditions. CRRT and a high degree of stress lead to the need for greater protein provision due to increased protein turnover and increased protein loss during CRRT therapies. Patients with ARF should receive 1.5 g/kg/d of protein if not being dialyzed, 2–3 g/kg/d if undergoing CRRT, and 3 g/kg/day if undergoing peritoneal dialysis.[4,37,38]

Most of the information regarding trace element support in ARF has been extrapolated from chronic renal failure in which oliguria is often present. The optimal trace element supplementation for patients with ARF has yet to be determined.[36] However, because serum water-soluble vitamin levels are depressed due to losses that occur during CRRT, water-soluble vitamins are often provided.[37] In ARF, both serum and erythrocyte levels of the antioxidant selenium are depressed. Selenium supplementation in critically ill patients receiving CRRT resulted in reduced occurrence of ARF and improved outcomes.[35,36] Optimal nutrition should be based on the type of illness causing ARF and the degree of catabolism in accordance with the type and frequency of CRRT.[35]

Chylothorax

Another complication of cardiac surgery in the neonate is the development of chylothorax during the postoperative period. The incidence of chylothorax has been reported to be 2.5%–4.7%.[5] Chylothorax or chylous fistula is defined as the accumulation of chyle in the pleural space. Injury or damage to the thoracic duct can lead to chyle leak.[39] Chyle is a mix of emulsified fat from intestinal lacteals and lymph from intestinal fluid, which is transported by the lymphatic system, resulting in a milky-white fluid.[39] For neonates, the diagnosis of chylothorax is made when the pleural effusion has a triglyceride level > 1.1 mmol/L, an absolute cell count > 1000 cells/μL, and a predominance of lymphocytes.[40] Nutrition management traditionally includes limiting the consumption of enteral long-chain triglycerides (LCTs).[41] Often, pediatric centers limit LCTs until 6 weeks after surgery.[5] Management of chylothorax after cardiac surgery may include a diet

enriched in medium-chain triglycerides (MCTs) for a neonate and/or PN to promote bowel rest. LCTs are transported in the form of chylomicrons via the lymphatic system, whereas MCTs are transported bound to albumin to the portal system.[41,42] If LCT intake is increased in conjunction with MCT intake, additional MCTs are transported by the lymphatic system.[39] Management of chylothorax also includes replacement of protein and electrolytes. A prolonged chylothorax can lead to deficiency of fat-soluble vitamins.[39]

Neurological Dysfunction

Mild forms of CHD in children are seldom associated with neurological dysfunction.[6] However, children with complex CHD, especially those undergoing cardiac surgery during the neonatal period, develop neurological and developmental abnormalities at an increased frequency.[6] The etiology of neurological dysfunction is multifactorial.[7] One possible mechanism for this is that newborn infants with CHD have impaired brain development.[6] Other potential causes include the effects of ischemia reperfusion injury related to CPB, resulting in compromised oxygen delivery to the tissues. In the immediate postoperative period, cardiorespiratory instability and cerebral vasoregulatory disturbances may also contribute to neurological dysfunction.[43] The effect of neurological dysfunction on nutrition in the neonate can include oral feeding difficulties, delayed development in major motor milestones, and abnormalities in the development of speech.[7]

Case Study

A female patient was diagnosed in utero at 25 weeks with HLHS by fetal echocardiogram. The patient was born by vaginal delivery at 38 weeks' gestation with Apgar scores of 8 and 9 and a birth weight of 3 kg. The patient remained in the neonatal intensive care unit (NICU) until day of life 6. On day of life 6, the patient had cardiac surgery requiring CPB. The patient underwent the stage I palliation Norwood procedure and arch reconstruction and 3.0 Blalock-Taussig shunt placement (BT-shunt). After surgery, the patient was transferred to the PICU with an open sternum, required mechanical ventilation, and received multiple medication infusions that included norepinephrine, epinephrine, vecuronium, fentanyl, milrinone, calcium gluconate, and furosemide.

On postoperative day 1 (day of life 7), the patient's weight was 3.2 kg. The patient's total fluid was limited to 7.5 mL/h for a 24-hour period; medication infusion volume was 4.5 mL/h, leaving the patient's IVF to run at 3 mL/h containing dextrose 10% and 34 mEq/L sodium chloride (see Table 3 for laboratory values). No potassium was added to the IVF solution due to marginal urine output at 0.5 mL/kg/h and a normal potassium level. The patient's IVF solution provided 5 kcal/kg/d.

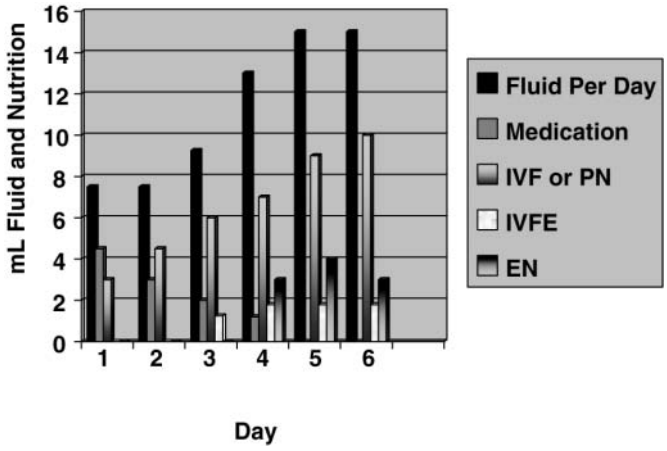

Figure 2. Case study: fluid advancement. Illustration of day 1 through 6 advancement in total fluids and its impact on nutrition provision. IVF, intravenous fluid; PN, parenteral nutrition; EN, enteral nutrition; IVFE, intravenous fat emulsion.

On postoperative day 2, a chest exploration was done for hemodynamic instability. After chest exploration, the patient's sternum remained opened. Mechanical ventilation continued; the patient's total fluid remained limited to 7.5 mL/h, and the patient's total medication infusions were reduced to 3 mL/h, allowing the patient's IVF rate to increase to 4.5 mL/h. Because the patient's glucose and potassium laboratory values indicated a downward trend, the dextrose in the patient's IVF solution was increased to 15% and potassium chloride added at 20 mEq/L. This IVF solution provided 16.0 kcal/kg/d. If delayed sternum closure persisted on postoperative day 3, IC would be performed.

On postoperative day 3, the patient's sternum was closed, and the weaning of the patient's mechanical ventilation was accelerated. IC was not performed due to the decision to close the patient's sternum and the impact of accelerated weaning of medication drips and mechanical ventilation on the patient's REE. Instead, the patient's REE was estimated between 50 and 55 kcal/kg/d. The patient's total fluid was increased to 9.25 mL/h, and the patient's medication infusion volume was reduced to 2 mL/h. Her IVF rate was advanced to 6 mL/h with dextrose 20% with no change in electrolyte composition. Intravenous fat emulsion (IVFE) 20% was started at 2 g/kg/d with a 24-hour infusion rate of 1.25 mL/h. Because the decision was made to close the patient's sternum, nutrition support was advanced to provide 52 kcal/kg/d.

On postoperative day 4, the patient's total fluid was further liberalized to 13 mL/h, and the patient's medication infusion volume was reduced to 1.2 mL/h.

Table 3. Case Study Laboratory Values Postoperative Days 1–5

Postoperative Day	Sodium, mEq/L	Potassium, mEq/L	BUN, mg/dL	Creatinine, mg/dL	Glucose, mg/dL	CRP, mg/dL
1	140	4.5	30	0.9	140	23
2	137	3.0	28	0.8	75	18
3	137	3.1	22	0.6	72	—
4	133	2.8	19	0.6	90	—
5	140	2.9	16	0.6	95	1.9

BUN, blood urea nitrogen; CRP, C-reactive protein.

After extubation and weaning of most medication drips, the patient's nutrition goals were estimated at 90–100 kcal/kg/d, with protein needs at 3–3.5 g/kg/d. PN was initiated at 7 mL/h and comprised 3 g/kg/d amino acids and dextrose 20%; 3 g/kg of IVFE was administered at 1.8 mL/h. Expressed breast milk (EBM) was infused at 3 mL/h over 24 hours via nasogastric (NG) tube. Potassium support increased to 25 mEq/L, and nutrition support advanced to 95 kcal/kg/d.

On postoperative day 5, the patient's weight was 2.99 kg. The patient was extubated and medication infusions were weaned off. The patient's furosemide drip was changed to intermittent dosing every 12 hours via enteral administration. The patient's total fluids increased to 15 mL/h. The patient's PN rate increased to 9 mL/h, and EBM increased to 4 mL/h with an advancement plan to increase by 3 mL/h every 6 hours up to 15 mL/h. As the patient's EBM intake was advanced, the patient's PN was reduced accordingly. No change was made in IVFE. Maximum nutrition support was ~110 kcal/kg/d.

On postoperative day 6, the patient was diagnosed with chylothorax; subsequently, chest tubes were placed. The patient had 150 mL of chylous output per day. The enteral feeding was changed from EBM to a 20-cal/oz formula enriched with MCT. The MCT-enriched formula mixture was reduced to 3 mL/h with PN restarted at 13 mL/h to allow healing of the lymphatic system. The formula at 3 mL/h was not included in total fluids allowed. The patient's chylous output declined over 3 days, and the patient's MCT-enriched formula mixture was advanced to 6 mL/h with no chylous output on postoperative day 9. The patient's chest tubes were removed and formula was advanced to 17 mL/h over 24 hours. The patient's PN was discontinued on postoperative day 10 after the patient's enteral formula was near 14 mL/h on postoperative day 10. IVFE continued until the enteral formula rate was near goal rate. The patient's nutrition goals advanced to 110–120 kcal/kg/d, and protein remained at 3–3.5 g/kg/d after being placed on full EN. On postoperative day 11, when EN was infusing at 17 mL/h, the EN was transitioned to 8 bolus feedings per 24 hours. On postoperative days 13 and 14, the patient's formula concentration was advanced to 24- cal/oz and 27-cal/oz, respectively. The goal was to meet the patient's nutrition needs while using less volume. Initially, the patient's feedings were given entirely via NG tube. On postoperative day 16, the patient's feedings advanced to an oral route with supplemental NG feedings. The patient required supplemental NG tube feeding support for several days, after which time the patient was able to consume all nutrition orally (postoperative day 19). Due to declining trends in serum levels of sodium and chloride, the patient continued to require furosemide with sodium chloride supplementation added to 1 feeding 2 times per day. Once able to meet full nutrition needs orally and gain age-appropriate weight, the patient was discharged from the hospital on postoperative day 22. The patient remained on the MCT-enriched formula mixture for 6 weeks. After 6 weeks, the patient's formula was changed to EBM fortified with term infant formula powder (final concentration of 27 cal/oz).

Summary

Neonates undergoing cardiac surgery experience the same metabolic response to stress seen in older children and adults, but their response is more profound. Most neonates experience a heightened increase in REE postoperatively that is short term and usually returns to basal energy requirements 12–24 hours after surgery. Neonates have lower metabolic reserves compared with older children and adults, putting them at greater risk for malnutrition. This necessitates a rapid initiation of nutrition support to minimize the negative metabolic consequences that can occur after cardiac surgery. However, the initiation of nutrition is hindered by fluid restrictions and the body's inability to use substrates in the immediate postoperative period as a result of decreased secretion of certain hormones. It is clear that energy expenditure is related to the phase of illness vs the severity of illness. Many factors influence the optimal timing of initiation of nutrition support and the composition of nutrition support.

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