Cardiology in the Young

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Disruptions in the development of feeding for infants with congenital heart disease

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Introduction

Congenital heart disease (CHD) is the most common birth defect for infants born in the United States, with approximately 36,000 affected infants born annually.[1,2] While mortality rates for children with CHD have significantly declined, there is a growing population of individuals with CHD living into adulthood prompting the need to optimise long-term development and quality of life. For infants with CHD, pre- and post-surgery, there is an increased risk of developmental challenges and feeding difficulties. Feeding challenges carry profound implications for the quality of life for individuals with CHD and their families as they impact short- and long-term neurodevelopment related to growth and nutrition, sensory regulation, and social-emotional bonding with parents and other caregivers.[1–6] Oral feeding challenges in children with CHD are often the result of medical complications, delayed transition to oral feeding, reduced stamina, oral feeding refusal, developmental delay, and consequences of the overwhelming intensive care unit (ICU) environment.[7–12] This article aims to characterise the disruptions in feeding development for infants with CHD and describe neurodevelopmental factors that may contribute to short- and long-term oral feeding difficulties.

Typical infant feeding

The main goal of feeding is the acquisition of sufficient nutrients for optimal growth and development. Oral feeding for any newborn is a complex process which requires the integration between sucking, swallowing, and breathing to conduct a bolus of milk into the esophagus while avoiding aspiration into the airway.[13] Nutritive sucking is initiated in utero and continues to develop in an organised pattern in the early weeks after birth. It involves the integration of multiple sensory and motor central nervous system functions.[14] The full-term healthy infant has the ability to organise their sucking patterns into productive periods of sucking and pausing that for the infant with perinatal distress is difficult to achieve.[14] At 3–6 months of age, the infant rooting reflex becomes neurologically integrated, and the nutritive sucking pattern transitions from a reflexive pattern to a voluntary action.[14,15] As primitive reflexes peel away, the infant can make choices about accepting or refusing oral feeding based on prior negative or positive experiences. The prevalence of pediatric feeding disorders in the general population is not well defined, but a general range of 3–20% of children in the United States present with oral feeding challenges.[16,17] The prevalence of feeding challenges among children with CHD ranges from 22 to 50%.[8,18,19] Understanding oral feeding and supporting the acquisition of oral feeding skills is so important as a delay in oral feeding may lead to malnutrition, failure to thrive and dysphagia, and could create dependence on tube feeding.[20]

Disruptive factors to feeding infants with CHD

Cardiac physiology

Infants with CHD, both with and without cardiac surgery, have structural and physiologic cardiopulmonary differences compared to infants without CHD, which can contribute to more frequent oral feeding challenges early in life. Most congenital heart defects can be classified into three broad categories, each posing challenges to feeding. Defects with increased pulmonary blood flow may lead to congestive heart failure with tachypnea, increased work of breathing, and growth failure necessitating tube feeding. Defects with decreased pulmonary blood flow result in cyanosis and hypoxia which may impede feeding until a reliable source of pulmonary blood flow is established. Defects with intracardiac mixing, including those with univentricle physiology, require a delicate balance to ensure adequate pulmonary and systemic blood flow. Circulatory imbalance can lead to decreased mesenteric blood flow with resultant feeding intolerance or intestinal ischemia. Introducing enteral feedings to a hypoxic gut presents a risk of necrotising enterocolitis. Depending on the severity of disease and institutional practices, efforts might be taken to avoid enteral feeding (formula or breastmilk in the gastrointestinal tract) of the newborn prior to surgical or medical intervention, thus disrupting the natural progression of oral feeding. Newborns with defects that cause obstruction to systemic blood flow rely on a ductus arteriosus maintained by prostaglandin infusion to ensure adequate blood flow to their body. These infants have intracardiac mixing, which results in decreased oxygen saturation as well. Potentially compromised mesenteric blood flow due to circulatory imbalance puts these infants at risk of developing necrotising enterocolitis which further delays and complicates enteral feeding. The decision to feed infants with ductal-dependent circulation preoperatively is controversial and enteral feeding may be avoided for fear of necrotising enterocolitis.[21–23] That said, roughly two-thirds of centres participating in the National Pediatric Cardiology Quality Improvement Collaborative reported feeding infants with hypoplastic left heart syndrome preoperatively, with varied routes.[24]

Necrotising enterocolitis

Infants with CHD are at a higher risk of necrotising enterocolitis due to perioperative physiology of over circulation and low cardiac output with an incidence rate of 5.1% for infants with CHD, 6.3% for infants with CHD and low birth weight, and 0.8% in non-CHD infants.[25] The nutritional composition of human milk reduces the incidence and severity of necrotising enterocolitis in preterm infants and human milk with fortifier provides optimal nutrition along with the associated lower risk of necrotising enterocolitis.[26] Preoperative use of unfortified human milk was associated with significantly lower risk of preoperative necrotising enterocolitis for infants with CHD. However, the stressors of cardiopulmonary bypass and cardiac surgical repair can elevate the endotoxins and cytokines increasing pro-inflammatory markers for necrotising enterocolitis.[25,27,28] Infants with higher Risk Adjustment for Congenital Heart Surgery Score have a higher risk of necrotising enterocolitis due to the increased complexity of surgery, inflammatory markers, need for longer respiratory support, and feeding intolerance.[29] Infants with univentricle physiology are at particularly high risk for developing necrotising enterocolitis in the perioperative period, with low cardiac output and diastolic runoff as potential contributing factors in addition to those mentioned above. While recovering from necrotising enterocolitis, infants typically receive prolonged periods where they receive no feedings enterally or orally. This is followed by very slow reintroduction of enteral feeding or trophic (minute volumes of human milk or formula through a feeding tube) feeding, which can impact gastrointestinal motility leading to gastrointestinal discomfort from the disruption of natural nutrition delivery.

Gastroesophageal reflux disease

The prevalence of gastroesophageal reflux disease is slightly higher in infants with CHD (25%) than infants without CHD (10–20%).[11,30–32] Clinical signs and symptoms of gastroesophageal reflux disease include choking, gagging, coughing with oral feeding, significant irritability, weight loss, and respiratory compromise.[32,33] The symptoms of gastroesophageal reflux disease overlap with symptoms of aspiration and gastroesophageal reflux disease is often overlooked. Adequate management of gastroesophageal reflux disease symptoms may positively impact long-term feeding by improving comfort, respiratory needs, and weight gain which all increase oral acceptance of nutrition.[11]

Surgical interventions

The timing of medical interventions for CHD can alter or postpone oral feeding with prolonged nils per os status, physiologic response to painful procedures, length of endotracheal intubation and all come at a neurodevelopmental cost. For example, infants with cyanotic univentricle heart disease are usually treated with staged surgical palliation. The first surgical intervention (stage I palliation) occurs within several days after birth, a time period typically dedicated to establishing parental bonding and harnessing feeding reflexes crucial to initial positive oral experiences. Timing of the second-staged surgical intervention (stage II palliation) between 3 and 6 months coincides with the neurological integration of reflexive sucking.[14,15] The timing of both of these surgeries can significantly impact feeding skill development.

Sedation and medication

Many of the medications used in the ICU may cause nausea, constipation, postoperative gastrointestinal ileus, and excessive drowsiness, which impact oral feeding and tolerance for enteral feeding. For example, prostaglandin infusion may be used to maintain the ductus prior to surgery; however, apnea and overall lethargy are significant side effects of prostaglandin administration, which can delay oral feeding. Prostaglandin use requires mechanical ventilation for 10–15% of infants, which also delays oral feeding.[34] Analgesia and sedation used to manage pain and facilitate invasive therapies can compromise haemodynamic stability and lead to respiratory depression, resulting in drug tolerance and physiologic dependence with prolonged administration.[35] Any weaning or discontinuation of medication may result in iatrogenic withdrawal and symptoms including tachycardia, tachypnea, hypertension, irritability, decreased alertness, tremors, increased muscle tone, diaphoresis, nausea and vomiting, diarrhea, and poor oral feeding.

Chylous pleural effusion

Chylous effusions occur when there is an interruption to the lymph ducts that transport chyle (milky fluid consisting of fat droplets and lymph) around the body.[36] The interruption can occur during heart surgery and cause chyle to drain from postoperative chest tubes with an incidence in 2–5% of cases.[37] The presence of chyle

in the pleural space can cause respiratory distress and often requires interventions such as low fat or no-fat diets (which promote ducts to close by decreasing the lipid volume of chyle and allow the ducts to heal) or surgery to repair the ducts.[38] Chylous effusion can lead to paused nutrition, withdrawal of human milk replaced by fat-free formulas, delayed oral feeding, painful procedures (chest tubes, surgery), and increased length of stay.

Respiratory support

Infants with CHD receive respiratory support through a variety of forms. Oxygen delivery can be through nasal canula, non-invasive positive pressure, invasive mechanical ventilation, or extracorporeal membrane oxygenation. In terms of feeding, there is inconsistency across centres regarding the start of positive oral stimulation and oral feedings within these various stages of respiratory support. One study found safe oral feeding for infants with bronchopulmonary dysplasia was achieved with nasal continuous positive airway pressure.[39] Another study with premature infants on nasal continuous positive airway pressure and infants requiring high flow respiratory support experience challenges with aspiration, poor tongue strength, and no sooner time to full oral feeding, which suggests that oral feeding might be contraindicated in the preterm infant receiving non-invasive positive pressure and further studies are needed specific to infants with CHD.[40–42] Respiratory support alone is not adequate to determine feeding readiness and initiation of positive oral experiences. Other factors such as the ability of the infant to demonstrate oral motor coordination, airway protection, physiologic stability, as well as the size and weight of the infant should also be considered.[43,44] Many institutions evaluate initiation of oral feedings with infants based upon criteria which include respiratory rate, respiratory support, level of tachypnea, size and age of the patient, haemodynamic stability, tolerance of oral or tube feedings in the gastrointestinal tract, oral motor readiness, and history of prior oral feedings.

Neurodevelopment

Full-term infants with CHD show decreased brain volume at birth (similar to that of a 36-week preterm infant), increased rates of brain injury such as white matter hypoxic-ischemic lesions, and delayed neurological maturation.[45–49] Recent preoperative studies revealed brain differences on conventional magnetic resonance imaging (MRI) revealing reduced regional functional connectivity involving critical brain regions (i.e., network hubs and/or rich club nodes) and evidence of severe central nervous system insults found in patients with abnormal placentas, and a significant correlation to aortic obstruction defects.[50,51] The neurological events which are common in the newborn period for infants with CHD due to innate immaturity of the brain, prenatal brain injury, and brain injury after surgery can result in neurobehavioural difficulties, such as seizures, hypotonia, hypertonia, or stroke, that impact the ability to coordinate oral feeding.[49,52,53] Specifically, in the newborn period, infants with CHD present with neurodevelopmental impairments, including abnormal motor tone, poor visual orientation, and the need for greater facilitation by the caregiver.[54–57] These neurological and developmental differences contribute to regulatory and feeding challenges in infancy. If an infant has difficulty with state regulation, this impacts oral feeding when they cannot calm or maintain an appropriate level of alertness to eat even with supports from a caregiver. In addition, delayed motor coordination and generalised weakness impact the infant's point of stability for efficient and safe oral feeding. An infant that struggles to maintain flexion and stability of the head, neck, and body will have difficulty taking the full volume needed for growth.

Genetic syndromes

Children with genetic syndromes frequently have feeding problems and swallowing dysfunction due to the complex interactions between anatomical, medical, physiological, and behavioural factors. Approximately 30% of infants with CHD have a related genetic syndrome compared to 0.5% in infants without CHD.[58,59] Specific risk factors for poor feeding in infants with genetic conditions can include structural anomalies (cleft-lip, glossoptosis), lack of experience (surgeries, intubation), neurologic abnormalities (hypotonia, brain malformation), or oral motor delays (poor tongue control, open mouth posture).[60] For example, neuromotor coordination impairments such as those associated with Down syndrome, frequently interfere with the acquisition of effective oral-motor skills and lead to feeding difficulties. Accurate genetic diagnoses are important to create an effective plan of care and initiate therapeutic services both inpatient and outpatient to support infant feeding.[60,61]

Noxious feeding environment

Infants in the cardiac ICU may experience a noxious and stressful environment with excessive stimulation and frequent painful procedures and interventions which impact overall development, including oral feeding. Environmental factors such as noise and light levels elicit physiologic changes in newborns, including increase in heart rate, respiratory rate and blood pressure, hearing loss, sleep disturbances, hypoxemia, decrease in endocrine and cardiovascular function, stress responses, and decrease in oxygen saturation, which all adversely affects growth, development, and the ability to orally feed.[62–65] Sights and sounds in the cardiac ICU have also been found to be a maternal stressor, impacting the mother/child relationship.[66] Current research advocates for adjustment to the hospital environment to reduce the detrimental effects of the noxious environmental stimuli in the cardiac ICU on the infant. For example, limiting noise by reducing loud talking, beeping of monitors and phones, closing doors quietly, decreasing or removing the sound level on music boxes and televisions, providing indirect lighting for infants, shielding infants' eyes, and bundling cares to allow time with no care giving for rest.[65]

Nerve paralysis/paresis

There are several nerves (vagus, recurrent laryngeal, phrenic) responsible for triggering a timely swallow response and simultaneous airway closure for the bolus of milk to be safely transported from the mouth to the oesophagus.[67] The vagus nerve and its branches are responsible for heart rate, gastrointestinal peristalsis, sweating, swallowing, sensation in the pharynx and larynx, and the gag reflex. The recurrent laryngeal nerve supplies most of the intrinsic muscles of the larynx, sensation below the vocal cords, and opening of the vocal cords. The phrenic nerve is vital for breathing, as it passes motor information to the diaphragm. Damage to these nerves results in poor airway closure and increased risk of aspiration (transport of milk into the lungs). Injury to the nerves complicates oral feeding after congenital heart surgery. Pham et al revealed 58% of Norwood and arch reconstructions had vocal cord paralysis correlating with dysphagia in 74% of Norwoods and 69% of Arch reconstructions.[68] Prompt diagnosis of

nerve immobility supports safety and sooner initiation of therapeutic oral feeding approaches.

Dysphagia

Dysphagia is defined as swallowing difficulties which are common in children receiving complex cardiac surgery, with prevalence ranging from 18 to 56% depending on the type of heart lesion. Univentricle physiology with stage 1 palliation, genetic abnormalities, and use of transesophageal echocardiography are risk factors for swallowing deficits and tracheal aspiration in infants.[69–71] Significant pharyngeal and esophageal dysmotility and abnormal upper esophageal sphincter opening are also factors which impact dysphagia and have been observed in infants post cardiac surgery.[72]

Safe swallowing requires intricate coordination of 31 paired muscle groups and 5 cranial nerves, influenced by the integrity of the respiratory, neurologic, musculoskeletal, and gastrointestinal systems.[69,70,73,19,74,75] Any disruption in the timing, strength, or movement of muscles in these systems can result in dysphagia. Dysphagia is diagnosed by a modified barium swallow study (MBSS) and evidenced by poor timing of pharyngeal swallow initiation, inadequate airway protection, tracheal aspiration, as well as poor suck, swallow, breathe coordination.[19,76]

The most common clinical finding for dysphagia in infants with CHD is poor coordination of sucking, swallowing, and breathing.[76] Clinical signs and symptoms of faulty coordination of sucking often manifest as an increase in oxygen desaturation, heart rate, respiratory rate, energy expenditure, coughing, choking, gagging, retching, and red/watery eyes with oral feeding. To compensate, infants display a multitude of disruptive strategies to reduce milk flow from the breast or bottle by allowing the liquids to spill laterally from the sides of the mouth, producing weak tongue patterns, holding the nipple without sucking, losing lip seal to the breast or bottle, or blocking their mouth with the tongue elevated.[77] Further, infants may shift alertness to drowsy or sleepy, even shutting down and thereby protecting their airway by refusing an oral feeding opportunity. They demonstrate uncoordinated oral patterns to avoid being fed, which can be due to increased work of breathing, aspiration, laryngeal penetration (liquids enter the larynx above the true vocal folds and are then ejected), intestinal discomfort, and fatigue. Poor sucking coordination may result in the infant with CHD burning more calories during an oral feeding trial than the calories received from the feeding attempt.

Consequences of feeding challenges

Nutritional interference

Infants with CHD have challenges with weight gain due to hypermetabolism and a greater demand on respiratory musculature for adequate ventilation, placing them at higher risk for malnutrition and growth failure.[78] Hypermetabolism has been reported to be three to five times higher in infants with CHD than typically developing infants.[30] In addition, infants with CHD present with tachypnea and poor endurance for the aerobic work required for oral feeding, thus presenting a challenge to consume enough volume to meet caloric needs. Often, the caloric density of fortified breast milk and formula is increased to 22–30 kcal/oz to meet that need, compared to 19–20 kcal for unfortified breast milk or formula. Higher caloric concentration demonstrates improved nutritional status for infants with hypoplastic left heart syndrome, but the long-term consequences have not been delineated.[24,79] Frequently, infants with CHD demonstrate poor intestinal tolerance of fortification and increased feeding volumes can be challenging for the poorly perfused gastrointestinal system.

Weight gain for infants with CHD, especially for infants requiring multiple surgeries, is optimised to promote positive surgical outcomes. Poor nutrition in newborns with CHD is negatively correlated to long-term brain development, motor skills, and higher parental stress.[78,80] Nutritional deficiency in infancy has a negative impact on the rapidly developing brain, which can alter brain size and neurodevelopment, as already mentioned for infants with CHD. In infants with CHD, malnutrition noted as a height-for-age or weight-for-age z-scores of <-2 is associated with a higher risk of mortality by 2.9 and 2.1%, respectively for each unit decrease in z-score.[81] Additionally, for each unit decrease in height-for-age z-score, there was a 1.2% of cardiac arrest, 1.1% of infection, 1.7 additional hours of mechanical ventilation, 6 hours longer ICU stay, and 13 hours longer hospital length of stay.[81]

Breastfeeding difficulty

Given the complex medical needs of infants with CHD at birth, breastfeeding is difficult for a myriad of reasons including separation of the mother and infant at birth, minimal privacy in the ICU, unknown volume of human milk the infant is consuming for fluid management, and ingrained myths regarding breastfeeding (more difficult than bottles, the mother ate something upsetting to the infant, etc.); however, research shows infants with CHD can engage in breastfeeding despite severity of heart defect.[82] Marino et al found infants with CHD had statistically significant higher oxygen saturation and less oxygenation variability during breastfeeding compared to a bottle feed, indicating less cardiorespiratory stress and better ability to regulate milk flow from a breast.[83] The central nervous system plays a role in creating neurological opportunities for swallowing at the breast within the sucking and breathing cycle that is not seen in bottle feeding.[84] This improves the potential for adequate ventilation during sustained feeding and supports energy conservation and weight gain.[9,85] Supporting early and consistent development of this unique motor planning and programming at the breast is essential for establishing the least restrictive feeding plan for children with CHD. While working toward exclusively breastfeeding, infants with CHD also benefit from additional feeding methods (tube or bottle) and fortification (additional calories added to the milk) to optimise growth.[86]

Infants with CHD are sometimes separated from their mothers in the initial transition period and there are limited opportunities for skin-to-skin contact and breastfeeding. Mothers of infants with CHD often express breast milk that can then be fortified. These mothers are at risk for late-onset milk production because of the delay in the opportunity for direct breastfeeding, stress of having an infant in an ICU, less restful sleep, and poor hydration/nutrition while navigating care in the ICU.[87,88]

Tube feeding

Infants with critical heart lesions requiring surgery in the newborn period typically benefit from feeding support such as total parental nutrition, nasogastric, nasojejunal feeding tubes, or gastrostomy or gastrojejunostomy to sustain growth and promote healing while recovering acutely from cardiac interventions.[89] Approximately, 30 to 64% of the time, infants with CHD discharge home with supplemental feeding using a feeding tubes. Infants with a genetic syndrome, preoperative feeding difficulty or a palliative procedure is more likely to discharge with a feeding tube.[11,12,72,90,91] Of note,

nasogastric supplemental feeding at home following discharge was not reported to increase mortality risk for univentricle patients.[92] Hospitals vary in their practice and use of tube feeding. Questions arise around whether to offer bolus tube feeding (volume given over a shorter time usually 0.5–1 hour) or continuous (volumes given over longer times, usually 18–24 hours) when supplementing oral nutrition to promote long-term feeding success. Additional questions arise regarding type of tube feedings (nasogastric, nasojejunal, gastrostomy). Children who initially require nasogastric/gastric tube placement are at risk of becoming "tube dependent". Tube dependency can be defined as either a medical need for the tube (i.e., inability to take in the required nutrition through oral means because of cardiorespiratory insufficiency or swallow dysfunction and risk of unsafe feeding from aspiration), or a perceived need (i.e., ongoing feeding tube use when the initial health issues are no longer necessitating a medical tube dependence). There is currently limited data available regarding best practices for tube weaning in children with complex CHD, but many cardiac ICU's have standardised feeding protocols with tube weaning programs and a formalised process to promote success.[89,93] Standardised protocols help therapists, families, and physicians support a team approach to tube weaning with the goal of full oral feeding when the infant demonstrates readiness. Tube weaning is indicated when the supplemental feeding tube is no longer needed to support the child medically. Parents should receive information on tube weaning and a full tube weaning plan before a tube is placed. Parents often are given an explanation as to why a tube is used for supplemental nutrition but given little guidance for how and when tube weaning will begin.

Oral aversion

Oral aversion is the reluctance, avoidance, or fear of feeding orally or accepting any sensation in or around the mouth. Many infants with CHD develop significant oral aversion resulting in negative refusal behaviors with attempted oral feeding.[72] Oral aversion is often triggered by oral intubation or surgery and not following the infants' cues regarding when to continue or discontinue feeding.[10] This oral feeding refusal is often distressing for both the infant and the caregiver.[94] The risk of oral aversion and not feeding orally at the time of hospital discharge is significantly related to the number of days intubated.[12,72]

Social emotional impact

The disruption to oral feeding impacts the social–emotional connection with infants and their caregivers. Feeding challenges in infancy change the psychosocial aspects of caregiving and contribute to parental stress.[95] Infants with complex CHD often require immediate postnatal medical attention contributing to altered holding, bonding, and early feeding experiences with their families. Medical and surgical interventions are often juxtaposed with a time typically dedicated to establishing parental bonding and developing feeding confidence. Both infants and their families perceive this lack of involvement as stressful. Studies of maternal stress in the cardiac ICU revealed the infant's appearance and behaviour is the greatest stressor for families, followed by parental role alteration and decreased time parenting their infant.[66] Postnatal feeding is altered for infants with CHD and their parents, resulting in difficulty with pre-feeding activities such as holding and skin-to-skin. Postnatal bonding experiences are altered for infants with CHD and their parents, resulting in difficulty with pre-feeding activities such as holding and skin-to-skin. Feeding and the precursors to feeding are an important part of parenting, attachment, and bonding for all infants.[96,97]

Feeding challenges often persist into childhood. A systematic review of parents' experience of long-term tube feeding in children with disabilities identified concerns for families related to oral feeding, including mixed feeding messages, pressure from health professionals, reduced support, difficulty with decision making, and continued feeling of inadequacy.[98] This research also found more negative interactions and/or parental dysfunction in comparison to families of healthy children.[99,100] Not surprisingly, concerns about infant feeding behaviours contribute to ongoing stress in families with children with CHD.[80,95] Parents of children with CHD report an initial worry about their child's survival, but over time their child's weight gain and feeding challenges become a more significant stressor. Mothers expressed concern about the time and energy involved in feeding with a tube, tube cleaning, how to replace a tube that is accidentally removed, long feedings, vomiting, nighttime feedings affecting sleep, confusing hunger cues, and concern around their child's ability to feed normally in the future.[95] Parents of children with cardiac disease who have feeding challenges have increased negative mood and spend significantly more time thinking about feeding than parents of children with cardiac disease that do not have feeding challenges or parents of children with no medical complications.[100]

Long term feeding outcomes

Children with CHD show persistent feeding challenges in early childhood.[8,101] Feeding difficulties in the newborn period and the need for multiple surgeries and hospitalisations are independent predictors of abnormal feeding behaviour at age 2 in 22% of patients who underwent newborn cardiac surgery.[8] The duration of intubation in the newborn period is positively associated with increased selective/restrictive feeding and identified feeding dysfunction in 50% of infants with complex CHD at 2–6 years. Families of children with univentricle physiology reported increased resistance to eating (13% of children) compared to children without CHD (1%).[102] They also reported increased mealtime aggression (16 versus 4%), choking/gagging/vomiting (29 versus 8%), and parental aversion to mealtime (11 versus 1%).[102] Children with CHD had significantly more problematic mealtime behaviours compared to healthy peers on the Paediatric Eating Assessment Tool, including selective/restrictive eating and oral processing dysfunction, indicating risk for oral aversion to age-appropriate textures and consistencies.[101] All these difficulties contribute to the long-term feeding challenges of children with CHD.

Feeding difficulties in infancy also correlate with poorer developmental outcomes. Children with early feeding difficulties resulting in tube feedings at hospital discharge and at 3 months of age were at higher risk for lower cognitive and motor development at 6 months of age.[6,103] Compromised nutrition and growth at 3 months of age have been associated with poorer cognitive and motor outcomes at 6 and 12 months of age.[80] Concerning scores in the areas of stress, newborn reflexes, and arousal on the neurobehavioral assessment of premature infants are associated with oromotor feeding impairments at 12 months of age.[104]

Feeding challenges and growth failure in early childhood are associated with not only long-term deficits in height and weight but also cognition, academic performance, executive functioning, and behaviour.[105] When looking at growth failure in children with cognitive and academic performance challenges at school age,

intelligence quotient scores that are lower than children with a history of adequate growth suggest that oral intake and growth impacts long-term cognitive performance.[106,107] Children with a history of feeding challenges are more likely to have regulatory problems such as sleeping, crying, and behavioural problems like internalising, externalising, and attention-deficit/hyperactivity disorder.[108] In children with CHD, this connection is less clear, yet research does show an impact of oral intake and growth on neurodevelopment in infancy.[91]

Conclusions

Infants born with CHD present with structural and physiologic differences to their heart function, brain development, and gut performance which can hinder the development of oral feeding skills. Many factors contribute to the feeding challenges, affecting not only the infant but also the family as well. Infants with CHD present with increased risk of malnutrition, feeding difficulty, need for tube feedings, dysphagia, neurodevelopmental issues, and disrupted attachment that can persist into childhood. Understanding the unique differences of infants with CHD is important in order to address specific interventions to support oral feeding. More research is also necessary in understanding feeding interventions for infants with CHD. Considering the significant negative impact that delayed or suboptimal feeding could have on infants with CHD, the authors suggest that inpatient teams include discussion of feeding as a critical element of the daily management of this fragile population, beginning at the time of admission. Further research in various treatment strategies to address oral feeding difficulties in infants with CHD is warranted to understand how to facilitate safe and efficient transitions to the highest level of oral feeding success by improving skill and function. More information is also needed to understand the direct influence of poor feeding, parental stress, and feeder response to infant cues during oral feedings and the impact on neurodevelopmental outcomes.

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