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Improving Maternal-Infant Bonding after the Prenatal Diagnosis of Congenital Heart Disease

Piers C.A. Barker¹, Gregory H. Tatum¹, Michael J. Campbell¹, Michael GW Camitta¹, Angelo S. Milazzo¹, Christoph P. Hornik³, Amanda French², and Stephen G. Miller¹

¹Division of Pediatric Cardiology, Department of Pediatrics, Duke University Medical Center, Durham, USA

²Advanced Practice Nursing, Duke University Medical Center, Durham, USA

³Duke Clinical Research Institute, Duke University, Durham, USA

Abstract

Background—Infants with prenatally diagnosed congenital heart disease are at high risk for adverse outcomes due to multiple physiologic and psychosocial factors. Lack of immediate physical postnatal contact due to rapid initiation of medical therapy impairs maternal-infant bonding. Based on expected physiology, maternal-infant bonding may be safe for select cardiac diagnoses.

Methods—Single center study to assess safety of maternal-infant bonding in prenatal congenital heart disease.

Results—157 fetuses with prenatally diagnosed congenital heart disease were reviewed. Based on cardiac diagnosis, 91 fetuses (58%) were prenatally approved for bonding and successfully bonded, 38 fetuses (24%) were prenatally approved but deemed not suitable for bonding at delivery, and 28 (18%) were not prenatally approved to bond. There were no complications attributable to bonding. Those who successfully bonded were larger in weight (3.26 versus 2.6 kg, $p < 0.001$) and at later gestation (39 versus 38 weeks, $p < 0.001$). Those unsuccessful at bonding were more likely to be delivered via Cesarean section (74% versus 49%, $p = 0.011$) and have additional non-cardiac diagnoses (53% versus 29%, $p = 0.014$). There was no significant difference in need for cardiac intervention prior to hospital discharge. Infants that bonded had shorter hospital (7 versus 26 days, $p = 0.02$) and ICU lengths of stay (5 versus 23 days, $p = 0.002$) and higher survival (98% versus 76%, $p < 0.001$).

Corresponding Author: Piers C.A. Barker, MD, CHC 1927A, DUMC, Durham, NC 27710, USA, Phone: 919-684-3574, piers.barker@duke.edu.

Conflicts of Interest:

The authors have no financial relationships or conflicts of interest relevant to this article to disclose.

Ethical Standards:

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national guidelines on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008, and was approved by the Duke University Institutional Review Board with waiver of informed consent.

Conclusion—Fetal echocardiography combined with a structured bonding program can permit mothers and infants with select types of congenital heart disease to successfully bond prior to ICU admission and intervention.

Keywords

Fetal Echocardiography; Congenital Heart Disease; Maternal-Infant bonding; Prenatal diagnosis

Introduction

The prenatal diagnosis of congenital heart disease creates a clinical and psychosocial paradox. Pregnancy management radically changes, grief for the loss of a “normal” pregnancy and “normal” child ensues, and social and financial stresses are placed upon the entire family¹. All this occurs in service of improving care of the child. Delivery represents a peaking of the conflict between infant well-being and maternal psychosocial support. Typically, the pregnancy is considered “high risk,” and delivery occurs in a medically intensive environment, with greater monitoring, fewer family members and more medical personnel present, and a greater likelihood of induction or Cesarean delivery². Immediately after birth the infant is handed to a waiting resuscitation team and often progresses very quickly to an intensive care unit (ICU) environment – potentially in a different hospital – after minimal physical contact with the parents. Once in the ICU intravenous access is established, prostaglandins started if necessary, and opportunities for parental-child bonding become even more limited.

This tremendous and well-intentioned effort stands in contradiction to the natural history of neonatal congenital heart disease^{3–7}. With relatively rare exceptions, newborns with congenital heart disease will not manifest hemodynamic instability immediately after birth^{3,4,7}. The ductus arteriosus maintains patency for hours after delivery^{5,6}, which supports the recommendation to perform pulse oximetry screening at 24 hours after birth to minimize false positive results^{8–11}. For the mother, immediate bonding after birth increases attachment and decreases maternal anxiety¹², while infants demonstrate greater temperature and cardiorespiratory regulation¹³. Successful breastfeeding is also more likely to occur^{13,14}. Most critically for infants both with and without congenital heart disease, all of these benefits occur within an ‘early sensitive period.’

The concept of an “early sensitive period” has been demonstrated both in human and animal studies^{12,13}, and in humans corresponds to a 2 hour window after birth of increased infant alertness and vocalization that prime reciprocal interaction¹³. Efforts to bolster this early sensitive period with early maternal-infant skin-to-skin contact and early breastfeeding improves both infant and mother long-term outcomes¹² while poor early bonding leads to worse later bonding up to 1 year of age¹⁵. These benefits are lost if maternal-infant bonding does not occur during this “early sensitive period” even if mother and infant are reunited later¹⁶, which is the scenario created by any urgent transfer of an infant with congenital heart disease to an ICU environment. Fortunately this “early sensitive period” corresponds with the same period during which ductal patency has been demonstrated to be preserved.

Knowledge of the natural history of congenital heart disease and the ductus arteriosus, coupled with knowledge of the benefits of maternal-infant bonding thus raises an important question: Is the rapid intensification of newborn medical care which limits maternal-child interaction successfully treating the infant, or instead palliating the anxiety of the medical team? Or asked another way: Is current medical practice interfering with an even more critical intervention for the health of the infant and mother, namely the opportunity for early bonding, immediate skin-to-skin contact, and early breastfeeding?

As a first step towards answering this question, this single center observational study evaluated the impact of initiating a formalized maternal-infant bonding program on the safety and early outcomes of infants with prenatally diagnosed congenital heart disease.

Methods

Bonding Algorithm

The Duke “Hearts for Bonding” algorithm was developed in collaboration between Advanced Practice Nursing, Pediatric Cardiology, Maternal-Fetal Medicine, Neonatology, Pediatric Cardiothoracic Surgery and Pediatric Critical Care (Figure 1). In the algorithm bonding duration is targeted to be 30 minutes beyond the time of initial resuscitation, with an ICU nurse always present in case of patient deterioration and to check infant oxygen saturation every 10–15 minutes during maternal-infant contact. Fetuses are designated as either “approved” or “not approved” for bonding by the attending cardiologist at the time of the initial Fetal Cardiology consultation based on the expectation for postnatal hemodynamic instability and need for elective or urgent neonatal intervention. Additionally, the intensive care unit for neonatal admission (Pediatric Cardiac ICU versus Neonatal ICU) is designated at the initial Fetal Cardiology consultation based on similar expectations and the presence of additional non-cardiac diagnoses. Bonding status and ICU admission location are confirmed or revised by the attending cardiologist at each subsequent Fetal Cardiology visit, and these designations are communicated to all the provider teams via a Fetal Cardiac Triage List.

Designation of Bonding Status

Based on natural history data, an infant in whom hemodynamic stability is expected for several hours after birth, with or without ductal patency, is approved for bonding immediately after delivery. In contrast, infants expected to be immediately unstable, regardless of ductal status, are not approved to bond and are transported directly to an ICU after neonatal resuscitation. While it is not possible to provide a truly comprehensive list of diagnoses “approved” or “not approved” for bonding, examples of diagnoses “approved” for bonding include hypoplastic left heart syndrome or other forms of functional single ventricle without atrial septal restriction (admit to Pediatric Cardiac ICU), tetralogy of Fallot in almost all of its variations (unit admission determined by expected degree of pulmonary stenosis), or balanced Atrioventricular Canal Defect with trisomy 21 (admit to Neonatal ICU). Examples of diagnoses “not approved” to bond include D-transposition of the great arteries with intact ventricular septum, tetralogy of Fallot with absent pulmonary valve, severe Ebstein’s anomaly of the tricuspid valve, total anomalous pulmonary venous

connection (all types), complete heart block, intractable fetal arrhythmia, heart disease of any type associated with hydrops fetalis, or infants with critical non-cardiac abnormalities such as congenital diaphragmatic hernia, open neural tube defect or craniofacial abnormalities at risk for immediate airway compromise.

Deferral of Approved Bonding

The algorithm includes strict criteria for making a decision at the time of delivery to defer bonding even when prenatally approved and planned. Examples of delivery scenarios in which the leader of the neonatal resuscitation team decides that approved bonding should not occur include respiratory distress associated with meconium aspiration, neonatal depression, prematurity (<35 weeks gestation), growth restriction (<2 kg at delivery) or significant hypoxemia. The neonatal resuscitation team is in attendance at all fetal cardiac deliveries.

Data Collection

Retrospective review was performed of the institutional Fetal Cardiology Quality Improvement Database to identify all patients who had a fetal echocardiogram from January 2011 to December 2013. The study was approved by the Duke University Institutional Review Board with waiver of informed consent. Inclusion criteria were any structural heart disease, arrhythmia, cardiac dysfunction, or hydrops fetalis. Exclusion criteria included in utero fetal demise, termination of pregnancy, delivery at another facility, planned palliative care, or absence of heart disease on post-natal assessment. For patients determined to have absence of heart disease (e.g.: potential coarctation of the aorta on fetal imaging, with normal aortic arch on postnatal assessment) the final diagnosis selected for analysis was based upon the last echocardiogram performed before neonatal discharge. Data collected included the fetal cardiac diagnosis, whether bonding was approved or not, if bonding occurred as planned, and reason bonding did not occur. We also collected data on gestational age at delivery, birth weight, date and time of delivery, method of delivery, and non-cardiac diagnoses. Complications attributable to bonding were assessed by review of delivery room, infant admission and discharge notes, and surrogate markers of instability. These markers included Apgar scores, if cardiac intervention (surgery or catheterization) was required prior to hospital discharge, need for non-elective intubation, number of days requiring mechanical ventilation, need for inotropic support prior to cardiac intervention, ICU and hospital lengths of stay, and survival to discharge. Bonding assignments were not changed for perinatal comorbidities that could not be reliably anticipated (prematurity, low birth weight, etc.), leaving bonding management to be determined by algorithm criteria.

Bonding Classification

Subjects were divided into three groups based on the Fetal Cardiology bonding plan and whether or not bonding successfully occurred. Group I: bonding both approved and occurred after delivery; Group II: bonding approved but did not occur due to the infant being deemed not suitable for bonding at the time of delivery; Group III: Not approved for postnatal bonding due to anticipated neonatal instability and/or expected need for emergency intervention. For analysis purposes, to be considered to have bonded the infant must have remained with the mother with an opportunity for skin-to-skin contact and breastfeeding for

at least 10 minutes beyond the time spent for neonatal resuscitation, evaluation and intermittent pulse oximetry.

Statistical Methods

We used standard summary statistics including counts (percentages) and medians (25th, 75th percentiles) to describe all categorical and continuous variables. The primary analysis compared subjects who underwent successful bonding to those in whom bonding was planned but did not occur (Group I versus Group II). We performed separate secondary analyses comparing: (1) subjects who successfully bonded to those who did not, regardless of prior bonding plan (Group I versus Groups II and III); and (2) subjects in whom bonding was planned but did not occur to those who were deemed prenatally not to be candidates for bonding (Group II versus Group III). We then repeated primary and secondary analyses in a cohort of patients with cyanotic and single ventricle lesions. We used Wilcoxon rank sum, Kruskal-Wallis, Fisher's exact, and Chi square tests of association to compare the distribution of study variables across Groups as appropriate.

Finally, we performed multivariable logistic regression to evaluate the association between bonding and the occurrence of non-elective intubation or inotropic support, controlling for the following a-priori determined covariates: type of heart disease (categorical variable), gestational age in weeks (continuous variable), birth weight in kg (continuous variable), method of delivery (categorical variable), and 5 minute Apgar (categorical variable). We performed all analyses in Stata 14.1 (Stata Corps, College Station, Texas) and considered a $p < 0.05$ statistically significant.

Results

We identified 227 abnormal fetal echocardiograms in 226 pregnancies, with one set of twins who both had congenital heart disease (Table 1). Seventy subjects were excluded due to: delivery at another facility (41); in utero demise (8); termination of pregnancy (8); planned palliative care (2); and absence of heart disease on postnatal assessment (11). Fetal diagnoses in which in utero demise occurred included atrioventricular canal defect, hypoplastic left heart syndrome or its variants, fetal cardiac rhabdomyomas with maternal Tuberous Sclerosis, hydrops fetalis associated with a large hepatic arteriovenous malformation, and fetal complete heart block. Elective termination was chosen for fetal diagnoses of hypoplastic left heart syndrome or its variants, unbalanced atrioventricular canal defect, or chromosomal abnormalities (trisomy 21 or trisomy 18). Postnatal palliative care was chosen for one fetus with severe cardiomyopathy and one fetus with in utero hepatitis A infection. Of the 11 fetuses without heart disease on postnatal assessment, 4 were suspected of having small to moderate-sized ventricular septal defects, 5 were considered at risk for coarctation of the aorta and 2 had frequent premature atrial contractions that resolved prior to delivery. A total of 157 infants were therefore included for analysis (Figure 2).

There were 91 infants (58%) in Group I (bonding planned and occurred), 38 infants (24%) in Group II (bonding planned but did not occur) and 28 infants (18%) in group III (bonding not approved). Data for Group II infants are presented in Table 2. Reasons why bonding did not

occur despite being planned in this group included respiratory distress requiring positive pressure ventilation (11), prematurity with gestational age < 35 weeks (9), persistently low oxygen saturations (below 70%) without distress (3), low 5 minute Apgar score without distress (3), and birth weight less than 2 kg (2). Choice of age and weight limits for prematurity and growth restriction as reasons for deferral of bonding were defined *a priori* as described in the Methods due to Neonatology concerns for lung disease or other abnormalities secondary to prematurity, or inadequate temperature/glucose regulation in the setting of growth restriction. Reason for deferral of bonding was unclear in 10 infants, with bonding time documented at less than 10 minutes without any instability in patient or other documented rationale for failure to follow protocol. In all subjects in whom bonding was planned but did not occur the decision to forgo bonding was made during the initial infant assessment and stabilization period, and these infants were transferred to the ICU within 10 minutes of birth. No mother requested to forego bonding for infants prenatally approved for bonding.

Types of heart diseases statistically differed when compared across the 3 groups (Table 3a) ($p=0.039$). Arrhythmias were more frequent in the group that bonded, while diagnosis of “other heart diseases” (Table 3b) was more common in the group in which bonding was not approved. When analysis was performed comparing infants who bonded successfully to those in whom bonding was approved but did not occur due to the infant being deemed not suitable for bonding at delivery (Groups I versus II), there was no significant difference in type of heart disease ($p = 0.093$). When Group I was compared to both II and III combined together, type of heart disease approached but did not reach statistical significance ($p = 0.055$).

Several birth characteristics were different between the groups (Table 4). Infants who successfully bonded were at older gestation (median Group I = 39 weeks, II = 38 weeks, $p < 0.001$) and higher birth weight than those who did not bond (median Group I = 3.26 kg, II = 2.6 kg, $p < 0.001$). Infants who bonded were less likely to be delivered by Cesarean-section (Group I = 49%, II = 74%, $p = 0.011$), but there was no significant difference between the groups in time of delivery; i.e.: during routine daytime hours or off hours (evenings, nights and weekends). Infants who successfully bonded were less likely to have an additional non-cardiac diagnosis than those who were deemed not suitable for bonding after birth (Group I = 29%, II = 53%, $p = 0.014$), but the types of non-cardiac diagnoses were not significantly different between the three groups (Table 5). Infants who successfully bonded had higher Apgar scores at 5 minutes ($p < 0.001$). When comparing Group II to Group III there were no statistically significant differences in these variables.

No complications attributable to maternal-infant bonding occurred in the group that successfully bonded. Surrogate markers for instability were also favorable for infants who successfully bonded (Table 6). Infants who successfully bonded (Group I) were less likely to need inotropic support prior to cardiac intervention (2% versus 24%, $p<0.001$), or require non-elective intubation (5% versus 45%, $p<0.001$), and had shorter ICU (median 5 days versus 23 days, $p = 0.002$) and hospital lengths of stay (median 7 days versus 26 days, $p = 0.02$) compared to infants who were deemed not suitable to bond at the time of delivery (Group II). However, the need for cardiac intervention prior to hospital discharge was not

different between the groups. The group that bonded (Group I) had a higher survival compared to Group II (98% versus 76%, $p < 0.001$), with both deaths in Group I occurring after cardiac intervention. One death was due to elective withdraw of care after Stage I palliation in an infant with hypoplastic left heart syndrome with late diagnosis of trisomy 13 and one death was in patient with unbalanced atrioventricular septal defect who had severe post-operative atrioventricular valve regurgitation requiring ECMO support and died from multiorgan failure at day of life 53. For Group II, 2 of the 9 deaths were after cardiac intervention, as were 4 of the 11 deaths in the group not approved for bonding (Group III). There were not any statistically significant differences in outcome measures between those in whom bonding was planned but did not occur (group II) and those in whom bonding was not approved (group III).

When only cyanotic and single ventricle infants were compared, infants who bonded (Group I) remained less likely to need inotropic support prior to cardiac intervention (3% versus 31%, $p=0.009$), or require non-elective intubation (6% versus 54%, $p<0.001$), and had a higher survival (94% versus 69%, $p=0.032$) compared to Group II. Differences in ICU (median 15 days versus 30 days) and hospital lengths of stay (median 30 days versus 44 days) were no longer significantly different. In this subgroup analysis there were no statistically significant differences between Groups II and III.

In multivariate analysis, the group that was deemed not suitable to bond at delivery remained more likely to require non-elective intubation or pre-operative inotropic support when compared to the group who successfully bonded (Odds ratio 3.47, 95% confidence interval (CI) 1.01, 11.92).

Discussion

This study highlights three important findings: First, immediate postnatal bonding between mothers and infants with prenatally selected types of congenital heart disease does not place those infants at higher risk of perinatal instability or short term complications. Second and reflective of this first finding, fetal echocardiography can successfully identify which infants can safely bond after delivery, assuming the absence of complications such as prematurity, growth restriction, respiratory distress due to meconium aspiration, or neonatal depression. For the cases in which bonding was approved but did not occur due to the infant being deemed not suitable for bonding at the time of delivery, the reasons were not related to the cardiac disease. Instead, decisions to defer bonding were typically secondary to non-cardiac problems, such as prematurity, respiratory distress due to meconium aspiration, etc.. A component of the choice to defer planned bonding also likely reflected provider discomfort with the initial implementation of the bonding algorithm. This is best and most specifically represented by one instance of an infant not being allowed to bond due to the documented reluctance of the Pediatric House Officer attending the delivery despite all bonding criteria being met. Third, infants who successfully bond are of greater gestational age at delivery, have a higher birth weight, less likelihood of Cesarean delivery, higher Apgar scores and less escalation of care prior to intervention or discharge.

The primary finding that postnatal bonding did not result in perinatal instability is not surprising given the natural history of the neonatal ductus arteriosus. In a study examining echocardiograms performed within 4 hours of delivery (median 3 hours 22 minutes), a patent ductus arteriosus was present in all healthy term infants⁵. A second study reviewed serial echocardiograms in infants divided into 3 groups by gestational age at delivery (30–33 weeks, 34–37 weeks, term). Only 1 ductus was found to be closed in each group on the first echo, performed at a mean 7.7 hours after delivery, with subsequent ductus closure rates of 50–58% (mean 31 hours), 81–88% (55 hours), and 99% (80 hours), and no difference in closure rates based on gestational age at delivery in these three cohorts⁶.

The natural history of neonatal congenital heart disease is well-established, and has been incorporated into fetal echocardiography standards used to stratify perinatal risk and to direct specialty care^{17,18,19,20, 21}. Hypoplastic left heart syndrome with restrictive atrial septal defect, D-transposition of the great arteries with intact ventricular septum, tetralogy of Fallot with absent pulmonary valve, Ebstein's anomaly of the tricuspid valve, persistent arrhythmias and heart disease causing hydrops fetalis are all high-risk lesions that reliably produce hemodynamic instability; however, most other congenital heart defects do not present immediately at birth. In a study reporting the prevalence of hypoplastic left heart syndrome in Oregon from 1979 to 1986, the median age at development of symptoms was 1 day, the median age at diagnosis was 4.1 days, and the greatest risk of death was at 3 days⁴. A similar study on the natural history of tetralogy of Fallot recorded that 68% of infants were visibly acyanotic at birth, 5% had intermittent or mild cyanosis and 19% had severe cyanosis³. This natural history is incorporated into the current pulse oximetry screening protocols for congenital heart disease, with the optimal timing for detection and minimizing false positives being approximately 24 hours after delivery^{8–10,22}.

Prenatal diagnosis of congenital heart disease has been demonstrated to create maternal stress, depression and anxiety^{1,23,24}. Prenatal stress can produce elevated maternal cortisol levels which overwhelm placental compensatory mechanisms and lead to elevated fetal/amniotic cortisol levels^{25,26}. Animals exposed to prenatal stress display increased fetal mortality, and decreased birth weight, structural brain abnormalities and cognitive impairments in offspring^{27,28}. In humans, increased maternal cortisol levels have correlated with delayed motor and mental development in infancy²⁵, which when coupled with the delayed brain maturation seen in fetuses with congenital heart disease, may create a negatively synergistic impact on neurodevelopment^{29–31}.

Postnatally, the process of normal maternal-infant bonding is at risk for infants with congenital heart disease, regardless of whether or not prenatal diagnosis occurred. Mothers of infants with congenital heart disease experience short and long-term depression and anxiety^{32,33,34}. This maternal depression may interfere with normal early bonding, and bonding problems may persist to 1 year¹⁵. Maternal psychological stress is exacerbated in the setting of infants with severe congenital heart disease, with mothers more likely depressed at 6 months post-partum, and the infants exhibiting maladaptive behaviors that may increase maternal depression and negate caretaking efforts, creating a cycle of maternal-child distraction³⁵.

Implementation of a perinatal bonding program creates an opportunity to favorably offset the negative factors associated with the prenatal diagnosis of congenital heart disease, and improve outcome beyond the perinatal period. Early skin-to-skin contact in premature infants has been associated with increased maternal attachment, decreased maternal anxiety, and improved autonomic functioning, cognitive development, sleep organization, stress response and executive function in children persisting up to 10 years of age¹². Mothers who experience early skin-to-skin contact with their full term infants have increased oxytocin release, and decreased cortisol levels, perceived pain after surgical delivery, bleeding time and time to placental expulsion. The infants also cried less, had slightly higher temperatures and slightly slower heart and respiratory rates; consistent with a safe experience for both mother and child¹³. In a similar meta-analysis, exclusive breastfeeding was more likely to occur when mothers and infants were maintained in the same room as opposed to being separated^{14,36}.

It is notable but not unexpected that infants in whom bonding was approved and successfully occurred were delivered at later gestational age with greater birthweight, and subsequently better perinatal and hospitalization outcomes, compared to the higher mortality in those who did not bond. This indicates the increase in risk when congenital heart disease is combined with prematurity³⁷, low birth weight³⁸ or additional non-cardiac diagnoses. There has been an evolving trend in fetal cardiac management towards later delivery with less inductions or surgical deliveries², with our practice following this trend during the study period. The importance of later gestational delivery is supported by registry data documenting worse outcomes for infants with congenital heart disease delivered prior to 39 weeks gestation (and even worse with delivery prior to 36 weeks)^{39,40}. Although associated non-cardiac defects did drive earlier deliveries in this study, severity of fetal heart disease (cyanotic versus single ventricle, or need for postnatal intervention) by itself did not, and was not associated with bonding success or overall outcome. Choice of ICU admission also was not related to bonding selection or success, but instead reflective of institutional architecture and care-team logistics.

Limitations of this study include the retrospective and single-center observation design limiting the sample sizes of each Group, and the selection-bias inherent to the assignment of bonding status with 10 out of 39 infants not having clear reasons for failure to bond. Although bonding duration was targeted for 30 minutes beyond neonatal resuscitation from the first use of the bonding algorithm, a bonding duration of at least 10 minutes beyond neonatal resuscitation was accepted for data analysis due to the challenges faced with initial provider comfort with implementing this new approach to perinatal care. It is therefore theoretically possible that the outcome data may be confounded in the group of infants bonding for greater than 10 minutes but less than 30 minutes, or for the 10 infants with unclear reasons for not being allowed to bond after delivery despite prenatal approval. Reassuringly, the absence of complications has persisted with the acceptance of a 30 minute duration for bonding as institutional standard of care. Similarly, while the intermittent assessment of infant oxygen saturation poses a risk for disruption of bonding, this assessment was performed with the infant in the mother's care and only for the time needed to obtain an accurate reading.

Statistical analyses were also chosen primarily to assess the predictors and risks of successful bonding. The retrospective design limited documentation of specific complications; however, this time period was selected for review as it coincided with the initial years of the Hearts for Bonding program. Heterogeneity of cardiac disease and small numbers of specific cardiac diagnoses prevented more detailed analysis of the trend seen in comparing type of heart disease between Group I versus Group II and III combined. Anecdotally, this trend likely reflects the critical granularity that exists between cardiac diagnoses (e.g.: the cyanotic heart disease category encompassing both D-transposition of the great arteries with intact ventricular septum versus D-transposition of the great arteries with ventricular septal defect) that is difficult to analyze in a single center study. While the prenatal assignment of bonding status created a selection bias favoring infants expected to be well after delivery, the expectations of postnatal stability are consistent with current Fetal Cardiology consensus, and the option to defer bonding based on the resuscitation team's judgement provided a maximal amount of safety. Lastly, the decision to exclude infants with suspected heart disease on fetal imaging but who were found not to have heart disease on postnatal assessment was made to minimize any bias favoring safety of bonding. Important future steps should include extension of the maternal-infant bonding period from 30 minutes to the full 2 hours of the "early sensitive period," cessation of intermittent pulse oximetry, use of an established bonding scale⁴¹ and connection of a prospective cohort to longer term neurodevelopmental follow-up.

Conclusion

Maternal-child bonding immediately after delivery can occur safely and without hemodynamic instability in appropriately selected infants with prenatally diagnosed congenital heart disease. This supports greater normalization of perinatal care for both mother and infant, and has the potential to improve infant and maternal physical, psychosocial and neurodevelopmental status.

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Hearts for Bonding Initiative

Figure 1.

Figure 2.

Table 1

Summary Cohort Data

Classification of Disease	Included	Delivered Elsewhere	IUFD/ Termination
Total	157	41	16
Shunt Lesion	36 (23 %)	19 (46%)	3 (19%)
Obstructive Lesion	27 (17%)	3 (7%)	1 (6%)
Cyanotic (2V) Lesion	24 (15%)	2 (5%)	1 (6%)
Single Ventricle	30 (19%)	5 (12%)	6 (38%)
Arrhythmia	19 (12%)	5 (12%)	1 (6%)
Other	21 (13%)	7 (17 %)	4 (25%)

Classification of Heart Disease:

- Shunt lesions: ventricular septal defects, atrial septal defects, or balanced atrioventricular septal defects
- Obstructive lesions: Amenable to a two ventricle repair, including aortic stenosis, pulmonary stenosis, or aortic arch obstruction
- Cyanotic lesions: Amenable to a two ventricle repair, including tetralogy of Fallot and all its variations, D-transposition of the great arteries.
- Single Ventricle: All defects requiring single ventricle palliation.
- Arrhythmias: Persistent and frequent atrial or ventricular ectopy, tachyarrhythmias and complete heart block
- Other heart disease: Cardiomyopathy (all types including non-compaction and complications of twin-twin transfusion syndrome), Ebstein's anomaly of the tricuspid valve, congenital mitral insufficiency, vascular ring, cardiac tumor, non-immune hydrops fetalis and ectopia cordis.

Group II Characteristics

Table 2

Reason for deferral of bonding	Gestational Age	Birth Weight (kg)	Cardiac Diagnosis	Non-Cardiac Diagnosis	Delivery Type
Inadequate time with parents	39 wk 2d	3.39	Hypoplastic aortic arch	None	Emergent Cesarean
Inadequate time with parents	38wk 1d	2.59	AVCD	FGR	Emergent Cesarean
Inadequate time with parents	39wk 3d	2.49	TOF	FGR	Induced
Intubated*	38wk 5d	1.91	Hypoplastic aortic arch	Trisomy 13	Elective Cesarean
Prematurity	34wk 2d	1.65	TTTS	None	Emergent Cesarean
Prematurity	33wk	1.55	AVCD	None	Elective Cesarean
Meconium aspiration*	36wk 4d	2.18	Unbalanced AVCD	Trisomy 21	Emergent Cesarean
Prematurity	31wk 6d	1.4	TOF	Cleft lip, 22q11 deletion	Emergent Cesarean
Continuous positive airway pressure*	37 wk	2.615	AVCD	None	Elective Cesarean
Unclear	36wk 3d	2.89	TGA with VSD	None	Emergent Cesarean
Low oxygen saturations	37wk 6d	3.11	Tricuspid Atresia	None	Emergent Cesarean
Intubated*	40wk	3.71	In utero ductal constriction	None	Emergent Cesarean
Low oxygen saturations	36wk 5d	2.49	TTTS	Multiple gestation	Elective Cesarean
Low Apgars	39wk 2d	3.11	HLHS	Renal anomaly	Induced
Inadequate time with parents	38wk 2d	2.32	Coarctation of aorta	None	Induced
Low Apgars	39wk 4d	3.09	VSD	None	Spontaneous
Bag-mask ventilation*	34wk 6d	2.11	Cardiac tumor	Gastroschisis	Elective Cesarean
Inadequate time with parents	38wk 4d	2.68	Pulmonary atresia	None	Induced
Inadequate time with parents	39wk 1d	3.78	AVCD	None	Elective Cesarean
Intubated*	39wk 6d	3.48	Ebstein's anomaly	None	Elective Cesarean
Bag-mask ventilation*	39wk 2d	3	VSD	SUA & tracheomalacia	Elective Cesarean
Bag-mask ventilation*	40wk 5d	3.21	AVCD	Trisomy 21	Elective Cesarean
Intubated*	38wk	2.8	Unbalanced AVCD	None	Induced
Continuous positive airway pressure*	37wk 3d	2.22	VSD	Chromosome 4 & 15 deletion	Elective Cesarean

Reason for deferral of bonding	Gestational Age	Birth Weight (kg)	Cardiac Diagnosis	Non-Cardiac Diagnosis	Delivery Type
Prematurity	33wk 4d	0.84	Pulmonary atresia	Trisomy 1	Elective Cesarean
Prematurity	24wk 1d	0.59	HLHS	None	Spontaneous
Inadequate time with parents	39wk 1d	3.78	AVCD	None	Elective Cesarean
Low Apgars	38wk 6d	2.97	DORV	None	Induced
Unclear	37wk 4d	4.55	Coarctation of aorta	Multiple extracardiac anomalies	Elective Cesarean
Prematurity	24wk	0.5	AVCD	Trisomy 18	Spontaneous
Prematurity	33wk 6d	1.81	Hypoplastic aortic arch	FGR	Elective Cesarean
Unclear	39wk	3.94	ASD	Maternal HELLP Syndrome	Emergent Cesarean
Prematurity	34wk 6d	1.88	ASD	Jacobson syndrome	Emergent Cesarean
Prematurity	30wk 2d	0.85	VSD	Oligohydramnios	Elective Cesarean
Nuchal cord *	39wk 2d	2.95	VSD	None	Emergent Cesarean
Low birthweight	35wk 1d	1.66	TOF	Trisomy 21	Emergent Cesarean
Low oxygen saturations	38wk 6d	3.06	DORV	None	Induced
Low birthweight	38wk	1.85	DORV	FGR	Emergent Cesarean

Inadequate time with parents = documentation of less than 10 minutes bonding time; Unclear = no indication in record of why bonding was deferred;

* = respiratory distress;

wk = week; d = day; AVCD = AtrioVentricular Canal Defect; TOF = Tetralogy of Fallot; TTTS = Twin-Twin Transfusion Syndrome; TGA = Transposition of Great Arteries; VSD = Ventricular Septal Defect; DORV = Double Outlet Right Ventricle; ASD = Atrial Septal Defect; HELLP = Hemolysis Elevated Liver enzymes Low Platelets; FRG = Fetal Growth Restriction, SUA = Single Umbilical Artery

Table 3

a: Heart Disease and Bonding Outcome			
Group	I – Approved and successfully bonded N=91	II – Approved but not suitable for bonding at delivery N=39	III – Bonding not prenatally approved N=28
Shunt Lesion	21 (23%)	12 (32%)	3 (11%)
Obstructive Lesion	13 (14%)	9 (24%)	5 (18%)
Cyanotic (2V) Lesion	14 (15%)	5 (13%)	5 (18%)
Single Ventricle	17 (19%)	8 (21%)	5 (18%)
Arrhythmia	17 (19%)	0 (0%)	2 (7%)
Other Heart Disease	9 (10%)	4 (10%)	8 (28%)

b: Other Heart Disease and Bonding Outcome			
Group	I – Approved and successfully bonded N=9	II – Approved but not suitable for bonding at delivery N=4	III – Bonding not prenatally approved N=8
Cardiomyopathy	3 (3.3%)	2 (5.1%)	3 (10.7%)
Ebstein's Anomaly/Tricuspid valve anomaly	2 (2.2%)	1 (2.6%)	1 (3.6%)
Hydrops	0	0	3 (10.7%)
Cardiac Tumor	1 (1.1%)	1 (2.6%)	0
Vascular Ring	3 (3.3%)	0	0
Ectopia Cordis	0	0	1 (3.6%)

Table 4

Birth Characteristics and Bonding

Bonding Group	I – Approved and successfully bonded N=91	II – Approved but not suitable for bonding at delivery N=38	III – Bonding not prenatally approved N=28
Gestation Age (weeks) Median(25–75%ile)	39 (38 – 40)	38(35 – 39)	37 (33 – 39)
Birth Weight (kg) Median(25–75%ile)	3.26 (2.84 – 3.55)	2.6 (1.85 – 3.11)	2.78 (1.81 – 3.3)
Delivered off hours	41 (45%)	12 (32%)	12 (43%)
Non-Cardiac Diagnosis	26 (29%)	20 (53%)	12 (43%)
Delivered by Cesarean	45 (49%)	28 (74%)	16 (57%)
5 Minute Apgar 1–3	0 (0%)	2 (5%)	3 (11%)
5 Minute Apgar 4–6	0 (0%)	8 (22%)	6 (22%)
5 Minute Apgar 7–10*	89 (100%)	27 (73%)	18 (67%)

Data shown are counts (%) unless otherwise indicated

* Apgar data was not documented for 2 patients in group I, and 1 each in groups II and III

Table 5

Non-Cardiac Diagnoses and Bonding Outcome

Type of Non-Cardiac Diagnosis	I – Approved and successfully bonded N=26	II – Approved but not suitable for bonding at delivery N=20	III – Bonding not prenatally approved N=12
Genetic Syndrome	11 (42%)	6 (30%)	0
Extra-cardiac malformation	8 (31%)	6 (30%)	7 (58%)
Multiple gestation pregnancy	3 (11%)	2 (10%)	2 (17%)
Fetal Growth Restriction	1 (4%)	4 (20%)	2 (17%)
Poly/Oligohydramnios	1 (4%)	1 (5%)	0
Single umbilical artery/Absent ductus venosus	2 (8%)	1 (5%)	1 (8%)

Table 6

Surrogate Markers for Instability and Bonding

Bonding Group	I – Approved and successfully bonded	II – Approved but not suitable for bonding at delivery	III – Bonding not prenatally approved
Cardiac Intervention before discharge N (%)	35 (38%)	14 (37%)	12 (43%)
Non-elective Intubation N (%)	5 (5%)	17 (45%)	16 (57%)
Total days intubated Median (25–75%)	0 (0–5)	2 (0–13)	8 (1–18)
Days intubated post-procedure Median (25–75%)	5 (2–7)	6 (2–9)	9 (5–24)
Inotropic support (prior to cardiac intervention) N (%)	2 (2%)	9 (24%)	11 (39%)
HLOS, days Median (25–75%)	7 (3–36)	26 (7–63)	34 (7–66)
ICU LOS Median (25–75%)	5 (1–24)	23 (7–52)	23 (5–58)
Survival N (%)	89 (98%)	29 (76%)	17 (61%)

ICU = Intensive Care Unit; HLOS = Hospital Length of Stay