Prenatal and Newborn Screening for Critical Congenital Heart Disease: Findings From a Nursery

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KEY WORDS
pulse oximetry, fetal echocardiography

ABBRVIATIONS
AAP—American Academy of Pediatrics
BCH—Boston Children’s Hospital
BWH—Brigham and Women’s Hospital
CCHD—critical congenital heart disease
CICU—cardiac ICU

Dr Johnson coordinated meetings with nursing staff to initiate pulse oximetry screening program adhering to American Academy of Pediatrics guidelines, collected data from pulse oximetry screening tests, analyzed the results, and wrote preliminary methods and results section; Dr Lieberman participated in organizational meetings to construct a pulse oximeter screening program, collected data from pulse oximetry screening tests, and supervised the statistical analysis of the pulse oximetry data; Dr O’Leary collected data from pulse oximetry screening tests, assisted with the data analysis, and wrote the first version of the introduction; Dr Geggel proposed the current investigation to determine the contribution of pulse oximetry in the detection of critical congenital heart disease in a tertiary-care level-1 nursery, participated in meetings with the nursery staff to construct a pulse oximetry protocol, reviewed the echocardiography reports performed on infants at Brigham and Women’s Hospital in the NICU and cardiac ICU at Boston Children’s Hospital for infants born at Brigham and Women’s Hospital, and patients referred from outside medical centers to Boston Children’s Hospital for management of critical congenital heart disease during the study period, and wrote the full first draft of the manuscript; and all authors approved the final manuscript.

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WHAT’S KNOWN ON THIS SUBJECT: The detection of critical congenital heart disease by fetal echocardiography or neonatal physical examination can have limitations. The addition of pulse oximetry screening in the newborn nursery increases the rate of diagnosis of these conditions before hospital discharge.

WHAT THIS STUDY ADDS: In a tertiary-care center with comprehensive fetal echocardiography, nearly all newborns with critical congenital heart disease are diagnosed prenatally. Pulse oximetry will identify more infants from settings with lower prenatal detection. Improving access to and training in fetal echocardiography should also improve detection of these conditions.

abstract

BACKGROUND: Delayed diagnosis of critical congenital heart disease (CCHD) in neonates increases morbidity and mortality. The use of pulse oximetry screening is recommended to increase detection of these conditions. The contribution of pulse oximetry in a tertiary-care birthing center may be different from at other sites.

METHODS: We analyzed CCHD pulse oximetry screening for newborns ≥35 weeks’ gestation born at Brigham and Women’s Hospital and cared for in the well-infant nursery during 2013. We identified patients with prenatal diagnosis of CCHD. We also identified infants born at other medical centers who were transferred to Boston Children’s Hospital for CCHD and determined if the condition was diagnosed prenatally.

RESULTS: Of 6838 infants with complete pulse oximetry data, 6803 (99.5%) passed the first screening. One infant failed all 3 screenings and had the only echocardiogram prompted by screening that showed persistent pulmonary hypertension. There was 1 false-negative screening in an infant diagnosed with interrupted aortic arch. Of 112 infants born at Brigham and Women’s Hospital with CCHD, 111 had a prenatal diagnosis, and none was initially diagnosed by pulse oximetry. Of 81 infants transferred to Boston Children’s Hospital from other medical centers with CCHD, 35% were diagnosed prenatally.

CONCLUSIONS: In our tertiary-care setting, pulse oximetry did not detect an infant with CCHD because of effective prenatal echocardiography screening. Pulse oximetry will detect more infants in settings with a lower prenatal diagnosis rate. Improving training in complete fetal echocardiography scans should also improve timely diagnosis of CCHD.

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Congenital heart disease is the most common cause of infant death associated with congenital malformations in the developed world.1–5 Approximately 9 of 1000 live births have congenital heart disease.4,6 Of these, ~25% have critical congenital heart disease (CCHD), defined as a condition requiring surgical or catheter intervention in the first year of life.2,4 Unfortunately, the diagnosis of CCHD is often delayed with negative impact on both morbidity and mortality.7–12 Some neonates with CCHD are diagnosed only after death.3,13 This deficiency in detection is contributed by limitations in physical examination,14,15 including difficulty in identifying cyanosis, especially in anemic or dark-pigmented neonates,7,16 early hospital discharge in duct-dependent lesions if the duct has not yet closed,7,13,17 and absence of murmurs in many patients with CCHD due to the specific anatomy, elevated pulmonary vascular resistance, or reduced contractility.11,15,16,18 Other modes of testing also have limitations. Electrocardiography and chest radiography lack sensitivity and specificity.4 Pediatric echocardiography is expensive and not readily available at many birthing centers.4 The detection of CCHD by prenatal ultrasound varies by lesion, experience of the operator, and protocol of views used with generally <50% of cases identified.2,4,5,21–24 Multiple studies over more than a decade have assessed the role of pulse oximetry to improve the detection of CCHD in neonates before hospital discharge. The results of these studies have led the Department of Health and Human Services and the American Academy of Pediatrics (AAP) to recommend universal pulse oximetry screening in the newborn nursery.25,26 Pulse oximetry has a high specificity and moderate sensitivity in the detection of CCHD,1,4,5,7–9,20 and has a lower false-positive rate if performed after 24 hours of age.1,4 Compared with physical examination alone, pulse oximetry increases the rate of detection of CCHD2,7,21,26–28 and contributes to improved clinical status at the time of diagnosis.

Although neonatal pulse oximetry screening increases the detection of CCHD, its impact may be less in a tertiary-care setting with extensive prenatal fetal imaging.2 We established a pulse oximetry program that fulfilled AAP guidelines in such a hospital setting and evaluated thoroughness of testing, screening outcomes, and whether CCHD was detected by pulse oximetry, fetal echocardiography, or postnatal clinical presentation.

METHODS

Patient Selection at Brigham and Women’s Hospital

We examined screening among neonates of ≥35 weeks’ gestation born from January 1 to December 31, 2013, at Brigham and Women’s Hospital (BWH) who were cared for in the well-infant nursery during the time frame when pulse oximetry screening would have been completed, or transferred to Boston Children’s Hospital (BCH) for management of specified congenital heart disease. BWH is a tertiary-care birthing center that provides extensive prenatal ultrasound with a protocol approved by the Committee on Clinical Investigation at both BWH and BCH.

Identification of Neonates Eligible for Pulse Oximetry Screening

We identified all neonates of ≥35 weeks’ gestation by using a hospital database. We excluded infants who spent their entire BWH hospital stay in the NICU or who were transferred to the NICU before 48 hours of age and had not received their CCHD screening before transfer.

Identification of Neonates With Prenatal or Postnatal Diagnosis of Congenital Heart Disease

Neonates born at BWH who were diagnosed prenatally or postnatally with CCHD consisting of 1 of the 7 lesions selected to be screened by the AAP guidelines, aortic arch obstruction, or other cyanotic heart disease (see Table 1 for list of diagnoses) were identified from echocardiography databases or admissions logs from the cardiac ICU (CICU) and NICU at BCH. Maternal obstetric records in the BWH NICU were reviewed to determine if the initial prenatal detection of congenital heart disease was made at BWH or a referring medical facility. Neonates with a fetal diagnosis of CCHD were initially admitted to the BWH NICU and had an initial echocardiogram either at BWH or BCH.

Identification of Neonates Born at BWH With Late Diagnosis of CCHD

The admissions logs from the CICU and NICU at BCH were reviewed for 2013 and the first 10 weeks of 2014 to determine any late admissions for previously undetected CCHD among infants born at BWH and discharged from the hospital before diagnosis. With few exceptions, neonates with CCHD in our region have surgical repair at BCH.

Determination of Prenatal Versus Postnatal Diagnosis of CCHD for Neonates Born at Medical Centers Other Than BWH

Neonates with CCHD lesions listed in Table 1 born at other medical centers during the study period who were transferred to BCH were identified from admissions logs to the CICU or NICU at BCH. These patients’ records were reviewed to determine if the diagnosis of CCHD was made before or after birth.
TABLE 1  Cyanotic Congenital Heart Disease or Aortic Arch Obstruction Diagnosed in Newborns at BWH or Referred From Other Hospitals to the NICU at BCH in the First 10 Weeks After Birth During the Study Period

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Born at BWH</th>
<th>Born at Other Hospitals</th>
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<tbody>
<tr>
<td></td>
<td>Prenatal</td>
<td>Postnatal</td>
</tr>
<tr>
<td>Lesions selected to be screened by AAP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypoplastic left heart</td>
<td>16</td>
<td>0</td>
</tr>
<tr>
<td>Pulmonary atresia-intact ventricular septum</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>17</td>
<td>0</td>
</tr>
<tr>
<td>Dextro-transposition of the great arteries</td>
<td>18</td>
<td>0</td>
</tr>
<tr>
<td>Tricuspid atresia</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Total abnormal pulmonary venous connection</td>
<td>0*</td>
<td>0</td>
</tr>
<tr>
<td>Truncus arteriosus</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>63</td>
<td>0</td>
</tr>
<tr>
<td>Aortic arch obstruction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coarctation of the aorta</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>Interrupted aortic arch, ventricular septal defect</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>26</td>
<td>1</td>
</tr>
<tr>
<td>Other cyanotic congenital heart disease</td>
<td>22*</td>
<td>0</td>
</tr>
<tr>
<td>Suspected aortic arch obstruction not confirmed by postnatal echocardiography</td>
<td>15</td>
<td>—</td>
</tr>
</tbody>
</table>

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* Two patients had critical pulmonary stenosis with near atresia.
* Eighteen with pulmonary stenosis, 11 with pulmonary atresia, 2 with absent pulmonary valve, 1 with complete atriopulmonary canal.
* Twenty-one with intact ventricular septum, 5 with ventricular septal defect, 6 with ventricular septal defect and pulmonary stenosis.
* Total abnormal pulmonary venous connection did occur in 3 patients with heterotaxy and atrioventricular canal defect that was detected prenatally, and are listed in the “Other cyanotic congenital heart disease” section.
* Heterotaxy (total 7) with atroventricular canal and total anomalous pulmonary venous connection (3), unbalanced atroventricular canal and hypoplastic left ventricle (2), or atrioventricular canal, double outlet right ventricle, pulmonary stenosis (2); complex congenital heart disease with single ventricle physiology (3); critical aortic stenosis with fetal intervention (4); unbalanced complete atrioventricular canal (2); Ebstein anomaly of the tricuspid valve (1); pulmonary atresia with large apical muscular ventricular septal defect (1); hypoplastic right ventricle, ventricular septal defect, pulmonary stenosis (1); double outlet right ventricle, superior-inferior ventricles, crisscross atrioventricular valves (1).
* Heterotaxy (total 2) with double outlet right ventricle, ventricular septal defect, crisscross atrioventricular valves, pulmonary stenosis (1), or tetralogy of Fallot and malposed atrial septum (1); Ebstein anomaly of the tricuspid valve (1); dysplastic tricuspid valve (1); right dominant unbalanced atrioventricular canal, small left ventricle, coarctation of the aorta (1); double outlet right ventricle, transposition of the great arteries, pulmonary stenosis (1).
* Critical aortic stenosis (1); arterial tortuosity syndrome and midaortic syndrome (1); pericardial teratoma compressing airway (1); pulmonary glycyogenosis and pulmonary vein stenosis (1); vascular ring (right aortic arch with aberrant left subclavian artery compressing airway and multiple ventricular septal defects) (1); Ebstein anomaly of the tricuspid valve (1); arcade mitral valve associated with severe mitral regurgitation (1).
* Left-ventricular–right-ventricular size discrepancy or aortic arch hypoplasia noted on fetal echocardiography.

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**Pulse Oximetry Screening Protocol**

We followed the pulse oximetry protocol approved by AAP,6 screening was performed by nurses who had undergone training to obtain simultaneous preductal (right hand) and postductal (either foot) oxygen saturations. Screening was targeted to occur between 24 and 48 hours of age. Infants discharged before 24 hours of age were screened as close to discharge as possible. Infants screened after 48 hours of age included those with an intravenous line in the right hand for antibiotic administration during the first 48 hours of life and those infants transferred to the well-newborn nurseries after 48 hours of age. In both of these groups, screening was performed as soon as feasible. The Radical-87 pulse oximeter with reusable probe LN0P Y1 (Masimo, Irvine, CA) was used for all initial screening tests in the well-newborn nurseries. Infants were considered to have passed the screening if the oxygen saturation was ≥95% in the right hand or foot and there was ≤3% difference between the 2 values. Neonates failing the first screening were transferred to the NICU triage area for clinical evaluation and further testing by using a DASH pulse oximeter with disposable LN0P probe (General Electric Medical Systems, Milwaukee, WI). Neonates with oxygen saturation values between 90% and 94% in both the right hand and foot, or >3% difference between the extremities required repeat testing in 1 hour; and if similar values were obtained, a third screening was performed 1 hour later. Patients were judged to fail the screening protocol if the oxygen saturation values were in this range on all 3 occasions or if any 1 screening yielded an oxygen saturation in either extremity of <90%. A failed screening protocol designation prompted a cardiology consultation.

**Results**

**Study Group for Pulse Oximetry Screening**

During the study period, there were 7328 live births of ≥35 weeks’ gestation at BWH, 442 of whom were excluded because pulse oximetry was not required according to our protocol. Excluded infants included those admitted to the NICU on the first day of life and who remained there for their hospital...
Polio patients who had prenatal diagnosis of CCHD (n = 126, Table 1), and those transferred to the NICU between 24 and 48 hours of life before pulse oximetry screening was performed (n = 23). Of the remaining 6886 infants, 48 (0.7%) were missing CCHD screening data (13 forms with missing oxygen values had no patient identifying information, 14 forms had uninterpretable or missing oximetry values, and 21 forms were missing from the medical record). The study group comprised the remaining 6838 infants (99.3% of eligible newborns).

Pulse Oximetry Screening Results

Of the infants who had complete pulse oximetry data, 6803 (99.5%) passed the first screening. One of these infants represented the only false-negative result in the study. This infant had a normal fetal cardiac examination as part of a routine ultrasound fetal survey at 18 weeks’ gestation at BWH. On pulse oximetry screening at 29 hours of age, pre- and postductal oxygen saturations were 97% and 96%, respectively. He developed a new murmur 10 hours and cyanosis 12 hours after the first oximetry screening (oxygen saturation 94% in the right hand, 85% in the leg), and was diagnosed to have interrupted aortic arch with ventricular septal defect. He underwent surgical repair with an excellent outcome.

Thirty-five infants (0.5%) failed the first pulse oximetry screening. Of these, 34 infants passed the second screening, 3 of whom had an uneventful subsequent hospital course, 3 of whom had subsequent desaturation associated with laryngomalacia (length of stay 5 days), neonatal stroke (length of stay 5 days), or unknown cause (length of stay 7 days), and 1 who had apnea of unknown cause (length of stay 7 days). One infant failed all 3 screenings and was diagnosed by using echocardiography to have persistent pulmonary hypertension of the newborn (length of stay 3 days); this was the only echocardiogram performed during the study period for a failed CCHD screening test.

Infants who failed the first screening did not differ from those who passed the first screening in terms of race, gestational age, or method of delivery (vaginal versus cesarean delivery). Both being large for gestational age (1.4% [7/520] vs 0.4% [28/6318], P = .006) and having a birth weight >4 kg (1.2% [7/599] vs 0.5% [28/6239], P = .02) were associated with failing the first CCHD screening. There was a trend toward a higher risk of failing the first screening among male infants compared with female infants (0.66% vs 0.36%, P = .08).

There were no infants born at BWH during the study period who were admitted to the BCH CICU within 10 weeks of their birth with previously unrecognized CCHD.

Prenatal Diagnosis of CCHD

During the study period, 112 infants (~1.5% of all live births) born at BWH had CCHD, 111 (99%) of whom had a prenatal diagnosis; the only postnatal diagnosis occurred in the patient with interrupted aortic arch described in the previous section. Of these infants, 83 (56%) had 1 of the 7 lesions selected to be screened by AAP; 27 (24%) had coarctation or interrupted aortic arch, and 22 (20%) had other complex congenital heart disease (Table 1). Thirteen mothers had detection of CCHD by prenatal ultrasound performed at BWH, and 98 mothers (42 from other Massachusetts medical centers, 29 from the other 5 New England states, 24 from other regions of the United States, and 3 from foreign nations) had ultrasound performed locally and were referred with concern about congenital heart disease that was confirmed or fully defined by staff at BWH or the Advanced Fetal Care Center at BCH. Prenatal screening raised concern about aortic arch obstruction in 15 other patients because of ventricular size discrepancy or aortic arch hypoplasia but postnatal echocardiography demonstrated normal cardiac anatomy and function.

During the study period, 81 infants born at other medical centers were transferred to the BCH CICU for management of CCHD. Of this group, 28 (35%) had a prenatal diagnosis and 53 (65%) had a postnatal diagnosis; 1 additional patient did not have postnatal confirmation of suspected aortic obstruction (Table 1). Thirteen of the patients with prenatal diagnosis had fetal echocardiography performed at BCH, 8 of whom were born at another tertiary-care birthing center 1 block from BCH and 5 of whom were born at other medical centers. Referral medical records documented a failed pulse oximetry screen in 4 infants; we could not determine how many of the referral centers had a neonatal pulse oximetry screening program. Five patients with postnatal diagnosis had been discharged from the hospital after birth and developed severe respiratory distress or profound cyanosis (2 infants with infradiaphragmatic total anomalous pulmonary venous connection) or shock (3 infants with coarctation) before diagnosis. Another infant with postnatal diagnosis developed severe acidosis at the birth hospital (dextro-transposition of the great arteries with restrictive atrial septum) before diagnosis.

DISCUSSION

We were able to establish a pulse oximetry screening program that successfully tested >99% of neonates cared for in the well-infant nursery. During the study period, ~1.5% of infants born at BWH had CCHD, a value ~7 times the expected incidence and associated with the tertiary referral status of our medical center. Of the patients diagnosed with CCHD, 56% had 1 of the 7 lesions...
targeted for detection by AAP, 24% had aortic arch obstruction (coarctation or interrupted aortic arch), and 20% had other cyanotic heart disease.

In our tertiary-care setting, each of the neonates with 1 of the 7 targeted lesions was diagnosed before birth by fetal echocardiography and none was initially detected by postnatal pulse oximetry. This finding is in contrast to infants born at other hospitals and referred for cardiac care, approximately two-thirds of whom had a postnatal diagnosis. Some of these neonates presented after hospital discharge in a compromised state and likely may have benefited from pulse oximetry screening. Previous studies also have noted a higher prenatal detection rate at tertiary-care centers. Approximately one-third of infants whose prenatal echocardiogram raised concern about aortic arch obstruction did not have the diagnosis confirmed after birth, so that some echocardiograms in healthy neonates were prompted by the fetal study.

Although we do not have data on how many of the infants born at other medical centers had fetal echocardiography, prenatal ultrasound is performed in the vast majority of pregnancies in our region. The detection of congenital heart disease by prenatal ultrasonography can be limited by fetal gestational age or position, maternal obesity, and operator experience. Lesions involving ventricular outflow tracts (transposition of the great arteries, tetralogy of Fallot, double outlet right ventricle), total anomalous pulmonary venous connection, and left-sided obstructive lesions other than hypoplastic left heart syndrome (coarctation, interrupted aortic arch) have lower detection rates.

Use of a fetal ultrasound protocol that includes multiple echocardiographic views, especially of the ventricular outflow tracts and aorta, improves detection.

Pulse oximetry screening is complementary to other modes of detection of CCHD and does not replace the utility of prenatal screening, physical examination, and continued observation. The importance of multiple diagnostic approaches is demonstrated in the 1 neonate in our study who had a false-negative initial pulse oximetry screening, developed new physical examination findings of murmur and cyanosis, and was subsequently diagnosed to have interrupted aortic arch. False-negative pulse oximetry screenings for CCHD are uncommon and have been mainly reported in patients with coarctation, some of which may not be duct dependent. False-negative tests also have been reported in patients with interrupted aortic arch, transposition of the great arteries, total anomalous pulmonary venous connection, truncus arteriosus, and double-outlet right ventricle.

Our study confirmed a low false-positive rate associated with pulse oximetry screening performed after 24 hours of age. As noted in previous studies, a false-positive result often has clinical utility in detecting noncardiac causes of cyanosis that require treatment, including sepsis, pulmonary hypertension, pneumothorax, transient tachypnea of the newborn, meconium aspiration, and seizures secondary to intraventricular hemorrhage. In our study, 1 echocardiogram was performed because of the false-positive results and identified a patient with pulmonary hypertension. Analysis of results of previous studies when pulse oximetry was performed after 24 hours of age has estimated that 1 additional echocardiogram is obtained per 3000 births.

We compared the characteristics of infants who failed with those who passed the initial screening and found that larger infants were more likely to fail. Given the small number of failures in our study, we were unable to determine whether this finding is truly related to infant weight or the result of other characteristics of the pregnancy or perinatal period. Whatever the underlying reason, even among large infants, the proportion who failed the initial screening was very low.

Our study has some potential limitations. The false-negative rate of screening may have been underestimated. We did not review records from the regional Medical Examiner’s Offices for out-of-hospital infant death secondary to undiagnosed CCHD. In addition, some infants born at BWH may have been admitted and treated for CCHD at other medical centers. However, in our region, with few exceptions, infants with CCHD are referred to and have surgical procedures at BCH. The prenatal detection of CCHD at other medical centers was underestimated, because some women transferred their care to BWH after CCHD was diagnosed at the facility where they initiated care.

CONCLUSIONS

The pulse oximetry protocol was effective in screening nearly all infants in the well-newborn nursery with a low false-positive and false-negative rate. Some patients with a failed initial screening had a noncardiac, clinically significant cause for desaturation. Neonatal pulse oximetry screening did not detect a single infant with CCHD in a 12-month period at a high-volume tertiary-care birthing center because of effective prenatal diagnosis by fetal echocardiography. Only 35% of patients with CCHD born at hospitals other than BWH and transferred to BCH for subsequent care had a prenatal diagnosis and some of these infants presented with severe symptoms after late postnatal detection. Neonatal pulse oximetry screening will identify more infants with CCHD in regions with a lower prenatal diagnosis rate. Improving patient access to and training of staff
in thorough fetal echocardiography protocols during routine fetal surveys also should improve timely diagnosis of CCHD. Finally, approximately one-quarter of patients with CCHD had aortic arch obstruction (coarctation or interrupted aortic arch) and consideration should be made for adding these diagnoses to the list of target lesions to be screened.

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REFERENCES


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