Role of Pulse Oximetry in Examining Newborns for Congenital Heart Disease

A Scientific Statement From the American Heart Association and American Academy of Pediatrics

William T. Mahle, MD, FAHA, FAAP, Chair; Jane W. Newburger, MD, MPH, FAHA, FAAP; G. Paul Matherne, MD, FAHA, FAAP; Frank C. Smith, MD; Tracey R. Hoke, MD, FAAP; Robert Koppel, MD, FAAP; Samuel S. Gidding, MD, FAHA, FAAP; Robert H. Beekman III, MD, FAHA, FAAP; Scott D. Grosse, PhD; on behalf of the American Heart Association Congenital Heart Defects Committee of the Council on Cardiovascular Disease in the Young, Council on Cardiovascular Nursing, and Interdisciplinary Council on Quality of Care and Outcomes Research; and the American Academy of Pediatrics Section on Cardiology and Cardiac Surgery, and Committee on Fetus and Newborn

Background—The purpose of this statement is to address the state of evidence on the routine use of pulse oximetry in newborns to detect critical congenital heart disease (CCHD).

Methods and Results—A writing group appointed by the American Heart Association and the American Academy of Pediatrics reviewed the available literature addressing current detection methods for CCHD, burden of missed and/or delayed diagnosis of CCHD, rationale of oximetry screening, and clinical studies of oximetry in otherwise asymptomatic newborns. MEDLINE database searches from 1966 to 2008 were done for English-language papers using the following search terms: congenital heart disease, pulse oximetry, physical examination, murmur, echocardiography, fetal echocardiography, and newborn screening. The reference lists of identified papers were also searched. Published abstracts from major pediatric scientific meetings in 2006 to 2008 were also reviewed. The American Heart Association classification of recommendations and levels of evidence for practice guidelines were used. In an analysis of pooled studies of oximetry assessment performed after 24 hours of life, the estimated sensitivity for detecting CCHD was 69.6%, and the positive predictive value was 47.0%; however, sensitivity varied dramatically among studies from 0% to 100%. False-positive screens that required further evaluation occurred in only 0.035% of infants screened after 24 hours.

Conclusions—Currently, CCHD is not detected in some newborns until after their hospital discharge, which results in significant morbidity and occasional mortality. Furthermore, routine pulse oximetry performed on asymptomatic newborns to detect critical congenital heart disease (CCHD).

The American Heart Association requests that this document be cited as follows: Mahle WT, Newburger JW, Matherne GP, Smith FC, Hoke TR, Koppel R, Gidding SS, Beekman RH 3rd, Grosse SD; on behalf of the American Heart Association Congenital Heart Defects Committee of the Council on Cardiovascular Disease in the Young, Council on Cardiovascular Nursing, and Interdisciplinary Council on Quality of Care and Outcomes Research; and the American Academy of Pediatrics Section on Cardiology and Cardiac Surgery, and Committee on Fetus and Newborn. The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention.

The American Heart Association and the American Academy of Pediatrics make every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on November 14, 2008, and by the American Academy of Pediatrics on March 25, 2009.

This article has been copublished in Circulation.


This article was copublished in Pediatrics.

Permissions: Multiple copies, modification, alteration, enhancement, and/or distribution of this document are not permitted without the express permission of the American Heart Association. Instructions for obtaining permission are located at http://www.americanheart.org/presenter.jhtml?identifier=4431. A link to the “Permission Request Form” appears on the right side of the page.


Circulation is available at http://circ.ahajournals.org

DOI: 10.1161/CIRCULATIONAHA.109.192576
newborns after 24 hours of life, but before hospital discharge, may detect CCHD. Routine pulse oximetry performed after 24 hours in hospitals that have on-site pediatric cardiovascular services incurs very low cost and risk of harm. Future studies in larger populations and across a broad range of newborn delivery systems are needed to determine whether this practice should become standard of care in the routine assessment of the neonate. (Circulation. 2009;120:0000-0000.)

Key Words: AHA Scientific Statements | diagnostic techniques and procedures | outcomes research | health policy | pulse oximetry | heart defects, congenital | tests

Congenital heart disease occurs in 9 of every 1000 livebirths.1 Approximately one quarter of these children will have critical congenital heart disease (CCHD), which by definition requires surgery or catheter intervention in the first year of life.2 Congenital malformations are one of the leading causes of infant death in the United States and other developed nations, and CCHD is responsible for more deaths than any other type of malformation.3,4 Most newborns with CCHD can be diagnosed by echocardiography, palliated with prostaglandin infusion, and treated with surgery or transcatheter interventions. In the current era, congenital heart surgery allows for repair or palliation of nearly all types of congenital heart malformations. Congenital heart surgery, together with transcatheter interventions, has resulted in a marked improvement in survival for those with CCHD.5 Intervention is typically performed in the first weeks of life to optimize hemodynamics and prevent end-organ injury associated with delayed diagnosis. Because timely recognition of CCHD could improve outcomes, it is important to identify and evaluate strategies to enhance early detection. Pulse oximetry has been proposed as one such strategy, and legislation has been proposed to support this practice.6

The present statement reviewed the existing data to evaluate the potential role of pulse oximetry in examining newborns for CCHD. A writing group was appointed by the American Heart Association (AHA) and the American Academy of Pediatrics to evaluate the available literature addressing current detection methods for CCHD, burden of missed and/or delayed diagnosis of CCHD, rationale of oximetry screening, and clinical studies of oximetry in otherwise asymptomatic newborns. Comprehensive searches of the MEDLINE database from 1966 to 2008 were done for English-language publications in scientific journals using the following search terms: congenital heart disease, pulse oximetry, physical examination, murmur, echocardiography, fetal echocardiography, and newborn screening. The reference lists of identified papers were also searched. Published abstracts from major pediatric scientific meetings in 2006 to 2008 were also reviewed. The AHA classification of recommendations and levels of evidence for practice guidelines were used. The classification of recommendations and levels of evidence are shown in Table 1.

| Prevalence and Scope of the Problem |

Currently, children with CCHD are diagnosed by a variety of mechanisms. Neonates with CCHD may be diagnosed in the newborn nursery on the basis of physical examination findings, such as heart murmurs, tachypnea, or overt cyanosis. These findings are not always evident before hospital discharge, which may occur before 48 hours of life. A recent study from the United Kingdom suggested that 25% of infants with CCHD were not diagnosed with heart disease until after discharge from the newborn nursery.7 The median age of diagnosis in these cases was 6 weeks. A recent publication from the United States suggested that delayed or missed diagnosis occurs in 7 per 100 000 livebirths.8 However, because these data are derived from a birth defect surveillance program with passive and thus incomplete case ascertainment, this calculation most likely represents a minimum estimate.

Newborns with CCHD are susceptible to profound, sudden worsening in clinical status in the first days and weeks of life. These acute physiological changes correspond to changes in pulmonary vascular resistance and closure of the ductus arteriosus. In neonates with CCHD, the ductus arteriosus is often essential for maintaining either pulmonary or systemic blood flow. These CCHD defects are considered ductus arteriosus–dependent lesions (Table 2). The newborn hospitalization provides a critical window for caregivers to identify CCHD lesions in order to avoid hemodynamic embarrassment. The timing of constriction or closure of the ductus arteriosus also explains why children with CCHD may be particularly vulnerable to cardiovascular collapse soon after discharge from the newborn nursery.

| Table 1. Classification of Recommendations and Level of Evidence |

| Classification of recommendations |

| Class I: Conditions for which there is evidence and/or general agreement that a given procedure or treatment is beneficial, useful, and effective and should be performed. Benefit >> risk. |

| Class II: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment. |

| Class IIa: Weight of evidence/opinion is in favor of usefulness/efficacy. It is reasonable to perform procedure/administer treatment. Benefit >> risk. Additional studies with focused objectives needed. |

| Class IIb: Usefulness/efficacy is less well established by evidence/opinion. Procedure/treatment may be considered. Benefit = risk. Additional studies with broad objectives needed; additional registry data would be helpful. |

| Class III: Conditions for which there is evidence and/or general agreement that a procedure/treatment is not useful/effective and in some cases may be harmful. Risk = benefit. No additional studies needed. Procedure/treatment should not be performed/administered because it is not helpful and may be harmful. |

| Level of evidence |

| A: Data derived from multiple randomized clinical trials or meta-analyses |

| B: Data derived from a single randomized trial or nonrandomized studies |

| C: Only consensus opinion of experts, case studies, or standard of care |

Downloaded from http://circ.ahajournals.org/ by guest on April 16, 2017
Morbidity and Sequelae

With the advent of prostaglandin therapy for ductus arteriosus–dependent lesions, many previously lethal congenital heart conditions that present with severe hypoxemia, shock, and acidosis in the newborn period are now survivable. The severity of organ damage is a function of the extent of insult, differential flow to organs as the neonatal circulation responds to the hypoxic/ischemic insult, and the oxygen requirement of each organ.

Among sequelae of neonatal hemodynamic compromise, the most important long-term effects relate to the consequences of brain injury from ischemia and reperfusion, because the brain has the highest oxygen requirement of any organ. Cerebrovascular pressure autoregulation and reactivity to CO2 are affected by hypoxic/ischemic injury, which renders the brain particularly vulnerable to hypotension and decreased cardiac output.9 Such hemodynamic instability is prevalent among neonates with CCHD who present with shock. Furthermore, preoperative events may interact with genetic mutations and both intraoperative and postoperative factors in determining later neurodevelopmental outcome.10

Using brain magnetic resonance imaging, a number of investigators have demonstrated acute brain injury in the newborn with CCHD before surgical intervention. Periventricular leukomalacia, which occurs secondary to vulnerability of the immature oligodendrocyte to hypoxia/ischemia, free radical attack, and excitotoxicity, and likely circulating cytokines, has been found on magnetic resonance imaging in up to 39% of neonates with CCHD.11–14

Children with CCHD are reported to experience more frequent impairments in motor function, speech and language, visual-motor-perceptual function, and executive function, as well as increased use of special services.10,15–22 The greatest frequency of adverse outcomes is found among those with a single ventricle with obstruction to systemic outflow, such as hypoplastic left heart syndrome.23 In this lesion, systemic perfusion occurs through the patent ductus arteriosus, and ductus closure results in shock and end-organ damage. Prenatal diagnosis of hypoplastic left heart syndrome has been reported in certain studies to reduce early neurological morbidity, with fewer adverse perioperative neurological events such as coma,24 although earlier age at surgery has not been shown to result in better long-term neurodevelopmental outcomes.23 One could infer that because delayed diagnosis is associated with damage to various end organs, it might also lead to hypoxic/ischemic brain injury; however, further studies are needed to demonstrate a true causal relationship.

Death Due to Delayed Diagnosis

A number of children with CCHD are so severely compromised at presentation that they die before surgical intervention. For example, investigators have reported that between 3% and 6% of neonates with dextro-transposition of the great arteries died because of hemodynamic compromise before surgical intervention could be offered.25,26 In a study from the Baltimore-Washington metropolitan area in the 1980s, Kuehl and colleagues27 reported that among 4360 children with any form of congenital heart disease, 76 (1.7%) died before the identification of heart disease. Delayed or missed diagnosis of CCHD accounted for 1.4 deaths per 10 000 livebirths in that series. In 1994, Abi-Habib and colleagues28 reported that 25% of CCHD lesions were not diagnosed until after hospital discharge, even in the most recent era. The data from these United Kingdom studies suggested that delayed or missed diagnosis of CCHD in infancy over a 6-year period in a region of northern England. Fifty-six of 185 children died in infancy, and 27 (48%) of these deaths resulted from sequelae of undetected CCHD. The great majority of these subjects had CCHD lesions that might have manifested hypoxemia. In another study from the United Kingdom, Wren and colleagues29 reported that among 4360 children with any form of congenital heart disease, 76 (1.7%) died before the identification of heart disease. Delayed or missed diagnosis of CCHD accounted for 1.4 deaths per 10 000 livebirths in that series. In 1994, Abi-Habib and colleagues28 reported that 25% of CCHD lesions were not diagnosed until after hospital discharge, even in the most recent era. The data from these United Kingdom studies suggested that delayed or missed diagnosis of CCHD accounted for 0.4 to 2.0 deaths per 10 000 livebirths.

With the increased use of prenatal ultrasound and a better understanding of the presentation of CCHD in the past decade, the risk of death before diagnosis has undoubtedly declined, although it is still likely to be important.30 Two recent studies have reported that the rate of mortality due to delayed diagnosis of CCHD is an order of magnitude lower than in the older studies discussed in the previous paragraph. First, a presentation from a study from metropolitan Atlanta, Ga, that used a population-based surveillance system reported

---

Table 2. CCHD Lesions and Associated Clinical Characteristics

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Prevalence*</th>
<th>Hypoxemia</th>
<th>Ductus Arteriosus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outflow tract defects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>6.1</td>
<td>Most</td>
<td>Uncommon</td>
</tr>
<tr>
<td>D-transposition of the great arteries</td>
<td>4.0</td>
<td>All</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Double-outlet right ventricle</td>
<td>1.7</td>
<td>Some</td>
<td>Some</td>
</tr>
<tr>
<td>Truncus arteriosus</td>
<td>1.0</td>
<td>All</td>
<td>None</td>
</tr>
<tr>
<td>TAPVC</td>
<td>1.2</td>
<td>All</td>
<td>None</td>
</tr>
<tr>
<td>Ebstein anomaly</td>
<td>0.6</td>
<td>Some</td>
<td>Some</td>
</tr>
<tr>
<td>Right obstructive defects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tricuspid atresia</td>
<td>0.5</td>
<td>All</td>
<td>Some</td>
</tr>
<tr>
<td>Pulmonary atresia, intact septum</td>
<td>0.8</td>
<td>All</td>
<td>All</td>
</tr>
<tr>
<td>Pulmonic stenosis, atresia</td>
<td>6.3</td>
<td>Some</td>
<td>Some</td>
</tr>
<tr>
<td>Left obstructive defects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypoplastic left heart</td>
<td>3.3</td>
<td>All</td>
<td>All</td>
</tr>
<tr>
<td>Coarctation of the aorta</td>
<td>4.7</td>
<td>Some</td>
<td>Some</td>
</tr>
<tr>
<td>Aortic arch atresia or hypoplasia</td>
<td>1.0</td>
<td>Some</td>
<td>All</td>
</tr>
<tr>
<td>Aortic valve stenosis (critical)</td>
<td>1.6</td>
<td>Uncommon</td>
<td>Some</td>
</tr>
<tr>
<td>Other major heart defects</td>
<td>12.4</td>
<td>Some</td>
<td>Some</td>
</tr>
</tbody>
</table>

TAPVC indicates total anomalous pulmonary venous connection.

*Per 10 000 livebirths. Data are derived from the Metropolitan Atlanta Congenital Defects Program.1
that death due to delayed diagnosis of CCHD occurred in 1.0
of every 100 000 livebirths and may be decreasing with
time, although this estimate could be understated, because
in that study, only deaths that occurred before arrival at a
hospital or before the child could be stabilized were attributed
to delayed diagnosis. Another preliminary study from Cali-
ifornia reported 2.0 deaths per 100 000 livebirths related to
delayed diagnosis of CCHD. Presumably, earlier recogni-
tion of CCHD in these patients could have prevented death in
at least some of these cases.

Impairment in cardiovascular function from delayed diagnosis
can also adversely impact survival during neonatal cardio-
vascular surgery and recovery. Certain studies that compared
outcomes in prenatal and postnatal diagnosis of CCHD have
reported better short-term results for those who were diagnosed
prenatally. However, numerous other studies have failed to
document any survival benefit of prenatal diagnosis among
infants undergoing congenital heart surgery.

In summary, delayed or missed diagnosis is associated with
significant morbidity, the most significant being hypoxic/ische-
mic brain injury. In addition, delayed diagnosis appears to lead
directly and indirectly to higher mortality in this population,
although the number of deaths that might be prevented through
pulse oximetry screening remains to be determined. Methods to
improve early detection of CCHD appear warranted.

**Customary Practice**

Children with CCHD are identified in a variety of ways. Since the late 1980s, prenatal ultrasound has been used to screen for congenital anomalies. An anatomic ultrasound is typically performed at 18 to 20 weeks’ gestation. During this process many, but not all, cases of CCHD can be identified by a methodical scan. When CCHD is identified by this approach, the patient is often referred to a pediatric cardiologist for confirmatory imaging and counsel-
ing. With knowledge that the fetus has CCHD, the newborn can be delivered in a hospital capable of providing intensive care, including prostaglandin, as well as mechanical ventilation. The newborn can be stabilized and transferred to a congenital heart center.

Prenatal ultrasound, performed by those with specific training in congenital heart disease, can identify a variety of CCHD lesions; however, numerous studies have reported that even when fetal ultrasound is routinely performed during pregnancy, fewer than 50% of cases of CCHD are identified. Most of the published literature comes from European countries, which tend to have more centralized healthcare systems and uniform practices vis-à-vis prenatal ultrasound. As such, these systems may represent the best-case scenario for population prenatal ultrasound screening. In the United States, many congenital surgery referral centers have reported prenatal detection rates > 50% for functional single-ventricle lesions, although the detection rate is generally < 30% for CCHD lesions with 2-ventricle circulation. These studies from referral centers may be biased toward higher detection rates, and population-based data on prenatal detection of CCHD in the United States are sparse.

There are several factors that might account for the relatively low prenatal CCHD detection rate. The quality of anatomic ultrasounds varies considerably. A number of medical professionals, including radiologists, perinatologists, and general obstetricians with varying degrees of training, as well as technicians, perform these ultrasounds. In addition to concerns about the quality, there may be limited access to prenatal ultrasound. In the United States, an anatomic ultrasound is not performed in all women. The availability of anatomic ultrasound is likely to be particularly limited in certain racial/ethnic or low-socioeconomic-status groups. Therefore, although prenatal ultrasound plays an important part in the timely identification of CCHD, population-based data demonstrate that this methodology by itself is insufficient to identify a high proportion of cases.

After birth, screening for congenital heart disease by primary care providers is currently accomplished by physical examina-
tion within the first 24 hours of life and on subsequent nursery
visits. Supplemental tests, including electrocardiograms, pulse
oximetry, and chest radiographs, are often obtained in suspicious
cases. Echocardiograms can be done either with or without pediatric cardiology consultation. This strategy blends diagnosis-
tic assessment approaches from the 1950s to 1970s with the
increasing availability of echocardiography. It results in substan-
tial case identification but is regarded as inefficient and costly
and misses a significant number of newborns with CCHD.

Skilled physical examination, a sensitive and specific screening
tool in older children, does not always distinguish between
neonates with and without congenital heart disease. Hypox-
emia is difficult to detect in newborns, and the transitional
circulation masks important clinical findings such as absent
femoral pulses while the ductus arteriosus remains patent. Reports of the late detection of coarctation of the aorta have been
published since the 1960s. Perhaps most importantly, physical examination skills are on the decline in current trainees.

Heart murmurs have a prevalence of between 0.6% and 4.2% in newborns and are mistakenly considered a hallmark of heart
disease. They often do not accompany critical heart defects,
particularly those with valve atresia and transposition. Flow
murmurs of the transitional circulation, transient tricuspid regur-
gitation, and small ventricular septal defects are common and of
no clinical importance in newborns. Conversely, murmurs of
many important complex heart defects, such as tricuspid atresia
with ventricular septal defect, double-outlet right ventricle,
and total anomalous pulmonary venous return, emerge only after the
decline in pulmonary resistance and after neonatal discharge and
are often heard but not considered pathological. Practicing
pediatricians currently have limited experience in discriminating
innocent from pathological murmurs. In a contemporary series
in which echocardiography was performed to evaluate for
possible heart disease based on suspicious physical examination,
fewer than 15% of subjects were found to have significant
congenital heart disease.

Clinical experience and epidemiological observations
suggest that although physical examination, electrocardiogram,
and chest radiograph are useful in identifying many
cases of serious congenital heart disease postnatally, they
do not have sufficient sensitivity and specificity to detect
all cases. Echocardiography, although an essential diag-
nontic tool, has serious limitations as a universal screening tool, particularly its cost.\(^6\) When used as a screening tool, echocardiography has a high frequency of either false-positive results (usually related to the transitional circulation) or recognition of clinically benign diagnoses (eg, small muscular ventricular septal defects). In addition, there may be an inadequate supply of trained personnel who could perform this screening with a reasonable degree of accuracy. Therefore, there is considerable interest in improving the detection of CCHD with novel diagnostic techniques.

**Pulse Oximetry and Detection of CCHD**

A common feature of many forms of congenital heart disease is hypoxemia. Hypoxemia results from the mixing of systemic and venous circulations or parallel circulations as one might see in dextro-transposition of the great arteries. Hypoxemia may result in obvious cyanosis. However, generally, 4 to 5 g of deoxygenated hemoglobin is needed to produce visible central cyanosis, independent of skin pigmentation.\(^5\) For the typical newborn with a hemoglobin concentration of 20 g/dL, cyanosis will only be visible when arterial oxygen saturation is <80%; if the infant only has a hemoglobin concentration of 10 g/dL, the saturation must be <60% before cyanosis is apparent.\(^5\) Importantly, those children with mild hypoxemia, with arterial oxygen saturation of 80% to 95%, will not have visible cyanosis. Moreover, the identification of cyanosis is particularly problematic in black and Hispanic neonates because of skin pigmentation.\(^5\)

The majority of CCHD lesions present with some degree of hypoxemia in the newborn period. Table 2 demonstrates the frequency of the most common forms of CCHD based on data from the Metropolitan Atlanta Congenital Birth Defects Surveillance Program\(^5\) and the likelihood of having some degree of hypoxemia in the newborn period. To improve timely detection of CCHD, a number of investigators have proposed that pulse oximetry be considered as a complementary modality to the newborn physical examination.\(^5\,\^9\,\^0\,\^1\)

Pulse oximetry was developed in the early 1970s based on the different absorption spectra between oxygenated and deoxygenated hemoglobin.\(^5\) Deoxygenated hemoglobin absorbs light in the red band (600 to 750 nm), whereas oxygenated hemoglobin absorbs light in the infrared band (850 to 1000 nm). The ratio of light absorbance at these 2 wavelengths correlates with the saturation of hemoglobin in the capillaries.\(^6\) Pulse oximetry has the potential to identify hypoxemia that might not otherwise produce visible cyanosis, especially among darkly pigmented newborns.

Pulse oximetry is used routinely in the assessment of young children in neonatal intensive care units and emergency departments and has been proposed as an adjunct to the assessment of the newborn in the delivery room.\(^5\) As such, some have proposed that pulse oximetry be considered as a vital sign equivalent in importance to pulse, respirations, and blood pressure.\(^6\,\^4\) Contemporary use of pulse oximetry has thus already contributed to heightened recognition of congenital heart disease in neonates.

**Clinical Studies of Oximetry Screening**

Pulse oximetry has gained wide acceptance as a noninvasive method to determine oxygen saturation (\(\text{SpO}_2\)). The method does not require calibration and is able to provide instantaneous data that correlate well with blood gas measurements. O’Brien and colleagues\(^6\,\^5\) have defined reference data for oxygen saturation in healthy full-term infants during their first 24 hours of life. The median value at 20 to 24 hours of life (97.8%) is similar to the results for healthy full-term infants between 2 and 7 days of age (97.6%).\(^6\) Other investigators have reported similar results.\(^6\,\^7\) Beginning in the 1990s, investigators began to explore the possible role of neonatal oximetry in identifying CCHD that might otherwise go undetected. Initially, investigators demonstrated that in neonates with known CCHD, pulse oximetry measurements were significantly lower than in age-matched control subjects. Using a cutoff of 95% in lower-extremity saturation, Hoke and colleagues\(^5\) suggested that 81% of neonates with CCHD could be identified. Given this association, the question arose as to whether oximetry can successfully identify CCHD in a population of newborns not otherwise suspected of having heart disease. To date, several published studies\(^6,\^5,\^9,\^0,\^4,\^6,\^7\) have used newborn oximetry to screen for CCHD (Table 3). Most studies were relatively small, and screening protocols differed with respect to both age at screening and cutoff levels for an abnormal screen. Nonetheless, the cumulative experience of these investigations provides a framework for evaluation of the test characteristics of newborn oximetry screening. The results of these studies and differences in study protocols are described below.

Because newborns with CCHD may have clinical deterioration in the first 48 hours of life, one would ideally use oximetry screening soon after delivery. However, arterial oxygen saturation varies considerably in the first 24 hours, with many healthy newborns having arterial saturations of less than 95%. As such, oximetry screening before 24 hours of life can result in a significant number of false-positive results. A study from the United Kingdom reported that the false-positive rate was as high as 5% when oximetry screening was performed in the first 24 hours compared with 1% at the time of hospital discharge.\(^76\) Therefore, to achieve an acceptable specificity, testing >24 hours after birth would appear to be the most reasonable strategy. This screening strategy assumes that the majority of newborns will not be discharged on the first day of life. With early discharge at less than 24 hours of age, many infants would not be screened.

The establishment of a cutoff threshold for an abnormal \(\text{SpO}_2\) is important. Other factors being constant, a higher threshold will increase sensitivity and at the same time decrease specificity. Setting the \(\text{SpO}_2\) cutoff value closer to the normal level will decrease the number of false-negative screening results at the cost of increasing the number of false-positive screening results. Conversely, a lower \(\text{SpO}_2\) threshold will lower sensitivity and raise specificity. Although a number of \(\text{SpO}_2\) thresholds have been proposed,
Many investigators believe that an \( \text{SpO}_2 \) of \( \geq 95\% \) is appropriate. In studies of healthy populations, the distribution of \( \text{SpO}_2 \) measured in a lower extremity at 24 hours was reported to be 97.3%\( \pm \)1.3%.68 One study suggested that \( \text{SpO}_2 \) to be 97.3%\( \pm \)1.2% at 24 h.51 The \( \text{SpO}_2 \) measurements in healthy newborns were lower in the lower extremity than in the upper extremity in the majority of studies,69 although some investigators have reported the normal \( \text{SpO}_2 \) values for different locations to be similar. In general, the mean difference between \( \text{SpO}_2 \) measurements in healthy newborns was shown to be less than 3% or 4% in some studies, although in most studies it was less than 1%.51,66,69

### Table 3. Results of Studies Examining Oximetry Screening for CCHD

<table>
<thead>
<tr>
<th>Study’s First Author</th>
<th>n</th>
<th>Age at Screening, h</th>
<th>Probe Location</th>
<th>Cutoff for Normal</th>
<th>FP</th>
<th>TP</th>
<th>FN</th>
<th>TN</th>
<th>PPV, %</th>
<th>NPV, %</th>
<th>Sensitivity, %</th>
<th>Specificity, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hoke(^{23})</td>
<td>2876</td>
<td>&lt;24</td>
<td>H+F</td>
<td>( \geq 95%)</td>
<td>53</td>
<td>1.84</td>
<td>4</td>
<td>0</td>
<td>2819</td>
<td>7.0</td>
<td>98.1</td>
<td>100.0</td>
</tr>
<tr>
<td>Richmond(^{21})</td>
<td>5626</td>
<td>11.7</td>
<td>F</td>
<td>( \geq 95%)</td>
<td>51</td>
<td>0.91</td>
<td>9</td>
<td>4</td>
<td>5621</td>
<td>15.0</td>
<td>99.9</td>
<td>69.2</td>
</tr>
<tr>
<td>Koppel(^{60})</td>
<td>11 281</td>
<td>72</td>
<td>F</td>
<td>( \geq 95%)</td>
<td>1</td>
<td>0.01</td>
<td>3</td>
<td>2</td>
<td>11 275</td>
<td>75.0</td>
<td>99.98</td>
<td>60.0</td>
</tr>
<tr>
<td>Reich(^{65})</td>
<td>2114</td>
<td>&gt;24</td>
<td>H+F</td>
<td>( \geq 95%)</td>
<td>2</td>
<td>0.09</td>
<td>1</td>
<td>1</td>
<td>2110</td>
<td>33.3</td>
<td>99.95</td>
<td>50.0</td>
</tr>
<tr>
<td>Bakr(^{72})</td>
<td>5211</td>
<td>31.7</td>
<td>H+F</td>
<td>( \geq 94%)</td>
<td>1</td>
<td>0.02</td>
<td>3</td>
<td>2</td>
<td>5211</td>
<td>75.0</td>
<td>99.9</td>
<td>60.0</td>
</tr>
<tr>
<td>Rosati(^{73})</td>
<td>5292</td>
<td>72</td>
<td>F</td>
<td>( \geq 96%)</td>
<td>1</td>
<td>0.02</td>
<td>2</td>
<td>1</td>
<td>5288</td>
<td>66.7</td>
<td>100</td>
<td>66.7</td>
</tr>
<tr>
<td>Arletta(^{69})</td>
<td>3262</td>
<td>8</td>
<td>F</td>
<td>( \geq 95%)</td>
<td>7</td>
<td>0.21</td>
<td>17</td>
<td>3</td>
<td>3235</td>
<td>70.8</td>
<td>99.9</td>
<td>85.0</td>
</tr>
<tr>
<td>Kawalec(^{70})</td>
<td>27 200</td>
<td>26</td>
<td>F</td>
<td>( \geq 95%)</td>
<td>13</td>
<td>0.05</td>
<td>7</td>
<td>1</td>
<td>27 179</td>
<td>35.0</td>
<td>99.9</td>
<td>87.5</td>
</tr>
<tr>
<td>Meberg(^{54})</td>
<td>50 008</td>
<td>6</td>
<td>F</td>
<td>( \geq 95%)</td>
<td>324</td>
<td>0.65</td>
<td>43</td>
<td>NA</td>
<td>NA</td>
<td>11.7</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Sendelbach(^{58})</td>
<td>10 976</td>
<td>4</td>
<td>F</td>
<td>( \geq 96%)</td>
<td>636</td>
<td>4.5</td>
<td>0</td>
<td>1</td>
<td>10 340</td>
<td>99.9</td>
<td>0</td>
<td>95.5</td>
</tr>
<tr>
<td>All studies</td>
<td>123 846</td>
<td></td>
<td></td>
<td></td>
<td>1089</td>
<td>0.87</td>
<td>89</td>
<td>15</td>
<td>122 762</td>
<td>16.4</td>
<td>99.9</td>
<td>75*</td>
</tr>
<tr>
<td>Studies &gt;24 h</td>
<td>51 098</td>
<td></td>
<td></td>
<td></td>
<td>18</td>
<td>0.035</td>
<td>16</td>
<td>7</td>
<td>51 063</td>
<td>47.0</td>
<td>99.9</td>
<td>69.6</td>
</tr>
</tbody>
</table>

*Excludes study by Meberg et al\(^{54}\) because false-negative data were not included.

FP indicates false-positive; TP, total positive; FN, false-negative; TN, total negative; PPV, positive predictive value; NPV, negative predictive value; H+F, hand and foot; F, foot; and NA, not available.

The results of published studies using oximetry screening to detect CCHD in a representative birth population are shown in Table 3. Ten studies with a total of 123 846 infants screened reported a mean of 0.87% of infants with false-positive screens but a false-positive rate of 0.035% when screening was done after 24 hours; however, there was remarkable dispersion in reported screening performance. Five studies reported a low false-positive rate (\( \leq 0.1\% \)) when measurements were made after 24 hours of life. The low false-positive rate is somewhat surprising given the reported variation of \( \text{SpO}_2 \) reported in normal newborn populations. It is not known whether there might be a publication bias in that only studies with favorable specificity might be published. A low false-positive rate would reduce the number of unnecessary echocardiograms. Nine of 10 studies listed in Table 3 reported sensitivity of \( \leq 90\% \), ranging from 0% to 87%. This is explained in part by the fact that hypoxemia is not present in some forms of CCHD (Table 2).

False-positive results can be a cause for concern in public health newborn screening programs that are based on the laboratory analysis of dried blood spot specimens collected.
on filter paper cards. These false-positive results typically require families to be notified to bring their child in for further testing, and there can be a delay of several days before the results of such testing become available. False-positive newborn screening results have been reported to sometimes result in lasting parental anxiety and possibly elevated use of healthcare services. In the case of pulse oximetry, this type of psychosocial risk of harm is very unlikely to be a problem in the typical hospital setting for infants not subject to early discharge. A positive test result leads to an immediate referral for an echocardiogram, and the results are reported before discharge. However, when the birth center does not have ready access to cardiac consultation, delay in hospital discharge or transfer to another facility may result in anxiety and added stress.

Oximetry screening may be less effective at identifying some CCHD lesions at greatest risk for acute cardiovascular compromise, namely, obstructive left heart lesions. A published analysis of oximetry has suggested that the difficulty in detecting hypoplastic left heart syndrome, aortic stenosis, and coarctation of the aorta limits the usefulness of this screening tool. However, it should be noted that nearly all forms of CCHD—even those unrelated to left heart obstruction—can result in serious morbidity and even death when diagnosis is delayed. Moreover, oximetry can detect a significant number of newborns with obstructive left heart lesions and right-to-left shunting at the ductus arteriosus (Table 4). In published series, hypoplastic left heart syndrome was detected in all cases, and coarctation of the aorta was detected in just over half of the studies. That studies have obtained SpO2 measurements on newborns with known CCHD have similarly reported that a lower-extremity SpO2 of \( \leq 95\% \) detected hypoplastic left heart syndrome in all cases and critical coarctation of the aorta in the majority of cases.

Several studies of screening oximetry have reported incidental findings of persistent fetal circulation, defined as elevated pulmonary vascular resistance and right-to-left shunting at the ductus arteriosus. In some reports, these cases have been reported as false-negative findings. In other studies, the investigators have emphasized the benefits of identifying these patients. The finding of persistent fetal circulation in otherwise healthy newborns may be of benefit to medical care. An understanding of the outcome of newborns who are asymptomatic with a decreased lower-extremity SpO2 will be needed to understand whether identification of this population is a true benefit of oximetry screening.

### Limitations and Challenges to Newborn Pulse Oximetry in Detection of CCHD

There are technical limitations to oximetry measurement in the newborn. As noted above, the mean SpO2 in the newborn at \( >24 \) hours of age is 97% to 98%; however, when continuous pulse oximetry is used, multiple investigators have demonstrated periodic and/or sustained desaturation below 95% during sleep, feeding, and crying. Sustained rather than variable hypoxemia is consistent with the diagnosis of cyanotic congenital heart disease. Low oximetry readings in the setting of normal arterial oxygen saturation have been reported by multiple investigators. In fact, falsely low oximetry readings in the newborn population are known to be associated with low peripheral perfusion and motion artifact, probe placement site and partial probe detachment, and hyperbilirubinemia or dysmoglobinemia. It is known that technical differences between the various types of oximeters in general use include measurement of functional or fractional oxygen saturation, preset signal-averaging times, and methods for the exclusion of motion artifact. There has been some research into the variability among various commercially available pulse oximeters; however, most of the variability occurs in the cyanotic range (<90%) and at the highest saturations (99% to 100%). The peak performance of the commercially available oximeters occurs in the range of 89% to 97%. Therefore, in the critical range for oximetry screening (94% to 97%), the variability of the most commonly used oximeters should be negligible.

### Table 4. Detection of CCHD Lesions From Screening Studies, Assuming a Positive Screen as SpO2 ≤95%

<table>
<thead>
<tr>
<th></th>
<th>Kao²</th>
<th>Hoke⁵</th>
<th>Richmond⁷¹</th>
<th>Koppel⁶⁰</th>
<th>Reich⁶⁵</th>
<th>Bakr⁷²</th>
<th>Rosati⁷¹</th>
<th>Arlettaz⁶⁹</th>
<th>Kawalec⁷⁰</th>
<th>Total</th>
<th>Percent</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>DORV</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0/1</td>
<td>0/1</td>
<td>0</td>
<td>0/1</td>
<td>1/1</td>
</tr>
<tr>
<td>HLHS</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0/1</td>
<td>0/1</td>
<td>0</td>
<td>0/1</td>
<td>1/1</td>
</tr>
<tr>
<td>PA</td>
<td>0</td>
<td>0</td>
<td>3/3</td>
<td>0</td>
<td>0</td>
<td>1/1</td>
<td>0</td>
<td>0/1</td>
<td>0/1</td>
<td>0</td>
<td>0/1</td>
<td>1/1</td>
</tr>
<tr>
<td>d-TGA</td>
<td>2/2</td>
<td>1/1</td>
<td>3/3</td>
<td>0</td>
<td>1/1</td>
<td>0</td>
<td>0</td>
<td>2/2</td>
<td>0/1</td>
<td>2/2</td>
<td>0/1</td>
<td>0</td>
</tr>
<tr>
<td>TAPVC</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2/2</td>
<td>1/2</td>
<td>1/1</td>
<td>1/1</td>
<td>0/1</td>
<td>1/1</td>
<td>0</td>
<td>0/1</td>
<td>1/1</td>
</tr>
<tr>
<td>Truncus</td>
<td>0</td>
<td>0</td>
<td>0/1</td>
<td>2/2</td>
<td>1/1</td>
<td>0</td>
<td>3/3</td>
<td>0</td>
<td>7/8</td>
<td>2/2</td>
<td>0/1</td>
<td>1/1</td>
</tr>
<tr>
<td>TA</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0/1</td>
<td>1/1</td>
<td>0</td>
<td>0/1</td>
<td>1/1</td>
</tr>
<tr>
<td>AA/AS</td>
<td>2/3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1/1</td>
<td>0</td>
<td>3/4</td>
<td>2/3</td>
<td>0/1</td>
<td>3/4</td>
</tr>
<tr>
<td>TOF</td>
<td>5/5</td>
<td>1/1</td>
<td>1/1</td>
<td>1/1</td>
<td>0</td>
<td>2/3</td>
<td>0</td>
<td>0</td>
<td>0/1</td>
<td>0</td>
<td>0/1</td>
<td>4/5</td>
</tr>
<tr>
<td>AVSD</td>
<td>2/2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1/1</td>
<td>0</td>
<td>0/1</td>
<td>0</td>
<td>1/1</td>
<td>0</td>
<td>0/1</td>
<td>1/1</td>
</tr>
<tr>
<td>CoA</td>
<td>0/3</td>
<td>1/1</td>
<td>2/3</td>
<td>0/1</td>
<td>0</td>
<td>1/1</td>
<td>1/1</td>
<td>2/2</td>
<td>3/4</td>
<td>8/15</td>
<td>5/5</td>
<td>0</td>
</tr>
<tr>
<td>PS</td>
<td>0</td>
<td>1/1</td>
<td>0/1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1/3</td>
<td>0</td>
<td>2/6</td>
<td>33.3</td>
<td>10–70</td>
<td>33.3</td>
</tr>
</tbody>
</table>

Cl indicates confidence interval; DORV, double-outlet right ventricle; HLHS, hypoplastic left heart syndrome; PA, pulmonary atresia; d-TGA, dextro-transposition of the great arteries; TAPVC, total anomalous pulmonary venous connection; Truncus, truncus arteriosus; TA, tricuspid atresia; AA/AS, aortic atresia/aortic stenosis; TOF, tetralogy of Fallot; AVSD, atrioventricular septal defect; CoA, coarctation of the aorta; and PS, pulmonary stenosis.
There has also been concern that pulse oximeters may not be as accurate in darkly pigmented adults and children. At low \( \text{SpO}_2 \) levels (<70%), commercially available oximeters appear to overestimate arterial saturation by 3% in darkly pigmented subjects.\(^9\) However, when \( \text{SpO}_2 \) is >90%, measurement bias related to skin pigmentation appears negligible (<0.2%). Lastly, the quality of oximetry measurements may be lower when performed in a screening setting.\(^9\)

When neonates are identified as having hypoxemia (\( \text{SpO}_2 \leq 95% \)), it is necessary to evaluate them for CCHD. Although physical examination, chest radiography, and electrocardiography can assist in this process, echocardiography is now considered the definitive diagnostic modality. Whenever possible, the echocardiograms should be interpreted by pediatric cardiologists; major errors in the interpretation of a newborn echocardiogram by trained pediatric cardiologists are rare.\(^9\)

Although the majority of metropolitan areas in the United States have access to pediatric subspecialists, such as pediatric cardiologists, availability in rural areas can be limited. Approximately 15% of births in the United States occur in nonmetropolitan areas.\(^9\) In these settings, echocardiograms are often performed by sonographers without formal pediatric training and are interpreted by adult cardiologists. Several investigators have found that the accuracy of pediatric echocardiograms interpreted by adult cardiologists is low.\(^9,9\) One alternative is to use telemedicine, in which echocardiograms are interpreted distantly at a pediatric referral center.\(^9,9\) The accuracy may be improved by direct guidance of the sonographers by a pediatric cardiologist via videoconferencing. This approach, which has been shown to be efficient and accurate, may be required to enhance detection of CCHD in rural or underserved areas. Another option is for newborns with suspected CCHD to be transported to a tertiary center. This strategy, however, would be expensive and impractical in many cases.

The cost of routine pulse oximetry performed on asymptomatic newborns after 24 hours of age includes both the direct cost of the pulse oximetry and the follow-up costs of any additional examinations and transfers. The largest direct cost component is staff time. At experienced centers, it may take a technician only 45 seconds on average to perform pulse oximetry on a newborn infant. The cost of diagnostic evaluation of infants who are referred for further examination after pulse oximetry depends on the frequency of referral, the duration of the diagnostic evaluation, and the ability for the evaluation to be performed without transfer to another center. A detailed cost accounting, to be reported elsewhere, indicates an average cost of approximately $1 per asymptomatic newborn infant, which includes the cost of diagnostic evaluations, in hospitals with moderate obstetric volume and ready access to pediatric echocardiography. Further work is needed to assess the cost and yield of routine pulse oximetry examination of newborns in a wider range of settings.

Oximetry to enhance the detection of CCHD has been considered previously in an evidence review sponsored by the United Kingdom’s National Health Service Health Technology Assessment program.\(^10\) The investigators observed that pulse oximetry is much more effective than current clinical practice in identifying infants with CCHD and more accurate and much less expensive than screening all newborns with echocardiography. The incremental cost per timely diagnosis of life-threatening congenital heart defects was calculated to be approximately $10 000 for pulse oximetry and $10 million for screening echocardiography. Although pulse oximetry was regarded as more promising than either the current practice or other options, the report called for further research to improve estimates of test performance and to inform timing, diagnostic, and management strategies and to “investigate the psychosocial effects of newborn screening for congenital heart disease” (p 127).\(^10\) Another report has suggested families were quite receptive to newborn screening with pulse oximetry, with 99.8% of a sample of parents in Poland reported to approve of the screening technique.\(^7\)

**Summary**

The association of delayed diagnosis of CCHD with mortality, morbidity, and disability provides a rationale for strategies such as pulse oximetry assessment to improve early detection. Some studies have reported a reasonable detection rate with pulse oximetry; however, the usefulness of oximetry in clinical practice is not well established (Class IIb, Level of Evidence C; Level of Evidence C corresponds to observational studies [case-control and cohort design]). Additional studies in larger populations and across a broad range of newborn delivery systems are needed to determine whether this practice should become the standard of care in the routine assessment of the neonate.

Currently, pulse oximetry is being performed routinely in some delivery centers in the United States and elsewhere.\(^9\) Because pulse oximetry cannot detect all cases of CCHD, the diagnoses in some infants will be missed until after discharge from the newborn nursery. Such cases will provoke the question of whether the newborn oximetry screen was performed accurately. Therefore, it is reasonable for centers that routinely use pulse oximetry to ensure the fidelity of oximetry measurements through periodic quality assessment. Parents and caretakers should also be informed that pulse oximetry cannot detect all cases of CCHD, and hence, a negative test result does not exclude the possibility of heart disease.

**Call for Future Studies**

Collaborative studies among hospitals conducting routine pulse oximetry should analyze pooled data and report detection, false-positive rates, and false-negative rates of CCHD. A pilot study of pulse oximetry screening has recently completed enrollment at 6 English hospitals by the National Institute for Health Research.\(^11\) In addition, a comprehensive assessment of the impact of pulse oximetry assessment and early detection of CCHD on morbidity, postoperative survival, and hospital costs will allow a more critical evaluation of the economic impact of efforts to improve timely diagnosis of CCHD.
# Disclosures

## Writing Group Disclosures

<table>
<thead>
<tr>
<th>Writing Group Member</th>
<th>Employment</th>
<th>Research Grant</th>
<th>Other Research Support</th>
<th>Speakers' Bureau/Honoraria</th>
<th>Expert Witness</th>
<th>Ownership Interest</th>
<th>Consultant/Advisory Board</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>William T. Mahle</td>
<td>Emory University School of Medicine</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Robert H. Beekman III</td>
<td>Cincinnati Children's Hospital Medical Center</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>AGA Medical Inc (Device Proctor)<em>; The Medicines Co (DSMC)</em></td>
<td>None</td>
</tr>
<tr>
<td>Samuel S. Gidding</td>
<td>Nemours Children's Clinic</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Scott D. Grosse</td>
<td>Centers for Disease Control and Prevention</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Tracey R. Hoke</td>
<td>University of Virginia</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Robert Koppel</td>
<td>Schneider Children's Hospital</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>G. Paul Matherne</td>
<td>University of Virginia</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Jane W. Newburger</td>
<td>Boston Children's Heart Foundation</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Frank C. Smith</td>
<td>Pediatric Cardiology Associates of Central New York</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (1) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (2) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

## Reviewer Disclosures

<table>
<thead>
<tr>
<th>Reviewer</th>
<th>Employment</th>
<th>Research Grant</th>
<th>Other Research Support</th>
<th>Speakers' Bureau/Honoraria</th>
<th>Expert Witness</th>
<th>Ownership Interest</th>
<th>Consultant/Advisory Board</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constance Cephus</td>
<td>Texas Children's Hospital</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Steven D. Colan</td>
<td>Children's Hospital Boston</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Rodney Howell</td>
<td>University of Miami</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Alex Kemper</td>
<td>Duke University</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>LuAnn Minich</td>
<td>Intermountain Healthcare</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Catherine L. Webb</td>
<td>Northwestern University</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Johnson &amp; Johnson*</td>
<td>None</td>
</tr>
</tbody>
</table>

This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be “significant” if (1) the person receives $10,000 or more during any 12-month period, or 5% or more of the person’s gross income; or (2) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be “modest” if it is less than “significant” under the preceding definition.

*Significant.
References


Role of Pulse Oximetry in Examining Newborns for Congenital Heart Disease. A Scientific Statement From the American Heart Association and American Academy of Pediatrics

William T. Mahle, Jane W. Newburger, G. Paul Matherne, Frank C. Smith, Tracey R. Hoke, Robert Koppel, Samuel S. Gidding, Robert H. Beekman III and Scott D. Grosse on behalf of the American Heart Association Congenital Heart Defects Committee of the Council on Cardiovascular Disease in the Young, Council on Cardiovascular Nursing, and Interdisciplinary Council on Quality of Care and Outcomes Research the American Academy of Pediatrics Section on Cardiology and Cardiac Surgery, and Committee on Fetus and Newborn

Circulation. published online July 6, 2009;
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2009 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/early/2009/07/06/CIRCULATIONAHA.109.192576.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/