

First Day of Life Pulse Oximetry Screening to Detect Congenital Heart Defects

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Objective To evaluate the efficacy of first day of life pulse oximetry screening to detect congenital heart defects (CHDs).

Study design We performed a population-based prospective multicenter study of postductal (foot) arterial oxygen saturation (SpO₂) in apparently healthy newborns after transfer from the delivery suite to the nursery. SpO₂ < 95% led to further diagnostic evaluations. Of 57,959 live births, 50,008 (86%) were screened. CHDs were prospectively registered and diagnosed in 658 newborns (1.1%), of whom 35 (5%) were classified as critical (ductus dependent, cyanotic).

Results Of the infants screened, 324 (0.6%) failed the test. Of these, 43 (13%) had CHDs (27 critical), and 134 (41%) had pulmonary diseases or other disorders. The remaining 147 infants (45%) were healthy with transitional circulation. The median age for babies with CHDs at failing the test was 6 hours (range, 1-21 hours). For identifying critical CHDs, the pulse oximetry screening had a sensitivity rate of 77.1% (95% CI, 59.4-89.0), specificity rate of 99.4% (95% CI, 99.3-99.5), and a false-positive rate of 0.6% (95% CI, 0.5-0.7).

Conclusions Early pulse oximetry screening promotes early detection of critical CHDs and other potentially severe diseases. The sensitivity rate for detecting critical CHDs is high, and the false-positive rate is low. (*J Pediatr* 2008;152:761-5)

Seemingly healthy newborn babies may be admitted to the nursery with hidden malformations or unrecognized symptoms of disease. Despite looking pink to health workers observing them, the babies may have low arterial oxygen saturation, a sign of disease. Studies have focused on congenital heart defects (CHDs) because a substantial percentage of severe CHDs are missed in the clinical screening of newborns before discharge home.¹⁻⁴ Screening with pulse oximetry has been put forth as a useful strategy for detecting defects with decreased arterial oxygen saturation (SpO₂) before heart failure and circulatory collapse develops.⁵⁻¹¹ Pulmonary diseases and other disorders may also be detected.⁵ In editorial comments,^{12,13} metaanalyses,^{14,15} and recent studies,¹⁶ the need for more extensive studies of the potential benefits of pulse oximetry screening programs has been emphasized.

The aim of this study was to evaluate the efficacy of a first day of life pulse oximetry screening program in detecting disorders with subnormal arterial oxygen saturation, especially CHDs, in apparently healthy babies after transfer from the delivery unit to the nursery.

METHODS

14 Norwegian hospitals with obstetric departments and pediatric services and neonatal special or intensive care units participated in the study during the years 2005 to 2006. These hospitals cover approximately 50% of all deliveries in Norway.

Postductal (probe on the foot) SpO₂ was consecutively measured when apparently healthy babies were admitted to the nursery from the delivery unit. Babies transferred

See editorial, p 747 and related article, p 756

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CHD	Congenital heart defect	SpO ₂	Arterial oxygen saturation
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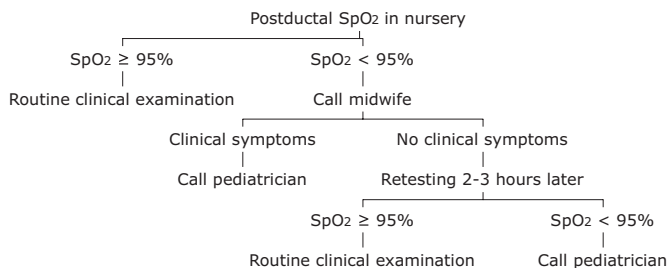


Figure 1. Algorithm for pulse oximetry screening of newborn infants in the nursery.

directly to a special or intensive care unit from the delivery suite or from the nursery before SpO₂ was measured or who had CHDs detected prenatally, were not included in the screening program. The algorithm for actions according to registered SpO₂ is shown in Figure 1.

The cutoff for SpO₂ (<95%) was based on measurements in 1000 consecutively admitted babies showing an SpO₂ of 95% to represent the 2.5 percentile for distribution of the measurements. This cutoff is also chosen in other studies that use the same technology in identifying infants with CHDs.^{5,8,9,11} Further investigations, such as echocardiography, radiographic studies, blood samples, and bacterial cultures were performed according to local routines and at the discretion of the pediatrician. Infants with SpO₂ < 95% on re-testing who were judged to be healthy and subsequently had normal SpO₂-measurements, were diagnosed as having a prolonged transitional circulation.

A pulse oximeter type RAD-5v (Masimo Corporation, Irvine, Calif) with a multi-site reusable sensor (LNOP YI) was used for the SpO₂ measurements. This new generation technology secures reliable oxygen saturation values in newborn infants, overcoming earlier limitations related to low perfusion states and motion artifacts.¹⁷ The probe was attached for at least 2 minutes, until a stable value was obtained.

CHDs in the populations were registered prospectively, until at least 6 months after the last infants were born. Prenatally diagnosed CHDs (routine fetal ultrasound screening at approximately week 18 of pregnancy) were verified with early postnatal echocardiography, invasive procedures, or both. Infants with syndromes or chromosomal disorders were routinely referred for echocardiography, preferentially before discharge. The same applied for babies with persistent murmurs, cyanosis, or symptoms compatible with heart failure, either detected in the routine pediatric examination of babies in the nursery (undertaken the first or second day after birth) or after transfer to a neonatal unit. After discharge, infants with findings suggestive of heart disease were referred for cardiologic examinations at the local pediatric department. All CHDs were verified with echocardiography. Excluded from the series were: 1) children with bicuspid aortic valve only (with no aortic stenosis or regurgitation); 2) children with cardiac arrhythmia only; 3) patent ductus arteriosus in all preterm infants, and in term infants unless the duct stayed open after 6 months of age; and 4) infants with isolated left to right interatrial shunt not present after the age of 6 months.

Table I. Disorders in newborns who failed the first day of life pulse oximetry screening (postductal SpO₂ < 95%), n = 324

Condition	n	Median SpO ₂ (%)	SpO ₂ range (%)	SpO ₂ interquartile range (%)
Congenital heart defects	43	88	47-94	9.8
Pneumonia/septicemia	55	90	75-94	8
Transient tachypnoea	54	91	70-94	7
Persistent pulmonary hypertension	6	83	75-94	15
Pneumothorax	6	79	70-94	10
Amniotic fluid aspiration	5	91	88-93	3.5
Miscellaneous*	8	88	72-93	10
Transitional circulation	147	93	82-92	3

*Hypoglycemia (3), pulmonary atelectasis (1), polycythemia (1), infantile pulmonary fibromatosis (1), respiratory distress syndrome (1), and cardiomyopathy (1).

The study was approved by the Regional Committee for Medical Research Ethics.

Statistical analyses were performed with the χ^2 test. A *P* value < .05 was regarded as significant.

RESULTS

Of 57,959 live births, 50,008 newborns (86%) were screened. In the first test, 1360 newborns (3%) had SpO₂ < 95%. Of these cases, 324 (0.6%) were classified as pathological, either because of symptoms of disease when first tested (3 CHD, 29 other disorders) or because of persistent SpO₂ < 95% when re-tested (292, all perceived as asymptomatic). Of infants not passing the screening, 43 (13%) had CHDs (transposition of the great arteries, 11; atrioventricular septal defect, 8; ventricular septal defect, 6; total anomalous pulmonary venous return, 5; coarctation of the aorta, 4; pulmonary atresia, 3; aortic stenosis, 2; pulmonary stenosis, 1; common arterial truncus, 1; tetralogy of Fallot, 1; single ventricle, 1). In 134 cases (41%), other disorders were diagnosed. The remaining 147 cases (45%) were classified as healthy newborns with prolonged transitional circulation. Table I shows the number and SpO₂ values in the different groups when failing the test.

In the 43 infants with CHDs who did not pass the pulse oximetry screening, 40 CHDs (93%) were diagnosed before the infants were discharged. The median age when a low SpO₂ initiated further investigations was 6 hours (range, 1-21 hours). In 39 of 43 babies (91%), the procedures were initiated within 12 hours after birth. Echocardiography was undertaken in 99 of 324 babies (31%) who did not pass the screening (40/43 [93%] with CHDs and 59/281 [21%] with other conditions; *P* < .0001).

658 cases (1.1%) of CHDs were diagnosed in the 57,959 live-born infants. Of these, 46 cases (7%) were detected prenatally, 40 cases (6%) were detected before discharge with the pulse oximetry screening program, 320 cases (49%) were detected with routine clinical examination of a

Table II. The accuracy of first day of life pulse oximetry screening (postductal SpO₂ < 95%) for detecting congenital heart defects in apparently healthy babies in the nursery (n = 50,008)

	Pulse oximetry		Pulse oximetry + clinical examination	
	Critical CHDs	CHDs in total	Critical CHDs	CHDs in total
Number of patients with CHDs detected	27/35	43/434	31/35	363/434
Sensitivity rate, % (true-positive rate; 95% CI)	77.1 (59.4-89.0)	9.9 (7.3-13.2)	88.6 (72.3-96.3)	83.6 (79.7-86.9)
Specificity, % (95% CI)	99.4 (99.3-99.5)	99.4 (99.3-99.5)	99.4 (99.3-99.5)	99.9 (99.8-99.9)
Positive predictive value, % (95% CI)	8.3 (5.7-12.0)	13.3 (9.9-17.6)	9.6 (6.7-13.4)	83.6 (79.7-86.9)
Negative predictive value, % (95% CI)	99.98 (99.97-99.99)	99.2 (99.1-99.3)	99.99 (99.98-100)	99.86 (99.82-99.89)
False-positive rate, % (95% CI)	0.6 (0.5-0.7)	0.6 (0.5-0.6)	0.6 (0.5-0.7)	0.1 (0.1-0.2)

pediatrician in the nursery, 178 cases (27%) were detected after admission to a neonatal special or intensive care unit, and 74 cases (11%) were detected during infancy after discharge. In the latter group, 3 patients had SpO₂ < 95%, also when re-tested, but CHD was not recognized until readmission with clinical symptoms (heart failure, murmur). Echocardiography had not been performed before discharge. 1 of these patients had critical aortic stenosis/coractation of the aorta, 1 patient had total anomalous pulmonary venous return, and 1 patient had pulmonary stenosis.

In CHDs detected prenatally, 34 of 46 cases (74%) had SpO₂ < 95% when tested after transfer to a neonatal intensive care unit. 5 of the 74 cases of CHD that were detected after discharge passed the pulse oximetry screening in the nursery (SpO₂ ≥ 95%) but were hypoxic when readmitted (SpO₂ ≤ 80%). 2 of these patients had coarctation of the aorta, 1 had an interrupted aortic arch, 1 had a transposition of the great arteries with ventricular and atrial septal defects, and 1 had atrioventricular septal defect. No deaths from unrecognized CHD were observed.

Of the 324 patients not passing the screening, 27 (8%) were diagnosed with critical CHD (ductus dependent left ventricle outflow obstruction [hypoplastic left heart syndrome, severe aortic stenosis, coarctation of the aorta or interrupted aortic arch], ductus dependent pulmonary flow obstruction [tricuspid atresia, pulmonary atresia], or other complex CHDs [d-transposition of the great arteries, total anomalous pulmonary venous return, single ventricle, double outlet right ventricle, common arterial truncus, Ebstein anomaly]). Of the 49,684 patients who passed the screening, 8 (0.2%) had critical CHDs (*P* < .0001), 4 of which were detected in the clinical routine examination before discharge. Table II shows the accuracy of pulse oximetry in diagnosing CHDs in total and for the subgroup of critical CHDs and for pulse oximetry combined with clinical routine examination.

Figure 2 summarizes the main findings in the study. In the group that did not undergo screening (transferred to a neonatal special or intensive care unit after birth, including prenatally detected CHDs), 224 of 7,951 babies (3%) had CHDs, compared with 434 of 50,008 apparently healthy babies (0.9%) screened in the nursery (relative risk, 3.2; 95% CI, 2.8-3.8). In the group that did not pass the screening

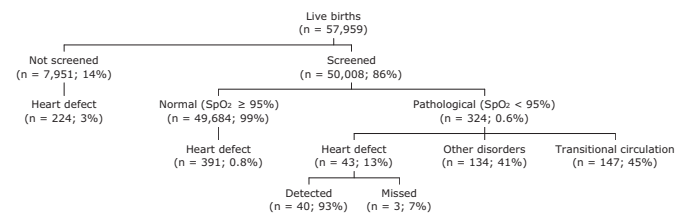


Figure 2. Results of pulse oximetry screening of newborn infants in the nursery.

(SpO₂ < 95%), 43 of 324 babies (13%) had CHDs, compared with 391 of 49,684 babies (0.8%) who passed the screening (relative risk, 16.9; 95% CI, 12.5-22.5).

DISCUSSION

In this large prospective multicenter study, first day pulse oximetry screening of apparently healthy babies admitted to the nursery alerted the staff to a considerable number of potentially severe disorders. CHDs and pulmonary diseases were the most common conditions detected. Most of the CHDs that were discovered might have been life-threatening if left undiagnosed. A low SpO₂ prompted early cardiologic examination. Valuable time thus may have been gained compared with detection with the pediatric routine examination or from later symptoms recognized clinically, in some infants probably after discharge from the hospital.

Among infants in need of cardiac surgery before 2 months of age, Mellander and Sunnegårdh³ found that 4% with CHDs with ductus dependent pulmonary circulation and 30% with CHDs with ductus dependent systemic circulation were missed and had to be readmitted. An even higher percentage (38%) of ductus independent severe CHDs was not detected before the infants were discharged. Reinhardt and Wren reported that as many as one third of babies with a potentially life-threatening CHD may leave the hospital with it undiagnosed.⁴ Among these are CHDs with critical obstruction for blood flow to the lungs (eg, atresia of the pulmonary or tricuspid valves) or systemic circulation (eg, hypoplastic left heart syndrome, severe aortic stenosis, coarctation of the aorta, or interrupted aortic arch). Such

CHDs depend on a patent ductus arteriosus for pulmonary and systemic perfusion, and severe hypoxia, acidosis, circulatory collapse, and possibly death will occur when the arterial duct closes.¹⁸ The risk of collapse and death may be particularly high with today's common practice of early discharge, which may result in ductal closure outside the hospital.

Despite not passing the pulse oximetry screening, a few patients had CHDs that were not detected before discharge, underscoring that scrupulous diagnostic procedures should be undertaken, including extensive use of echocardiography, in babies with persistent subnormal SpO₂.

Some infants who passed the SpO₂-screening were hypoxic when readmitted with CHD. They had large left-to-right shunts or left-sided outflow obstructions. Coarctation or interruption of the aorta may especially be at risk for being overlooked,^{5,14} as was also found in this study.

Fetal ultrasound scanning screening programs improve detection of complex CHDs.¹⁹ However, in routine screening of large populations, the detection rate may be low.²⁰ This may also be the case in our study, although we did not have access to data on pregnancies terminated because of malformations. However, the high prevalence of CHDs (1.1%) in the population of live-born infants suggests that few pregnancies were terminated because of CHD. When complex CHDs are unrecognized at birth, pulse oximetry screening may represent a complementary strategy for early diagnosis because a low SpO₂ was found in most cases that were detected prenatally.

The largest group that did not pass the SpO₂ screening was infants with prolonged transitional circulation. The speed with which the physiologically high pulmonary arterial pressure decreases after birth because of decreasing pulmonary vascular resistance varies considerably in healthy babies.²¹ Thus, a right-to-left or bi-directional shunt through a patent ductus arteriosus may persist for some hours and cause a transiently low postductal SpO₂. We have found that the median time to reach postductal SpO₂ \geq 95% is 20 minutes in healthy babies, but has a range of 3 to 90 minutes.²² This is in accordance with other studies.²³⁻²⁵ To avoid too many false-positive results from babies with a prolonged phase of transitional circulation, it may be reasonable to perform the pulse oximetry screening when the baby arrives in the nursery a couple of hours after birth. Then the basic routines in the delivery room and the first undisturbed period of parent-infant attachment have taken place.

In a meta-analysis of 8 studies, most with a SpO₂ cutoff $<$ 95%, Thangaratnam et al¹⁵ found pulse oximetry to be a highly specific tool in detecting critical CHDs, with very low false-positive rates. The mean summary estimates of sensitivity and specificity rates were 63% and 99.8%, respectively, yielding a false-positive rate of 0.2%. Our data showed a somewhat higher sensitivity rate (77%) and a higher false-positive rate (0.6%). However, in half the studies in the meta-analysis, the infants were screened 24 hours after birth or just before discharge, thus avoiding many of the transitional

phenomena of this study. We suggest, however, that the potential advantage of early detection of hypoxemic CHDs and severe extracardiac disorders with first day testing may outweigh the disadvantage of a somewhat increased false-positive rate.

Shifting the cutoff point of SpO₂ from $<$ 95% to higher levels (eg, 96% as used in the study by Koppel et al)⁷ will possibly increase sensitivity, but at the expense of a lower specificity. Other studies have indicated that SpO₂ $<$ 95% is an appropriate cutoff in screening for CHDs.^{11,14,26} Increased sensitivity may also be obtained by measuring the SpO₂ both preductally and postductally, emphasizing the hand-foot difference in saturation.²⁶ The simplicity of postductal measurement is, however, an advantage when screening large populations. Because right-to-left shunting through a patent ductus arteriosus characterizes the important group of obstructive left heart malformations, of which coarctation of the aorta and interruption of the aortic arch are most likely to remain undiagnosed at discharge,⁴ postductal measurements may be preferred for screening.

In apparently healthy babies who did not pass the SpO₂ screening, the risk for CHD was 17-times higher than that of the group that passed. However, nearly 60% of all CHDs occurred in babies with a normal SpO₂ in the nursery. Most of these acyanotic CHDs were detected at the routine clinical screening before discharge, underscoring the importance of this examination. For detecting CHDs in general the sensitivity of pulse oximetry is low, because most CHDs present with a normal SpO₂. For detecting critical CHDs, however, the sensitivity is high and further improved when combined with clinical routine examination.

Until now pulse oximetry screening has focused on detecting CHDs. However, in this study, pulmonary diseases and other disorders were detected in a substantial number of cases. Lowest SpO₂ values were found with pneumothorax and persistent pulmonary hypertension. Although respiratory distress or depression might have been present from birth, the symptoms may have been subtle and first recognized when a low SpO₂ called attention to the disorder. Detecting such disorders may be a major added advantage of the screening program, possibly as important as detecting CHDs. The low rate of babies failing the SpO₂ screening, of whom more than half had CHDs or extracardiac disorders, indicates that this program does not generate a marked increase in consumption of healthcare resources.

The sensitivity rate for detecting critical CHDs is high, and the false-positive rate low. A confirmed SpO₂ $<$ 95% must lead to scrupulous diagnostic investigations. Some complex CHDs, however, may still be missed. The ease of performing the pulse oximetry measurements, the minimal discomfort for the baby, low costs,⁷ and the significance of a low SpO₂ in alerting the staff of potentially severe diseases, are strong arguments for implementing pulse oximetry screening of newborn babies as a basic routine in the nursery.

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