OXYGEN SATURATION AS A SCREENING TEST FOR CRITICAL CONGENITAL HEART DISEASE:

A PRELIMINARY STUDY

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Oxygen Saturation as a Screening Test for Critical Congenital Heart Disease: A Preliminary Study

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Abstract. The aim of this study was to assess the utility of arm and leg oxygen saturation as a candidate screening test for the early detection of ductal-dependent left heart obstructive disease. We measured arm and leg oxygen saturation in 2876 newborns admitted to well baby nurseries and 32 newborns with congenital heart disease. Fifty-seven newborns in the well baby nurseries (0.02%) had an abnormal test (leg saturation less than 92% in room air or 7% lower saturation in the leg than in the arm). Four of the 57 had critical congenital heart disease, including 1 with coarctation of the aorta. Of the 32 newborns with congenital heart disease, 11/13 (85%) with left heart obstructive disease had abnormal oxygen saturation tests, as did 15/19 (79%) with other forms of congenital heart disease. Pulse oximetry deserves further study as a screening test for critical congenital heart disease.

Key words: Newborn screening — Congenital heart disease — Early discharge — Oxygen saturation

The timely diagnosis of serious congenital heart disease (CHD) remains a challenge. Among newborns that die with CHD, representative population-based studies have shown that up to 30% have unrecognized cardiac defects at the time of death [1, 5]. Early detection is complicated by many factors, including a rapid change in patency of the ductus arteriosus in the first 24 hours of life [4, 6, 10], limited detection of cardiac malformations on newborn examination [2,18], the subtlety of physica! findings associated with impending clinical deterioration [12, 13, 16], and the trend in many countries toward shorter lengths of hospital stay for apparently healthy newborns [5, 16, 18].

Ideally, critical CHD should be detected before the advent of acidosis and cardiovascular collapse, changes that can be abrupt and fatal in ductal-dependent lesions. These lesions include coarctation of the aorta, interrupted aortic arch, hypoplastic left heart syndrome, and critical aortic stenosis–collectively defined as left heart obstructive disease (LHOD). In the setting of early discharge, newborns with LHOD (and pulmonary or systemic blood flow dependent on patency of the ductus arteriosus) might be discharged after an apparently normal exam on the first day of life only to return in critical condition as the ductus closes, leading to loss of systemic vascular perfusion or severe cyanosis [1, 2, 5, 12, 13, 16, 18, 19].

In this study, we explored the utility of arm and leg oxygen saturation as a candidate screening test for the early detection of critical CHD. Because of the obligate right-to-left shunt at the level of the ductus arteriosus in newborns with LHOD, we hypothesized that these newborns would have abnormally low leg saturation before the onset of symptoms associated with closure of the ductus arteriosus. The test would also potentially detect those newborns with complete mixing defects, such as total anomalous pulmonary venous return, in which early detection could lead to improved survival.

The objectives of this study were to obtain data on the feasibility of performing arm and leg saturation in usual clinical circumstances in a large cohort of healthy newborns, to assess the prevalence of abnormal test results in the first week of life in both healthy full-term newborns and those with suspected CHD, and to generate preliminary estimates of the

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utility of pulse oximetry as a means of identifying newborns with CHD.

Materials and Methods

Study Design and Setting

This was a case-control study of oxygen saturation measurement in newborns. The study was conducted from August 1993 to February 1995 at an urban tertiary care institution [Johns Hopkins Hospital (JHH)] and from November 1993 to April 1994 at a suburban community hospital [Greater Baltimore Medical Center (GBMC)].

Subjects

Two groups of newborns were eligible for inclusion in this study: (1) healthy newborns admitted to the well baby nursery at JHH or GBMC who were at least 34 weeks gestation at birth and (2) newborns who were at least 34 weeks gestation at birth and who were born at JHH, or transferred at less than 7 days of life to JHH, for evaluation of suspected CHD.

Interventions

Nursery personnel (nurses or nurse practitioners) made all oxygen saturation measurements by pulse oximetry. For each newborn, the right arm and either leg saturation and heart rate were obtained using a Nellcor 50 pulse oximeter with a soft, reusable Nellcor probe adapted to clip onto the toe or finger. The Nellcor pulse oximeter was set to read eight pulses before calculating a final measurement. A single oximeter was used for both measurements in order to make the screening test as easy as possible for nursery personnel to carry out. Personnel recording pulse oximeter measurements were not necessarily blinded to the physician's level of suspicion for CHD.

Main Outcome Measures

Measurements were recorded at less than 6 hours of life, at 24 hours of life, and/or at discharge. Care was taken to minimize factors known to interfere with pulse oximetry readings, such as patient movement, patient hypothermia, and phototherapy for hyperbilirubinemia [11, 17]. An important limitation of pulse oximetry is failure to detect pulse and saturation in the setting of low blood pressure or peripheral vasoconstriction [15]. All newborns had normal skin temperature and blood pressure at the time of data collection. Exposure to phototherapy and/or other sources of ambient light interference has been shown to decrease the reliability of oximeters in both the recognition of pulse and the calculation of oxygen saturation [14]. No recordings in this study were performed in the presence of phototherapy. We recorded arm and leg saturation and required that the reported heart rate differ by less than 20 beats per minute for each pair of oximetry readings in order for the test to meet criteria for an acceptable measurement. This exclusion criterion was intended to maximize data reliability [10]. An abnormal test was defined as an oxygen saturation 7% lower in the leg than in the arm or a saturation less than 92% in the leg.

Study personnel reviewed the medical charts of all newborns with an abnormal test to record symptoms, physical findings, and results of additional studies in order to determine if these newborns would have been otherwise suspected to have CHD. Oxygen saturation and heart rate from the pulse oximeter, site of probe placement (right or left side and hand or foot), instrument number, and date and time of evaluation were recorded. The most recent respiratory rate was noted from the bedside flow sheet. Birth weight, gestational age, and time of birth were recorded from the medical record.

Among newborns referred for evaluation of suspected CHD, the first saturation and vital signs (temperature, heart rate, respiratory rate and blood pressure) obtained at the hospital of birth were recorded. In all cases, these measurements were recorded before the initiation of prostaglandin therapy.

Statistical Analysis

Descriptive statistics were used to evaluate the utility of oximetry as a screening tool for CHD. Box plots indicating the mean, quartiles, and extreme values of oxygen saturation were constructed to demonstrate the change in leg saturation over time and the difference between arm and leg saturation between healthy children and those with CHD. The sensitivity and specificity of the test were determined using various definitions of an abnormal saturation in order to determine the most appropriate criteria for initiating further diagnostic evaluation. Likelihood ratios were calculated for each paired specificity and sensitivity [3].

Results

During the study period 3697 newborns were admitted to the well baby nursery (WBN) at JHH, of whom 1581 (43%) were enrolled, and 1695 newborns were admitted to the WBN at GBMC, of whom 1391 (82%) were enrolled. Those enrolled in the study at JHH did not differ from those not enrolled with regard to gestational age, birth weight, or gender. Three sets of parents refused entrance into this study. The remaining newborns were not enrolled because they were born on days when study personnel were off duty.

Because of a heart rate discrepancy of greater than 20 beats per minute between arm and leg measurement, 96 of the 2972 enrolled newborns were excluded from the analysis. These were the only exclusions from the initial 2972 enrollees. Of the 1496 newborns enrolled at JHH, the mean birth weight was 3245 g (SD-505), the mean gestational age was 38.98 weeks (SD-1.35), and 49% were male and 79% were non-white. Of the 1380 newborns enrolled at GBMC, the mean birth weight was 3423 g (SD-502), the mean gestational age was 39.32 weeks (SD-1.44), and 51% were male and 19% were nonwhite.

In addition, 32 newborns with known or suspected CHD were studied by pulse oximetry. In general, these newborns were born at outside hospitals and transferred to JHH for further evaluation and care. Acceptable saturation measurements re-

Table 1. Oxygen saturations of newborns in the well baby nurseries discovered to have congenital heart disease

	Oxygen Saturation %			
Patient No.	Arm	Leg	Diagnosis	Presentation
1	98	90	CoA	Decreased femoral pulses at 22 hours of life
2	77	77	TGA	Cyanosis
3	81	84	PS/RVH	Cyanosis with feeding
4	87	87	TOF/PAT	Cyanosis with crying at 16 hours of life

CoA, coarctation of the aorta; TGA, transposition of the great arteries; PS/RVH, pulmonary stenosis with right ventricular hypertrophy; TOF/PAT, tetralogy of Fallot with pulmonary atresia.

corded at some point during the hospital stay were thus available for 2876 healthy newborns and 32 newborns with suspected CHD.

Of the 2876 healthy newborns enrolled, 57 (0.02%) met study criteria for an abnormal test. The newborns with normal and abnormal tests did not differ with respect to birth weight, gestational age, or gender. The first test was performed at less than 24 hours of life for 37 of these, at 1 day of life for 8 these, and at the time of discharge for 12 newborns. Four of the newborns with abnormal oxygen saturation screening tests had otherwise unsuspected heart disease, whereas a fifth had pulmonary hypertension. All 5 of these newborns had persistently abnormal tests.

Of the remaining 52 newborns with at least one abnormal test, 31 had a test that was abnormal at less than 24 hours of life but normal at 1 day of life or at the time of discharge. These newborns were therefore considered healthy. Ten had normal measurements at less than 24 hours of life but an abnormal measurement at 1 day of life or at the day of discharge. Follow-up data on cardiac outcome was available for 3 of these newborns; none were believed to have CHD. Eleven newborns had only one pair of measurements but were judged to be normal at discharge by the nursery staff despite the abnormal test. Cardiac outcome was available for 5 newborns in this group and none had CHD. We cannot exclude the possibility that the remaining 13/21 children had CHD. Four of the remaining 13/21 newborns had a normal leg saturation but an abnormal arm saturation and were unlikely to have CHD.

The results of oximetry screening in the four newborns initially admitted to the WBN and subsequently categorized as having CHD are presented in Table 1. One newborn had a webbed neck and hypoplastic nails thought to be consistent with Turner's syndrome but had a normal cardiac exam on admission to the nursery. Screening oximetry at the time of a discharge evaluation (22 hours of age) confirmed the oxygen saturation in the leg to be 8% lower than that in the arm. Repeat physical examination identified decreased femoral pulses, and an echocardiogram confirmed coarctation of the aorta. A second newborn had an oxygen saturation of 77% in both the arm and the leg at <24 hours of life, a finding that was associated with obvious cyanosis. Echocardiography in this newborn demonstrated transposition of the great arteries. A third newborn had a saturation of 81 and 84% in the arm and leg, respectively. Echocardiography revealed pulmonary stenosis. A fourth newborn was first tested at 36 hours of life after developing seizures. Saturation was 89 and 87% in the arm and the leg, respectively. Further studies confirmed a diagnosis of persistent pulmonary hypertension in association with incontientia pigmenti. The fifth newborn was shown to have CHD after cyanosis was noted with crying and saturation was 87% in both the arm and the leg. The echocardiogram demonstrated pulmonary atresia and tetralogy of Fallot.

Thirty-two newborns were referred with suspected CHD, confirmed in all cases by further evaluation (Table 2). For the purposes of this analysis, they were categorized as having LHOD or other CHD. Five of these newborns were not identified as having CHD by pulse oximetry: two with pulmonary stenosis (patients 10 and 31), 1 with double-inlet left ventricle (patient 32), and 1 with aortic valvular stenosis (patient 3), and 1 with an interrupted aortic arch and choanal atresia (patient 18). Of the 13 newborns with LHOD, the earliest oxygen saturation available revealed an abnormality in 11 (85%).

Figure 1 shows a comparison of mean oxygen saturation over time for newborns with and without CHD. All but 2 of 13 newborns with LHOD had an oxygen saturation less than 92% in room air. Only 14 of the 2872 healthy newborns (0.05%) had an oxygen aturation in the leg below 92% in any circumstance. At all three time points (3–6 hours, 24 hours, or more than 24 hours or at discharge) newborns with LHOD had mean oxygen saturation in the leg well below that of healthy newborns. Furthermore, as mean saturation increased for healthy newborns over the first 24 hours of life, it decreased into a clearly abnormal range for newborns with LHOD by 24 hours of life or at discharge (time of presentation with abnormal saturation in the leg, median day 1 (day of birth); range, 1–5 days). Newborns with other CHD had mean saturation comparable to those with LHOD but demonstrated a wider range of oximetry measurements.

Table 2. Saturations of newborns with suspected CHD (n = 32)

Patient No.	Initial saturation (%)		Subsequent saturation (%)		Diagnosis	Presentation
	Arm	Leg	Arm	Leg		
1			90		TOF	Murmur on DOL 1
2		83	91		TGA	Prenatal diagnosis of TGA, cyanosis
3			100		AS	Murmur on DOL 1
4		85	82	82	PAT	Murmur, cyanosis at 1 hour of age
5			84		AVC	Poor feeding, trisomy 21
6		75	85		Ebstein anomaly	Cyanosis at 3 hours of age
7		56	70		TOF	Cyanosis at 4 hours of age
8			79	84	TAT	Cyanosis with feeding
9			81	82	TGA	Cyanosis at delivery
10	97	97			PS	Murmur on DOL 1
11	85	85			DILV/CoA	Prenatal diagnosis of CHD
12	98	97			AVC	Prenatal diagnosis of CHD
13	85	88	91	90	TAPVR	Prenatal diagnosis of CHD
14	85	82			TAT	Cyanosis, murmur at 3 hours of age
15	86	81	87	88	TGA	Murmur on DOL 1
16			82	76	TGA	Cyanosis at 13 hours of age
17			91	85	IAA	Murmur, decreased femoral pulses
18			97	92	IAA	Apnea on DOL 1, Choanal atresia
19	85	79	80	85	HLH	Prenatal diagnosis of HLH
20			98	90	AS	Respiratory distress on DOL 1
21	94	87			TGA,VSD,PS	Prenatal diagnosis of CHD
22	100	90			CoA	New murmur on DOL 2
23			99	88	CoA	Decreased femoral pulses
24			96	82	HLH	Poor perfusion at 18 hours of age
25			97	82	CoA	Decreased femoral pulses
26			97	85	HLH	Murmur, tachpnea at the office
27			98	78	IAA	Tachypnea, poor perfusion
28			97	75	IAA	Decreased femoral pulses
29				68	PS	Murmur, poor feeding
30				87	PS	Murmur, cyanosis, poor feeding
31				95	PS	Murmur
32				99	DILV	Murmur on DOL 1, tachypnea

TOF, tetralogy of Fallot; TGA, transposition of the great arteries; AS, aortic stenosis; PAT, pulmonary atresia; AVC, atrio-ventricular canal; TAT, tricuspid atresia; PS, pulmonary stenosis; DILY, double-inlet left ventricle; CoA, coarctation of the aorta; TAPVR, total anomalous pulmonary venous return; IAA, interrupted aortic arch; HLH, hypoplastic left heart; VSD, ventricular septal defect; CHD, congenital heart disease; DOL, day of life.

Figure 2 shows the difference in arm and leg oxygen saturation for healthy newborns and for newborns with CHD. Healthy newborns were found to have a negligible difference between arm and leg saturation whereas newborns with LHOD exhibited a marked difference. Newborns with other CHD had lower saturation overall but did not have a difference in arm and leg measurements.

Varying the definition of abnormal leg oxygen saturation resulted in the sensitivity, specificity, and likelihood ratios shown in Table 3. In this table, measurements of leg saturation are shown as recorded at 24 hours of life or at discharge. If the criterion for an abnormal oximetry test for LHOD is a leg saturation of less than 92%, the test has a sensitivity of 0.81 (95% confidence interval 0.66–0.96) in this sample.

Discussion

In this study we examined the utility of pulse oximetry as a screening test for the detection of ductus arteriosus-dependent CHD in newborns. An ideal screening test should detect the latent or early symptomatic period of a disease when early treatment can prevent progression. Early detection of LHOD has been shown to decrease mortality and result in better operative outcomes [7, 8, 12, 19]. For newborns with critical LHOD the latent period can be brief and can go undetected by clinicians because newborns often present with nonspecific symptoms [2, 18]. The classic physical findings LHOD–the absence of femoral pulses and upper extremity hypertension–are late signs that herald impending cardiovascular collapse. Our study suggests that

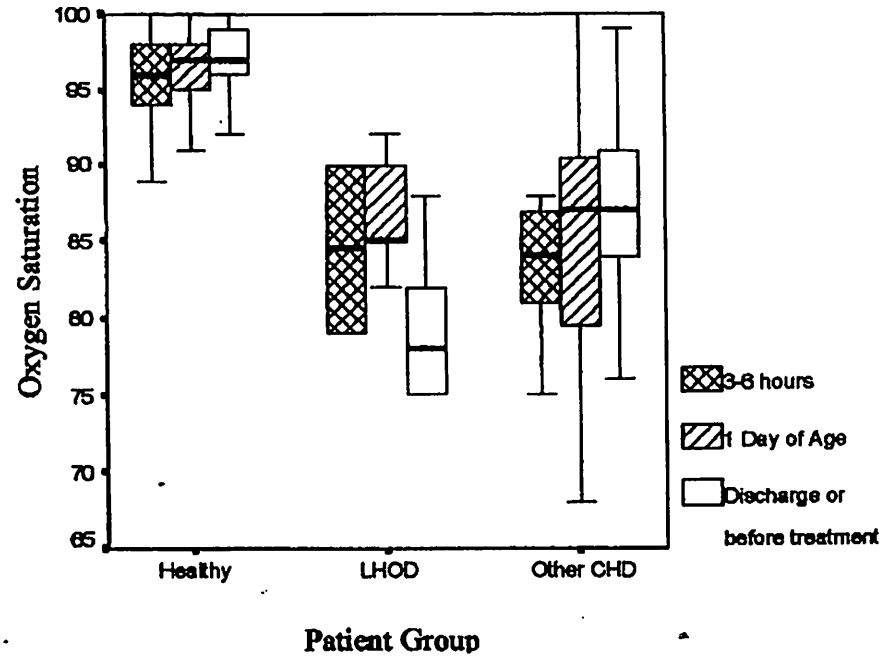

Fig. 1. Comparison of mean saturation in healthy newborns and those with left-heart obstructive disease (LHOD) and other forms of congenital heart disease (CHD). The boxes represent the 25th–75th percentile measurements. The horizontal lines within the boxes use the median value and the "whiskers" reach the 95th percentile values.

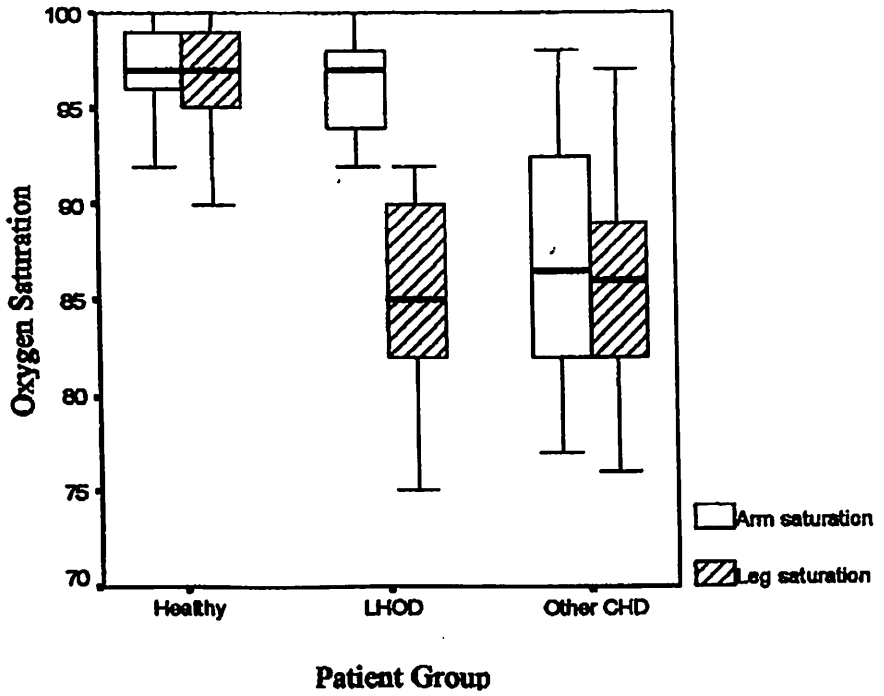

Fig. 2. Comparison of the difference between arm and leg saturation between healthy newborns and those with left heart obstructive disease (LHOD) and other forms of congenital heart disease (CHD). The boxes represent the 25th–75th percentile measurements. The horizontal lines within the boxes are the median values and the "whiskers" reach the 95th percentile values.

Table 3. Sensitivity and specificity of pulse oximetry at 24 hours of life or discharge

	Newborns with CHD (n = 36)	Newborns without CHD (n = 2697)	Sensitivity	Specificity	LR$^+$	LR$^-$
Criteria for an abnormal test						
Leg SaO_2 < 95	27	327	0.75	0.88	6.25	0.28
Leg SaO_2 < 94	26	144	0.72	0.95	14.4	0.29
Leg SaO_2 < 93	26	48	0.72	0.98	36.0	0.29
Leg SaO_2 < 92	25	15	0.69	0.99	115	0.31
Leg SaO_2 < 91	23	6	0.64	0.99	320	0.36
Leg SaO_2 < 90	19	0	0.53	1.0	530	0.47

LR$^+$ positive likelihood ratio; LR$^-$ negative likelihood ratio; CHD, congenital heart disease; SaO_2, oxygen saturation.

screening pulse oximetry performed at 24 hours of age or before hospital discharge may result in earlier identification of newborns with LHOD or complete mixing defects.

During the study period, arm or leg pulse oximetry was abnormal in 85% (11/13) of newborns admitted to our hospital with suspected CHD and later diagnosed with LHOD. One newborn with normal oxygen saturation and aortic stenosis presented with a loud murmur but was otherwise well on the day of birth. By 24 hours of age, however, saturation was 98% in the arm and 91% in the leg. The newborn underwent balloon valvuloplasty on day 2 of life. Among newborns in the WBN, the test also detected 1 newborn at approximately 24 hours of age with previously unsuspected coarctation of the aorta. This newborn had a normal cardiac examination on admission the nursery, but abnormalities on the screening test prompted a reevaluation.

Conversely, the potential utility of an effective early screening test was emphasized during the study by the case of a newborn discharged from the WBN with a normal cardiac examination. This newborn did not undergo oxygen saturation screening, and LHOD was missed until 15 days of age when the baby was readmitted to the hospital with absent femoral pulses and 2-day history of poor feeding. Echocardiography confirmed critical coarctation of the aorta. Future studies will help determine whether all babies with similar defects could be detected by newborn screening oximetry.

Although not the focus of this study 81% of newborns with any CHD had abnormal screening pulse oximetry using a leg saturation cutoff point of less than 92%. This test has the potential to improve early detection of cyanotic CHD, some of which is sufficiently mild to escape detection in the first several days after birth. Newborns with LHOD and associated malformations leading to a large left-to-right shunt might have normal oxygen saturation in the leg, and it is important to recognize that this test will miss some newborns with LHOD.

An important limitation of this study was that not all newborns with abnormal screening tests underwent echocardiography, nor was clinical follow-up available for many of these children. We therefore are unable to confirm the true number with false positive oximetry screening tests. These data will have to be generated in a larger study that includes echocardiographic assessment of those with abnormal oxygen saturation in the leg. Despite the incomplete follow-up data, we believe it is unlikely that a significant number of newborns with CHD went undetected. As the primary referral center for pediatric cardiac surgery in this region, we anticipate that newborns would have returned to this institution if subsequently found to have CHD. We did not identify any children who presented with CHD after being discharged with a normal screening test.

This study leaves several other important questions unanswered. The optimal time to perform screening pulse oximetry is still unclear. In a recent study of 90 newborns, O'Brien et al. [9] reported that median baseline oxygen saturation remained stable until 20 to 24 hours of age, when it became significantly lower. Our data show that oxygen saturation in the leg increases during the first day of life, consistent with a reduction in right-to-left flow through the ductus arteriosus as it closes (Fig. 1).

Echocardiography studies have shown that complete closure of the ductus arteriosus occurs in less than 10% of full-term newborns before 12 hours of age, in 50% of newborns by about 24 hours, and in 81% of newborns by 48 hours [4, 6, 10]. Performing pulse oximetry screening at less than 6 hours of age when some newborns may still have persistent ductal shunting, could result in false positives and in unnecessary expenditures for cardiac consultation and echocardiographic studies. These data emphasize the need for more detailed study of newborns with abnormal screening tests before the utility of screening oximetry can be confirmed. In the course of a larger study, it will be important to evaluate the psychological burden of screening on the families of normal newborns.

In summary, we have shown that the near simultaneous measurements of arm and leg oxygen saturation require little extra time and can be performed during routine care by nursery staff. The procedure is noninvasive and safe. A large prospective study of universal newborn screening would indicate if pulse oximetry could serve as a suitable screening test for LHOD and CHD.

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