REVIEW

Should pulse oximetry be used to screen for congenital heart disease?

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Ten studies (44 969 newborns, 71 severe defects) evaluating the usefulness of neonatal pulse oximetry (PO) screening in timely detection of congenital heart disease (CHD) were reviewed. PO showed a high specificity (99.9–99.99%), and the overall rate of detection of 15 individual defects with PO was 72% (range 46–100%), exceeding that of the clinical examination 58% (9–86%). Similar results were obtained for cyanotic CHD (89% *v* 69%, respectively). Without PO, discharge of apparently healthy infants with unknown CHD was 5.5 times and 4.1 times more likely in cyanotic CHD and all serious CHD, respectively. The paper describes the technical and practical details of first day and later screening. Diagnosis is reached earliest with first day screening, but it requires more resources. PO screening is not sensitive enough to serve as an independent screen, but along with the clinical examination it helps minimise the morbidity and mortality associated with discharge without diagnosis. Further research is needed for precise delineation of populations that would benefit from PO screening.

Timely diagnosis of congenital heart disease (CHD) is challenging but critical. Children with such life-threatening defects may not initially have symptoms or the symptoms may be vague, and the condition is not detected on routine clinical examination in the majority of cases.[1–3] In the UK alone, up to 100 otherwise healthy infants may be dying every year from undiagnosed CHD.[4]

Strategies that have been suggested to improve early detection of CHD include prenatal ultrasound screening, prolonged hospital stay after delivery,[5] one or more postdischarge examinations during the first[5 6] or the following two weeks of life,[2] and training clinicians[5] to detect ''silent'' CHD. However, all these strategies are cumbersome and costly and their value is doubtful. Prenatal ultrasound detects only 25% of CHD (16% without syndromes),[7] and the effectiveness of all postnatal interventions remains questionable.[1] Despite the apparent need, until recently, there were no other means for early detection of CHD.

Pulse oximetry is the first, appropriately simple method to have been used for universal screening of CHD, and the earliest reports (abstracts) on pulse oximetry screening were published in 1995.[8 9] Currently, a wealth of experience based on screening of 45 000 newborns is available, making it timely to review the usefulness of the screen.

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PULSE OXIMETRY IN THE NEONATAL PERIOD

Every baby is cyanotic until birth. The first breaths and increasing pulmonary circulation induce a rapid rise in pulse oximetry oxygen saturation (Spo_2). The mean preductal (right hand) and postductal (foot) Spo_2 of healthy newborns at the age of 2 min has been shown to be 73% (range 44–95%) and 67% (34–93%), respectively.[10] In that study of 50 newborns, these values increased at 10 min to 92% (65–99%) and 89% (62–99%), respectively, and in every newborn both measurements reached 95% within 1 h. The reduction in the difference between preductal and postductal measurements reflects the diminishing ductal right-to-left shunt.[10] However, a slower disappearance of the shunt has been seen in 4–5% among larger populations.[11] Thus, use of Spo_2 for screening is not practical before the age of 1–2 h. Thereafter, the normal baseline Spo_2 is quite stable at about 98%, except for short periods when it is lower, in particular during feeding and apnoeic spells.[12]

PULSE OXIMETRY IN CHD

Byrne *et al* found that cyanotic CHD with obligatory or mixing cyanosis was associated with either a generally low level of Spo_2 (<88%) or a continuous preductal/postductal difference of at least 7%.[8] Ductal-dependent obstructive left heart defects often have a preductal/postductal Spo_2 difference of at least 4–5%.[13 14] However, a pure left-to-right shunt does not affect systemic oxygenation and is not detectable by pulse oximetry. These phenomena form the basis of pulse oximetry screening for CHD.

TYPES OF OXIMETER

Two types of pulse oximeter are available on the market. One measures functional Spo_2 (oxyhaemoglobin/oxyhaemoglobin + reduced haemoglobin) and the other displays fractional Spo_2 (oxyhaemoglobin/oxyhaemoglobin + reduced haemoglobin + approximated carboxyhaemoglobin and methaemoglobin). Functional Spo_2 is thought to be about 1.6–2% higher at saturation levels used as cut-offs in pulse oximetry screening.[15] If this is not taken into consideration while screening, the actual cut-off might shift by 1–2%, thus potentially increasing the false-positive or false-negative rates. However, the exact algorithms used for the calculation of fractional Spo_2 are more complex,

Abbreviations: CHD, congenital heart disease; Spo_2, pulse oximetry oxygen saturation

otherwise all fractional pulse oximeters would display a maximum of 98%, which is not the case.

Other technical differences between the various types of oximeter include signal averaging times and methods for excluding movement artefacts.[15] These are dependent on product developments aiming to minimise bias, which probably makes them less noteworthy. Adequate, up-to-date comparisons of the continually changing selection of oximeter brands, models and software upgrades on the market are not usually available. Despite the variations described above, the results of pulse oximetry screening seem rather consistent at cut-off levels of about 94–96%,[3 11 13 14 16–20] allowing for minor technical differences (table 1).

In the case of—for example, large numbers of false-positive findings, the oximeter should be exchanged for one that complies with the chosen reference; alternatively the cut-off can be revised. Future developments may produce more precise oximeters, further increasing the effectiveness of screening.[21]

PRACTICAL ISSUES

For the purpose of this review, pulse oximetry screening studies identified by a MEDLINE and other literature search until February 2006 were classified into three groups according to the method used (table 1). ''Preliminary screens'' showed wide variation in practical details such as timing, cut-offs and probe placement. In studies in the two other groups, the method used was more accurately defined and also adjusted for two slightly different populations of newborns. ''First day screens'', designed for the first 24 h of life, include measures for controlling bias related to ductal right-to-left shunting during early postnatal pulmonary hypertension. ''Later screening'' after the first 24 h allows for further practical simplification of the screening in the absence of such shunting. The wide spectrum of methods among the preliminary screen studies (table 1) is helpful for distinguishing the optimal techniques.

Age at screening

The first hour of life is not suitable for pulse oximetry screening owing to the large number of false-positive findings.[11 17] Thereafter, infants can be screened at any age, but somewhat more reliably after 2 h.[11 16] First day screening identifies CHD and some other neonatal problems, whereas later screening is more specific for CHD. The effect of the timing of pulse oximetry screening is discussed further below.

Probe site

Newborns who have obligatory or mixing cyanotic CHD have reduced Spo_2 both preductally and postductally. Defects with ductal-dependent systemic circulation exhibit higher preductal than postductal Spo_2 due to right-to-left shunting of unoxygenated blood to the lower body.[13] Pulse oximetry screening is thus most effective with postductal probe placement.

Signal quality and newborn behaviour

Technical errors are minimised by accepting only high-quality pulse oximetry signals confirmed by a good pulse signal displayed simultaneously.[15 20] Measurements should not be made when the infant is moving, crying, eating or has an apnoeic spell.[12 15] The heart rate displayed by the oximeter should be within the range expected for a calm, regularly breathing newborn (about 90–160/min). The highest stable Spo_2 value, maintained longer than the averaging time of the pulse oximetry, is recorded. In practice, the measurement usually takes a few minutes[3 11 19 20]

Cut-off

Early preliminary screen studies used Spo_2 cut-offs lower than 95% to avoid false-positive findings due to ductal shunting. Repeating the Spo_2 measurements minimises these findings,[11] making it possible to use a higher and more sensitive cut-off of 95%, also in first day screening.[11 16 20] In later screening, a cut-off of 95% (94–96%) is adequate (table 1). Cut-offs of 95–96% seem high enough to make the measurement of a preductal/postductal Spo_2 difference[8] unnecessary.

RESULTS OF SCREENING

Table 1 presents a summary of the published studies of pulse oximetry screening. Comparison of the studies was complicated by differences in the target CHD, technical details and way of expressing the results. Data from the original reports were therefore rearranged for several calculations as indicated below.

In all studies, screening detected CHD. The overall proportion of CHD detected by pulse oximetry screening was 17% (29/168), or 21% (35/168) when including cases identified by a higher

Table 1 Summary of studies on neonatal pulse oximetry screening for congenital heart defects (CHD)

			Screening technique				Identification of CHD			
								Birth hospital		
Study	No.	Target defects (n)	Age, mean or median (h)	Probe site	Normal range (%), hand–foot difference (%)	Oximeter type*	Prenatal (excluded)	Spo_2	Other	After discharge
Preliminary screens										
Gnanalingham *et al*[17]	1421	Cyanotic (1)	1.7	Foot	⩾91	Fractional	NA	1	0	NA
Hoke *et al*[13]	2876	Critical†(4)	<24	Arm + foot	⩾92, <7	Functional	NA	4	NA	0
Reich *et al*[14]	2114	Marked†(3)	>24	Arm or foot	⩾95, <4	Functional	(1)	1	1	1
Lapland Central Hospital 2000–03 (unpublished)	4354	Serious (17)	2	Arm or foot	⩾90 (⩾95)	Fractional	0	2 (6)	13 (9)	2 (2)
First day screens										
Richmond *et al*[11]	5626	All (50)	11.7	Foot	⩾95	Fractional	4	6	30	10
Arlettaz *et al*[20]	3262	All (40)	8	Foot	⩾95	Functional	11	6	NA	NA
Meberg and Brun[16]	3532	Serious (7)	6.6	Foot	⩾95	Functional	NA	0 (2 ASD)‡	6	1
Later screens										
Koppel *et al*[18]	11 281	Critical (11)	72	Foot	⩾96	Fractional	(9)	3	6	2
Rosati *et al*[19]	5292	Critical (3)	72	Foot	⩾96	NA	(NA)	2	Excluded	1
Bakr *et al*[8]	5211	All (32)	31.7	Arm + foot	⩾94	Fractional	NA	4	25	3
Total	44 969	168	1.7–72	Arm + foot	90–96		15 (+10)	29 (35)	81+	20+

ASD, atrial septal defect; NA, not available.
*Oximeter type (functional/fractional; not clearly indicated in some studies) may slightly influence the cut-offs, see text.
†''Critical'', (duct-dependent/requires operation <1 month); marked (necessitating treatment <5–12 month)
‡Defect not sought for in the study.

Table 2 Sensitivity of pulse oximetry and routine follow-up with clinical examination to detect serious congenital heart defects

Defect	Pulse oximetry, screened newborns* Positive/all (%)	Pulse oximetry, all data available Positive/all (%)	Routine follow-up and clinical examination[1] Positive/all (%)
Hypoplastic left heart	3/3 (100)	15/15 (100)	23/35‡ (66‡)
Pulmonary atresia	5/5 (100)	14/14 (100)	47/64‡ (73‡)
Ebstein's anomaly	0	1/1 (100)	NA
Transposition of great arteries	9/10 (90)	35/36 (97)	65/76‡ (86‡)
Common arterial trunk	5/6 (83)	8/9 (89)	11/18 (61)
Total anomalous pulmonary venous return	4/5 (80)	7/8 (88)	2/22 (9)
Tricuspid atresia	1/1 (100)	6/7 (86)	NA
Double outlet right ventricle	4/5 (80)	6/7 (86)	NA
Double inlet left ventricle	0	4/5 (80)	NA
Interrupted aortic arch	0	8/10 (80)	10/19‡ (53‡)
Aortic atresia or stenosis	2/2 (100)	8/10 (80)	32/58‡ (55‡)
Atrioventricular septal defect	3/4 (75)	6/8 (75)	3/19 (16)
Tetralogy of Fallot	6/11 (55)	15/21 (71)	54/72‡ (75‡)
Coarctation of aorta	5/10 (50)	13/26† (50 (62)†)	30/95‡ (32‡)
Pulmonary stenosis	4/9 (44)	6/13 (46)	65/112‡ (58‡)
Left-to-right shunts, see text			
Total	51/71 (72)	152/190 (80)	342/590 (58)

NA, not available.
*References as in table 1; †References as in table 1 and references 9, 21.
†13/21 or 62% by exclusively postductal Spo_2.
‡Includes deaths due to missed diagnosis.

alternative cut-off level of 95% at our institution and those identified outside the intended scope of the screen[16] (table 1).

Sensitivity of pulse oximetry screening

The overall sensitivity of pulse oximetry screening in serious CHD was 72%, higher than that of the clinical examination (58%, table 2). As a group, defects with obligatory cyanosis or cyanosis with mixing lesions (the first eight entities in table 2) were more often detected by pulse oximetry than by the clinical examination: 31/35 (89%, range 80–100%) and 148/215 (69%, range 9–86%), respectively.

Defects in which murmurs are predominantly absent (murmur present at examination in <50%; total anomalous pulmonary venous return (TAPVR)), atrioventricular septal defect, coarctation of aorta; table 2) are especially interesting, because they are most often missed clinically. Pulse oximetry clearly detected these defects more often: 63% (12/19) compared with 26% (35/136) by clinical examination. Even the chances of detecting silent coarctation of aorta were doubled when using pulse oximetry with confirmed postductal probe placement compared with clinical examination. However, in a third of newborns, coarctation of aorta was still missed. Auscultation performed better than pulse oximetry in tetralogy of Fallot and pulmonary stenosis, probably because neonates with milder disease have murmurs without any hypoxaemia, so they cannot be screened with pulse oximetry.

All 15 infants with prenatally detected CHD whose Spo_2 was reported would have been detected by pulse oximetry[11 20] but only 4/11 had a finding permitting clinical diagnosis.[20] In one study, all instances of CHD that were diagnosed with pulse oximetry were missed clinically and vice versa,[3] although some overlap was usually present. Pulse oximetry alone was more sensitive than the routine examination but a combination of these two provided the highest diagnostic sensitivity. All defects found with a sensitivity approaching 50% or greater are included in table 2. They are later referred to as ''screenable'' forms of CHD.

Pure left-to-right shunts such as ventricular septal defect, atrial septal defect, or patent ductus arteriosus should not be detected by pulse oximetry, but some of these defects (5–17%) proved to be screen positive. This happened almost at all times within the first 24 hours of life,[3 11 16 20] probably due to bidirectional shunting during early postnatal pulmonary hypertension.

Discharges with undiagnosed heart disease

The impact of pulse oximetry on discharge without diagnosis can be directly assessed only in late screening, because the number of infants in whom CHD was detected by first day screening and would have been missed clinically remains unknown. Importantly, late pulse oximetry screening reduced the numbers of discharges with an unknown serious defect in the studies with pertinent data available.[3 18] In six of the eight newborns in whom asymptomatic cyanotic CHD was not diagnosed on clinical examination (three with total anomalous pulmonary venous return; one each with pulmonary atresia, tricuspid atresia, and common arterial trunk), the condition was detected by late screening.[3 18]

All screening studies reporting discharge data were compared with an extensive clinical study (table 3).[1] Discharge with unknown cyanotic CHD was 7.8 times more common without oximetry. Although CHD was suspected in a minority of these infants, such discharge was still 5.5 times more likely without pulse oximetry when the clinical examination was normal. The corresponding figures for all severe CHD were 5.9 and 4.1, respectively (table 3).

Even the usefulness of immediately available ultrasound facilities is marginal if the primary level of detection is poor.[22] Auscultation no longer seems to ''offer the only real opportunity''[22] to detect CHD early. Pulse oximetry screening enhances the chances of early echocardiography of many CHD presenting with no murmurs. As pulse oximetry screening reduces the risk of discharge with undiagnosed CHD by at least 75%, universal screening may reduce the numbers of deaths due to unknown CHD from 100[4] to 25 or fewer every year in the UK alone. Indeed, only one such death occurred in the screening studies—an infant who was not screened.[14]

Age at diagnosis in newborns screened and not screened

The age at diagnosis was retrieved from two studies using pulse oximetry screening. In a Swiss series of 40 infants[20] (table 1),

Table 3 Frequency of diagnostic failure in congenital heart disease with and without pulse oximetry screening

Diagnostic methods	No.	Category of diagnostic failure	Cyanotic defects*		All comparable defects†	
			No.	Frequency	No.	Frequency
Clinical follow-up and routine examination[1]	300 102	Discharged undiagnosed	116	1:2587	351	1:855
		Discharged undiagnosed (normal routine examination)	82	1:3660	245	1:1224
		Death without diagnosis after routine examination	5‡		15*	1:20 006
		Death without diagnosis after discharge	2		11	1:25 008
Pulse oximetry screen, clinical follow-up and routine examination¶	40 286	Discharged undiagnosed	2	1:20 143	8	1:5036
		Death without diagnosis after routine examination	0	0	0	0
		Death without diagnosis after discharge	0	0	0	0

*Transposition of great arteries, Tetralogy of Fallot, pulmonary atresia, hypoplastic left heart, total anomalous pulmonary venous return, common arterial trunk.
†Transposition of great arteries, Tetralogy of Fallot, pulmonary atresia, hypoplastic left heart, total anomalous pulmonary venous return, common arterial trunk, pulmonary stenosis, coarctation of aorta, aortic atresia or stenosis, interrupted aortic arch, atrioventricular septal defect.
‡Includes two newborns (hypoplastic left heart, coarctation of aorta) without exact data from routine examination.
¶References as in table 1 except for 17, 20.

CHD was detected prenatally in 11, on the first day of life in 6 (by pulse oximetry), and between 2 days and 9 months (median 7 days) in the remaining 23, with approximately 60% of postnatal diagnoses being made within the first week. The Finnish sample from Lapland Central Hospital, Rovaniemi (table 1) included 48 infants with CHD (0 prenatal diagnoses, 15 first year operations, 28 simple ventricular septal defects). The age at clinical examination was 48–72 hours. Despite the lack of prenatal detection, the final diagnosis was made during the first 3 days of life in 29 (60%), before 1 week in 34 (71%), within 6 weeks in 38 (79%) and during the first year in 44 (92%). The median age at diagnosis was 3 days. The age at diagnosis in both two studies was less than when diagnoses were made without pulse oximetry.[1 6 22] Pulse oximetry screening did enhance early detection of CHD, but the overall swiftness of the diagnosis was more due to immediate echocardiography.

Two other studies report early and more accurate suspicion[23] or diagnosis[24] of CHD than usual. They also used pulse oximetry, but only when CHD was suspected. One study used experienced clinicians and a strict investigative protocol with prompt ultrasound on suspicion of CHD.[24] The most recent study found a decrease in discharges with unsuspected CHD.[23] This may be partly due to improved prenatal screening or coincidence, because the number of the clinically most challenging cyanotic defects decreased from eight (three of which probably did not present with murmurs; table 2) to two (both with probably audible murmurs; table 2) between the study periods.[23]

Table 4 Comparison of first day and later screens in detection of screenable heart defects

Findings	First day screen[11 16 20] n = 12 419		Later screen[3 18 19] n = 21 783	
Screenable defects*	36	(1:345)	11	(1:1980)
True positive	25	(1:497)	8	(1:2723)
False negative	11		3	
False positive, of these	26		3	
Other cardiac disease	5		0	
Non-cardiac disease	19		0	
Healthy infants	2		3	
True negative	12 357		21 769	
Sensitivity	69%		73%	
Specificity	99.79%		99.99%	
Positive predictive value	49%		73%	
Negative predictive value	99.91%		99.99%	
Repeat Spo_2 measurements	461 (3.7%)		0	
Negative ultrasound examination	15†		3	
Discharged undiagnosed	2–5‡		3	

*Listed in table 2.
†12 from an early study phase; similar ultrasound studies later replaced by repeat Spo_2.[11]
‡Exact data not available.

FIRST DAY OR LATER SCREENING

The results of first day and later screening (table 1) are not comparable as such. First day screening yielded more diagnoses of CHD because it is done before the routine clinical examination, and often before symptoms appear, soon leading to a diagnosis. The sensitivity of pulse oximetry screening calculated for a fixed set of severe defects was the same, regardless of the timing (table 4).

First day screening detects CHD earliest. The age at screening seems to be of little importance for the overall results (tables 1 and 4) but this comparison ignores the condition of the patient at diagnosis. A diagnosis made as early as possible is of utmost importance for some newborns, because rapid deterioration during the first day(s) of life worsens the prognosis of surgical treatment, and even that of survival.[1 13 25 26]

First day screening is done so early that even many acute or life-threatening non-cardiac problems may still be asymptomatic. Many such diseases have been detected as ''false-positive'' findings on early screening (box 1). Most of these are respiratory problems (some later requiring mechanical ventilation) and persistent pulmonary hypertension (some necessitating specific therapy).[11 20] Early-onset sepsis[16 17] and conditions inducing seizures[11 17] have also been detected as a byproduct of first day screening (box 1). Even ''non-screenable'' cardiovascular conditions such as left-to-right shunts[11 16 20] and conditions with impending circulatory failure are occasionally identified by first day screening (box 1). These ''false-positive'' findings can be regarded as an advantage, allowing earlier treatment of severe, potentially deteriorating conditions.[20]

First day screening must be repeated in 4–5% of newborns to exclude healthy babies with delayed transitional circulation.[11 20] Use of a two-point cut-off to form three groups permitted immediate echocardiography (with only 1/24 normal hearts) for all newborns with Spo_2 <90% and those with symptoms from the 90–94% group.[20] Repeat measurement within six hours showed 97% of the asymptomatic newborns with an Spo_2 of 90–94% as false positives.[11 20] Further repeat measurements for asymptomatic newborns in the following days would reduce the number of seemingly unnecessary echocardiographies indicated on the first day (table 4).

Later screening is simple because a single measurement is sufficient.[18 19] Repeat measurements were included in only one study of later screening (at a low average age of 32 hours),[3] but it was not indicated if that was necessary. Reliable Spo_2 signals

Box 1 Diseases other than screenable heart defects, identified by low Spo_2 in first day screening

Cardiac disease

- Patent ductus arteriosus (some necessitating surgery)
- Ventricular septal defect
- Atrial septal defect
- Paroxysmal supraventricular tachycardia*
- Myocardial tumour

Respiratory

- Transient tachypnoea (some later ventilated)
- Persistent pulmonary hypertension
- Spontaneous pneumothorax
- Pneumonia

Other

- Sepsis
- Septo-optic dysplasia
- Intracranial haemorrhage
- Symptomatic hypoglycaemia

*Heart rate 271/min, oximeter (programmed not to accept rates >250/min) displayed a false normal rate of 135/min (author's observation).

may be more rapidly obtained in later screening than during the first hour(s) of life.[17]

BENEFITS AND DRAWBACKS OF PULSE OXIMETRY SCREENING

Benefits

As shown above, pulse oximetry screening enhances timely diagnosis of CHD and minimises the risk and sequelae of delayed detection.[1 2 4 5] Emergency transfer and surgery carry increased risks, not to mention parental anxiety. Screening allows safer transfer and treatment before clinical deterioration.[25 26] Early diagnosis improves operative outcomes and/or decreases mortality related to most of the severe defects listed in table 3.[13 14] A UK-based study reported in 2006 confirmed this and emphasised the need for optimal screening procedures.[26] In some instances, diagnostic delay (that could be avoided by pulse oximetry) may even make the infant inoperable owing to severe heart failure or development of chronic pulmonary hypertension (Eisenmenger's syndrome).[27]

Unlike other approaches attempting an earlier diagnosis of CHD, pulse oximetry screening is an easy, quick, non-invasive and cheap test,[11 18 28] suitable for large-scale use. In the usual case (negative screen) the result is immediately available. Nurses carry out pulse oximetry reliably and are accepting of the screen as a safety improvement for newborns in their care.[20] Pulse oximetry screening fulfils the general criteria of a screening test[19 28] and is more cost effective than several established neonatal screening programmes.[11 18] The human resources and material costs of a single measurement in our unit are comparable with those of a single diaper change.

The present tendency towards earlier postnatal discharge is feared to increase discharges with undiagnosed CHD.[1 5 6 25] Pulse oximetry screening offers an effective way to minimise this risk.

Drawbacks

The main drawback of pulse oximetry screening is its limited sensitivity. Pulse oximetry does not detect left-to-right shunts or other CHD not affecting oxygenation. Its sensitivity for most of the severe defects is good, but coarctation of aorta is often missed (table 2). A recent study has suggested that the more accurate newest generation of oximeters reveal smaller ductal shunts than previously, allowing for more sensitive detection of coarctation of aorta.[21] This remains to be confirmed in true screening conditions.

Pulse oximetry screening cannot be relied on as a sole screening test. However, researchers judge both first day and later screening as valuable adjuncts to the routine clinical examination.[3 11 18–20] As such, the associated ethical and psychological concerns are similar to those of the routine examination itself. However, if an ultrasound evaluation of a positive screening result is not readily available, such issues should be specifically dealt with.[23 28] Parents should be given adequate information and follow-up[2 5 7 23 27] ensured in such circumstances.

COMBINED CLINICAL EXAMINATION AND PULSE OXIMETRY SCREENING: MANAGEMENT ASPECTS

Optimal management varies depending on the goals of pulse oximetry screening. One must first choose between prevention of discharges with unknown CHD alone (later screening) and the same combined with earliest possible diagnosis (first day screen). The latter aims at safer transfer and better operative condition in rapidly deteriorating cases, but at the price of more complicated management. Both options are acceptable. The choice may be made at organisational level to fit best with local circumstances such as postnatal treatment times, resources, or distances to surgical centres.

Management with late pulse oximetry

This is the simplest and cheapest form of pulse oximetry screening. If treatment times exceed one to two days, only a single measurement at or near discharge is needed. The lower pulmonary resistance will then have reduced the false-positive rate close to 1:10 000 (table 4), and the ratio of true and false positives to 3:1 (tables 1, 4). As shown in table 4, only 11 ultrasound examinations were needed to detect eight silent but potentially life-threatening[14 23] defects. Such rates indicate the necessity of immediate echocardiography. If this is not available, the infant may be followed up as described below.

If discharge is earlier, more false-positive readings will be obtained. These can be managed by an ambulatory repeat pulse oximetry measurement one to two days later (see below).

Management with first day pulse oximetry

Clinical examination may be best as late as possible before discharge.[6 24] Early pulse oximetry at about 2 h of age before transfer to the maternity ward fits well here, revealing CHD that would rapidly deteriorate (as well as several other conditions, box 1). The measurement can be made any time thereafter, if later time is more acceptable.[11 20] Most false positives are excluded by repeat measurements over the following days; postdischarge measurements can be made on an ambulatory basis. A single cut-off is valid,[11] and a double cut-off[20] (below) may be helpful for more rapid diagnosis:

- $Spo_2 \geq 95\%$—no further actions
- Spo_2 90–94%—check-up by attending midwife/nurse[23] (including respiratory rate, heart rate, feeding):
  - no symptoms—repeat Spo_2 (eg at 6, 12–24, 48 and 72 hours) until $\geq 95\%$

- suspicion of CHD—examination by physician. If no non-cardiac explanation for symptoms (box 1)—cardiac work-up
- if symptoms appear or Spo_2 remains <95% at 3 days—cardiac work-up

- Spo_2<90%—cardiac work-up immediate

A full cardiac work-up usually includes blood pressure measurements (all limbs), electrocardiogram (ECG), *x* rays, and oxygen tests, but only echocardiography is mandatory. Where this is not immediately available and symptoms do not call for prompt referral, clinical follow-up (eg days 2, 4, 7, 10, 14, etc; until echocardiography) can be organised as suggested for other high-risk patients.[5 6 23 27] It must be borne in mind that most of these patients have critical or serious CHD (table 4). Parents must be well informed about how to recognise symptoms of CHD and respond promptly.[23 27]

Much of the clinical follow-up could perhaps even be done by trained nurses,[23] using also blood pressure and Spo_2 measurements. Perhaps a simple blood test might become available in the future. Serum B-type natriuretic peptide concentration reveals CHD causing volume or pressure overload,[29] but its sensitivity is not known as yet, and neonatal reference values need to be refined.

CONCLUSION

Pulse oximetry screening meets the general requirements for a screening test,[18] provided the prevalence of severe CHD is not negligible due to successful prenatal screening. It has limitations, which must be borne in mind, but it does improve early detection of CHD. As such, it is a suitable adjunct to the routine clinical examination. However, randomised studies are needed to confirm and clarify the performance of the pulse oximetry screen in different conditions—for example, variable levels of prenatal screening.

Management and material costs are especially low in late screening and higher with the early screen. Although the available data permit temporary or experimental organisational decisions on pulse oximetry screening, sound cost–benefit analyses for large-scale, established screening cannot be done without further research. Increasing accuracy of prenatal screening reduces the need and usefulness of pulse oximetry screening.[18] More detailed data from a variety of circumstances and ''second-look'' decisions during the development of prenatal screening may be needed for preventing wastage of resources. However, as long as newborns are discharged with an unknown CHD, risks will be there. Pulse oximetry screening cannot eliminate all the present risks, but it can prevent most at an acceptably low cost.

Competing interests: None declared.

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