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Accelerated pulse oximetry in the neonate: a proposal

Abstract

Pulse oximetry is used as a screening test for critical congenital heart disease. Current algorithms involve one or to re-screenings for some patients. That may take 2-8 hours before a diagnosis is made. I propose that after the initial screen, patients who do not pass the test be given a short exposure to high oxygen concentration in order to eliminate lung disease as a cause of the desaturation.

Keywords: oxygen, lung disease, pulmonary hypertension, critical congenital heart disease

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Introduction

Critical congenital heart disease (CCHD) refers to those lesions that if untreated cause early death, usually under 1 month of age. Most of these are ductus-dependent lesions, and they include most of the cyanotic congenital heart diseases (principally transposition of the great arteries) and the severe left ventricular outflow tract obstructions (principally coarctation of the aorta. Fetal ultrasound detects congenital heart disease in many infants, or they will present soon after birth with severe symptoms, abnormal murmurs, or cyanosis. However, some congenital cardiac lesions have unimpressive murmurs, and the rest of the physical examination may apparently be normal; cyanosis is often undetectable unless the arterial oxygen saturation is under 85%, or even lower if the neonate is anemic. These neonates appear well, are sent home, and either die or become severely ill when the ductus arteriosus closes. Even if they do eventually have corrective surgery, delayed diagnosis makes their risk of dying in hospital high or they may require longer periods of expensive intensive care. Because in these neonates arterial oxygen saturation in the descending aorta is often below normal, pulse oximetry offers a way of detecting otherwise inconspicuous CCHD.[1–3] The standard algorithm as adapted from Kemper et al.,[4] is presented in Figure 1.

The one difference from the original algorithm is that the delay between screening periods has been varied from 1 to 4 hours, a variant frequently used. The horizontal line in the figure will be discussed later. This algorithm is effective, and yields a very small number of false positive and false negative tests when implemented correctly. The algorithm is often not followed correctly or its results may be misinterpreted.[5,6] What is apparent from the algorithm is that it is entirely passive. Apart from setting up the apparatus, all the medical personnel do is wait and observe those patients with indeterminate results. This takes up time that can be as much as 8 hours from start to finish.

It might be possible to shorten the time to a decision if we intervened in this process. If the first screen is not negative, thereby excluding all CCHD except for some left heart obstructive lesions,[7] we can give the neonate a short (1-2 minute) exposure to a high concentration of oxygen. This can be done by adding 100% oxygen to the isolette, putting the infant's head in a disposable plastic hood, or using blow-by oxygen with a T piece resuscitator device. With any of these methods of delivery the inspired oxygen will not be 100%, but will still be above normal, The effect of blowing oxygen into the isolette will vary with the isolette and how much leakage there is from it, but one study[8] found that giving oxygen at 7 l/min produced an oxygen concentration in the isolette of about 50%.

The consequences of giving supplemental oxygen can be estimated from the alveolar air equation:

$PAO_2=(PB-PH_2O) \times FiO_2-PCO_2/R$

Where PB = barometric pressure at sea level ≈76- torr, PH_2O-water vapor pressure =47 torr,

FiO_2 = inspired oxygen fraction, PCO_2=carbon dioxide tension, assumed to be 40 torr, and R=gas exchange ratio assumed to be 0.8. The two last components have some variability, but have little effect on the final result.

With these assumptions, when FiO_2=0.5 (50%), PAO_2=(760-47) x0.5–40/0.8≈300 torr. This is sufficient to produce an arterial oxygen tension of at least 250 torr, and the resultant oxygen saturation will be almost 100%. Small differences in the variables used above will not materially affect the oxygen saturation of arterial blood. In fact, an arterial oxygen tension of 150 torr will fully saturate fetal or adult hemoglobin, and this can be achieved with an alveolar oxygen concentration using an FiO_2 of 0.28 (28%).

What effect will this increased ambient oxygen have?

An arterial oxygen saturation below 95% at sea level is likely to be due to congenital heart disease with a right-to-left shunt, to lung disease (usually parenchymal due to pneumonia or retained lung fluid but possibly due to hypoventilation), or persistent pulmonary hypertension of the neonate. If hypoventilation is causing the desaturation, increased alveolar oxygen concentration will cause oxygen saturation to rise to 100% The same will occur with moderate. parenchymal lung disease although the arterial oxygen tension will still be below normal.[9] Both pulmonary hypertension and most forms of CCHD have right to left shunts that bypass the alveoli, so that the

resulting arterial desaturation is insensitive to alveolar concentration. There might be a slight increase in arterial oxygen saturation because of an increase in dissolved oxygen in the plasma, but failure of arterial saturation to rise to 100% will indicate strongly the presence of a fixed right to-left shunt due to CCHD or severe persistent pulmonary hypertension. By adding brief oxygenation to the procedure, we can terminate the test after the first results are obtained (above the horizontal line in Figure 1) and obtain enough information to proceed to discharge or echocardiography.

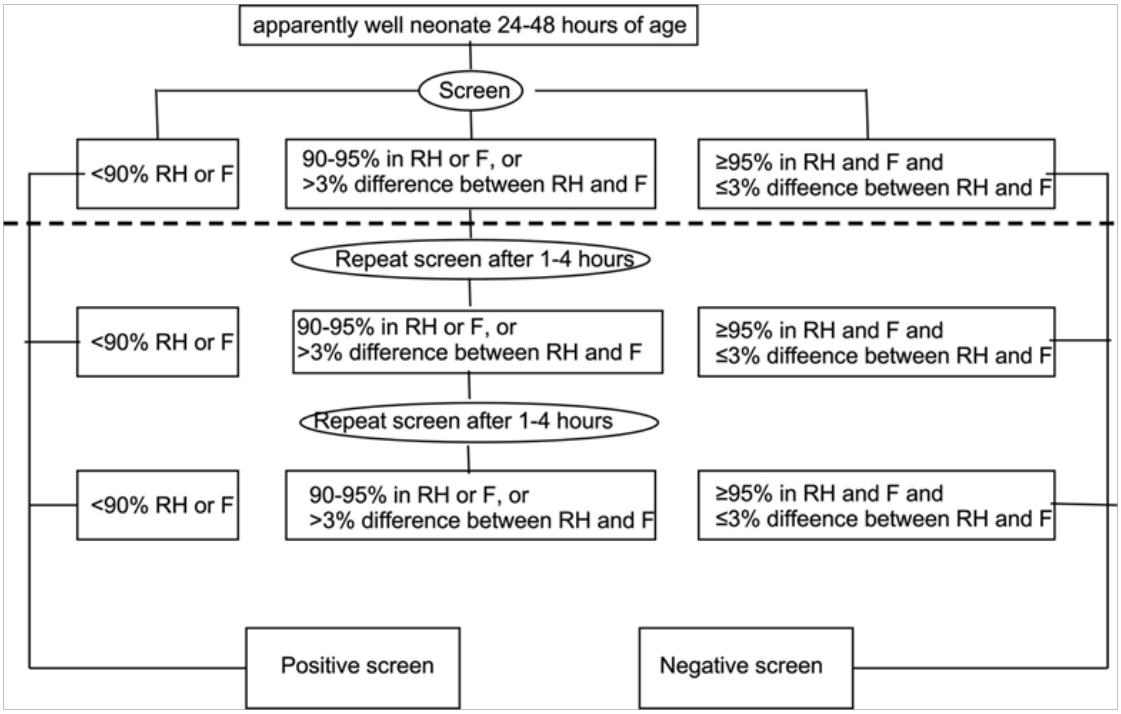

Figure 1 Pulse oximetry algorithm.

Conclusions

After the initial pulse oximetry screen, the tine to diagnosis can be shortened by giving brief exposure to a high oxygen concentration. The advantages of an accelerated method are:

a. The protocol is simpler and therefore less likely to be implemented incorrectly.

b. A course of action is determined without having to wait 2-8 hours. This is often helpful for parents.

c. Less nursing time is required than to complete the standard algorithm.

Practical issues

a) Pulse oximetry is an addition to careful clinical examination.

b) Some parenchymal lung disease is so severe that the response to oxygen administration is unimpressive. These neonates, however, will deeply cyanotic and display abnormal signs on clinical examination such as tachypnea, retractions, and rales. These are not usually candidates for diagnostic pulse oximetry.

c) False negative pulse oximetry does occur, either because of small amounts of right-to-left shunting, with some left heart obstructive lesions (coarctation of the aorta, interrupted aortic arch or critical aortic stenosis), or occasionally for technical reasons. See Hoffman.[10]

d) The choice of using an isolette, hood or T piece depends on the ease with which nurses can use these systems.

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None.

Conflicts of interest

The authors declare there are no conflicts of interest.

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References

1. De-Wahl Granelli A, Wennergren M, Sandberg K, et al. Impact of pulse oximetry screening on the detection of duct dependent congenital heart disease: a Swedish prospective screening study in 39,821 newborns. *BMJ*. 2009;338:a3037.

2. Meberg A, Andreassen A, Brunvand L, et al. Pulse oximetry screening as a complementary strategy to detect critical congenital heart defects. *Acta Paediatr*. 2009;98(4):682–686.

3. Thangaratinam S, Brown K, Zamora J, et al. Pulse oximetry screening for critical congenital heart defects in asymptomatic newborn babies: a systematic review and meta-analysis. *Lancet*. 2012;379(9835):2459–2464.
4. Kemper AR, Mahle WT, Martin GR, et al. Strategies for implementing screening for critical congenital heart disease. *Pediatrics*. 2011;128(5):e1259–1267.
5. Kochilas LK, Lohr JL, Bruhn E, et al. Implementation of critical congenital heart disease screening in Minnesota. *Pediatrics*. 2013;132:e587–594.
6. Mahle WT, Newburger JW, Matherne GP, et al. American Heart Association Congenital Heart Defects Committee of the Council on Cardiovascular Disease in the Young CoCN, Interdisciplinary Council on Quality of C, Outcomes R, American Academy of Pediatrics Section on C, Cardiac S, Committee on F, Newborn: Role of pulse oximetry in examining newborns for congenital heart disease: a scientific statement from the American Heart Association and American Academy of Pediatrics. Circulation 2009;120:447–458.
7. Mellander M, Sunnegardh J. Failure to diagnose critical heart malformations in newborns before discharge--an increasing problem? *Acta Paediatr*. 2006;95(4):407–413.
8. Clair S, Touch SM, Greenspan JS. Supplemental oxygen to the nonventilated neonate. *Neonatal network*. 2001;20(6):39–46.
9. Comroe JHJ, Forster RE, Dubois AB, et al. The Lung, 2nd ed. Chicago, Year Book Medical Publishers, Inc, 1962.
10. Hoffman JIE. False negative diagnoses of critical congenital heart disease with screening neonatal pulse oximetry. *Journal of Neonatal-Perinatal Medicine*. 2019.