How Cyanotic Is Too Critical?

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이 형 두

- Critical
  - 비평(criticism)의, 평론의; 비판적인; 비판력이 있는; 혹평의, 흠을 잡는
  - 위기(crisis)의, 위급한, 아슬아슬한; 결정적인, 중대한; (병이) 위독한, 위급한
  - 임계(臨界)의

- How Cyanotic Is Too Critical?
- 허용되는 청색증의 한계는?
- 얼마나 심한 청색증은 너무 위험한가?
- 얼마나 새파래지면 너무 비판적인가?

Cyanosis or Hypoxemia?

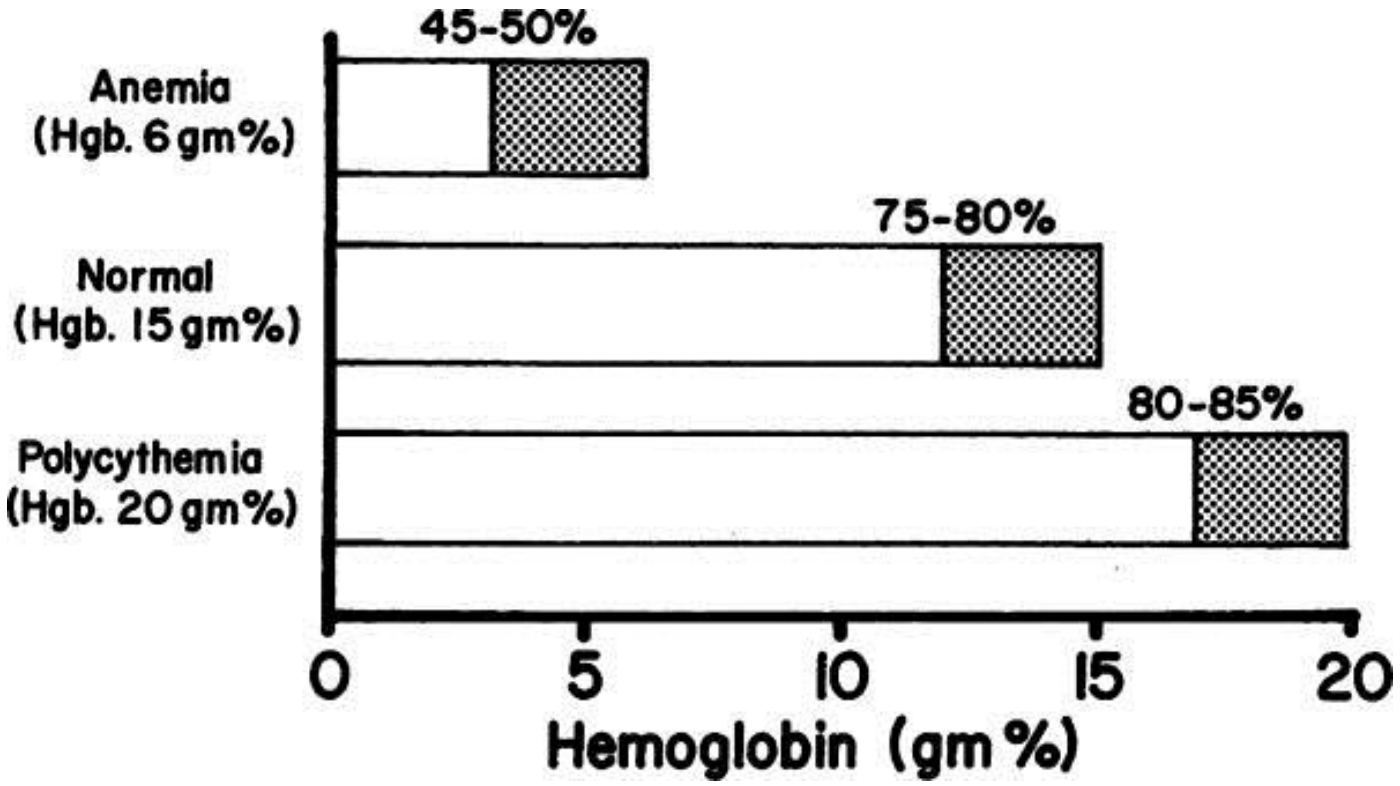
- Cyanosis : bluish discoloration of skin or mucosa resulting from the presence of deoxygenated hemoglobin in the circulation.
- Hypoxemia : low PaO_2 than normal (80-100 mmHg)
- Hypoxia : failure of oxygenation at the tissue level, metabolic acidosis due to anaerobic metabolism.

- CYANOSIS
  - derived from the Greek, dark blue
  - Bluish discoloration of skin and mucous membrane
- Descriptions of cyanopathia or *Morbus caeruleus* (cyanosis) - medical literature since the time of Hippocrates
- De Senac (1749), personal physician to King Louis XV - the first explanation of the pathogenesis of cyanosis
- Christen Lundsgaard(1919) : actually quantified the amount of deoxygenated hemoglobin, required to produce that bluish discoloration

Cyanosis

- Resulting from inc conc of reduced Hb to about 5 g/100ml in cutaneous vein
  - Normally about 2 g/100ml of reduced Hb is present in the venules so that an additional *3 g/100ml of reduced Hb in arterial blood* produces clinical cyanosis.

Influence of Hb levels on clinical recognition of cyanosis



- Cyanosis as a tool for detecting arterial hypoxemia is neither sensitive nor specific .
- Comroe and Botelho studied a group of normal subjects breathing various concentrations of oxygen. Definite cyanosis was not apparent to 25% of observers even at arterial oxygen saturations of 71 to 75% (PaO_2 35 to 40 mm Hg).
- Am J Med Sci 1947 ;214 :1-6.

- Sg & Sx of hypoxemia other than cyanosis
  - Increased ventilation with dyspnea and tachypnea
  - Sympathetic nervous system stimulation → restlessness, sweating, elevation of blood pressure, and tachycardia
  - In severe hypoxemia, impaired cerebral oxygenation → confusion or coma

How Cyanotic Is Too Critical?

How hypoxemic is too critical?

How hypoxemic is too critical in cyanotic congenital heart disease?

- Normoxemia in PaO_2, SaO_2
  - while breathing air at sea level
  - PaO_2 : 80-100 mmHg, *NEJM* 1998; 339:1063–1072
  - SaO_2 : > 94%, *Thorax* 2008; 63(Suppl 6):1–68
  - Considerable interindividual variability may exist
- To make a diagnosis of hypoxemia
  - PaO_2 : 60-75 mmHg
- Refractory hypoxemia
  - PaO_2/FiO_2 ratio < 100
  - e.g., PaO_2 < 60 mmHg while receiving 60% oxygen

TABLE 1.

Causes of Hypoxemia in Children

- **Pulmonary venous desaturation**
  - With high A-a gradient
    - Severe parenchymal lung disease (eg, acute respiratory distress syndrome, pulmonary hemorrhage)
    - Lung disease with diffusion impairment (eg, interstitial lung disease)
    - Intrapulmonary right to left shunting (eg, atelectasis, pulmonary arteriovenous malformations
  - With low A-a gradient (eg, central hypoventilation, opioid overdose, parenchymal lung disease such as pneumonia)
- **Extrapulmonary right to left shunting**
  - Cyanotic congenital heart disease with decreased pulmonary blood flow and right to left shunting (eg, tetralogy of Fallot, pulmonary atresia)
  - Pulmonary hypertension, primary or secondary, with right to left shunt at the level of ductus or patent foramen ovale (eg, primary pulmonary hypertension, Eisenmenger syndrome)
- **Transposition physiology**
  - (eg, D-transposition of great arteries, double outlet right ventricle with malposed great vessels and subpulmonary ventricular septal defects)
- **Hemoglobin disorders with decreased oxygen affinity**
  - (eg, methemoglobinemia)

Pediatr Ann. 2015;44(2):76-80

TABLE 2.

Diagnostic Testing of Cyanosis in Children

Test	Pulmonary Parenchymal Disease	Intra- or Extrapulmonary Right to Left Shunt	Central Hypoventilation	Transposition Physiology	Hemoglobin Disorders
Respiratory distress	Present; may have fever	No	No; apnea/hypoventilation	Mild distress, usually tachypnea due to increased PBF	No
Cardiac examination	Normal	May have single S2, RV heave, thrill, and murmurs	Normal	Single S2, flow murmur, RV heave	Normal
Chest X-ray	Pulmonary pathology	Variable cardiac silhouette; usually clear lung fields	Normal	Egg-on-end appearance, pulmonary venous congestion +/-	Normal
Differential saturation (preductal vs postductal)	Absent	Present if right to left shunt at ductus; postductal < preductal	Absent	Postductal > preductal	Absent
Complete blood count	Elevated white cell count	Polycythemia if chronic	Normal	Polycythemia if chronic	Normal
Arterial blood gas on 100% FiO_2	PaO_2>150 mm Hg PCO_2 variable	PaO_2<150 mm Hg Normal PCO_2	PaO_2>150 mm Hg, usually much higher; elevated PCO_2	PaO_2<150 mm Hg, usually <50 mm Hg; normal PCO_2	Normal PaO_2 and PCO_2

Abbreviations: FiO_2, fraction of inspired oxygen; PaO_2, partial pressure of oxygen in a systemic artery; PBF, pulmonary blood flow; PCO_2, partial pressure of carbon dioxide; RV, right ventricle.

TABLE 1. Causes of Hypoxemia and Their Effect on the Alveolar-Arterial Oxygen Difference

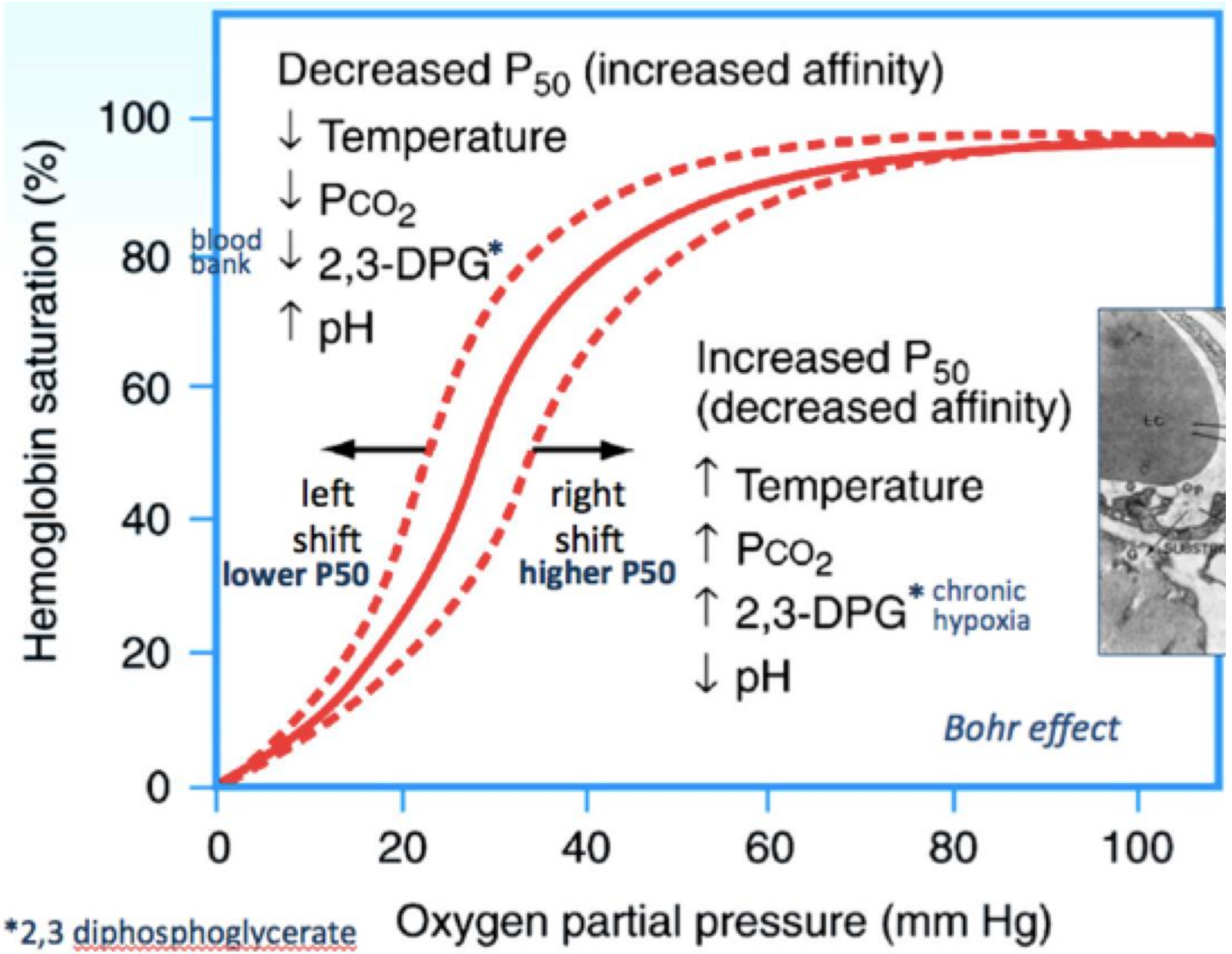
Cause of Hypoxemia	Effect on $P(A\text{-}a)O_2$	Comment
Reduced FIO_2 (or PIO_2)	Unchanged or reduced	Resolved by increasing FIO_2 (or PIO_2)
Hypoventilation	Unchanged	Causes included pharmacological, neurological, and muscular weakness. Alleviated by increasing FIO_2
Ventilation–perfusion mismatch	Increased	Commonest cause of hypoxemia in the critically ill. Alleviated to some extent by increasing FIO_2
Right-to-left shunt	Increased	Anatomical or physiological. Cannot be alleviated by increasing FIO_2
Diffusion limitation	Increased	Rare cause of hypoxemia that can be alleviated by increasing FIO_2

TABLE 2. Proposed Terms for the Categorization of Hypoxemia Based on Physiological Responses to the Duration of Its Development

Term	Description
Acute hypoxemia	A rapid decline in arterial oxygenation developing over < 6 hrs (e.g., acute upper airway obstruction)
Subacute hypoxemia	Reduced arterial oxygenation occurring in 6 hrs to 7 days (e.g., pneumonia)
Sustained hypoxemia	Reduced arterial oxygenation for 7–90 days (e.g., prolonged acute respiratory distress syndrome, high altitude climbing expeditions)
Chronic hypoxemia	Prolonged reduction of arterial oxygenation for over 90 days (e.g., chronic obstructive pulmonary disease)
Generational hypoxemia	Cross-generational reduced arterial oxygenation (e.g., Tibetan highland residents)

In the absence of any universally accepted terminology describing the time-related differences in responses to hypoxemia, the proposed criteria are based upon human physiological adaptations to hypoxemia.

Crit Care Med 2013; 41:423–432


Decreased P_{50} (increased affinity)
↓ Temperature
↓ P_{CO_2}
↓ 2,3-DPG*
↑ pH
blood bank
Increased P_{50}
(decreased affinity)
↑ Temperature
↑ P_{CO_2}
↑ 2,3-DPG*
↓ pH
* chronic hypoxia
left shift lower P50
right shift higher P50
Bohr effect
Hemoglobin saturation (%)
100
80
60
40
20
0
0
20
40
60
80
100
Oxygen partial pressure (mm Hg)
*2,3 diphosphoglycerate

- Normal partial pressure of oxygen in alveoli (P_AO_2) $\doteqdot$ 100 mmHg at sea level in room air
- Partial pressure of oxygen in systemic artery (PaO_2) $\doteqdot$ 80 mmHg at sea level in room air
- Normal alveolar-arterial oxygen gradient (A-a gradient) $\doteqdot$ 20 mmHg

- With 100% oxygen, the alveolar P_AO_2 at sea level is greater than 500 mmHg (P_AO_2 = 760 × fraction of inspired oxygen [FiO_2] − 1.2 × partial pressure of carbon dioxide [$PaCO_2$)]). If the A-a gradient is normal (approximately 20 mmHg), then the pulmonary venous partial pressure of oxygen (PO_2)should be close to 500 mmHg.

- $P_AO_2 = FiO_2 \times (Pb - PH_2O) - (P_ACO_2/R)$.

  P_AO_2 : the mean alveolar oxygen pressure.

  FiO_2 : the fractional concentration of inspired oxygen. 0.21 at room air

  Pb : the barometric pressure (760 mmHg at sea level).

  PH_2O : the water vapor pressure (47 mmHg at 37°C).

  $PaCO_2$: the alveolar carbon dioxide tension. assumed to be equal to arterial PCO_2 .

  R : the respiratory quotient, approximately 0.8 at steady state on standard diet.

- Normal $P_AO_2 = FiO_2 \times (Pb - PH_2O) - (PACO_2/R)$
  $= 0.21 \times (760 - 47) - (40/0.8) = 100$ mmHg.
- In young person, the A-a oxygen difference <10 mmHg. The A-a oxygen difference increases with age.
- A normal $P(A\text{-}a)O_2$ is a value less than one half your age, with a maximum of 25 mmHg.
- Respir Care 2014;59(10):1590 –1596

- PaO_2 /FiO_2 is a rough estimate of shunt fraction.

  If PaO_2 /FiO_2 is < 200, shunt fraction > 20%,

- PaO_2 /FiO_2 of > 200 indicates a shunt fraction of <20%.

- Crit Care Med. 1983;11:646–9

Global compensatory mechanism in tissue hypoxia

- Acute
  - regional pulmonary vasoconstriction to improve V/Q matching
  - hyperventilation to improve P_AO_2
  - acidosis related right shifting of the oxyhemoglobin dissociation curve to facilitate O_2 unloading
  - increase in cardiac output
- Chronic
  - increase in red blood cell production → polycythemia
    - polycythemia → blood viscosity↑ → impair blood flow and tissue oxygen delivery through capillaries

- Respir Care 2014;59(10):1590 –1596

Cellular level compensatory mechanism in tissue hypoxia

- RBC 2,3-DPG↑ → facilitate O_2 unloading in the tissues
- hypoxia-inducible factors (HIFs) activation → upregulation of erythropoietin, angiogenic factors, and vasoactive mediators
- glycolytic enzymes activation → anaerobic metabolism (with resulting lactate production)
- mitochondrial hibernation-like phenomenon → oxygen demands↓
  - deactivation of mitochondrial biogenesis and down-regulation of mitochondrial uncoupling → improved efficiency of ATP production *FASEB J* 2012;26: 1431–1441

- Following exposure to moderately prolonged hypoxia, cultured cells demonstrate a 40 to 60% reduction in cellular oxygen consumption (VO_2) secondary to the down-regulation of “non-essential” cellular processes
- *Am J Physiol* 1993; 265:L395–L402, *Am J Physiol* 1996; 270:L44–L53

- Severe hypoxemia → cellular hypoxia, organ dysfunction, and death.
- The degree of organ dysfunction
  - ← rapidity of onset
    severity
    duration of hypoxemia
    individual susceptibility

- With proper compensatory mechanisms, mammalian cells can thrive with a PaO_2 lower than the traditional clinical thresholds of 55–60 mmHg.
- Classic examples
  - The ability of humans to live at very high altitudes with a PaO_2 of 50 mmHg
  - Stories of stowaways surviving in airplane landing gear assemblies for many hours at an altitude of 35,000 feet (CNN website, April 21, 2014).


Courtesy The Maui News/Chris Sugidono
COURTESY THE MAUI NEWS/CHRIS SUGIDONO
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DEPARTMENT OF TRANSPORTATION
DEVELOPING STORY
THE SITUATION ROOM
TEEN SURVIVES FLIGHT TO HAWAII IN LANDING GEAR?
LIVE CNN
CIVILIANS KILLED IN STRIKE ON SATURDAY, OFFICIAL SAYS ▶ GOVT


San Jose
CALIFORNIA
COLORADO
NEBRAS
OKLA
PACIFIC
OCEAN
MAUI
HAWAII


HAWAIIAN
HAWAIIAN AIRLINES FLIGHT45: 38,000 FT.
MT. EVEREST SUMMIT: 29,029 FT.
ALTITUDE

- *JAMA*1970; 211:1815–1817
- 22 clinical cases of profound hypoxemia (PaO_2 < 20.3 mmHg), 13 of the patients survived, ten of whom were seemingly unaffected by the event.
- The lowest reported PaO_2 was 7.5 mmHg, in a 20-yr-old male patient breathing room air following a heroin overdose yet he made an unremarkable recovery.

- Hypoxic brain injury in the absence of hypoperfusion
- Ischemic cerebral injury in post-cardiac arrest.

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Brain Volumetrics, Regional Cortical Thickness and Radiographic Findings in Adults with Cyanotic Congenital Heart Disease ☆

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- Adults with chronic neurocyanosis due to congenital heart disease
- Reduced brain volume as well as specific foci of cortical thickness reduction

Critical level?

RELATIONSHIPS OF PYRUVATE AND LACTATE DURING ANAEROBIC METABOLISM. III. EFFECT OF BREATHING LOW-OXYGEN GASES [1]

By WILLIAM E. HUCKABEE

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(Submitted for publication August 3, 1957; accepted September 26, 1957)

When the oxygen supply to tissues is impaired progressivly, as in continuous reduction of the oxygen content of alveolar air, oxygen tensions in various other parts of the gas transport system proceed to fall progressively also. At some stage in the course of this unbroken continuum a state of "hypoxia" presumably may be said to exist. It is, in any event, a useful concept to suppose that it does, inasmuch as symptoms and dysfunction of organs may appear in the course of diseases of the oxygen transport systems which might be accounted for by hypoxia.

If hypoxia is induced by all diminutions of allfallen below the rate of oxygen requirement. It seemed possible that if this discrepancy between rates of supply and demand in tissues were taken as the proper indication of physiologic hypoxia, a distinct starting point of hypoxia could be identified in the course of progressive pulmonary or circulatory impairment. In the present study, experimental subjects and animals were given various low oxygen gas mixtures to breathe while anaerobic metabolism, as previously defined (1), was estimated at various blood oxygen tensions.

METHODS

J Clin Invest 37:264-271, 1958

SUMMARY AND CONCLUSIONS

1. Blood total lactate concentration bears no relationship to severity of respiratory hypoxia, but calculated "excess lactate" corresponds closely to the magnitude of the O_2-debt developed in this as in other types of hypoxia.

2. Progressive reduction of oxygen content of inspired air in human subjects and anesthetized animals leads to gradual alteration in blood oxygen, pH, lactate and pyruvate; but excess lactate is absent until $F_{I_{O_2}}$ is reduced to a critical value of about half (arterial blood oxygen saturation reduced to 60 to 74 per cent, P_{O_2} to 26 to 32 mm. Hg) and thereafter is produced in increasing amounts as oxygen supply diminishes.

3. It is suggested that a wide range of hypoxemia, including the range of visible cyanosis in many subjects, is not associated with hypoxia or deficiency of oxygen in body tissues, and that caution should be exercised in ascribing symptoms or signs to this particular aspect of pulmonary or circulatory disease.

METABOLIC ACIDOSIS IN CHILDREN WITH SEVERE CYANOTIC CONGENITAL HEART DISEASE

Norman L. Gootman, M.D., Emile M. Scarpelli, M.D., *and* Abraham M. Rudolph, M.D.

Department of Pediatrics, Albert Einstein College of Medicine, New York 61

SEVERE DEGREES of right ventricular outflow obstruction may result in marked decrease of pulmonary blood flow with, in the presence of a right-to-left shunt, little associated decrease in systemic blood flow. A similar decrease of effective pulmonary flow may occur when there is an inadequate communication between the systemic and pulmonary circulations, as in transposition of the great arteries. Although several investigators have studied various aspects of acid-base balance in older children with cyanotic congenital heart disease who were in no distress,[1-4] little information is available prior to catheterization. The catheterization procedure was carried out from the right saphenous vein, as previously described.[5] In all instances it was possible to manipulate the catheter into one of the pulmonary veins during the procedure. At this stage a needle was inserted into the previously exposed right femoral artery. Blood samples were obtained simultaneously from pulmonary vein and femoral artery in oiled syringes, heparinized with a neutral heparin solution.

The diagnoses after cardiac catheterization and angiography were tetralogy of Fallot (Patient J.C.); pulmonary atresia, ven-

Pediatrics 31:251-4, 1963

TABLE I

Data on Four Children with Cyanotic Congenital Heart Disease

Patient			Diagnosis	Systemic Arterial					Pulmonary Venous					Flows (ml/min/m²)	
Initials	Sex	Age		pO_2 (mm Hg)	pCO_2 (mm Hg)	pH	HCO_3^- (meq/l)	% Sat.	pO_2 (mm Hg)	pCO_2 (mm Hg)	pH	HCO_3^- (meq/l)	% Sat.	Pulmonary	Systemic
J.C.	M	4 yr	Tetralogy of Fallot	34	37	7.24	15.2	40.0	..	..	7.4	..	..	1,100	4,500
D.G.	F	6½ mo	Pulmonary atresia; ventricular septal defect; patent ductus arteriosus	21	36	7.05	9.8	22.0	100	18	7.4	10.8	96.6	1,500	5,000
G.C.	M	5 mo	Transposition of the great vessels with patent foramen ovale	24	36	7.20	13.6	25.0	93	27	7.4	15.8	97.4	800*	4,400
E.S.	M	5 da	Severe pulmonary stenosis with patent foramen ovale	37	34	7.28	15.2	48.5	98	26	7.4	15.3	100	750	1,950

- PaO_2 < 35mmHg in severe cyanotic CHD – uncompensated acidosis

JACC Vol. 24, No. 5
November 1, 1994:1376–81

Balancing the Circulation: Theoretic Optimization of Pulmonary/Systemic Flow Ratio in Hypoplastic Left Heart Syndrome

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WILLIAM P. SANTAMORE, PHD*

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Objectives. **This study examined the effects of the pulmonary (Q_P)/systemic (Q_S) blood flow ratio (Q_P/Q_S) on systemic oxygen availability in neonates with hypoplastic left heart syndrome.**

Background. **The management of neonates with hypoplastic left heart syndrome is complex and controversial. Both before and after surgical palliation and before heart transplantation, a univentricle with parallel pulmonary and systemic circulations exists. It is generally assumed that balancing pulmonary and systemic blood flow is best to stabilize the circulation.**

Methods. **We developed a mathematical model that was based on the simple flow of oxygen uptake in the lungs and whole-body oxygen consumption to study the effect of varying the Q_P/Q_S ratio. An equation was derived that related the key variables of cardiac output, pulmonary venous oxygen saturation and the Q_P/Q_S ratio to systemic oxygen availability.**

Results. **The key findings are 1) as the Q_P/Q_S ratio increases, systemic oxygen availability increases initially, reaches a maximum and then decreases; 2) for maximal systemic oxygen availability, the optimal Q_P/Q_S ratio is ≤1; 3) the optimal Q_P/Q_S ratio decreases as cardiac output or percent pulmonary venous oxygen saturation, or both, increase; 4) the critical range of Q_P/Q_S, where oxygen supply exceeds basal oxygen consumption, decreases as cardiac output and percent pulmonary venous oxygen saturation decrease; 5) the relation between oxygen availability and Q_P/Q_S is very steep when Q_P/Q_S approaches this critical value; and 6) the percent oxygen saturation of systemic venous blood is very low outside the critical range of Q_P/Q_S and high within the critical range.**

Conclusions. **This analysis provides a theoretic basis for balancing both the pulmonary and systemic circulation and suggests that evaluating both systemic arterial and venous oxygen saturation may be a useful way to determine the relative pulmonary and systemic flows. When high systemic arterial and low systemic venous oxygen saturation are present, pulmonary blood flow should be decreased; conversely, when both low systemic arterial and venous oxygen saturation are present, more flow should be directed to the pulmonary circulation.**

(J Am Coll Cardiol 1994;24:1376–81)

Hypoplastic left heart syndrome is currently the most common cardiac malformation that results in death of the newborn infant (1). Without treatment, 95% of these infants die during the 1st

must be stabilized for an extended period. Unfortunately, many neonates die while awaiting heart transplantation.

Proper medical management is crucial if these critically ill

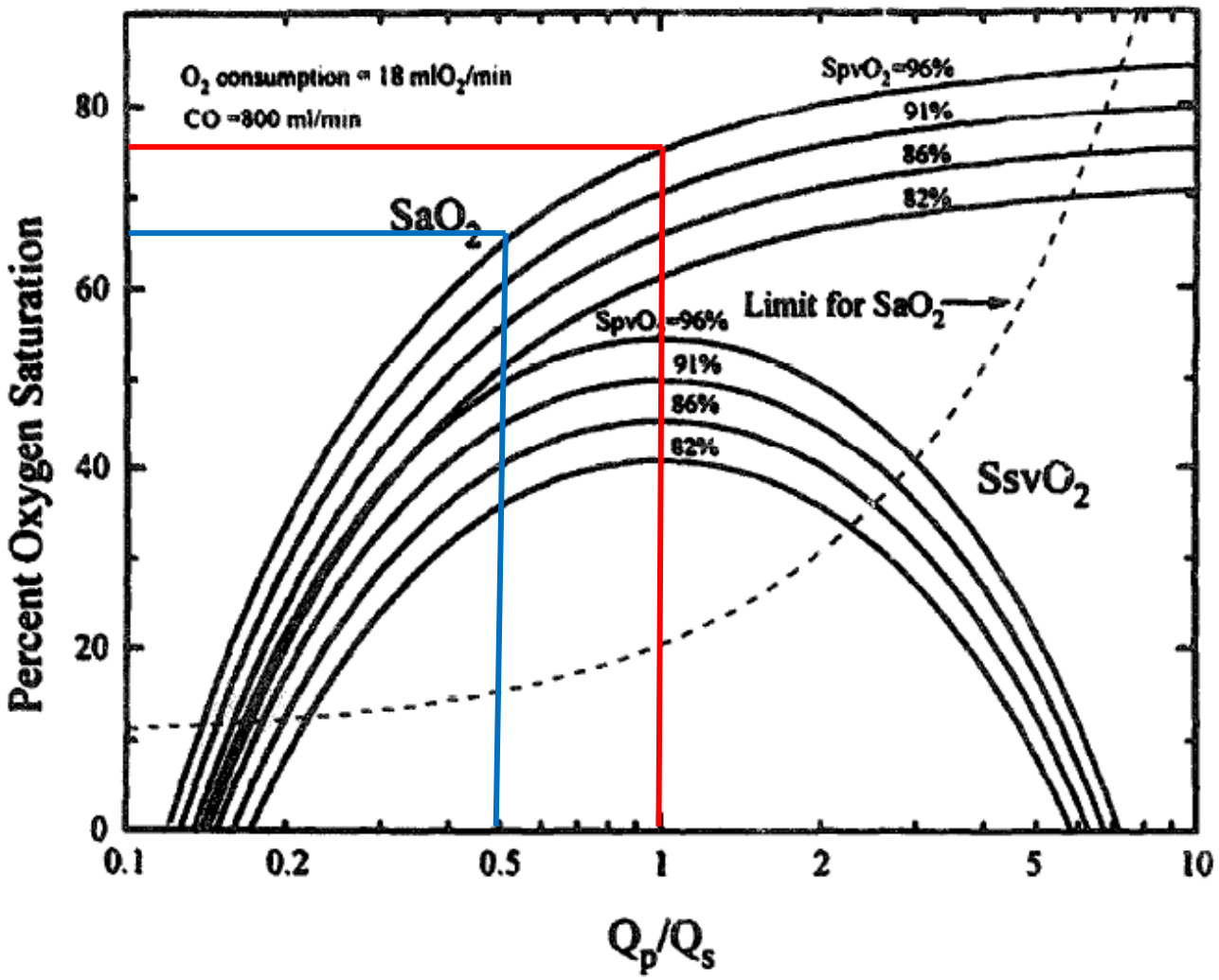

Figure 3. Percent oxygen saturation for systemic arterial (Sao_2) and venous ($Ssvo_2$) blood as a function of the pulmonary/systemic flow ratio (Q_P/Q_S) for different values of percent oxygen saturation of the pulmonary venous blood ($Spvo_2$). **Dashed line** indicates the critical range for Q_P/Q_S, where systemic oxygen availability exceeds basal oxygen demands. Note that percent Sao_2 continually increases as Q_P/Q_S increases, whereas percent $Ssvo_2$ is very low outside this critical range for Q_P/Q_S. CO = cardiac output.

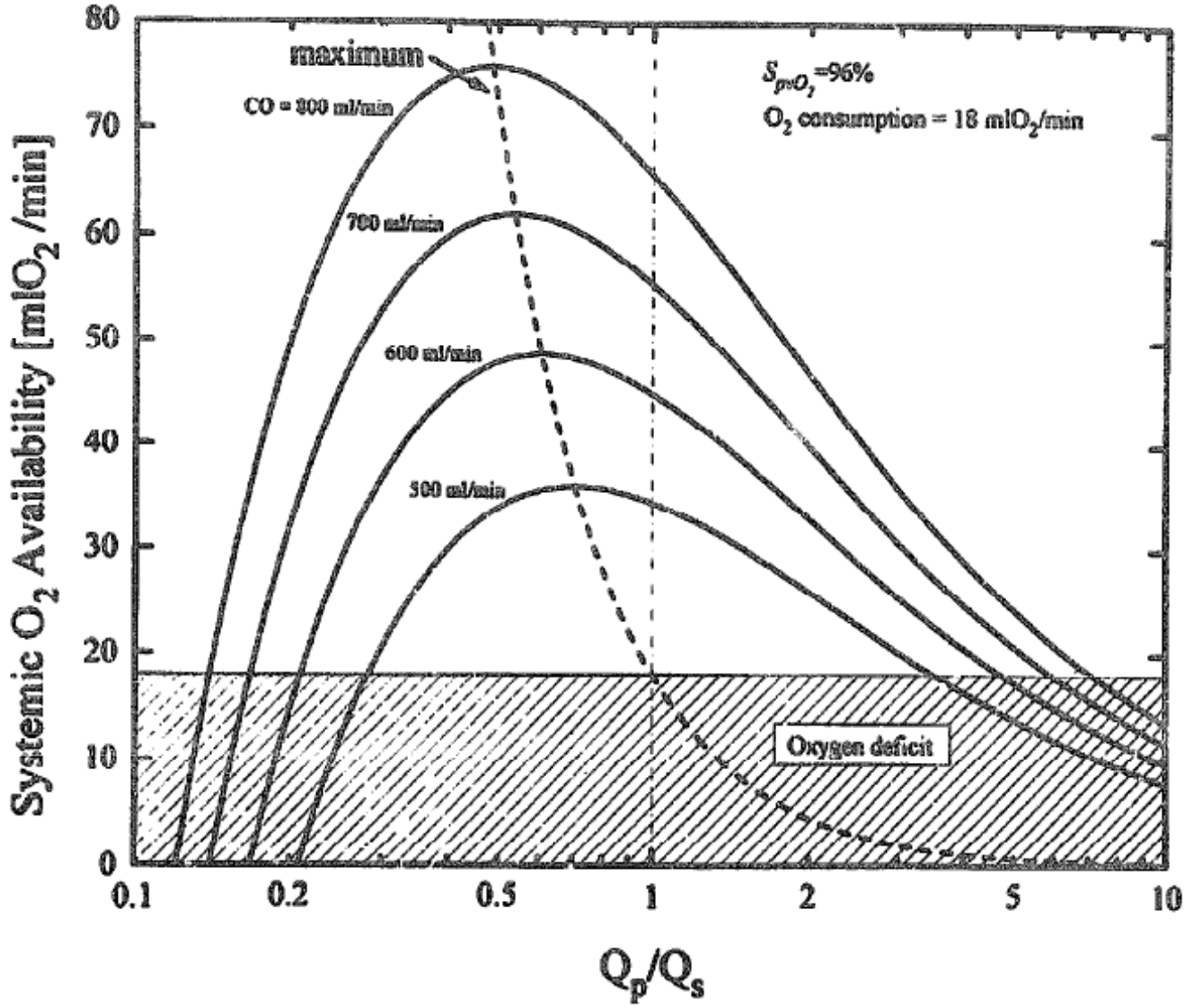

Figure 4. Oxygen balance as a function of the pulmonary/systemic flow ratio (Q_P/Q_S) for different values of cardiac output (CO). S_{PVO_2} = percent oxygen saturation of pulmonary venous blood.

The optimal ratio of Qp/Qs decreases as cardiac output or percent pulmonary venous oxygen saturation, or both, increase.

- Univentricular hearts : complete mix of systemic and pulmonary venous circulations at the ventricular level.
- As a rule of thumb, values ≥ 85% and < 75% signify increased and decreased pulmonary blood flow, respectively.
- *J Am Coll Cardiol*. 1986;7:1420–1423

Table 1. Normal Oxygen Saturation Values in the Term Newborn

	Before delivery	5 minutes old	10 minutes old	12 hours old
O_2 saturation	60	81-87	90-91	94+
(Approximate) PaO_2	*20	35	50	65+
Clinical examination	--	Mild cyanosis	No visible cyanosis	No visible cyanosis

**Oxygen content (PaO_2) = O_2 bound to hemoglobin + dissolved O_2= Hb(g/dL) x %sat x 1.36 mL O_2/g Hb +pO_2 x .0031 O_2/dL/mm Hg*

Adapted from Arikan, 2000; Kamlin, O'Donnell, Davis, & Morley, 2006; Rabi, Yee, Chen, & Singhal, 2006.

Canadian Journal of Cardiology 33 (2017) 199–208

Society Position Statement

Canadian Cardiovascular Society/Canadian Pediatric Cardiology Association Position Statement on Pulse Oximetry Screening in Newborns to Enhance Detection of Critical Congenital Heart Disease

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Canadian Journal of Cardiology 33 (2017) 199-208

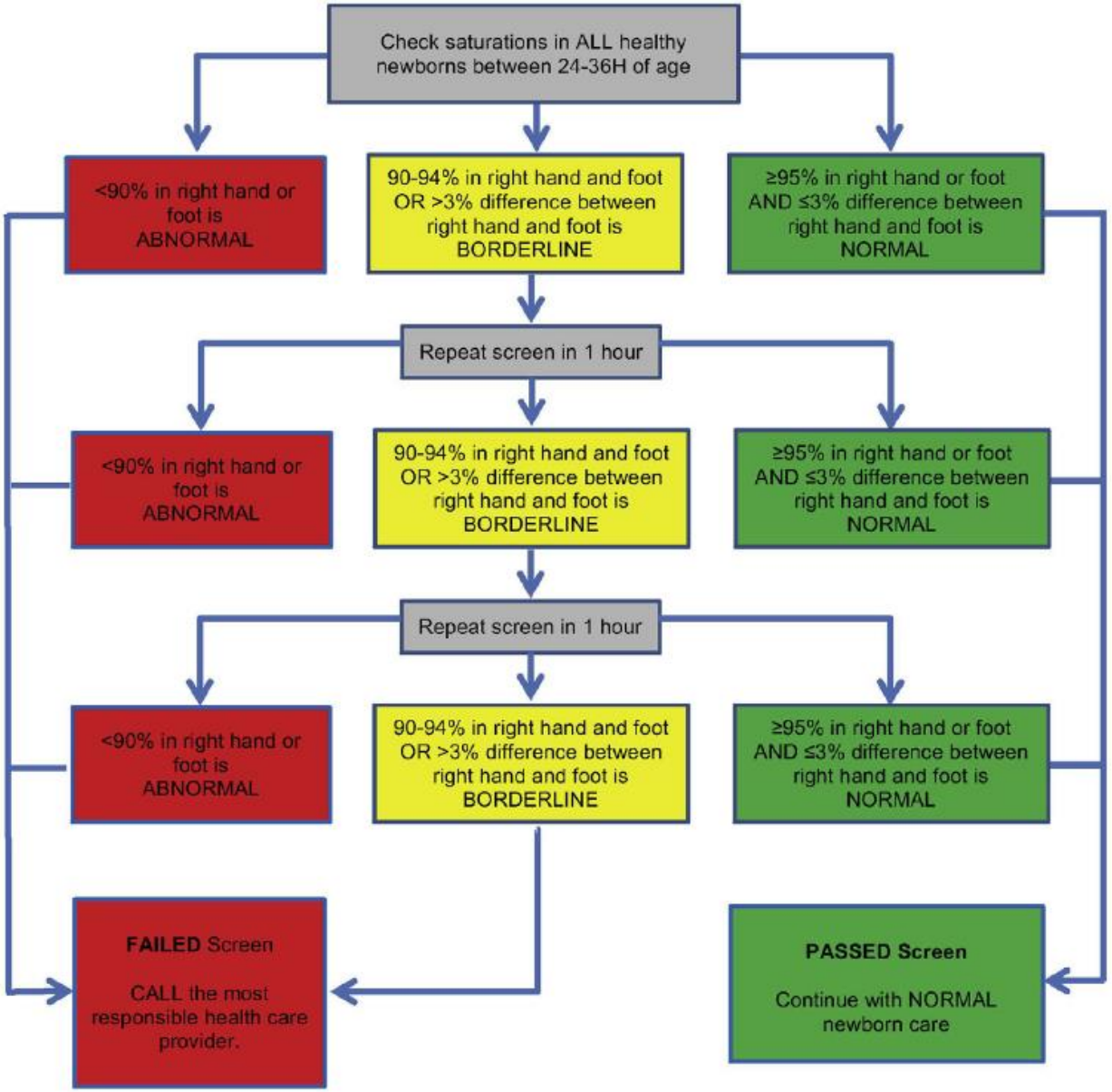


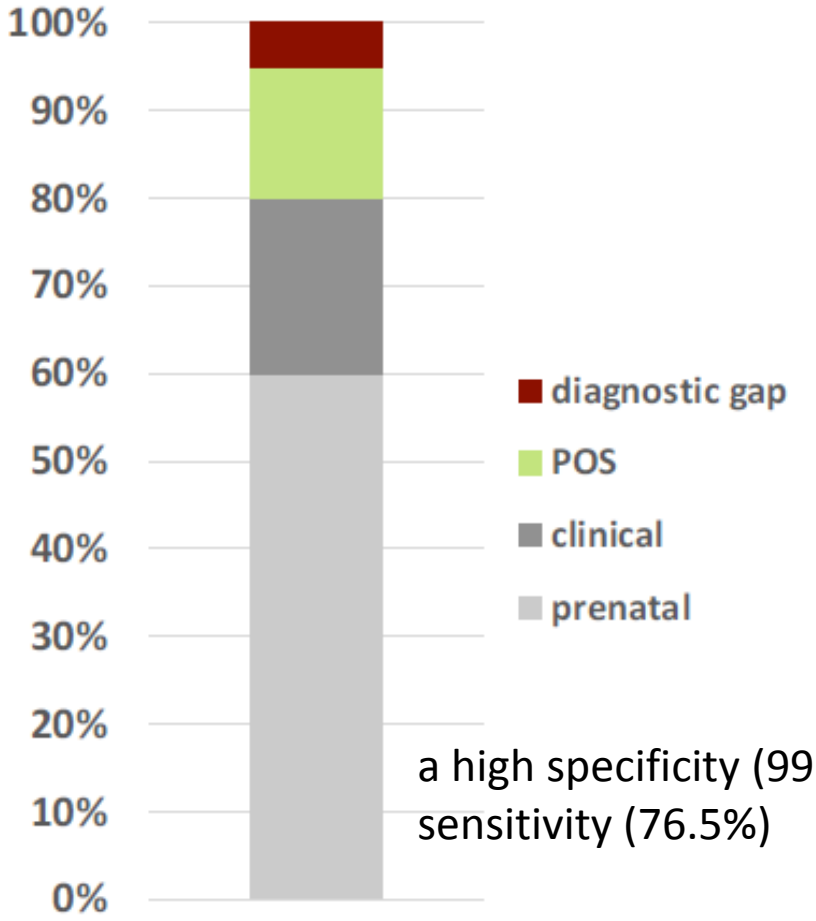
Figure 2. Pulse oximetry screening protocol. Modified from Kemper et al.[20]. Reproduced with permission from *Pediatrics*, Vol. 128:e1259-67. Copyright © 2011 by the AAP.

Table 1. Examples of CCHD lesions detectable using pulse oximetry screening

Most consistently cyanotic
Hypoplastic left heart syndrome
Pulmonary atresia with intact ventricular septum
Total anomalous pulmonary venous return
Tetralogy of Fallot
Transposition of the great arteries
Tricuspid atresia
Truncus arteriosus
Might be cyanotic
Coarctation of the aorta
Double outlet right ventricle
Ebstein anomaly
Interrupted aortic arch
Other single ventricles

CCHD, critical congenital heart disease.


141
97
64
PHILIPS
36.8
10%
Giraffe
Calliope



a high specificity (99.9%) and moderately high sensitivity (76.5%)

Figure 1. Reducing the diagnostic gap for detecting critical congenital heart disease with the addition of pulse oximetry screening (POS). Reproduced from Reide et al.[18] under the Creative Commons Attribution Noncommercial License.

Summary

- Cyanosis의 정도로 hypoxemia의 정도를 정확히 반영할 수 없다.
- Hypoxia의 한계는 아직 확실하지 않으나, PaO_2 < 35mmHg의 경우 uncompensated metabolic acidosis가 발생한다.
- Single ventricle physiology의 palliation 후 SO_2는 75~84%를 목표로 한다.
- 생후 24시간 후 신생아 산소포화도의 측정은 critical CHD의 screening으로 유용하다.