# Pulse Oximetry in Comparison to Arterial Blood Oxygen Saturation in Respiratory Distress in Neonates

***Samiha Samuel Wissa Doss, Dalia Sayed Mosallam, Enas Abdallah Ali, Marwa Mohamed Mohamed Moro***

Departments of Pediatrics, Faculty of Medicine, Cairo University, Egypt

## Abstract:

*Monitoring oxygen saturation is important to the newborns receiving supplemental oxygen so to decrease the incidence of exposure to hyperoxemia and the risk of potential deleterious effects of radical oxygen species. Pulse oximetry is a non invasive, medical device that indirectly measures the oxygen saturation of a patient's blood. An arterial blood gas (ABG) is a blood test that is performed using blood from an artery. ABG testing is used to determine the pH of the blood, the partial pressure of carbon dioxide and oxygen($PaO_2$), and the bicarbonate level.: This study aimed at studying the relationship of PaO2 and pulse oxygen saturation values in newborn with respiratory distress to evaluate whether pulse oxygen saturation value can reflect significant changes in PaO2. Prospective comparison of PaO2 and pulse oxygen saturation values in 80 patients (mean gestational age 35 ± 3.6 weeks) was performed. The PaO2 measurements were obtained from blood gas analyzer; simultaneous pulse oxygen saturation values were recorded. We evaluated PaO2/ pulse oxygen saturation values in the 80 neonates. Of the 80 samples, 30 (37.5 %) of cases were breathing supplemental oxygen by CPAP, 27 (33.7 %) of cases were on nasal prong, 21 (26.3 %) were on ventilator and 2 (2.5 %) were on head box. A mean pulse oxygen saturation of (93.7 ± 7.1%) and a mean PaO2 of (34.9 ± 10) mmHg was observed. There was a statistically significant positive (direct) correlation between $PO_2$ and $SpO_2$. An increase in $PO_2$ is associated with an increase in SpO2 ($p<0.001$). In conclusion: pulse oximetry measured from neonates and infants with respiratory distress has statistically significant positive (direct) correlation with PO2.*

Key words: pulse oximetry, blood gases, neonates, respiratory distress.

## Introduction:

Respiratory distress is a common emergency in neonatal period. Prolonged distress leads to hypoxaemia, hypercarbia and acidosis. These changes lead to pulmonary vasoconstriction and persistence of foetal circulation with right to left shunting which leads to multi system organ dysfunction [(1)]

Monitoring oxygen saturation is important to the newborns receiving supplemental oxygen so to decrease the incidence of exposure to hyperoxemia and the risk of potential deleterious effects of radical oxygen species [(2)]

Pulse oximeter is a non invasive, medical device that indirectly measures the oxygen saturation of a patient's blood, it is often attached to a medical monitor so staff can see a patient's oxygenation at all times. Most monitors also display the heart rate. Portable, battery-operated pulse oximeters are also available for home blood-oxygen monitoring [(3)]. The accuracy of pulse oximetry is limited when the saturation drops to lower than eighty percent [(4)].

Oximetry is not a complete measure of respiratory sufficiency. A patient suffering from hypoventilation (poor gas exchange in the lungs) given 100% oxygen can have excellent blood oxygen levels while still suffering from respiratory acidosis due to excessive carbon dioxide [(3)]. It is also not a complete measure of circulatory sufficiency. In severe anemia, the blood will carry less total oxygen, despite the hemoglobin being 100% saturated [(5).]

More data are needed for how far the accuracy on depending on pulse oximetry which is not invasive, easy to use, has no side effects, is accurate, allows continuous monitoring and is the preferred method of oxygen monitoring in neonates comparing to arterial blood gases which is invasive ,needed frequent sampling leading to anemia, risk for arterial occlusion, need heparinized syringe to prevent coagulation [(6)].

The aim of this work was to define the relationship between $PaO_2$ and pulse oxygen saturation values during routine clinical practice and to evaluate whether pulse oxygen saturation values are related to PaO2 level.

## Subjects and Methods:

### Study Design

Eighty newborn infants (80) who suffered from respiratory distress of different causes in neonatal intensive care unit (NICU), obstetrics and gynecology department, Kasr Al-Aini hospital, Cairo University were enrolled in our prospective comparison between noninvasive pulse oximetry ($SpO_2$ readings) and $PaO_2$ values obtained from arterial samples.

### Study Population and Data Collection

*Inclusion criteria of the cases:* Newborn infants with respiratory distress requiring oxygen supplementation with or without ventilatory support within the first two weeks of life were included in this study. They were clinically diagnosed as having respiratory distress by using the Downes' score [7] and radiologically by X-ray [8].

For the samples to be included, the newborn needs to be without rapid deterioration during sampling. Blood gas samples were ordered for clinical indications (no additional blood samples were obtained). The $SpO_2$ monitor reading was observed and recorded. $SpO_2$ readings have to remain stable for 60 seconds before and 60 seconds after the blood gas sampling, with an accepted maximal variation of $SpO_2$ of no more than 1%.

*Exclusion criteria were:* Major congenital anomalies and acute clinical changes or changes in $SpO_2$ of >1% during arterial blood sampling, jaundice, or dark skin pigmentation, haemodynanic instability as all these factors affect $SpO_2$ reading [9].

*All Patients were subjected to:*

History taking with particular emphasis on: maternal parity and illnesses, mode of delivery and any complications during pregnancy and delivery, gestational age, weight on admission and diagnosis on admission.

Full physical examination of all cases was done with particular emphasis on: Assessment of gestational age using the criteria of the new Ballard score system[10], assessment of the severity of the respiratory distress using the Downes' score [7], and the need for oxygen supplementation, nasal CPAP, or intermittent positive pressure ventilation.

Laboratory and radiological investigations of all cases were done including pulse oximetry, analysis of blood gases using arterial samples [11] and chest X-Ray.

### Statistical Analysis

Numerical data were presented as means and standard deviation (SD) values. Student's t-test was used to compare between means of two groups.

Qualitative data were presented as frequencies and percentages. Pearson's correlation coefficient was used to determine significant correlations between different variables. The significance level was set at $P \leq 0.05$. Statistical analysis was performed with SPSS 16.0® (Statistical Package for Scientific Studies) for Windows.

## Results:

The mean gestational age of cases was 35 ± 3.6 weeks. Forty one (51.3%) neonates were males and 39 neonates (48.8 %) were females (Figure 1). Mean birth weight was 2.3 ± 0.8 kg. Mean respiratory rate was (49.4 ± 7.9). As regards the mode of delivery we found that CS in neonates with respiratory distress were (76.2%) of cases, compared to normal vaginal delivery (NVD) were (23.8%) of cases (Figure 2). Regarding the maternal illness we found that most of cases were without history of maternal illness (75%), while history of Pre-eclampsia was observed in (10%) of neonates with respiratory distress, PROM was (6.3%) and ante partum hemorrhage was (3.7%) (Figure 3).

The diagnosis on admission included RDS (53.8 %), TTN (28.75 %) and others(table 1) (17.75%). The mean Downes' score was (4.4 ± 1.3). Regarding complications during hospital stay in NICU, we found that (87.5%) of neonates had no complication (Table 1).

Comparing between Oxygen devices used in our study; we found that: (37.5%) of neonates were on CPAP, (33.8 %) were on nasal prongs, (26.3%) were on ventilators and (2.5%) were on head box (Figure 4).

Results of CBC showed a mean hemoglobin of (14.9 ± 3.2 g/dl); with a minimum of (6.2 g/dl) and a maximum of (21 g/dl). There was no significant statistical correlation between $SpO_2$ and birth weight ($p< 0.483$) or gestational age ($p< 0.657$).

When $SpO_2$ was correlated with hemoglobin concentration, no statistically significant correlation was found ($P < 0.10$).

The mean pulse oximetry reading was (90.7 % ± 7.1), with a minimum of (72 %) and a maximum of (99 %) (Table 2) . The mean pH was (7.4 ± 0.1) with a minimum of (7.2) and a maximum of (7.6) (Table 3). There was no statistically significant correlation between $SpO_2$ and pH, $PCO_2$, HCO3. The mean of $PaO_2$ was (64.9 ± 15.9) with a minimum of (40 mmHg) and a maximum of (96 mmHg).

There was a statistically significant positive (direct) correlation between PaO2 and SpO2 (Table 4 and Table 5) (Figure 5 and Figure 6). An increase in PaO2 was associated with an increase in SpO2 ($p< <0.001$)

Table (1): Descriptive data of diagnosis and complications

| Variable | Frequency | % |
|---|---|---|
| Diagnosis | | |
| Respiratory distress (RDS) | 43 | (53.75) |
| TTN | 23 | (28.75) |
| Pneumonia | 3 | (3.75) |
| MAS | 2 | (2.5) |
| Apnea | 1 | (1.25) |
| Choanal atresia | 1 | (1. 25) |
| IUGR + RDS | 1 | (1.25) |
| Low birth weight + TTN | 1 | (1.25) |
| Pierre Robin Syndrome | 1 | (1.25) |
| Apnea + RDS | 1 | (1.25) |
| Prematurity + RDS | 1 | (1.25) |
| Low birth weight + RDS | 1 | (1.25) |
| Preterm + RDS | 1 | (1.25) |
| Complications | | |
| Hypertension | 4 | (5) |
| Apnea | 1 | (1.25) |
| Apnea + Pneumonia | 1 | (1.25) |
| Convulsions | 1 | (1.25) |
| Dehydration | 1 | (1.25) |
| Pneumonia | 1 | (1.25) |
| Pneumothorax | 1 | (1.25) |
| No complications | 70 | (87.5) |

Table (2): Descriptive data of ABG

| | Mean ± SD | Minimum | Maximum |
|---|---|---|---|
| Ph | 7.4 ± 0.1 | 7.2 | 7.6 |
| $PO_2$ | 64.9 ± 15.9 | 40 | 96 |
| $PCO_2$ | 34.9 ± 10 | 18 | 61 |
| $HCO_3$ | 20.3 ± 5.6 | 8.8 | 39.3 |

Table (3): Descriptive data of SpO2

| | Mean ± SD | Minimum | Maximum |
|---|---|---|---|
| $SpO_2$ | 90.7 ± 7.1 | 72 | 99 |

Table (4): Pearson's correlation coefficient results for the correlation between PO2 and SpO2 in the whole sample

| Correlation coefficient | *P*-value |
|---|---|
| 0.436 | <0.001* |

**: Significant at P ≤ 0.05*

Table (5): Pearson's correlation coefficient results for the correlation between PO2 and SpO2 in neonates with RDS

| Correlation coefficient | *P*-value |
|---|---|
| 0.507 | <0.001* |

**: Significant at P ≤ 0.05*

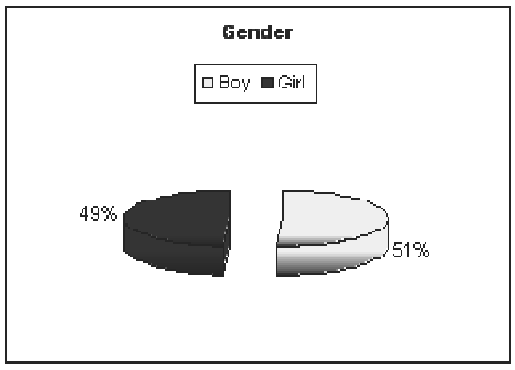


Figure (1): Gender distribution in the studied group

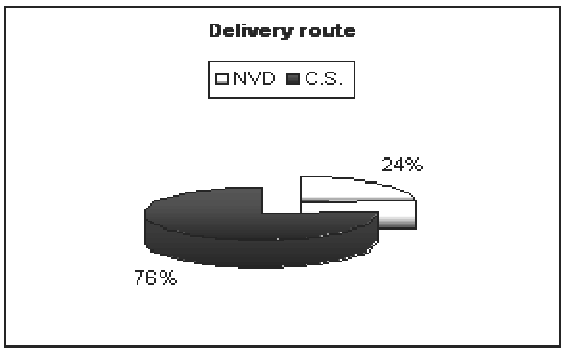


Figure (2): Delivery mode in the studied group

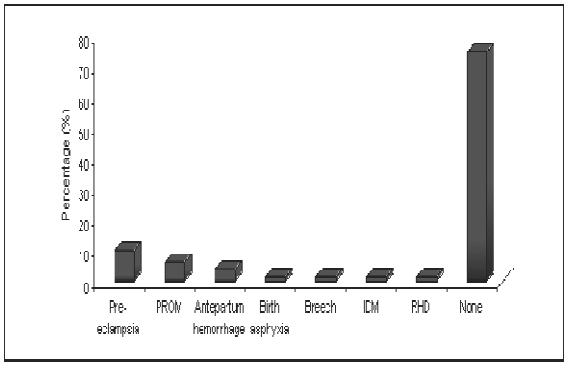


Figure (3): maternal illness in the studied group

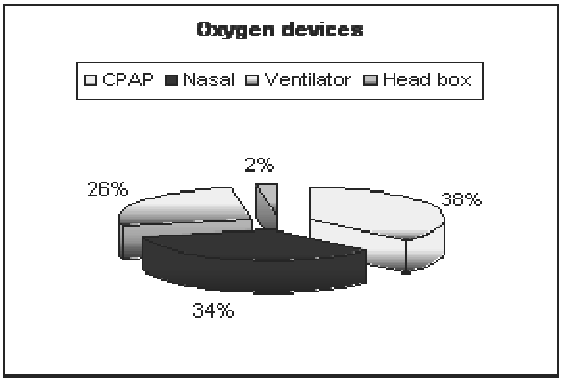


Figure (4): Oxygen delivery method used in the studied group

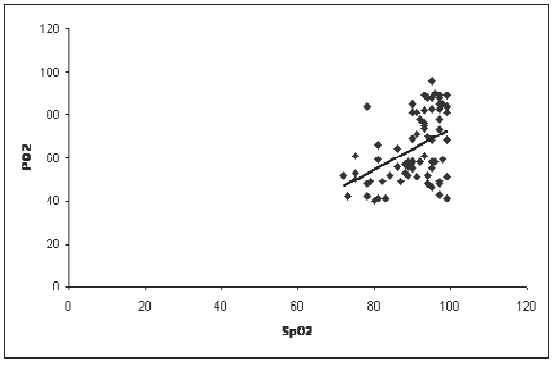


Figure (5): Scatter diagram showing positive correlation between $SpO_2$ and $PO_2$ in the whole sample

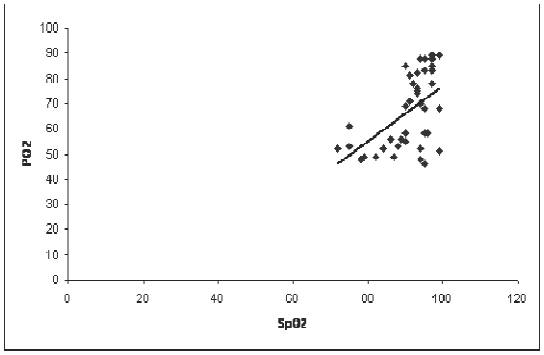


Figure (6): Scatter diagram showing positive correlation between $SpO_2$ and $PO_2$ in neonates with RDS

## Discussion

Pulse oximetry arterial oxygen saturation ($SpO_2$) has become the fifth vital sign in the examination of every newborn and infant with respiratory system presentation [12].

The normal oxygen saturation in healthy newborns breathing room air is 93% and varies according to postnatal age [13].

In addition, in vivo intravascular arterial saturation was reported by Castillo, 2008 to be between 85% and 95% in infants with pulmonary disease who were in stable condition and were breathing spontaneously [2].

(In addition, we do not know exactly what "normal" $PaO_2$ is. $PaO_2$ of >40 mmHg should be adequate for tissue needs during early neonatal life, given normal hemoglobin concentrations, cardiac output, blood flow, and cellular conditions) [2].

The American Academy of Pediatrics suggests that $PaO_2$ values above 80 to 90 mmHg may be considered hyperoxemia, but these values are not based on systematically performed studies [14].

This study was conducted on newborn babies admitted to neonatal intensive care unit (NICU), Kasr Al-Aini hospital, Cairo University with respiratory distress requiring oxygen supplementation with or without ventilatory support within the first two weeks of life. Our study included 80 neonates who were clinically diagnosed as respiratory distress by using the Downes' score and X- ray was done to confirm the diagnosis [7]. Forty one of neonates were boys (91.9%), while thirty nine were females (89.5%).The mean gestational age among babies was (35 ± 3.6) weeks in which the maximum was (43) weeks and the minimum was (26) weeks, and the mean birth weight was (2.3± 0.8) kg.

There was no statistically significant correlation ($p< 0.982$) between $SpO_2$ and respiratory rate and this was in agreement with the results of a study done by Mower et al., (1998) who examined how well respiratory rate correlated with arterial oxygen saturation status as measured by pulse oximetry and they found that respiratory rate measurements correlate poorly with oxygen saturation measured by $SpO_2$ ($p< 0.379$) and do not screen reliably for desaturation. Patients with low $SaO_2$ do not usually exhibit increased RR. Similarly, increased RR is unlikely to reflect desaturation [15].

Comparing between methods of oxygen delivery, CPAP constituted 37.5%, nasal prongs 33.8 % while ventilators were used in 26.3% and head box in 2.5%.

Clinical trials on "To tube or not to tube in RDS" done by Sekar and Corff, (2009) indicated that optimal management of neonatal RDS could be improved by early surfactant treatment (within 2□h of birth) followed immediately by extubation and stabilization on CPAP with a significant reduction in the need for mechanical ventilation and its associated morbidities [16].

The hemoglobin consentration of our neonates range between (6.2g/dl) and (21g/dl) with a mean of (14.9g/dl±3.2g/dl); there was no statistically significant correlation between $SpO_2$ and hemoglobin ($P < 0.10$), and this was in agreement with the results obtained by Pamela and Shiao., (2005) who found that the correlation between $Spo_2$ and hemoglobin was 0.44 ($P < 0.1$) [18].This was also in agreement with Perkins et al., (2003) who found that changes in $SpO_2$ do not depend on hemoglobin changes [18].

Similarly, there was no significant correlation between $SpO_2$ and birth weight. This was in agreement with the results obtained by Røsvik et al., (2009) [19].

As regards Pulse oximetry reading in our study; the mean was (90.7 % ± 7.1), with a minimum of (72 %) and a maximum of (99 %).

As regards the mean pH of neonates included in our study it was (7.4 ± 0.1) with a minimum of (7.2) and a maximum of (7.6).

As regards ABG results, we found that; there was no statistically significant correlation between $SpO_2$ and pH, $PCO_2$, HCO3.This was also in agreement with the results obtained by Muñoz et al., (2008) who found that bicarbonate had no significant effect on $SpO_2$ and Lee et al., (2000) who assessed the accuracy of pulse oximetry in the emergency department and analysis of several variables including age, sex and levels of hemoglobin, bicarbonate, pH, $Pa,O_2$, $Pa,CO_2$, and concluded that the effect of pH, although statistically significant, it was small [21, 22].

The mean of $PO_2$ was (64.9 ± 15.9) in our study with a minimum of (40) and a maximum of (96). There was a statistically significant positive (direct) correlation

between PO2 and SpO2. An increase in PO2 was associated with an increase in SpO2 (p< <0.001).
Castillo et al., (2008) found that pulse oxygen saturation values of >93% are frequently associated with $PaO_2$ values of >80 mmHg, which may be of risk for some newborns receiving supplemental oxygen [23].

## Conclusion:

There is relationship between SpO2 and PaO2, Changes in SpO2 was associated with changes in paO2 in the respiratory distress syndrome.
Neonates with RDS showed higher SpO2 than patients without RDS. However the difference was not statistically significant.
Neonates delivered by CS showed higher SpO2 than patients delivered vaginally. However the difference was not statistically significant.

## Recommendations:

The pulse oximeter remains a valuable tool in the care of intensive care patients, but an awareness of its limitations is an important component of enhancing the quality of care.
In condition with oxygen saturation (SpO2 <80%) and in critical status, SpO2 is not sufficient neither accurate to replace PaO2 and SaO2 measured by arterial blood gases analyzer.
Future studies are needed to find safer SpO2 limits or improved technology that can assist clinicians in eliminating both hyperoxemia and hypoxemia.

## References:


1. Mathai SS, Raju CU, Kanitkar CM. Management of Respiratory Distress in the Newborn. Emerg Med MJAFI 2007; 63: 269-272.
2. Castillo A, Sola A, Baquero H, et al. Pulse Oxygen Saturation Levels and Arterial Oxygen Tension Values in Newborns Receiving Oxygen Therapy in the Neonatal Intensive Care Unit: Is 85% to 93% an Acceptable Range? Pediatr 2008; Vol. 121 No. 5, pp. 882-889.
3. Brand TM, Brand ME, Jay GD. Enamel nail polish does not interfere with pulse oximetry among normoxic volunteers. J Clin Monit Comput 2002; 93-6.
4. Shiao RN, Pamela K, Ching-Nan Ou. Validation of Oxygen Saturation Monitoring in Neonates. Ameri J Critical Care 2007; 16: 168-178
5. Keller G, Cassar E, Desebbe O, Lehot JJ, Cannesson M. Ability of pleth variability index to detect hemodynamic changes induced by passive leg rising in spontaneously breathing volunteers. Crit Care 2008; 12 (2): R37.
6. Baillie JK. Simple, easily memorised "rules of thumb" for the rapid assessment of physiological compensation for acid-base disorders". Thorax 2008; 63 (3): 289–90.
7. Downes JJ, Wood DW, Locks HI. A clinical score for the diagnosis of respiratory failure. Am J Dis Child 1970; (123): 227-9.
8. Whitsett JA, Wert SE, Xu Y. Genetic disorders of surfactant homeostasis. Biol Neonate 2005; 87(4): 283-7.
9. Pierson DJ. Long term $O_2$ therapy. Lung. 2007; 782-788.
10. Ballard JL, Khoary JC, Wedig K, et al. New Ballard score; expanded to include extremely premature infants. Pediatr 1991; 119: 417-423.
11. Royal Australian College of Pathologists of Australasia. Manual of Use and Interpretation on Pathology Tests. Maryborough, Victoria: Australian Print Group.1997.
12. Kugelman A, Wasserman MD, Mor F, Goldinov L, et al. Reflectance Pulse Oximetry from Core Body in Neonates and Infants: Comparison to Arterial Blood Oxygen Saturation and to Transmission Pulse Oximetry. J Perinatol 2004; 24:366–371.
13. Rabi Y, Yee W, Chen SY, Singhal N. Oxygen saturation trends immediately after birth. J Pediatr 2006; 148 (5): 590 –594.
14. American Academy of Pediatrics and American College of Obstetricians and Gynecologists. Guidelines for Perinatal Care. 5th ed. Elk Grove Village, IL: American Academy of Pediatr 2002; 246.
15. Mower WR, Sachs C, Nicklin EL, Baraff LJ. A comparison of pulse oximetry and respiratory rate in patient screening. Respir Med. 1998; 92 (7): 989-990.
16. Sekar KC and Corff KE. To tube or not to tube babies with respiratory distress syndrome To tube or not to tube in RDS. J Perinatol 2009; 29, S68-S72.
17. Pamela K and Shiao PhD. Accurate Measurements of Oxygen Saturation in Neonates: Paired Arterial and Venous Blood Analyses. Newborn and Infant Nursing Review; Problems of Infancy. 2005; 170-178.
18. Perkins GD, McAuley DF, Gile S et al. Do changes in pulse oximeter oxygen saturation predict equivalent changes in arterial oxygen saturation? Critical Care. 2003; 7: R67-R71.
19. Røsvik A, Øymar K, Kvaløy JT and Berget M. Oxygen saturation in healthy newborns; influence of birth weight and mode of delivery. J Perinat Med 2009; 37: 403–406.
20. Muñoz X,Torres F, Sampol J. Accuracy and reliability of pulse oximetry at different arterial carbon dioxide pressure . E R J 2008; (32): 1053-1059.
21. Lee WW, Mayberry K, Crapo R, Jensen RL. The accuracy of pulse oximetry in the emergency department. Am J Emerg Med 2000; 18: 427–431.
22. Linhartova L, kurtansky A, Suksa P. Correlation between fetal blood oxygen saturation and the neonatal umbilical blood pH values. Bratisl Lek Listy.2009; 110 (11): 684-687.
23. Pamela K, Shiao PhD, NanOu C. Validation of Oxygen Saturation Monitoring in Neonates. Am J crit care 2007; (16): 168-178.