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#### <u>Original Research Article</u> Room-air Pulse Oximetry: effects of Smoking, Age, Gender, Blood pressure, Respiratory rate and Body mass index

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#### Abstract

**Background:** Hypoxemia is a serious situation; physical examination alone is insufficient to detectoxygenation failure. As oxygenation requirements are largely determined by pulse oximetry, vs. arterial blood gas, which is invasive and not a real-time technique. Various parameters are known to have effects on pulse oximetry values other than various disease processes. Before interpreting an abnormal pulse oximetry value, it will be prudent to know the effects of those personal parameters.

**Methods:** We sought to determine the distribution of oximetry  $(SpO_2)$  values in awake, asymptomatic adults, and the effect of personal characteristics like smoking, gender, respiratory rate, blood pressure, and body mass index (BMI) on these values.

It is a prospective cross-sectional study, sampling oximetry readings in adults without acute cardiac or pulmonary disease, who had no history of chronic lung disease. Participants were invited to participate if they were in normal health, as we excluded anyone with respiratory or cardiac symptoms.

**Results:** Data from 338 people in the southern region of Saudi Arabia were collected; seventy-five of them (22.2%) were female and 263 (77.8%) were male, mean age was 29.6 years. Smoking status: never smoked, 277 (82%); previous smoker, 7 (2%); current smoker, 54 (16%). Room-air SpO<sub>2</sub> values of less than 92% were noted in 6% of asymptomatic volunteers.

Statistical analysis showed no effects due to age (p = 0.27), smoking history (p = 0.69), gender (p = 0.33), blood pressure (p = 0.3), respiratory rate (p = 0.819), body mass index (p = 0.45) on SpO<sub>2</sub> levels as assessed by room air pulse oximetry.

**Conclusion:** Personal characteristics, such as age, gender, smoking history, blood pressure, respiratory rate, and BMI had no effect on room-air  $SpO_2$  values.

Keywords: pulse oximetry; BMI; saturation; oxygen.

#### Introduction

Accurate and quick detection of hypoxemia is critical in preventing serious complications in critically ill patients. Clinical examination alone is not adequate to detect oxygen desaturation since cyanosis does not develop until the level of deoxyhemoglobin reaches 5 g/dL, which corresponds to arterial oxygen saturation (SaO<sub>2</sub>) approximately 67%<sup>[1]</sup>. Furthermore, the of threshold at which cyanosis becomes apparent is multiple variables, including affected bv peripheral perfusion, skin pigmentation, and haemoglobin concentration<sup>[2]</sup>.

Arterial blood gas analysis was the optimal technique for detecting hypoxemia in critically ill patients for many years, but it has potential complications and is unable to provide a continuous measurement<sup>[3]</sup>. Oxygen requirements can be determined using pulse oximetry, instead of arterial blood gas sampling. Pulse oximetry is a technique used to measure oxygen saturation (SpO<sub>2</sub>) non-invasively. In addition, oxygen saturation is considered as the "fifth vital sign," as discussed in some of the literature and in clinical practice, which is documented in most patients while in the emergency room (ER)<sup>[4]</sup>. Pulse oximetry is also helpful with many respiratory disorders, including pneumonia and pulmonary embolism<sup>[7,8]</sup>. In clinical practice, oxygen functions saturation as complementary a parameter to vital signs, if it is not included as a fifth one. Our knowledge of pulse oximetry reference values in normal adults is somewhat limited. Therefore, should we consider different ranges of what constitutes normal for smokers and nonsmokers?

#### **Materials and Methods**

The Research and Ethics Committee approval was obtained to conduct the study. Using a crosssectional design, after obtaining consent, the subjects were initially asked whether they had any cardiac or respiratory symptoms: if they answered positive, they were excluded. Initially, we asked participants about coughing, shortness of breath, and chest pain, while pulse rate, respiratory rate, blood pressure, weight, and height were measured as well. Lastly, oxygen saturation was measured with a portable pulse oximeter (Smart signs Minipulse, Huntleigh Healthcare), and used for all subjects, thereby avoiding inter-device discrepancy. Smoking history was defined as someone being a current smoker, a previous smoker, which also included the type of smoking (cigarettes or other types).

Exclusion criteria for the study were strict, as we excluded those with temperature  $> 38^{\circ}$ C or  $< 35^{\circ}$ C, respiratory rate > 25 breaths/min, systolic blood pressure < 90 or > 220 mm Hg, or diastolic blood pressure > 120 mm Hg.

The information was collected by one group of investigators, who used the same scale and height measurement device, as well as the same pulse oximeter device.

The team was instructed to avoid measuring oxygen saturation at the fingers with any nail polish or dye, as they can interfere with accurate measurements.

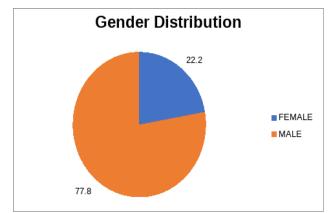
We agreed on acceptable readings for oxygen saturation after 30 s of the pulse oximeter being attached to the subjects' fingers (covered to avoid ambient light interference).

The primary goals of this study were to determine the distribution of oxygen saturation in asymptomatic adults and to find the effect of personal characteristics in SpO<sub>2</sub> values. Statistical significance was defined as p < .05. Analyses was performed with statistics software Statistical Package for the Social Sciences (SPSS).

#### Results

Data from 338 healthy volunteers from the southern region of Saudi Arabia were collected over a 10-month period: of these, 75 (22.2%) were female and 263 (77.8%) were male, **Fig 1**.

#### Fig 1: Gender Distribution



# The mean age was 29.6 years, **Table.1**. **Table 1**: Age Distribution

| Age in years | Freq. | %       |  |
|--------------|-------|---------|--|
| 1-20         | 68    | 20.12%  |  |
| 21-41        | 205   | 60.65%  |  |
| 42-62        | 58    | 17.16%  |  |
| Above 63     | 7     | 2.07%   |  |
| Total        | 338   | 100.00% |  |
| Mean         | 29.58 |         |  |
| SD           | 12.74 |         |  |

277 (82%) subjects werenever smokers, 7 (2%) were previous smokers while 54 (16%) were current smokers. BMIs were as follows: 18.5-24.9 (38%), 25-29.9 (28.00%), or more than 30 (26%), with a mean BMI of 26.7, **Table.2**. **Table 2**: BMI

| BMI              |            |     |      |
|------------------|------------|-----|------|
| Underweight      | 15-18.5    | 24  | 7%   |
| Normal           | 18.5-24.99 | 130 | 38%  |
| Overweight       | 25-29.99   | 96  | 28%  |
| Obese grade 1    | Above 30   | 55  | 16%  |
| Obese grade 2    | above 35   | 33  | 10%  |
| Total            |            | 338 | 100% |
| Mean 26.7,SD 6.7 |            |     |      |

The average room-air SpO<sub>2</sub> was 95.8 (SD = 2.74) Room-air SpO<sub>2</sub> values less than 92% were noted in less than 6% of asymptomatic volunteers. The average respiratory rate, pulse rate, systolic and diastolic blood pressure were 21 breaths/min, 81 beats/min, 128 mm Hg, 79 mm Hg, respectively. As depicted in **Table 3**, SpO<sub>2</sub>has no significant relationship with age (p=0.275), RR (p=0.819) with SBP (p= 0.305), with DBP (p=0.724), and with BMI (p= 0.453).

## **Table 3:** Relationship between Patient parameters and SpO<sub>2</sub>

| Respiratory Rate |       |         |
|------------------|-------|---------|
| C.I              | Freq. | %       |
| 10-18            | 246   | 72.78%  |
| 19-25            | 85    | 25.15%  |
| above 25         | 7     | 2.07%   |
| Total            | 338   | 100.00% |
| Average= 21.3    |       |         |
| S.D= 22.5        |       |         |
| SBP              |       |         |
| C.I              | Freq. | %       |
| 1-120            | 114   | 33.73%  |
| 121-139          | 151   | 44.67%  |
| above 140        | 73    | 21.60%  |
| Total            | 338   | 100.00% |
| Average= 128.4   |       |         |
| S.D=16.2         |       |         |
| DBP              |       |         |
| C.I              | Freq. | %       |
| below 40         | 4     | 1.18%   |
| 40-60            | 12    | 3.55%   |
| 61-80            | 180   | 53.25%  |
| 81-90            | 85    | 25.15%  |
| above 90         | 57    | 16.86%  |
| Total            | 338   | 100.00% |
| Average= 78.9    |       |         |
| S.D=13.4         |       |         |
| SpO <sub>2</sub> |       |         |
| Range            | Freq. | %       |
| 61-91            | 14    | 4.14%   |
| 91-100           | 324   | 95.86%  |
| Total            | 338   | 100.00% |
| Average= 95.8    |       |         |
| S.D= 2.74        |       |         |

#### Discussion

In this study, we found that room-air  $SpO_2$  less than 92% was quite rare in awake, adult asymptomatic healthy volunteers; we suspect this was related to how the older volunteers were excluded if they had symptoms of cough, shortness of breath, or chest pain. By default, this left us with a younger population. Increasing age was a strong predictor of lower values of oxygen saturation, as reported in previous studies<sup>[9]</sup>, which included BMI, male gender, and smoking tendencies. We could not demonstrate this in our own study with a younger cohort, with a lower incidence of smoking. A healthy survivor effect and a decreased representation of those with poor health may have led to a healthier sample. This may explain why aging did not lead to decreased

SpO2. Smoking is a strong predictor of acquiring chronic obstructive pulmonary disease and lower oxygen saturation<sup>[10]</sup>. Previous data on room-air pulse oximetry<sup>[11]</sup> suggested that smoking was not associated with lower SpO<sub>2</sub> values, which was consistent with our findings, although we were limited in the comparison between smokers (7%) and nonsmokers (82%), as most smokers were excluded in our study. We rarely found a smoker without cough, shortness of breath, or chest pain. We were surprised by the increased obesity in the community see Table 2. (Body mass index distribution, the prevalence of obesity was more than 70 % in this cohort). This alerted us to the need for general health education about this disease. A similar finding was noted with the 'silent killer' hypertension, as its prevalence in healthy volunteers exceeded 68%. Witting and his group did similar work to this study evaluating the room-air SpO<sub>2</sub> values in asymptomatic, awake adults and they found that a white race and male sex are associated with lower  $SpO_2$  readings<sup>[11]</sup>. Obesity has a definite effect as per the observation by Kapur et al<sup>[12]</sup>. Smoking history and obesity are associated with low SpO<sub>2</sub>, while age and gender have no effect as per Vold et al<sup>[13]</sup>. Smoking reduces oxygen saturation as per the study by Ozdal et al<sup>[14]</sup>. Age influences pulse oximetry but not gender as per a study by Bhogal et  $al^{[15]}$ .

#### Conclusion

Personal characteristics such as age, gender, smoking history, blood pressure, respiratory rate, and BMI had no effect on room-air  $SpO_2$  values.

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