Reference values for pulse oximetry at high altitude

M J Gamponia, H Babaali, F Yugar, R H Gilman

Abstract

***Objective*—To determine reference values for oxygen saturation (Sao_2) in healthy children younger than 5 years living at high altitude.**

***Design*—One hundred and sixty eight children were examined for Sao_2 at 4018 m during well child visits. Physiological state was also noted during the examination.**

***Results*—The mean Sao_2 was 87.3% (95% confidence intervals (CI) 86.7%, 87.9%) with a median value of 87.7%. A significant difference was observed in Sao_2 between children younger than 1 year compared with older children, although the difference was no longer demonstrable when sleeping children were excluded.**

***Conclusions*—This study has provided a reference range of Sao_2 values for healthy children under 5 years old so that pulse oximetry may be used as an adjunct in diagnosing acute respiratory infections. Younger children were also shown to have a lower mean Sao_2 than older children living at high altitude, which suggests physiological adaptation to high altitude over time. In addition, sleep had a lowering effect on Sao_2, although the clinical importance of this remains undetermined.**

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Pulse oximetry measures beat to beat arterial oxygen saturation (Sao_2) transcutaneously, and is related to the partial pressure of arterial oxygen (Pao_2) through the oxyhaemoglobin dissociation curve.[1] In the USA and other developed countries, pulse oximetry is used for rapid assessment of children who present to emergency rooms in respiratory distress. An accurate correlation between the measurement of oxyhaemoglobin saturation of blood and transcutaneous oxygen tension has been well documented in previous investigations.[2–8] However, as a diagnostic tool, pulse oximetry has the advantages that it is non-invasive, easy to use, and gives an Sao_2 reading within seconds after application of the transducer. Certainly, its use merits consideration in less developed countries, where acute respiratory infection accounts for a large proportion of childhood mortality.

In Bolivia, where most of the population lives at altitudes between 3000 and 4200 m above sea level, the under 5 childhood mortality rate is 75–90 deaths/1000 live births, with acute respiratory infection as a leading cause of death.[9] Although pulse oximetry as a diagnostic tool for acute respiratory infection in a high altitude setting is currently under evaluation, reference values in healthy children must be described first. Four previous studies have shown an adaptation to low oxygen tension by describing oxygen saturation reference values for children living at high altitude: in Denver, Colorado at an altitude of 1610 m above sea level[10]; in Bogata, Columbia at 2640 m[11]; in Summit County, Colorado at 2800 m[12]; and in the Peruvian Andes at 3700 m.[13] In the latter study, Reuland *et al* noted a significant disparity between the mean Sao_2 values for infants and children 1–5 years old.[13] In this study, our objective was to determine the reference values of oxygen saturation, respiratory rate, and heart rate in a sample of healthy children living in El Alto, Bolivia. Located 10 km from the centre of La Paz, El Alto is a rapidly growing city of 500 000 people at 4018 m above sea level.[14] Additional goals were to establish further hypothesised relations between age and oxygen saturation, and different physiological activity states and oxygen saturation at high altitude. These relations might contribute to the increased susceptibility of infants to hypoxia during acute respiratory infection in the high sierras.

Subjects and methods

This study was approved by the institutional review boards of Johns Hopkins University and PROSALUD, a Bolivian non-governmental organisation that provides primary and preventive health care services. One hundred and sixty eight healthy children who presented to four PROSALUD clinics in the city of El Alto were enrolled in this descriptive observational study from July 1994 to October 1994. All of the children were lifetime residents of El Alto and nearly 100% were Aymara Indian. They ranged in age from 1 day to 60 months and were visiting the ambulatory outpatient clinics for well child check ups, immunisations, or other minor non-pulmonary problems. Enrollment was stratified into five age groups: 0–5, 6–11, 12–23, 24–35, and 36–60 months. Exclusion criteria were based on the following factors: a history of chronic illness, such as asthma or congenital cardiac anomalies; the presence of chest retractions, rhonchi, wheezing, rales, or heart murmur on physical examination; a history of respiratory illness within the last two months; a history of blood transfusionwithin the last six months; and obvious malnutrition.

Table 1 Summary of oxygen saturation, respiratory rate, and heart rate for different age groups

Group (age in months) (mean age)	n	Oxygen saturation (%)		Respiratory rate (breaths/min)	Heart rate (beats/min)
		Mean (95% CI)	Median	Mean (95% CI)	Mean (95% CI)
1 (0–5) (1.85)	40	86.9 (85.4, 88.4)	87.0	45.5 (41.4, 49.5)	145.0 (139.4, 150.7)
2 (6–11) (8.87)	31	85.7 (84.3, 87.2)	86.3	41.5 (37.7, 45.2)	130.4 (124.0, 136.8)
3 (12–23) (17.6)	33	87.5 (85.9, 89.1)	88.7	37.0 (33.3, 40.6)	127.4 (120.4, 134.4)
4 (24–35) (28.7)	32	88.1 (87.1, 89.0)	87.7	33.5 (31.2, 35.9)	111.8 (104.9, 118.8)
5 (36–60) (47.5)	32	88.5 (87.6, 89.3)	88.8	32.0 (29.7, 34.3)	109.1 (102.6, 115.7)
Total	168	87.3 (86.7, 87.9)	87.7	36.2 (34.6, 37.9)	125.7 (122.3, 129.2)

No significant difference comparing all groups individually to each other (p = 0.10; Kruskal-Wallis). Infants (age 0–11 months) had a lower mean oxygen saturation than older children (12–60 months) (mean 86.4% *v* 88%), this difference was not significant when sleeping children were excluded (mean 87.2% *v* 88.1%, p = 0.07; Mann-Whitney). Comparing all groups individually with each other, increasing age was associated with a decrease in mean heart rate (p < 0.05; ANOVA) and respiratory rate (p < 0.05; ANOVA).

Table 2 Summary of oxygen saturation, respiratory rate, and heart rate for different age groups exluding sleeping children

Group (age in months)	n	Oxygen saturation (%)		Respiratory rate (breaths/min)	Heart rate (beats/min)
		Mean (95% CI)	Median	Mean (95% CI)	Mean (95% CI)
1 (0–5)	29	87.8 (86.2, 89.4)	88.0	46.0 (40.9, 51.1)	147.0 (140.0, 154.0)
2 (6–11)	28	86.7 (85.5, 87.8)	87.0	42.2 (38.2, 46.2)	130.6 (125.2, 136.0)
3 (12–23)	33	87.5 (85.9, 89.1)	88.7	37.0 (33.3, 40.6)	127.4 (120.4, 134.4)
4 (24–35)	31	88.2 (87.2, 89.2)	87.7	33.6 (31.3, 36.0)	112.1 (105.0, 119.2)
5 (36–60)	31	88.6 (87.8, 89.5)	89.0	32.3 (29.9, 34.6)	109.5 (102.8, 116.3)
Total	152	87.8 (87.1, 88.4)	88.0	38.0 (36.3, 39.8)	125.0 (121.4, 128.6)

Informed consent was obtained before examination. Respiratory rate, heart rate, and three pulse oximetry readings were obtained for each child using a Nellcor N-10 self-calibrating pulse oximeter and an appropriately sized adhesive transducer placed on the index finger, thumb, or large toe of each subject. In most instances, the subject was awake and quietly resting. Bright lights and excessive motion were avoided during transducer application to minimise artefacts. For the saturation of oxygen to register, the oximeter had to track the peripheral pulse for at least 10 seconds. Measurements were discarded if not consistent with other readings.

Respiratory rate, heart rate, and mean oxygen saturation recordings for each subject were entered into an Epi Info 6 database and a descriptive statistical analysis was performed to calculate the mean and median values and 95% confidence intervals (CI). In addition, Kruskal-Wallis and ANOVA tests (or Mann-Whitney or Student's *t* tests where appropriate) were completed to compare the different means among age stratified groups.

Results

Mean and median values were obtained for oxygen saturation, respiratory rate, and heart rate in the different age groups (table 1). The mean Sao_2 for all age groups was 87.3% (95% CI 86.7%, 87.9%) with a median of 87.7%. No significant differences in Sao_2 were observed by the Kruskal-Wallis test (p = 0.10, 7.67), or Mann-Whitney tests among age stratified groups. Although, as a group, children less than 1 year old (groups 1 and 2) had a lower mean Sao_2 (86.4%; 95% CI 85.4%, 87.4%; median 87%). In contrast to the mean Sao_2 of older children (groups 3, 4, and 5) (88%; 95% CI 87.3%, 88.7%; median 88.3%) (Mann-Whitney, p = 0.02, 5.80), the difference was not significant when sleeping children were excluded (Mann-Whitney, p = 0.07, 3.19) (table 2). Increasing age was associated with a decrease in mean heart rate (ANOVA, p = 0.00; F statistic = 22.40) and mean respiratory rate for each group (ANOVA, p = 0.00; F statistic = 11.79).

Physiological states with corresponding mean values for oxygen saturation, respiratory rate, and heart rate are shown in table 3. Relatively lower mean values for oxygen saturation were observed in children who were either breast or bottle feeding or asleep, compared with children who were awake and quiet, or agitated. These differences in Sao_2 were not significant. In addition, no significant difference (by Mann-Whitney test) was detected in the mean Sao_2 of the group that was either breast or bottle feeding compared with the other groups, which could be a result of our small sample. Comparisons of the sleep state were made with other physiological states only in group 1 (0–5 months old) because it was the only subgroup in which there were adequate

Table 3 Summary of oxygen saturation, respiratory rate, and heart rate by physiological state

State	n	Oxygen saturation (%)		Respiratory rate (breaths/min)	Heart rate (beats/min)
		Mean (95% CI)	Median	Mean (95% CI)	Mean (95% CI)
Awake, quiet	128	88.0 (87.8, 88.1)	88.3	38.0 (36.2, 39.9)	121.6 (118.2, 125.1)
Breast or bottle feeding	4	83.5 (81.0, 86.0)	83.0	38.5 (30.0, 47.0)	149.8 (108.4, 191.1)
Crying	19	87.5 (86.2, 88.8)	87.5	38.1 (31.2, 45.0)	143.0 (129.2, 156.8)
Asleep	16	83.0 (80.0, 85.9)	83.3	40.1 (34.3, 46.0)	132.7 (119.6, 145.9)

Children who were asleep had a significantly lower mean oxygen saturation thant children in other physiological states (p < 0.05; Mann-Whitney).

Table 4 Summary of oxygen saturation, respiratory rate, and heart rate for sleeping children (aged 0–5 months) compared with other physiological states

State	n	Oxygen saturation (%)		Respiratory rate (breaths/min)	Heart rate (beats/min)
		Mean (95% CI)	Median	Mean (95% CI)	Mean (95% CI)
Sleeping	12	84.6 (81.3, 87.9)	84.7	43.3 (36.7, 49.9)	140.6 (131.1, 150.1)
Other states	28	87.9 (86.3, 89.5)	88.2	46.4 (41.2, 51.4)	146.9 (139.6, 154.9)

Group 1 (age 0–5 months) was the only group with a substantial proportion of children in a non-quietly awake state.
No significant difference (p = 0.14; Mann-Whitney).

Table 5 Summary of oxygen saturation, respiratory rate, and heart rate for different age groups excluding crying children

Group (age in months)	n	Oxygen saturation (%)		Respiratory rate (breaths/min)	Heart rate (beats/min)
		Mean (95% CI)	Median	Mean (95% CI)	Mean (95% CI)
1 (0–5)	32	86.9 (85.1, 88.7)	87.0	44.3 (39.8, 48.8)	143.0 (137.5, 148.5)
2 (6–11)	30	85.6 (84.1, 87.1)	86.3	41.4 (37.6, 45.2)	131.2 (124.7, 137.7)
3 (12–23)	28	87.6 (85.7, 89.5)	89.5	38.6 (34.8, 42.4)	124.2 (118.1, 130.3)
4 (24–35)	27	87.9 (86.8, 89.0)	87.7	34.4 (31.9, 36.9)	108.1 (102.4, 113.8)
5 (36–60)	32	88.5 (87.6, 89.3)	88.8	32.0 (29.7, 34.3)	109.1 (102.5, 115.7)
Total	149	87.3 (86.9, 87.7)	87.7	38.2 (36.4, 40.0)	123.5 (120.1, 126.9)

No significant differences were found in different age groups when crying children were excluded (p = 0.11; Kruskal-Wallis). No or minimal differences were found in different age groups comparing results in tables 1 and 5 (with or without including crying children).

numbers of infants sleeping. Sleeping infants tended to have lower Sa_{O_2} mean and median values (table 4), but this difference did not achieve statistical significance (Mann-Whitney, p = 0.14, 2.14). Crying does not appear to have an effect on Sa_{O_2}. When we excluded crying children from our analysis, there were no (or minimal) changes in Sa_{O_2} values (table 5).

Discussion

The findings of this study confirm mean lower oxygen saturations in children living at high altitudes, as has been shown previously.[10–13] It appears that this phenomenon occurs only at altitudes above 1600 m.[10–13 15 16]

In addition, we found a significant difference in oxygen saturation between infants and older children living at high altitudes. The trend was similar to an observation by Reuland *et al* that, in general, younger children have lower mean Sa_{O_2} values—a relation seen only at altitudes above 3000 m.[13] However, we also found that sleeping was associated with a decrease in mean Sa_{O_2}. When we excluded sleeping children from the study, the difference, although present, no longer reached statistical significance. Further studies with larger numbers of awake children will be needed to determine if the difference in Sa_{O_2} in younger versus older children at high altitude is indeed present. We had expected infants to have a lower mean Sa_{O_2} than older children for the following reasons: during the first year of life, many of the physiological compensations stimulated by low oxygen tension may not yet be developed; infants have comparatively less functional residual capacity than older children; and their smaller airways generate a higher airway resistance.[17]

During the first few months of life, the neonate may be able to compensate for low oxygen tension because fetal haemoglobin (HbF), which has a greater affinity for the oxygen molecule, is still present. At birth, fetal haemoglobin comprises 80% of an infant's total haemoglobin, but diminishes to 5% after 140 days.[18] Because 2,3-diphosphoglycerate (2,3-DPG) interacts less strongly with HbF than with haemoglobin A, the result is a shift to the left of the oxygen dissociation curve.[19] Although the presence of fetal haemoglobin may lead to poorer unloading of oxygen in the venous blood, it is advantageous in oxygen loading in the lungs,[20] which serves as a possible explanation for our observation. After reaching 12 months of age, the child may begin to develop functional compensatory mechanisms for long term adaptation to high altitude. These compensatory mechanisms include polycythaemia, enhanced alveolar growth during the first eight years of life, an increase in capillary proliferation in the peripheral tissues, and changes in oxidative enzymes.[20] Because the adaptive response in respiratory physiology appears to be environmental rather than genetic, these mechanisms are probably not yet present in neonates.[21] This relation, however, was not a finding of the study by Lozano *et al* in Bogata, Columbia, which provided reference values for children 5 days to 24 months old at an altitude of 2640 m and found a significant difference in mean Sa_{O_2} only for the subgroups of children less than 1 month old and those 13–18 months old.[11] In addition, a recent study of children born in Lhasa, Tibet showed that Tibetan infants had a higher arterial oxygen saturation at birth and during the first four months of life than Han newborns. The authors concluded that genetic adaptations might permit adequate oxygenation and confer resistance to the syndrome of subacute infantile mountain sickness.[22]

In any case, we believe that children younger than 1 year old are particularly vulnerable to acute respiratory infection; they are multiply disadvantaged because they have less mature immune systems,[23] less physical ability to clear secretions, and less verbal communicability to convey distress.

The 1994 Encuesta Nacional Demografia y Salud for Bolivia also supports the idea that children less than 1 year old might be more susceptible to acute respiratory infections:

during interviews for the survey, the prevalence of acute respiratory infection over the past two weeks was 13% for children 0–5 months, 25% for children 6–11 months, 18% for children 12–23 months, and 17% for children 24–35 months.[9]

With regard to levels of activity, as noted previously, the sleep state confers a significantly lower mean oxygen saturation than other physiological states.[11 17 24] We speculate that because recumbency occurs with sleep, it is usually associated with mildly decreased functional residual capacity, owing to pulmonary atelectasis, which results in the child taking more frequent and shallower breaths. In newborns, it is also well documented that during active sleep, there is loss of diaphragmatic and intercostal muscle tone.[24–26] In addition, Nicholas *et al* have observed reductions in Sa_{O_2} during sleep and crying, but only in the presence of either upper or lower respiratory tract infections.[12] The clinical significance of this observation is uncertain and perhaps may play a role in sudden infant death syndrome.

The limitations of pulse oximetry, which is not reliable when the subject is experiencing hypothermia, hypovolaemia, shock, extreme hyperoxia ($Pa_{O_2} > 100$), or extreme hypoxia ($Pa_{O_2} < 70$),[1] were considered in this investigation. However, based on our exclusion criteria, we believe our subjects to be a fairly good representation of healthy children living in El Alto. Other conditions known to interfere with oxygen saturation detection by pulse oximetry, such as smoke inhalation with carbon monoxide poisoning, the use of intravenous dyes, nitroglycerin, nail polish, direct sunlight, and xenon arc surgical lamps,[1] were also negligible in this study.

Many pulse oximeters used in the emergency room setting are similar to the model used in this study, which has a beat to beat averaging mode. When an infant or child is asleep and periodically breathing, the averaging mode may reflect an artificially low measurement of oxygen saturation. Over a duration of 10 seconds, a sleeping child will have apnoeic pauses as part of the periodic breathing, which is then averaged into the final measurement, which could also partially explain our findings of lower mean Sa_{O_2} values in sleeping children. Whether the decrease in Sa_{O_2} in sleeping children is a true finding or an artefact of pulse oximetry will require further study.

Although we were unable to evaluate the effect of polycythaemia on oxygen delivery, mean haematocrit values of a similar population (H Babaali, unpublished data, 1995) were compared with those of populations living at sea level. Based on these observations, polycythaemia does not appear to be present in this population at these ages. In addition, Reuland *et al* have shown that children living at high altitude do not have raised haematocrits until they reach about 3–4 years of age.[13]

CONCLUSION

This study provides a definition of normal Sa_{O_2} in a healthy population of children under 5 years old living at high altitude. With the use of pulse oximetry, Sa_{O_2} might eventually be considered a fifth vital sign[16 27] in the World Health Organisation diagnostic algorithm,[28] which provides guidelines for the treatment of acute respiratory infection based on the presence of chest retractions and tachypnoea. We have also shown that younger children have a lower mean Sa_{O_2} than older children living at high altitude, which suggests physiological adaptation to high altitude over time. Although sleep had a lowering effect on Sa_{O_2}, the clinical importance of this effect remains undetermined.

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