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Pulse Oximetry at High Altitude

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Abstract

Luks, Andrew M., Erik R. Swenson. Clinician's corner: pulse oximetry at high altitude. *High Alt. Med. Biol.* 12:109–119, 2011.—Pulse oximetry is a valuable, noninvasive, diagnostic tool for the evaluation of ill individuals at high altitude and is also being increasingly used to monitor the well-being of individuals traveling on high altitude expeditions. Although the devices are simple to use, data output may be inaccurate or hard to interpret in certain situations, which could lead to inappropriate clinical decisions. The purpose of this review is to consider such issues in greater detail. After examining the operating principles of pulse oximetry, we describe the available devices and the potential uses of oximetry at high altitude. We then consider the pitfalls of pulse oximetry in this environment and provide recommendations about how to deal with these issues. Device users should recognize that oxygen saturation changes rapidly in response to small changes in oxygen tensions at high altitude and that device accuracy declines with arterial oxygen saturations of less than 80%. The normal oxygen saturation at a given elevation may not be known with certainty and should be viewed as a range of values, rather than a specific number. For these reasons, clinical decisions should not be based on small differences in saturation over time or among individuals. Effort should also be made to minimize factors that cause measurement errors, including cold extremities, excess ambient light, and ill-fitting oximeter probes. Attention to these and other issues will help the users of these devices to apply them in appropriate situations and to minimize erroneous clinical decisions.

Key Words: high altitude; hypoxemia; pulse oximetry; acute mountain sickness; high altitude cerebral edema; high altitude pulmonary edema

Introduction

For medical providers working at remote high altitude clinics or as part of trekking or climbing expeditions, the pulse oximeter, which noninvasively measures arterial blood oxygenation, often serves as a valuable piece of diagnostic equipment, particularly in the evaluation of people with symptoms suggestive of acute altitude illness. Pulse oximeter use also appears to be increasing in other settings; anecdotal reports suggest guides, trekkers, and climbers are increasingly using the devices to monitor the well-being of expedition members. Although these devices yield information within seconds of being applied to the traveler's finger and are simple to use, data output may be inaccurate or difficult to interpret in certain situations. Failure to recognize such problems could lead, in turn, to inappropriate decisions regarding medical care or travel planning.

The purpose of this review is to consider the use of pulse oximetry at high altitude in greater detail. We describe the operating principles of pulse oximeters and then examine the

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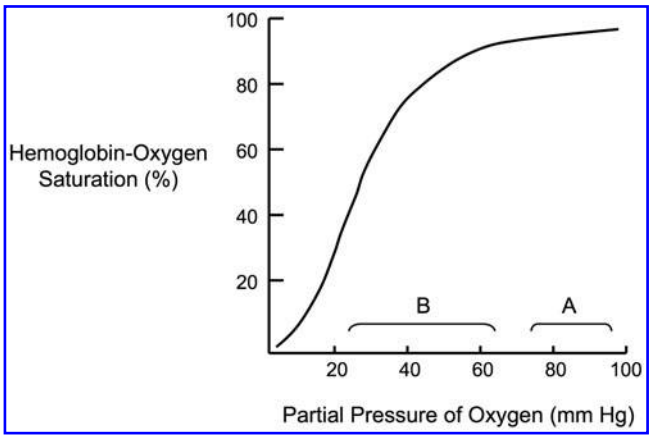

FIG. 1. This hemoglobin–oxygen dissociation curve depicts how hemoglobin–oxygen saturation varies as a function of the partial pressure of oxygen (Po_2). At sea level, individuals with normal gas exchange have arterial Po_2 values that fall in the range depicted by bar A. This places them on the flat portion of the dissociation curve whereby saturation changes minimally in response to changes in Po_2. At high altitude, individuals will have arterial Po_2 values in the range depicted by bar B. In this range, the dissociation curve is steep and, as a result, saturation changes significantly in response to small changes in the Po_2.

types of devices that are available to travelers and medical providers and the potential uses of oximetry at high altitude. We then consider the potential pitfalls of pulse oximetry at high altitude to help all users to avoid erroneous data interpretation and application of the devices in inappropriate situations.

How Pulse Oximeters Work

Complete descriptions of the operating principles behind pulse oximetry are available elsewhere (Schnapp and Cohen 1990; Sinex 1999). Pulse oximeters provide an estimate of arterial hemoglobin oxygen saturation, that is, the percentage of hemoglobin binding sites that are occupied at any one time by oxygen. In general, the saturation is a function of the arterial partial pressure of oxygen (Po_2), a relationship best graphically described by the hemoglobin–oxygen dissociation curve (Fig. 1). Pulse oximeters provide an estimate of oxygen saturation by taking advantage of the fact that oxygenated and deoxygenated hemoglobin absorb light differently from each other. Two light-emitting diodes (LEDs) project light of specific wavelengths (660 and 940 nm in most oximeters) through a cutaneous vascular bed. The fingers are typically used, although the earlobe may be used in certain situations. As the light of each wavelength passes through the vascular bed, a portion of it is absorbed by hemoglobin, while the remainder passes to the other side of the cutaneous vascular bed, where a photodiode detector measures the intensity of the transmitted light at each wavelength. The technique subtracts out the constant absorption of light by all nonvascular structures and the nonpulsatile capillary and venous blood, leaving only the arterial signal. This analyzed amount of transmitted light will be a function of the relative prevalence of oxygenated and deoxygenated hemoglobin. Adequate pulsatile blood flow is necessary for proper device operation, because it is only with pulsatile flow that the oximeter can distinguish light that is absorbed by hemoglobin in arterial blood from that absorbed by other elements. The pulse oximeter then uses an internal algorithm to translate the intensity of transmitted light at the different wavelengths to determine oxygen saturation. This value is referred to as the Spo_2, where p refers to the fact that oxygen saturation is measured by pulse oximetry, rather than by co-oximetry on an actual arterial blood sample (Sao_2).

When considering the utility of pulse oximeters and data in the literature regarding how well they function, three important variables should be taken into consideration: accuracy, precision, and bias (Fig. 2). Accuracy refers to how close the measured value is to the true value. In pulse oximetry, this refers to how close the pulse oximeter value is to the saturation measured from arterial blood by co-oximetry. Precision refers to how close the measured values are to each other. That is, with repeated measurements, will you obtain similar or different values? Bias refers to the difference between the average of the measurements made by the device and its true value. For example, a pulse oximeter that consistently reads 2% below the Sao_2 value is a biased instrument. If the bias is known, the user or device manufacturer can correct for it, although the lack of readily available data about the bias of many of these devices makes this a difficult task for device users. The ideal pulse oximeter is one with high accuracy and precision, but low bias.

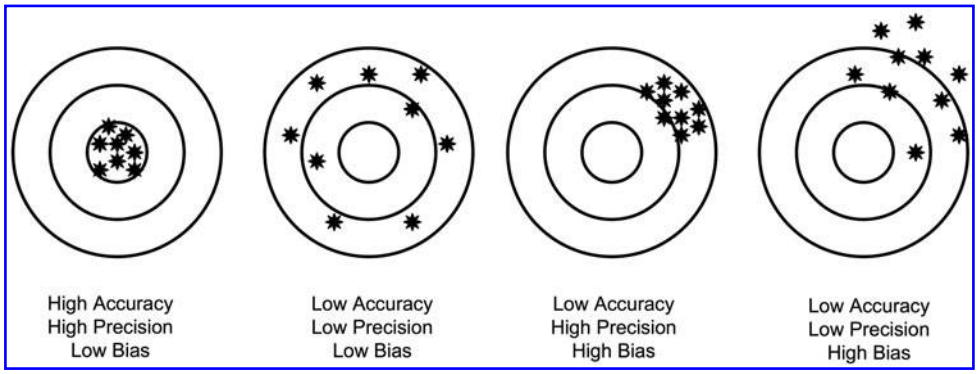

FIG. 2. Visual description of the terms accuracy, precision, and bias that are used throughout the literature to evaluate pulse oximeters and other monitoring devices. Each diagram represents a bull's-eye target, and the location of the markers relative to the center of the target demonstrates the meaning of each term.

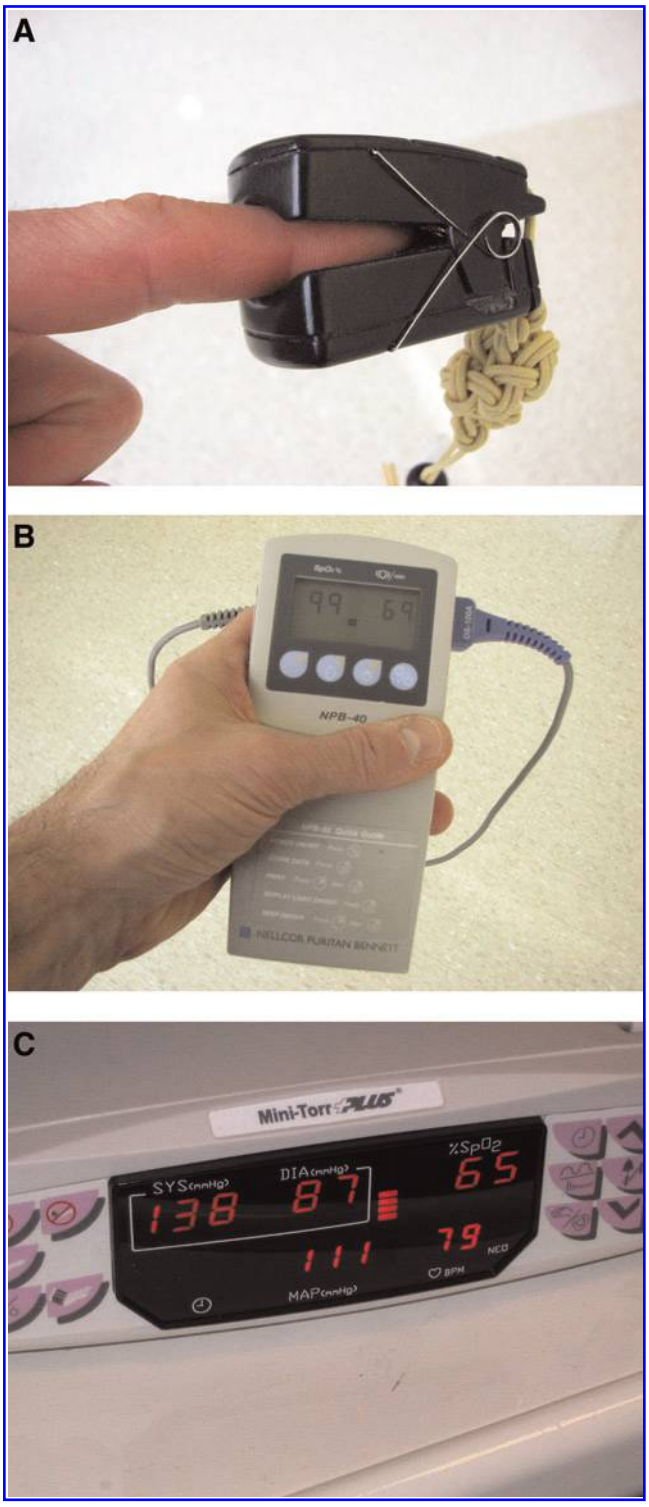

FIG. 3. Images of pulse oximeters from each of the three main categories of devices that may be used at high altitude: (**A**) pocket oximeter, the Nonin Onyx® 9500, Nonin Medical, Inc.; (**B**) handheld oximeter, the Nellcor NPB-40; (**C**) table-top oximeter with blood pressure monitoring capabilities, the Mini-Torr Plus.

Types of Pulse Oximeters for Use at High Altitude

Several categories of pulse oximeters might be used in a variety of contexts at high altitude. Images of representative devices from each category are shown in Fig. 3.

Pocket pulse oximeters

This category includes a series of battery-powered devices that are small and light enough to fit in pants or jacket pockets, such as the Nonin Onyx®9500 (Nonin Medical, Inc., Plymouth, MN, USA) or the SPO Medical PulseOx series devices (SPO Medical, Inc., Simi Valley, CA, USA). Given their size and relatively low cost (many of the devices other than the Nonin Onyx 9500 retail for less than $100), the pocket oximeters, a term originally coined elsewhere (Torre-Bouscoulet et al., 2006), are the devices most likely to be used by trekkers, climbers, or guides. While there is an exceptionally large array of U.S. Food and Drug Administration (USFDA)-approved pocket devices, what is lacking is adequate, readily accessible documentation about the accuracy, bias, and other operating characteristics. The Nonin Onyx 9500 is perhaps the only device for which one can easily find such information (Table 1). There is also a distinct lack of information in the medical literature about how well the various devices perform in comparison with co-oximetry, nonportable pulse oximeters, or other small, lightweight devices, both at sea level and, in particular, at high altitude. In one of the few studies of pocket oximeters at high altitude, Torre-Bouscoulet and colleagues (2006) measured pulse oximetry in 96 patients at 2240 m using the Nonin Onxy 9500 and found a mean Spo_2 of $86.7 \pm 8.6\%$, compared with a mean Sao_2 of $87.2 \pm 11\%$, with a corresponding bias of $0.28 \pm 3.1\%$.

Hand-held portable pulse oximeters

The second category is the larger, battery-powered, portable devices, such as the Nonin PalmSat® 2500 (Nonin Medical, Inc.), Nellcor OxiMax™-N-65 (Covidien, Dublin, Ireland), or Masimo Rad-57 (Masimo Corp., Irvine, CA, USA), that can be carried in the palm of the hand, but are too big for easy storage in clothing. These devices are heavier and more expensive than the pocket devices and are probably better suited for use as part of a medical kit with larger groups, in a medical tent on a large expedition, or at a remote clinic. Accuracy data are not reported for $Sao_2 < 70\%$ because the USFDA does not require accuracy reporting for saturations in this range. Similarly to the pocket devices, there is a lack of data about how these devices perform at high altitude. Operating characteristics for a representative sample of these devices are provided in Table 1.

Table-top pulse oximeters

A large number of pulse oximeters are better designed for use in a clinic setting or medical tent and are considered to be too big to carry in a pack or as part of a mobile medical kit. Among the many available devices, representative examples include the Masimo Radical-7 (Masimo Corp., Irvine, CA, USA), Nellcor OxiMax N-600x™ (Covidien), and the Nonin Avant® series (Nonin Medical, Inc.). Some devices, such as the Mini-Torr Plus (Smiths Medical, Kent, UK), function as combined blood pressure and pulse oximetry monitors. These are the most expensive of the oximeters discussed in this article, with costs ranging in the several thousands of US dollars, and are also the devices for which there are the most data in the literature that compare them to other pulse oximeters, as well as co-oximetry. Also, these devices have not been studied at terrestrial high altitude, although data do suggest that many of them overestimate Sao_2 in subjects when it is <80% (Feiner

Table 1. Operation Characteristics of Representative Pulse Oximeters

Device	*Accuracy*[a]	*Maximum altitude of operation (m)*	*Operating temperature (°C)*	*Power features (source and run time)*	*Weight (kg)*	*Dimensions (cm)*
Nonin Onyx® 9500 (pocket)	Sao_2 70%–100% ± 2%	12,000	0 to 40	2 AAA batteries; 18-h life	0.06	3.5×5.8×3.3
Nonin PalmSat® 2500 (handheld)	Sao_2 70%–100% ± 2%–3%	12,000	−20 to 50	4 AA batteries; 8-h life; 40-h life on rechargeable batteries	0.21	7×13.8×3.2
Nellcor OxiMax™ N-65 (handheld)	Sao_2 70%–100% ± 2%	3,012	5 to 40	4 AA batteries; 15–40 h	0.28	7.3×15.9×3.5
Masimo Rad-57 (handheld)	Sao_2 70%–100% ± 2%–3%	5,486	−18 to 54	4 AA batteries; up to 10 h	0.37	15.8×7.6×3.6
Smiths Medical Mini-Torr Plus (tabletop)	Sao_2 70%–100% ± 2%	Not reported	0 to 50	105–230 V ac; 6-h run time off internal nickel–cadmium battery	1.6	8.2×25.4×14
Nonin Avant® 9600 (tabletop)	Sao_2 70%–100% ± 1%–2%	12,000	0 to 50	100–240 V ac; 12-h life when running on charge	1.00	18.4×14×11.4
Nellcor OxiMax™ N-600x (tabletop)	Sao_2 70%–100% ± 2% Sao_2 60%–80% ± 3%	3,658	5 to 40	100–240 V ac; 7-h run time running off internal lead–acid battery	2.6	8.4×26.4×17.3
Masimo Radical-7 (tabletop)	Sao_2 70%–100% ± 2% Sao_2 60%–80% ± 3%	5,486	5 to 40	100–240 V ac; 10-h run time running off internal NiMH battery	1.95	8.9×26.7×19.6

[a]Reported accuracy may vary based on motion, type of sensor, adequacy of perfusion, and type of patient (e.g., pediatric vs. adult).
Information obtained from device manufacturers' product brochures:
- Masimo oximeters: http://www.masimo.com/rad-57/
  http://www.masimo.com/rainbow/Radical7.htm
- Nellcor oximeters: http://www.nellcor.com/prod/product.aspx?id=295
  http://www.nellcor.com/prod/product.aspx?id=294
- Nonin oximeters:http://www.nonin.com/PulseOximetry/Fingertip/Onyx9500
  http://www.nonin.com/PulseOximetry/Handheld/PalmSAT2500
  http://www.nonin.com/ProductDetail.aspx?ProductID=12
- Smiths medical oximeter: http://hwww.smiths-medical.com/upload/products/pdf/Mini-TorrPlus3.pdf

et al., 2007). Operating characteristics for some these devices are provided in Table 1.

Uses for Pulse Oximetry at High Altitude

In several situations, pulse oximetry has either a clear indication for use at high altitude or has been proposed as a way to monitor people traveling in this environment.

Evaluation of the ill patient

Clearly, the most important situation in which pulse oximetry should be used is in the evaluation of patients presenting with symptoms of altitude illness or other medical problems. A low Spo_2 relative to the expected value for a given altitude, an important concept discussed later, should always raise suspicion of high altitude pulmonary edema (HAPE), because multiple reports have documented markedly lower Spo_2 in these individuals compared with healthy people at the same elevation (Bartsch et al., 1987; Scherrer et al., 1996; Fagenholz et al., 2007). It is important to remember, however, that this finding is not specific for HAPE, since inappropriately low values may be seen in patients with other pulmonary problems, such as pneumonia, venous thromboembolism, pneumothorax, pleural effusions, or disorders associated with hypoventilation.

Monitoring the patient with underlying cardiopulmonary disease

Patients with severe underlying cardiopulmonary diseases, such as chronic obstructive pulmonary disease, cardiomyopathy, or congenital heart disease, may be at risk for severe hypoxemia following ascent to high altitude (Luks and Swenson, 2007; Luks et al., 2010). Because high altitude simulation testing (Dine and Kreider, 2008) may not always be feasible prior to their planned sojourn and because travel with supplemental oxygen is logistically difficult, many of these patients may wish to monitor their oxygen saturation using a portable device or by periodic clinic visits upon arrival and use the measured values as a guide for further action (Luks, 2009). No data suggest this strategy improves outcomes at high altitude, but it is a prudent approach in light of the logistical issues previously noted and for the potential of detecting clinical deterioration in certain patients following arrival.

Assessing the likelihood of acute altitude illness following arrival at high altitude

Numerous studies have attempted to determine whether there is a link between oxygen saturation at high altitude and the likelihood of developing acute mountain sickness (AMS), with some studies reporting a link between hypoxemia and the development of AMS (Roeggla et al., 1996; Roach et al., 1998; Basnyat et al., 1999; Tannheimer et al., 2002; Burtscher et al., 2004; Karinen et al., 2010; Koehle et al., 2010) and others reporting no relationship (Roach et al., 1995; O'Connor et al., 2004). No studies have examined a link between hypoxemia upon arrival and subsequent development of high altitude pulmonary edema (HAPE), although Sonna and colleagues (2000) did report a case in which routine pulse oximetry identified a case of HAPE in a person who had yet to present to medical providers. In considering the AMS studies, it is important that only a few of the studies used a prospective approach and asked whether the presence of hypoxemia early in the trip predicts development of AMS at a later time (Karinen et al., 2010; Roach et al., 1998; Tannheimer et al., 2002); the remainder of the studies measured Spo_2 at the same time the subjects were being assessed for AMS (Roeggla et al.,1996; Basnyat et al., 1999; O'Connor et al., 2004; Koehle et al., 2010).

The question of a link between a low Spo_2 and AMS is important, because a strong positive relationship could serve as a basis for screening trekkers or climbers with pulse oximetry during the course of their ascent. Although the preponderance of data suggests a link between a low Spo_2 and the development of AMS, it is still not clear that routine monitoring of pulse oximetry during ascent is of any utility. There is often significant interindividual discrepancy between the measured saturation values and physical well-being, a phenomenon well-demonstrated in a recent report of arterial blood gas results from four climbers at 8400 m on Mt. Everest (Grocott et al., 2009); one individual had an arterial oxygen saturation of only 34% yet felt well, performed normally, and successfully climbed the mountain. There are also no randomized studies of hypoxemic individuals to determine if starting them on AMS prophylaxis after they are identified as having a low Spo_2 prevents AMS later in the trip. If we take screening to an extreme and turn around those people with low saturations, it would become a self-fulfilling prophecy that such people would not do well at high altitude. Finally, when we consider the potential pitfalls of pulse oximetry at high altitude (described in detail later), there is a significant potential for misidentification of those at risk for AMS, with routine screening practices leading to unnecessary use of pharmacologic prophylaxis or changes in travel plans. Even if people are not placed on prophylaxis or continue their itinerary, routine monitoring of pulse oximetry runs the risk of causing unnecessary worry in those whose values fall in the lower end of the normal range or a false sense of security in those with values in the upper portion of that range. The issue of what constitutes a "normal" Spo_2 for a given altitude is considered below.

Predicting summit success

Related to the studies examining a link between Spo_2 on arrival and the development of AMS, several studies have sought to determine whether Spo_2 is predictive of summit success, with discrepant results. Davies and colleagues (2009) reported no link between Spo_2 and summit success on the Marangu route on Kilimanjaro (5895 m). Tannhemier and colleagues (2002) noted that Spo_2 measured on arrival at base camp was predictive of the maximum altitude reached on Broad Peak (8047 m), and Lazio and colleagues (2010) reported that Spo_2 measured following a 6-min walk test was predictive of summit success on Aconcagua (6962 m). Even if these studies were in agreement, this is another situation in which routine assessment of Spo_2 creates either a self-fulfilling prophecy, whereby those with lower than expected saturations are turned around without being able to make a summit attempt, or causes unnecessary worry on the part of those with measured values that fall in the lower part of the normal range for a given altitude. In the end, it should be the person's symptoms and physical condition that dictate whether they go higher or abort their planned excursion, rather than a pulse oximetry measurement.

Table 2. Sources of Error in Pulse Oximetry Readings with Digital Probes

Excess ambient light
Low cardiac output (e.g., hypovolemia)
Hypothermia with peripheral vasoconstriction
Motion artifact (e.g., shivering)
Dyshemoglobinemias (methemoglobinemia; carboxyhemoglobinemia)
Nail polish (black, green, blue)
Acrylic fingernails
Dark skin pigmentation
Digital clubbing

Sources: Sinex, 1999; Van Ginderdeuren et al., 2006.

Pitfalls of Pulse Oximetry at High Altitude

Many factors are known to affect the accuracy of pulse oximetry (Table 2) (Sinex, 1999; Jubran, 2004). Regardless of which type of oximeter is employed to measure Spo_2, users should be aware of these and other potential pitfalls of the technique at high altitude. Given data that suggest that trained providers, including physicians and nurses, have limited knowledge of the sources of error in pulse oximetry (Howell, 2002), it is reasonable to suspect that nonmedical persons using pulse oximetry at high altitude may also be unaware of these issues. Efforts should be made to educate users of these devices to minimize the risk of improper clinical decisions.

Shape of the hemoglobin–oxygen dissociation curve

The hemoglobin–oxygen dissociation curve is sigmoidal in shape whereby oxygen saturation changes minimally in response to changes in arterial oxygen tension at high partial pressures of oxygen, but varies to almost a 10-fold greater extent in response to small changes in arterial oxygen tension at the lower end of the range (20 to 60 mmHg). (Fig. 1). This feature of hemoglobin–oxygen binding can have an important effect on pulse oximeter readings at high altitude. With travel to elevations $\geq$3000 m, average barometric pressure will fall to a point where the arterial Po_2 in most individuals will fall on the steep portion of the hemoglobin–oxygen dissociation curve whereby small changes in oxygen tension lead to large changes in oxygen saturation. Any transient hyper- or hypoventilation on the part of testing subjects owing to, for example, talking, recent physical movement, deep breathing, or holding their breath will cause transient changes in the alveolar Po_2 that will translate into changes in the arterial Po_2 and subsequent significant changes in the measured saturation. This problem may be particularly difficult when monitoring oxygen saturation in sleeping individuals with periodic breathing, because the minute-to-minute changes in minute ventilation will lead to large fluctuations in the alveolar and arterial oxygen tensions. For this reason, pulse oximeter readings to assess the awake patient's clinical status should only be taken after the individual has remained silent and quietly breathing at rest for at least a period of several minutes. The probe should then be left on for several minutes, and the most frequent saturation value should be taken as the relevant saturation, rather than the initial displayed value at the start of monitoring.

Accuracy of pulse oximeter devices at high altitude

The high altitudes at which trekkers and climbers might be using these devices are significant for pulse oximeter function in another important respect. As noted earlier, pulse oximeters actually measure absorbency ratios, and these ratios are converted to an oxygen saturation value using calibration algorithms derived from experiments in healthy volunteers. In these experiments, the volunteers were exposed to hypoxic conditions, and oxygen saturation was then measured by pulse oximeter and in vitro laboratory co-oximetry. This approach was limited by the fact that the volunteers could only be exposed to modest degrees of hypoxia with nadir oxygen saturations of roughly 75% to 80%. At oxygen saturations below these levels, the data output from pulse oximeters is no longer based on actual comparisons between pulse oximetry and co-oximetry and, instead, is based on extrapolation from the data obtained at the higher saturation values. As a result, the accuracy, precision, and bias of these devices fall off at saturation values below 70% to 75% (Sinex, 1999) and may vary considerably between devices with these degrees of hypoxia (Severinghaus et al., 1989). In fact, as noted previously, many manufacturers do not report accuracy data for their devices at saturation values below 70%, since this is not required by the USFDA. (Locating any accuracy data for small pocket oximeters other than the Nonin Onyx 9500 is particularly difficult.) These accuracy problems may be exacerbated in dark-skinned individuals traveling at high altitude; it has been reported that several pulse oximeter models overestimate arterial oxygen saturation with exposure of such individuals to hypoxia (Bickler et al., 2005; Feiner et al. 2007). Given these issues, climbers and trekkers moving to very high altitudes (>4500 m), where baseline oxygen saturation in healthy individuals may fall below 75%, should avoid making clinical decisions based on small differences in oxygen saturation compared with other travelers in the group or with earlier values for a particular individual.

What is a normal saturation for a given altitude?

Clearly, oxygen saturation should decline with increasing altitude, but what constitutes a "normal" saturation for a given elevation is not entirely clear. We can get some sense of the expected values for particular elevations by reviewing oxygen saturation data from research studies conducted across different elevations (Table 3, Fig. 4). Although these values provide a reasonable estimate of the expected values, several important issues must be taken into consideration. First, although the saturation values across studies at a given elevation are often in agreement, other studies report discrepant values.

Second, in many studies, the standard deviations, 95% confidence intervals, or interquartile ranges are very large. Fagenholz and colleagues (2009), for example, reported a mean Spo_2 of 87.6 and a standard deviation of 4.5% at 4240 m. And Dehnert and colleagues (2010) reported a mean Spo_2 of 72% and standard deviation of 7% at 4559 m. These large standard deviations fit with what we expect from a physiologic standpoint. Because of the nearly l0-fold difference in hypoxic ventilatory response (HVR) among healthy individuals (Weil, 2003), there will be a large range of Sao_2 at any altitude. The effect of HVR on the wide range of saturation in normal individuals is magnified by the fact that those with a brisk HVR will have a more prominent respiratory alkalosis.

Table 3. Reported Oxygen Saturation Values at Various High Altitude Locations

Study	*Location and altitude*	*Number of subjects*	*Measured saturation (mean ± SD)*	*Timing of measurements*
Kaminsky et al., 1996	5 Clinics in Summit County, Colorado, USA, ≥2720 m	10[H]	Median: 93 IQR: 90–94	Not specified
Kriemler et al., 2008	Jungfrau-Joch, Switzerland, 3450 m	20[a]	89.4 ± 3.1	4–5 h after arrival
Martin et al., 2010	Namche Bazaar, Nepal, 3500 m	24	90.4 ± 2.3	Upon arrival (day 4–6 of trek)
Hupperets et al., 2004	Barcroft Laboratory, California, USA, 3800 m	5	93.2 ± 0.8	24 h after arrival
McElroy et al., 2000	Barcroft Laboratory, California, USA, 3800 m	24	87.6 ± 2.2 88.2 ± 2.3[b]	Nights 1 and 2 after arrival
Fagenholz et al., 2007	Pheriche, Nepal, 4240 m	7[H]	87 ± 2.8	Not specified
Fagenholz et al., 2009	Pheriche, Nepal, 4240 m	218[H]	87.6 ± 4.5	Within 1 to 2 days of arrival
Basnyat et al., 2008	Pheriche (4250 m) and Dingboche (4350 m), Nepal	177[P]	85.9 ± 4	Average of 5 nights since start of trek
Baggish et al., 2010	Pikes Peak, Colorado, USA, 4300 m		83 ± 4	90 min after arrival after 7 days at 2200 m
Bosch et al., 2009	4500 m during climb of Muztagh Ata, China	27	84 ± 3	Measurements on day 2 or 4 of climb[c]
Agostini et al., 2010	Capanna Margherita, Italy, 4559 m	23	Median: 80 IQR: 78–81	Day after arrival after ascent over 36 h
Bartsch et al., 1991	Capanna Margherita, Italy, 4559 m	7[H]	70 ± 2.6	4 h after arrival after ascent over 22 h
Bartsch et al., 1991	Capanna Margherita, Italy, 4559 m	7[H]	78 ± 2.0	Morning of day 4 at the hut
Dehnert et al., 2010	Capanna Margherita, Italy, 4559 m	10[H]	72 ± 7	4 h after arrival after ascent over 22 h
Dehnert et al., 2010	Capanna Margherita, Italy, 4559 m	10[H]	80 ± 5	44 h after arrival after ascent over 22 h
Scherrer et al., 1996	Capanna Margherita, Italy, 4559 m	17[H]	81 ± 5	18–36 h after arrival after ascent over 22 h
Davies et al., 2009	4700 m during climb of Mt. Kilimanjaro, Tanzania	43[d]	79. 2 No SD reported	Day 4 of climb
Kayser et al., 2008	4740 m during climb of Mt. Kilimanjaro, Tanzania	16[P]	87 ± 4	Day 5 of climb
Bailey and Davies, 2001	Everest Base Camp, Nepal, 5180 m	9[P]	85 ± 5	Morning after arrival at base camp (day 11 of trek)
Ghofrani et al., 2004	Everest Base Camp, Nepal, 5180 m	14	Median: 83 95% CI: 80%–86%	Measurements made after 6 days at base camp following 8-day trek
Martin et al., 2010	Everest Base Camp, Nepal, 5300 m	24	80 ± 6.1	Upon arrival (days 15-17)
Agostini et al., 2010	Everest Base Camp, Nepal, 5350 m	16	Median: 81 IQR: 78–85	Over 2 days after arrival after 10 day trek
Bosch et al. 2009	5533 m during climb of Mustagh Ata, China	27	75 ± 6	Measurements on day 6 or 7 of climb[c]
Bosch et al., 2009	6265 m during climb of Mustagh Ata, China	27	73 ± 6	Measurements on day 13 or 17 of climb[c]
Martin et al., 2010	6400 m during climb of Mt. Everest, Nepal	14	72 ± 5.5	Upon arrival (days 34 to 35 of expedition)
Bosch et al., 2009	6865 m during climb of Mustagh Ata, China	15	66 ± 3	Measurements on day 18 of climb

Altitudes listed are those reported in the study.
Superscript P: reported values for subjects in the placebo group in a randomized study.
Superscript H: reported values for subjects who were AMS- or HAPE-free (i.e., healthy) during a study comparing AMS or HAPE patients with healthy patients.
IQR: Interquartile range.
[a]Data are from the adult subjects in the study.
[b]Data are from a cross-over design study. Each value given represents the saturation prior to each of the testing situations in the study independent of the order in which participants were tested.
[c]Subjects in this study followed one of two different ascent profiles. Spo_2 data for groups using each ascent profile were not separated.
[d]Data from successful summiteers not taking acetazolamide during 5-day ascent to summit.

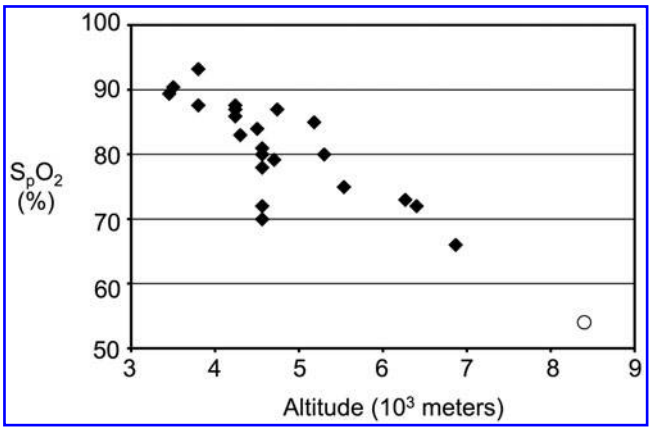

FIG. 4. Graphical display of the average hemoglobin oxygen saturation measured by pulse oximetry in different research studies conducted at high altitude. Data are reported for only those studies that report mean values. Error bars have been omitted from the individual data points. In studies comparing outcomes between treatment and control groups, data are reported for the control group only. In studies comparing sick and healthy (AMS- or HAPE-free) individuals, data are reported for the healthy individuals only. The open circle represents the average oxygen saturation measured by co-oximetry on arterial blood samples taken from 4 climbers at 8400 m on Mt. Everest (Grocott et al., 2009).

This leads to a further shift in their hemoglobin–oxygen dissociation curve to the left relative to those with more blunted responses and, as a result, a higher saturation for any Po_2. When viewed together, this physiology and the reported standard deviations in the literature suggest that the range of "normal" values at a particular elevation among individuals feeling well may actually be very broad.

Third, it is important to consider the timing of the measurements relative to arrival at high altitude, because, given the effects of acclimatization and maturation of ventilatory responses, the expected value on day 1 at a given elevation will not necessarily be the same as that on day 3 or 4. This is demonstrated nicely in data from Bartsch and colleagues (1991), who measured oxygen saturation in climbers on arrival and over the course of 4 days at 4559 m and noted an increase in Spo_2 from $70\% \pm 2.8\%$ to $78\% \pm 2\%$ over this period. Finally, barometric pressure for a given elevation is lower at higher latitudes (West et al., 1983). As a result, the expected value at 4200 m on Denali (62°N latitude) may not be the same as at 4200 m in Pheriche (27°N latitude).

Given all these issues, it is clear that reliable normal values for a given elevation may not be available to people making these measurements. Lacking such data, guides and other travelers may need to rely on the predominant value in their group at a given altitude as the expected value, provided these measurements were taken in an appropriate manner. The problems defining the normal or expected saturation provide further evidence that clinical decisions based on oxygen saturation should not rely on subtle distinctions of only a few percentage points when compared with either other people in a group, earlier readings for a given individual, or known normal values. Instead, clinicians, guides, or other people taking measurements should be looking for large changes or deviations from expected values before implementing action plans.

Other environmental conditions at high altitude

Important environmental conditions at high altitude, including cold temperature and increased sunlight, may affect the accuracy of pulse oximeter readings. Pulse oximeters must distinguish signal from noise and will not generate accurate readings when the signal-to-noise ratio is low (Sinex, 1999). Although many potential sources of low signal-to-noise ratio, such as hypotension, use of vasoconstricting agents, and peripheral vascular disease, are more likely in a hospital setting than among travelers at high altitude, another factor, cold-induced peripheral vasoconstriction, may affect device accuracy. Intense vasoconstriction may limit blood flow to the extremities and, as a result, decrease the pulsatility of flow necessary to detect an accurate signal. Also, the reported temperature range for operation of many devices is above 0°C (Table 1). For these reasons, people traveling in cold environments should ensure that they are taking measurements within conditions appropriate for their oximeter and that the individuals have warm extremities prior to any measurements. Caution should also be used when interpreting values in travelers assessed during hypothermia.

Ambient light interference from a variety of sources has been shown to affect the accuracy of pulse oximeters by causing what is referred to as false signal (Sinex, 1999). Given that sun exposure is increased at high altitude, particularly when individuals are traveling on snow-covered terrain, it is reasonable to ask whether this could be a source of false signal at high altitude. Several reviews on pulse oximetry mention this possibility (Schnapp and Cohen, 1990; Sinex, 1999), but primary literature supporting this claim is difficult to find. When using a pulse oximeter in any light conditions, care should always be taken to ensure that the device fits well on the finger, and finger devices should not be applied to earlobes or toes, because the poor fit in these situations allows excessive ambient light into the device sensor. Given the possible interference from excessive sunlight, it is also worth considering doing measurements indoors, in a tent, or by covering the hand in a manner that blocks much of the ambient light.

Dyshemoglobinemias

Dyshemoglobinemias, such as methemoglobinemia or carboxyhemoglobinemia, can cause misleading pulse oximeter values that do not reflect the true level of oxyhemoglobin in the blood. Although the former would be extremely unlikely among high altitude travelers, unless they were using sulfonamide antibiotics or other medications known to cause this problem, the latter is potentially a concern in certain groups, particularly those cooking inside tents or other small, enclosed places. Thomassen and colleagues (2004), for example, have demonstrated that the mean carboxyhemoglobin level rose to 21.5% in seven health volunteers using a cooking stove in a small tent for 120 min, and Foutch and Henrichs (1988) have documented a case of fatal carbon monoxide poisoning in two climbers on Denali. Because the absorbance patterns of carboxyhemoglobin resemble those of oxyhemoglobin at one of the wavelengths used by pulse oximeters (660 nm), the pulse oximeter overestimates the true oxygen saturation in a person with significant carbon monoxide exposure. Although the diagnosis of carbon monoxide poisoning may be apparent in many cases when adequate history is available, in cases where the risk of carbon monoxide expo-

Table 4. Potential Problems with Pulse Oximetry Readings at High Altitude and Proposed Solutions

Potential problem	*Solution*
Oxygen saturation changes readily in response to small changes in arterial Po_2 at high altitude.	Ensure subjects are at rest and breathing quietly for several minutes before taking measurement. Measure value for several minutes to determine the (time-weighted average) predominant value.
Accuracy, bias, and precision of pulse oximeter declines when the arterial saturation is below 80%.	Avoid making clinical decisions or large changes in travel plans based on small differences in pulse oximetry values relative to other individuals or earlier values obtained in a particular individual. Avoid routine screening of individuals for acute mountain sickness or potential for summit success.
The normal value for a particular elevation may not be known with certainty.	Expect a wide range of normal values for a particular elevation. Expect the normal value to increase over time at a given elevation. Expect the normal value for a given elevation to be lower at the extremes of latitude. Do not base clinical or travel planning decisions on small differences in oxygen saturation. Avoid routine screening of individuals for acute mountain sickness or potential for summit success.
Cold-induced vasoconstriction and other sources of poor perfusion may decrease pulsatility of blood flow in the digits and cause erroneous values.	Warm the distal extremity as best possible prior to making measurements and do not trust data output unless the device provides evidence of a good pulsatile signal. Use caution when interpreting data in hypothermic patients.
Excess ambient light may cause false signal and lead to erroneous values.	Ensure oximeter probe fits securely on the finger. Do not place on earlobe or toe. Consider protecting from excess sunlight by performing measurement indoors or in a tent or by shielding the extremity during the measurement.
Dyshemoglobinemias can cause misleading pulse oximetry readings	Use caution when interpreting pulse oximetry values in travelers with risk factors for carbon monoxide exposure (e.g., cooking in their tent). Ill patients taking sulfa antibiotics with oxygen saturation 87% to 89% that does not improve with supplemental oxygen should be suspected of methemoglobinemia.

sure is not readily appreciated, the falsely elevated pulse oximeter values can lead to inappropriate assessment of the patient who presents with dyspnea on exertion or symptoms resembling those of acute mountain sickness.

Summary

Pulse oximetry will continue to be widely used at high altitude. Clinics and other medical providers depend on it for the evaluation of people with respiratory and other complaints, and the increasing availability of low-cost pocket devices makes it highly likely that trekkers, climbers, and guides will also use them on a frequent basis. The ease of use makes these devices inherently appealing but, as discussed throughout this review, unrecognized potential pitfalls of the technique could lead to inaccurate measurements and inappropriate clinical decisions. These important considerations are summarized in Table 4. Users of these devices should familiarize themselves with these issues in order to minimize erroneous measurements, use in inappropriate situations, and misinterpretation of data that could lead to unnecessary disruption in high altitude travel plans.

Disclosures

The authors have no conflicts of interest or financial ties to disclose.

References

Agostoni P., Caldara G., Bussotti M., Revera M., Valentini M., Gregorini F., Faini A., Lombardi C., Bilo G., Giuliano A., et al. (2010). Continuous positive airway pressure increases haemoglobin O2 saturation after acute but not prolonged altitude exposure. Eur. Heart J. 31:457–463.

Baggish A.L., Fulco C.S., Muza S., Rock P.B., Beidleman B., Cymerman A., Yared K., Fagenholz P., Systrom D., Wood M.J., et al. (2010). The impact of moderate-altitude staging on pulmonary arterial hemodynamics after ascent to high altitude. High Alt. Med. Biol. 11:139–145.

Bailey D.M., and Davies B. (2001). Acute mountain sickness: prophylactic benefits of antioxidant vitamin supplementation at high altitude. High Alt. Med. Biol. 2:21–29.

Bartsch P., Maggiorini M., Ritter M., Noti C., Vock P., and Oelz O. (1991). Prevention of high-altitude pulmonary edema by nifedipine. N. Eng. J. Med. 325:1284–1289.

Bartsch P., Waber U., Haeberli A., Maggiorini M., Kriemler S., Oelz O., and Straub W.P. (1987). Enhanced fibrin formation in high-altitude pulmonary edema. J. Appl. Physiol. 63:752–757.

Basnyat B., Hargrove J., Holck P.S., Srivastav S., Alekh K., Ghimire L.V., Pandey K., Griffiths A., Shankar R., Kaul K., et al. (2008). Acetazolamide fails to decrease pulmonary artery pressure at high altitude in partially acclimatized humans. High Alt. Med. Biol. 9:209–2016.

Basnyat B., Lemaster J., and Litch J.A. (1999). Everest or bust: a cross sectional, epidemiological study of acute mountain sickness at 4243 meters in the Himalayas. Aviat. Space Environ. Med. 70:867–873.

Bickler P.E., Feiner J.R., and Severinghaus J.W. (2005). Effects of skin pigmentation on pulse oximeter accuracy at low saturation. Anesthesiology. 102:715–719.

Bosch M.M., Merz T.M., Barthelmes D., Petrig B.L., Truffer F., Bloch K.E., Turk A., Maggiorini M., Hess T., Schoch O.D., et al. (2009). New insights into ocular blood flow at very high altitudes. J. Appl. Physiol. 106:454–460.

Burtscher M., Flatz M., and Faulhaber M. (2004). Prediction of susceptibility to acute mountain sickness by SaO2 values during short-term exposure to hypoxia. High Alt. Med. Biol. 5:335–340.

Davies A.J., Kalson N.S., Stokes S., Earl M.D., Whitehead A.G., Frost H., Tyrell-Marsh I., and Naylor J. (2009). Determinants of summiting success and acute mountain sickness on Mt. Kilimanjaro (5895 m). Wilderness Environ. Med. 20:311–317.

Dehnert C., Luks A.M., Schendler G., Menold E., Berger M.M., Mairbaurl H., Faoro V., Bailey D.M., Castell C., Hahn G., et al. (2010). No evidence for interstitial lung oedema by extensive pulmonary function testing at 4,559 m. Eur. Respir. J. 35:812–820.

Dine C.J., and Kreider M.E. (2008). Hypoxia altitude simulation test. Chest. 133:1002–1005.

Fagenholz P.J., Gutman J.A., Murray A.F., and Harris N.S. (2007). Treatment of high altitude pulmonary edema at 4240 m in Nepal. High Alt. Med. Biol. 8:139–146.

Fagenholz P.J., Gutman J.A., Murray A.F., Noble V.E., Camargo C.A. Jr., and Harris N.S. (2009). Optic nerve sheath diameter correlates with the presence and severity of acute mountain sickness: evidence for increased intracranial pressure. J. Appl. Physiol. 106:1207–1211.

Feiner J.R., Severinghaus J.W., and Bickler P.E. (2007). Dark skin decreases the accuracy of pulse oximeters at low oxygen saturation: the effects of oximeter probe type and gender. Anesth. Analg. 105:S18–S23, tables of contents.

Foutch R.G., and Henrichs W. (1988). Carbon monoxide poisoning at high altitudes. Am. J. Emerg. Med. 6:596–598.

Ghofrani H.A., Reichenberger F., Kohstall M.G., Mrosek E.H., Seeger T., Olschewski H., Seeger W., and Grimminger F. (2004). Sildenafil increased exercise capacity during hypoxia at low altitudes and at Mount Everest base camp: a randomized, double-blind, placebo-controlled crossover trial. Ann. Intern. Med. 141:169–177.

Grocott M.P., Martin D.S., Levett D.Z., McMorrow R., Windsor J., and Montgomery H.E. (2009). Arterial blood gases and oxygen content in climbers on Mount Everest. N. Engl. J. Med. 360:140–149.

Howell M. (2002). Pulse oximetry: an audit of nursing and medical staff understanding. Br. J. Nurs. 11:191–197.

Hupperets M.D., Hopkins S.R., Pronk M.G., Tiemessen I.J., Garcia N., Wagner, P.D., Powell, F.L. Increased hypoxic ventilatory response during 8 weeks at 3800 m altitude. Respir Physiol Neurobiol 142(2-3):145–152.

Jubran A. (2004). Pulse oximetry. Intensive Care Med. 30:2017–2020.

Kaminsky D.A., Jones K., Schoene R.B., and Voelkel N.F. (1996). Urinary leukotriene E4 levels in high-altitude pulmonary edema: a possible role for inflammation. Chest. 110: 939–945.

Karinen H.M., Peltonen J.E., Kahonen M., and Tikkanen H.O. (2010). Prediction of acute mountain sickness by monitoring arterial oxygen saturation during ascent. High Alt. Med. Biol. 11:325–332.

Kayser B., Hulsebosch R., and Bosch F. (2008). Low-dose acetylsalicylic acid analog and acetazolamide for prevention of acute mountain sickness. High Alt. Med. Biol. 9:15–23.

Koehle M.S., Guenette J.A., and Warburton D.E. (2010). Oximetry, heart rate variability, and the diagnosis of mild-to-moderate acute mountain sickness. Eur. J. Emerg. Med. 17:119–122.

Kriemler S., Jansen C., Linka A., Kessel-Schaefer A., Zehnder M., Schurmann T., Kohler M., Bloch K., and Brunner-La Rocca H.P. (2008). Higher pulmonary artery pressure in children

than in adults upon fast ascent to high altitude. Eur. Respir. J. 32:664–669.
Lazio M.P., Van Roo J.D., Pesce C., Malik S., and Courtney D.M. (2010). Postexercise peripheral oxygen saturation after completion of the 6-minute walk test predicts successfully reaching the summit of Aconcagua. Wilderness Environ. Med. 21:309–317.
Luks A.M. (2009). Do lung disease patients need supplemental oxygen at high altitude? High Alt. Med. Biol. 10:321–327.
Luks A.M., Stout K., and Swenson E.R. (2010). Evaluating the safety of high-altitude travel in patients with adult congenital heart disease. Congenital Heart Dis. 5:220–232.
Luks A.M., and Swenson E.R. (2007). Travel to high altitude with pre-existing lung disease. Eur. Respir. J. 29:70–92.
Martin D.S., Goedhart P., Vercueil A., Ince C., Levett D.Z., and Grocott M.P. (2010). Changes in sublingual microcirculatory flow index and vessel density on ascent to altitude. Exp. Physiol. 95:880–891.
McElroy M.K., Gerard A., Powell F.L., Prisk G.K., Sentse N., Holverda S., and West J.B. (2000). Nocturnal O2 enrichment of room air at high altitude increases daytime O2 saturation without changing control of ventilation. High Alt. Med. Biol. 1:197–206.
O'Connor T., Dubowitz G., and Bickler P.E. (2004). Pulse oximetry in the diagnosis of acute mountain sickness. High Alt. Med. Biol. 5:341–348.
Roach R.C., Greene E.R., Schoene R.B., and Hackett P.H. (1998). Arterial oxygen saturation for prediction of acute mountain sickness. Aviat. Space Environ. Med. 69:1182–1185.
Roach R.C., Houston C.S., Honigman B., Nicholas R.A., Yaron M., Grissom C.K., Alexander J.K., and Hultgren H.N. (1995). How well do older persons tolerate moderate altitude? West. J. Med. 162:32–36.
Roeggla G., Roeggla M., Podolsky A., Wagner A., and Laggner A.N. (1996). How can acute mountain sickness be quantified at moderate altitude? J. Royal Soc. Med. 89:141–143.
Scherrer U., Vollenweider L., Delabays A., Savcie M., Eichenberger U., Kleger G.R., Firkle A., Ballmer P., Nicod P., and Bartsch P. (1996). Inhaled nitric oxide for high-altitude pulmonary edema. N. Engl. J. Med. 334:624–629.
Schnapp L.M., and Cohen N.H. (1990). Pulse oximetry: uses and abuses. Chest. 98:1244–1250.
Severinghaus J.W., Naifeh K.H., and Koh S.O. (1989). Errors in 14 pulse oximeters during profound hypoxia. J. Clin. Monitoring. 5:72–81.
Sinex J.E. (1999). Pulse oximetry: principles and limitations. Am. J. Emerg. Med. 17:59–67.
Sonna L.A., Kain J.E., Hoyt R.W., Muza S.R., and Sawka M.N. (2000). Ambulatory physiological status monitoring during a mountaineering expedition. Milit. Med. 165:860–866.
Tannheimer M., Thomas A., and Gerngross H. (2002). Oxygen saturation course and altitude symptomatology during an expedition to Broad Peak (8047 m). Int. J. Sports Med. 23:329–335.
Thomassen O., Brattebo G., and Rostrup M. (2004). Carbon monoxide poisoning while using a small cooking stove in a tent. Am. J. Emerg. Med. 22:204–206.
Torre-Bouscoulet L., Chavez-Plascencia E., Vazquez-Garcia J.C., and Perez-Padilla R. (2006). [Precision and accuracy of "a pocket" pulse oximeter in Mexico City]. Rev. Invest. Clin. 58:28–33.
Van Ginderdeuren F., Van Cauwelaert K., and Malfroot A. (2006). Influence of digital clubbing on oxygen saturation measurements by pulse-oximetry in cystic fibrosis patients. J. Cystic Fibrosis. 5:125–128.
Weil J.V. (2003). Variation in human ventilatory control: genetic influence on the hypoxic ventilatory response. Respir. Physiol. Neurobiol. 135:239–246.
West J.B., Lahiri S., Maret K.H., Peters R.M. Jr., and Pizzo C.J. (1983). Barometric pressures at extreme altitudes on Mt. Everest: physiological significance. J. Appl. Physiol. 54:1188–1194.

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