the trachea, controlled ventilation with high inspired concentrations of oxygen, CPAP, and the iv administration of furosemide.

REFERENCES


Evaluation of Pulse Oximetry

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Continuous assessment of arterial oxygenation is important in clinical management of critically ill or anesthetized patients. Analysis of arterial blood gases is reliable but is invasive and only provides intermittent information. Transcutaneous oxygen tension measurement provides continuous information but requires special site preparation, airtight probe mantling, and a potentially harmful local heat source to induce “arterialization.” Even then transcutaneous oxygen monitoring fails to perfectly reflect true arterial oxygenation.1 Analysis of arterial blood gases and transcutaneous oxygen measurements both provide oxygen tension (PO2) data from which the oxygen content and percentage of hemoglobin saturated with oxygen can be estimated. Arterial oxygen saturation of hemoglobin can be determined directly and continuously in vivo by using spectrophotometric oximetric techniques.2–5 The wave-length dependence of reduced versus oxyhemoglobin is evident from the prominent color differences in spectral light absorbance of “red” oxyhemoglobin and “blue” reduced hemoglobin. The light absorbances differences between reduced and oxyhemoglobin are described quantitatively by the molecular extinction coefficients in Beer’s Law. Other methods measure saturation using relative light absorption of two or more wavelengths directed through the ear vasculature. “Arterialization” is achieved by combinations of heat and chemical treatment to dilate the vascular bed in the measurement area. Such devices offer limited clinical utility because they are inconvenient and because it is technologically difficult to consistently differentiate light absorbance of the desired arterial blood from the absorbance of tissue and venous blood.6 This deficiency can be circumvented by measuring light absorbance changes time coherent with arterial pulsation.7

Pulse oximetry functions by positioning any pulsating arterial vascular bed between a two-wavelength light source and a detector. The pulsating vascular bed, by expanding and relaxing, creates a change in the light path length that modifies the amount of light detected. The familiar plethysmograph waveform results. The amplitude of the varying detected light depends upon the size of the arterial pulse change, the wavelength of
light used, and the oxygen saturation of the arterial hemoglobin. Because the detected pulsatile waveform is produced solely from arterial blood, using the amplitude at each wavelength and Beer's law allows exact beat-to-beat continuous calculation of arterial hemoglobin oxygen saturation with no interference from surrounding venous blood, skin, connective tissue, or bone.

A pulse oximeter recently has become commercially available.‡ A light source generated by two LEDs, wavelengths at approximately 660 and 940 nm, and a photodiode are mounted in a finger receptacle. No heating or "arterialization" techniques are required. Circuit control, saturation calculation, and display are managed by a microprocessor instrument. No user calibration procedure is required.

A study was conducted to evaluate the accuracy of the pulse oximeter over a broad range of arterial saturations. The protocol follows well-considered guidelines for ethical evaluation of devices, which requires clinically induced hypoxemia for meaningful assessment.

METHODS

A standard Ohio recirculating anesthesia machine was modified to supply continuous steady state inspiratory oxygen concentrations using a 6-l reservoir bag. An Instrumentation Laboratory Model 402 Oxygen Monitor (IL402) was inserted in the inspiratory limb, and the fresh oxygen supply hose was rerouted to enter the circle at the expiratory limb to maximize gas mixing before inspiration. The anesthesia circle initially was filled with room air; the circle then was closed and the subject instructed to breathe normally. By carefully adjusting oxygen inflow from below the metabolic consumption rate (ca. 100 ml/min) to well above the metabolic rate (300 ml/min), exact steady state inspired oxygen concentrations could be altered, monitored, and maintained for indefinite periods of time.

Five subjects were selected with the use of the following guidelines approved by our local institutional review board. All were healthy nonsmoking college students ranging in age from 18–25 years. Each was interviewed individually, asked to complete a detailed medical history questionnaire, received a complete physical examination, and had a hemoglobin determination and urinalysis performed. Each was informed of the complete procedure with the possible risks involved and was not asked to sign the permit until he or she returned on the day of the study. Each received monetary compensation for participation in the study.

For the study, an IV infusion and a radial arterial catheter were inserted. ECG and transduced arterial mean arterial pressure were displayed on an oscilloscope monitor. The pulse oximeter was placed on the ipsilateral index finger. Each subject was placed in the supine position and asked to hold the breathing mask himself and to breathe normally. The inspired oxygen concentration was changed in step decrements, each producing approximately a 5% decrease in arterial blood saturation. The inspired oxygen percentage was maintained constant at each plateau until pulse oximeter readings were stable. Then arterial blood, 5 ml, was withdrawn and analyzed immediately on a IL282 CO-Oximeter. The inspired oxygen concentration thereafter was decreased stepwise in like manner to 10%, which corresponded to an arterial saturation of approximately 70%.

Throughout the study, verbal contact was maintained with the subject who was reminded continually that if he felt too uncomfortable, he could terminate the procedure himself immediately by removing the breathing mask. (None voluntarily terminated the procedure.) From the lowest saturation point, the inspired oxygen concentration was increased in a similar stepwise manner to return to room air concentration. A few points then were taken breathing high oxygen concentrations.

The CO-Oximeter and saturation valves were compared with the results of the pulse oximeter. Percentage saturation was defined as the ratio of hemoglobin bound to oxygen divided by the total amount of hemoglobin available for reversibly binding to oxygen (oxymoglobin/reduced hemoglobin + oxyhemoglobin). Dyshemoglobins such as carboxyhemoglobin and methemoglobin were considered dysfunctional and were not considered in the comparison. The values from analysis of arterial blood were modified appropriately to be consistent with this definition.

RESULTS

The 79 pooled data points from the five subjects were analyzed by analysis of linear regression and student's t test. Correlation coefficient (R) was 0.98, the intercept −2.33, and the slope 1.03, with a standard deviation Syx of 1.83 (fig. 1). A P < 0.0001 was calculated. All subjects tolerated the procedure well without adverse sequelae.

DISCUSSION

In vivo oximetry is a clinically useful method of determining arterial hemoglobin oxygen saturation. The pulse oximeter technique allows complete elimination of artifact from the light absorption of tissue and venous blood, something not possible with traditional ear ox-
imetry techniques. Our data indicate that the pulse oximeter is linearly accurate and precise over the range evaluated (70–100%) in noninvasive measurement of arterial hemoglobin oxygen saturation.

The definition of hemoglobin saturation depends upon the treatment of the various hemoglobin species present. Hemoglobin may be unbound, may be bound to oxygen, or may be functionally inert. Oxygen saturation can be defined as the ratio of oxyhemoglobin to the sum of oxyhemoglobin and reduced hemoglobin, (functional or reversible saturation) or alternatively as the ratio of oxyhemoglobin to the sum of all hemoglobin species present, whether available or not for reversible binding to oxygen (total saturation). Hemoglobin not available for reversible oxygen binding is effectively removed from the functional hemoglobin pool and appears inert with changes in oxygen partial pressure. Excluding such dyshemoglobin species from the definition of oxygen saturation provides a more physiologic indication of arterial oxygen content versus oxygen saturation. However, the two definitions are interrelated mathematically.

Because pulse oximetry utilizes light absorbance changes produced by arterial pulsations, any event that significantly reduces vascular pulsations will reduce the instrument’s ability to calculate saturation. Adequate finger pulsation generally is lost with 1) hypothermia of a few degrees, 2) hypotension (mean blood pressure less than 50 mmHg), and 3) infusion of vasoconstrictive drugs. Pulsation of the nasal septal anterior enthrmoid artery (supplied by the internal carotid) persist under greater extremes than the finger pulse. A special nasal probe can be used in those circumstances.

Extensive studies have been made on errors and differences occurring with oxygen saturation measurements on in vitro hemolyzed blood, in vitro nonhemolyzed blood, and in vivo blood flowing in vessels. A detailed discussion of these variances is beyond the scope of this article. The pulse oximeter employed in this study utilizes two wavelengths (660 and 940 nm) generated by light-emitting diodes. Neither wavelength is an isobestic point.

Because only two wavelengths are used in this pulse oximeter, some error in measurement inherently will occur if dyshemoglobin species are present. The degree of error depends upon the quantity of dyshemoglobin present, as well as the relative spectral extinction of the
dyshemoglobins versus reduced hemoglobin and oxyhemoglobin at the specific wavelengths used. For this particular instrument, the maximum measurement error resulting from carboxyhemoglobin, the only significant dyshemoglobin present, is 10% of the carboxyhemoglobin percentage or less than 0.4%. Also, saturations no lower than 70% were achieved. The 70–100% range is certainly the most common clinically, but clinical patients inevitably will drift into lower saturations on occasion, and confirmed accuracy of oximetric devices in this lower range is desirable. Ethically, however, evaluation of accuracy at less than 70% saturation only can be obtained in the serendipitous situation when no deliberate hypoxemia has been induced. No error is introduced nor is the instrument limited by skin pigmentation.

The pulse oximeter accurately and precisely measures arterial hemoglobin oxygen saturation in the evaluated range of 70–100%.

References


An Unusual Complication of Nasotracheal Intubation

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Avulsion of the turbinate bones is not mentioned by Stoelting1 or Snow2 as one of the complications of nasotracheal intubation. This case report documents such a complication.

Report of a Case

A healthy, 21-year-old woman underwent third-molar extraction under general anesthesia during which nasotracheal intubation was utilized. Both the procedure and anesthesia were uneventful by history. Two days later, the patient went to her family physician with epistaxis of several hours duration. Anterior nasal packs were inserted, but posterior nasal bleeding persisted. She then was transferred to our hospital.

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She appeared to be a healthy young woman with anterior nasal packs. Vital signs were stable. A slow but persistent trickle of blood could be seen in the oral pharynx. Her hematocrit was 31%. The anterior packs were removed without revealing the bleeding site. Examination of the nasopharynx by mirror suggested the bleeding was predominantly left sided. Also, a midline, mucosally covered mass was observed that appeared to be pedicled superiorly to the basiocciput. A posterior nasal pack was applied, which resulted in control of the bleeding. A computerized axial tomogram taken with the pack in place verified the presence of a soft-tissue nasopharyngeal mass containing some bone (fig. 1). A diagnosis of osteosarcoma or teratoma was considered. We speculated that the nasal intubation traumatized the lesion, causing the epistaxis.

Five days later, a transpalatal exploration of the nasopharynx was performed. Upon entering that space, a mobile, mucosally covered mass was evident. Its lateral attachment to the pharynx was sectioned and the entire lesion removed. The mass was the left middle turbinate, which was verified histologically. The patient had an uneventful recovery. When seen 4 months later she had had no further epistaxis and the nasal passages were moist without exudate or crusting.

Discussion

In retrospect, during the nasotracheal intubation, the tube must have engaged the anterior end of the middle