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The Fifth Vital Sign: Pulse Oximetry in Noninvasive Respiratory Monitoring

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The fifth vital sign, pulse oximetry, routinely is used in every emergency department (ED) throughout the country. It is used to determine the baseline oxygenation of a patient in respiratory distress, to assess a patient's response to therapeutic decisions, and to monitor a child during a conscious sedation or resuscitation. It is important to understand, how the device functions and the limitations of this routinely used technology.

Understanding that pulse oximetry measures functional saturation will help the clinician understand the limitations of this technology in the setting of a carbon monoxide exposure. It is also very important clinically to understand the limits of pulse oximetry in the setting of high venous pressures (congestive heart failure) or anemia.

Certain clinical factors, such as sickle cell anemia, do not affect pulse oximetry, and the results provide meaningful information. As with every diagnostic test that a clinician performs, the information obtained is useful only if it can be interpreted accurately and applied appropriately to the individual patient.

The author reviews a technology that every clinician uses on a daily basis with special attention to appropriate uses, interpretation of results and limitations that may affect accurate interpretation of the data provided.

—The Editor

Introduction

Pulse oximetry is a technology that allows continuous noninvasive monitoring of the arterial oxygen saturation (SaO_2) level,[1] which is calculated from estimates of the oxygenated hemoglobin ($HgbO_2$) and the reduced hemoglobin (Hgb) levels.[2]

The oxygenated Hgb and reduced Hgb measurements are derived from photodetector measurements of two wavelengths of light. The two wavelengths of light (red and infrared) are transmitted from two light-emitting diodes (LEDs) through a pulsatile vascular bed (e.g., the finger) and strike a photodetector. (*See Figure 1.*) The pulsatile components of red (660 nanometers [nm]) and infrared (990 nm) light absorbencies as they pass through tissue may be used to determine the ratio of $HgbO_2$ to reduced Hgb.[3] (*See Figure 2.*) From this ratio of $HgbO_2$

to reduced Hgb, the SaO_2 level can be calculated using calibration curves.[4]

Clinical Uses of Pulse Oximetry

Pulse oximetry has been suggested as the fifth vital sign.[5,6] It is a form of point-of-care testing that allows for continuous noninvasive bedside monitoring of a patient's oxygenation status.[7]

Indications for pulse oximetry are categorized into three groups: 1) a baseline indicator or monitor of a patient's oxygenation status, 2) evaluation of response to therapy, and 3) monitoring during procedures. (*See Table 1*.) Pulse oximetry is indicated in patients with cardiopulmonary disease; unstable or critically ill patients; patients in cardiopulmonary arrest; and patients with or the potential for apnea, hypoxia, respiratory distress/failure, or shock.[1,7-13] Pulse oximetry has been used in the diagnosis of numerous cardiopulmonary diseases and to evaluate the response to treatment in various cardiopulmonary disorders ranging from asthma, chronic obstructive pulmonary disease, bronchiolitis, and reactive airway disease, to pneumonia, airway obstruction, heart failure, and cyanotic congenital heart disease.[7-16]

Examples of the use of pulse oximetry to monitor a patient's clinical status include apnea monitoring, evaluation of periodic breathing (in infants), during transport (in the hospital or prehospital setting), and in critical care areas (e.g., the intensive care units and the ED).[1,7,8,17-20] Pulse oximetry is used during airway procedures (e.g., intubations) and during lumbar punctures and other invasive procedures, (e.g., central lines), especially in infants.[8,21] Pulse oximetry is used for monitoring patients on mechanical ventilation.[22-24] In patients with an endotracheal (ET) tube, pulse oximetry may be a clue to a misplaced or blocked ET tube.

Pulse oximetry has become a standard of care during anesthesia since its recommendation by the American Society of Anesthesiologists in 1986.[3,25] The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) cited a need for pulse oximetry in locations other than in the operating room in 2003.[26] Multiple medical specialty societies, including the American College of Emergency Physicians, the American Academy of Pediatrics, and the American Society of Anesthesiology, have guidelines that suggest pulse oximetry be part of the monitoring of patients undergoing sedation and analgesia.[27-31]

Advantages of Pulse Oximetry

Pulse oximetry's advantages over arterial blood gases measurements are obvious: noninvasive, less pain and risk than arterial puncture, less expensive, more real-time or point-of-care testing method (immediately available vs time for obtaining/transporting/ running/reporting a blood gas), and a continuous monitor (rather than an isolated point[s] in time).[1,7,8,12,14,18,32] (*See Table 2*.) The advantages of pulse oximetry include use as a continuous noninvasive marker or warning signal for adverse patient events that can result in hypoxia/arterial desaturation.[2-4,16] By detecting those events early, treatment can be initiated sooner with the goal of improving patient outcomes.[7,8] Because clinical detection of cyanosis is unreliable, use of pulse oximetry should allow earlier detection of hypoxemia.[20,33] Reports from the recovery room and the operating room do indicate a faster detection of hypoxic episodes, a lower incidence and shorter duration of arterial desaturation, and fewer adverse events in the recovery room in cases where pulse oximetry is used.[34-36] However, several studies of pulse oximetry in anesthesia/post anesthesia care and in general care units failed to show a difference in patient outcome with its use.[36-39] The consensus and expert opinion are that pulse oximetry can and should be used,[26-30] and there are many clinical indications for its use.[1,2,4,6,11-14]

Pulse oximetry is easy to use and requires no special training. It can be applied easily and quickly and is inexpensive. It is

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Figure 1. Pulse Oximetry with Light-Emitting Diodes and Photo Detector

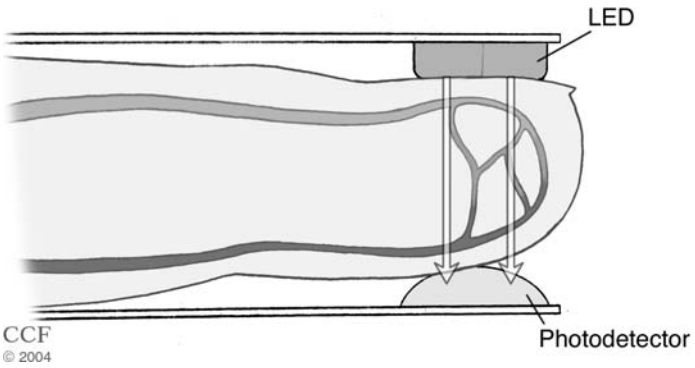


Figure 1. The two wavelengths of light (red and infrared) are transmitted from two light-emitting diodes through a pulsatile vascular bed and strike a photodector.

Illustration courtesy of Cleveland Clinic Foundation, Department of Medical Illustration.

Figure 2. Absorbance Spectra Curves for Various Hemoglobins

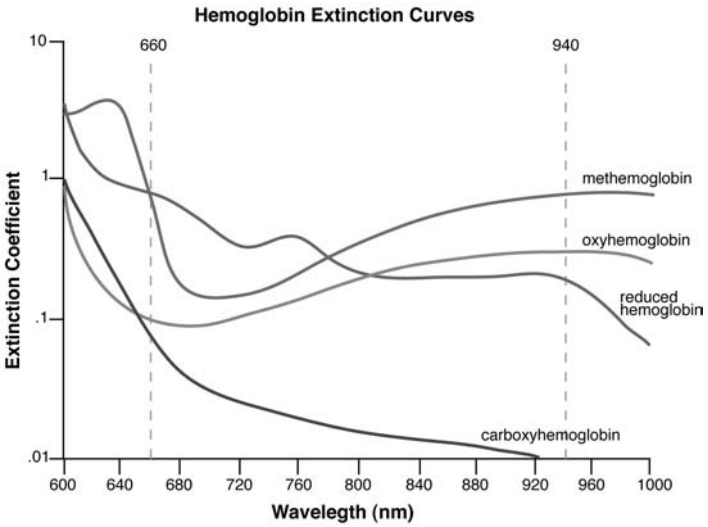


Figure 2. The pulsatile component of red (660 nanometers) and infrated nanometers light absorbencies as they pass through tissue may be used to determine the ratio of $HbgO_2$ to reduced Hgb.

Illustration courtesy of Cleveland Clinic Foundation, Department of Medical Illustration.

small, lightweight, and portable; therefore, it can be used in almost any area and requires little space. It can be stand-alone equipment or incorporated into a bedside monitoring unit. It can be used in any age or type of patient from newborns to geriatric patients, in the ED, intensive care units, inpatient floors, operating suite, recovery room, and in prehospital care.

It has become the fifth vital sign and is a standard of care during procedures requiring general anesthesia and during procedural sedation and analgesia.[5,26-31]

Mechanism of Pulse Oximetry

Pulse oximetry is based upon spectrophotometric principles and on the Beer-Lambert law. All substances have a unique absorbency spectrum. Oxyhemoglobin ($HgbO_2$) and deoxyhemoglobin absorb light differently. (*See Figure 2.*) $HgbO_2$ absorbs more infrared light (wavelength = 940 nm) and less red light (wavelength = 660 nm) than reduced Hgb.[40] The light intensity transmitted (It) or passing through a solution is dependent upon the intensity of the incident light (Io) minus the light absorbed. The amount of light absorbed depends upon the concentration of solute (C), the distance or path length through the solution (D), and the molar extinction coefficient of the solute (k) (a constant for Hgb at a given wavelength), or absorbance = $k \cdot C \cdot D = \log Io/It$. (*See Figure 3.*) Blood is the solvent and hemoglobin, the solute.

Because the amount of tissue is constant, the amount of light transmitted (and the amount of light absorbed) varies with the arterial blood flow. Arterial blood creates a pulsatile flow—and a pulsatile signal—with each heartbeat. The venous blood and nonpulsatile arterial blood flow are fairly constant throughout the cardiac cycle, and the tissue is constant; therefore, the only variation during the cardiac cycle (e.g., one heartbeat) is the arterial pulsatile flow. [22,41,42] This pulsatile arterial blood flow increases the distance or path length through which the red and infrared wavelengths travel. (*See Figure 4.*) According to the Beer-Lambert Law, this results in increased absorbance of light (of both the red and infrared wavelengths) with less light transmitted and striking the photodetector. The pulse oximeter calculates the ratio of the pulsatile and nonpulsatile absorbencies at the 660 nm (red) and 940 nm (infrared) wavelengths. (*See Figure 2.*) From the absorbencies, the pulse oximeter's microprocessor calculates the ratio of the $HgbO_2$ and the reduced Hgb levels, and then, plots this ratio against a standardized calibration curve to derive the SaO_2 measurement.

Pulse Oximetry Equipment

All pulse oximeters contain a probe that consists of two light-emitting diodes (LEDs), a photodetector, and an onboard microprocessor/computer.[41] (*See Figure 1.*) The SaO_2 measurement is derived from the ratio using the calibration curves in the computer software.[22] The standardized calibration curve is derived from healthy adult volunteers. Across from the LEDs is a photodetector that is placed across a pulsatile vascular bed, (e.g. a finger, toe, earlobe, nose, or forehead).[2] (*See Figure 1.*) If the forehead is used, then reflectance (not transmittance) is used. (*See Figure 5.*) With reflectance oximetry, the light from the LEDs is reflected from tissue, and the photodetector is located on the same side as the LED.[2] Reflectance oximetry is technically more difficult with less accuracy; therefore, it is not used widely.

Pulse oximetry probes usually are configured into a disposable patch or, more commonly, a reusable clip.[7] In adults, the fin-

Table 1. Clinical Uses of Pulse Oximetry

MONITORING DURING:

- Anesthesia
- Procedural sedation and analgesia (e.g., reduction of fractures)
- Lumbar puncture, other invasive procedures, especially in infants
- Transport (e.g., prehospital, within hospital)

EVALUATION OF CLINICAL STATUS

- Apnea monitoring (in infants)
- Periodic breathing
- Obstructive sleep apnea

MONITORING OF THE AIRWAY:

- During mechanical ventilation
- During weaning from oxygen therapy
- During noninvasive positive pressure ventilation
- For preoxygenation prior to and during airway procedures (e.g., intubation)
- For evaluation of the need for increased FiO_2 and/or a definitive airway
- To detect problems with a definitive airway (e.g., misplaced/obstructed endotracheal tube)

MONITORING THE OXYGENATION IN:

- Unstable or critically ill patients (e.g., hypotension, shock, respiratory disease/failure, cardiopulmonary arrest)
- Patients with the potential for hypoxia, apnea, respiratory distress/failure, shock
- Patients with pulmonary disease (e.g., asthma, COPD, bronchiolitis, reactive airway disease, pneumonia, or airway obstruction)
- Patients with cardiac disease (e.g., heart failure, cyanotic congenital heart disease, acute coronary syndrome, or cardiomyopathy)

Table 2. Advantages of Pulse Oximetry

- Fifth vital sign
- Requires no special training to use
- Noninvasive
- Inexpensive
- Continuous real-time measurements (point-of-care testing)
- Avoids pain/risks of arterial puncture
- Standard of care during procedures requiring general anesthesia
- Standard of care during procedural sedation and analgesia
- Small, requires little space, portable
- Can be used almost anywhere
- Can be stand-alone equipment or incorporated into a multipurpose monitor
- Easy to apply and use

ger is the most common probe site, whereas in infants and small children, the great toe is used frequently. Adhesive sensors, which often are used in infants and small children, can be placed over the heel or lateral aspect of the foot and secured with a gauze or Coban wrap.[9] To avoid interference with high intensity ambient light, a light barrier (e.g. a dark cloth) can be placed around the probe. However, the author of a recent article found that ambient light had no statistically significant effect on pulse oximetry readings, and any difference due to ambient light was small and clinically unimportant.[43] If motion artifact is a problem, then using a different site may help. Also, correlating an electrocardiogram (ECG) monitor waveform with a pulse oximeter signal may help determine whether a reading is a valid pulse oximetry record or a motion artifact.

Pulse oximetry is noninvasive and considered safe, although rare reports of pressure necrosis and burns due to defective probes have been reported.[44-47] Pulse oximetry probes can be a source of nosocomial infection if they become contaminated with pathogenic bacteria. However, there are many methods to avoid this possibility: Disposable probes can be resterilized, and protective sheaths can be placed on the probe to allow for multiple uses.

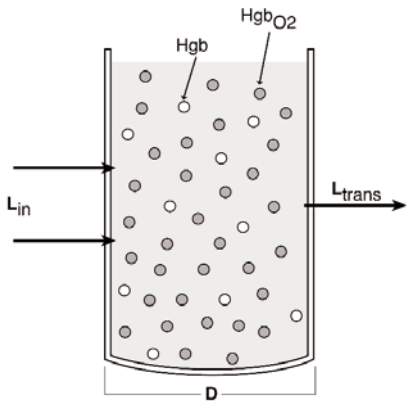
Pulse oximetry has a low failure rate. In one operating room study, the failure rate was less than 5% with a trend toward failure occurring in elderly and sicker patients and during longer surgical procedures.[48] Accidental disconnection or probe misplacement are probably the most common causes of pulse oximetry failure.

Pathophysiology

Oxygen Saturation. Oxyhemoglobin ($HgbO_2$) and deoxyhemoglobin absorb light differently, forming the basis for pulse oximetry oxygen saturation measurements. (*See Figure 2*.) Erythrocytes containing hemoglobin (Hgb) pick up oxygen in the pulmonary capillary beds and become oxyhemoglobin ($HgbO_2$). They travel back to the left side of the heart, then, are transported throughout the body to the systemic capillary beds. Oxygen is released down a gradient to the tissues when the tissue partial pressure of oxygen is lower than that of the arterial blood. After releasing oxygen to the tissues, some of the Hgb—which is now deoxygenated Hgb—may pick up some of the carbon dioxide (CO_2) formed as a byproduct of cellular aerobic metabolism to form carbaminohemoglobin. Approximately 5%-10% of CO_2 is transported as carbaminohemoglobin; the majority of CO_2 (90%-95%) is transported as bicarbonate. CO_2 is transported to the lungs where it is released into the alveoli and eliminated in the process of ventilation.[49]

The SaO_2 measurement is the percentage of Hgb bound to oxygen. In normal systemic arterial blood, the SaO_2 level is about 97% ($\geq$ 95%). Thus, the amount or concentration of Hgb bound to oxygen ($HgbO_2$) is 97% of capacity. The hemoglobin saturation (SaO_2) (in percent) equals the amount of oxygenated Hgb divided by the total amount of Hgb available for oxygena-

Figure 3. Schematic Diagram of Absorption Signal Passing through a Solution (e.g. blood)



$$\text{Absorbance} = \log\left(\frac{L_{in}}{L_{trans}}\right) = k \cdot C \cdot D$$

L_{in}	–	incident light
L_{trans}	–	light transmitted
D	–	distance light is transmitted
C	–	concentration of Hgb_{O2}
k	–	constant

Figure 3. The amount of light absorbed depends upon the concentration of solute, the distance or path through the solution, and the molar extinction coefficient of the solute.

Illlustration courtesy of Cleveland Clinic Foundation, Department of Medical Ilustration.

Table 3. Oxyhemoglobin Disassociation Curve

SHIFT TO LEFT	SHIFT TO RIGHT
• Increased affinity of oxygen to Hgb	• Decreased affinity for oxygen to Hgb
• Decreased availability of oxygen to tissues	• Increased availability of oxygen to tissues
• Factors causing shift to left - increased pH level (e.g., alkalosis) - decreased pCO_2 level (e.g., hypocarbia, respiratory alkalosis) - decreased 2,3 DPG level	• Factors causing shift to right - decreased pH level (e.g., acidosis) - increased pCO_2 level (e.g., hypercarbia, respiratory acidosis) - increased temperature (fever) - increased 2,3 DPG level - chronic hypoxia - chronic alkalosis - high altitude adaptation - hyperthyroidism - anemia

tion (the oxygenated Hgb plus the reduced Hgb) times 100.[8]

$$SaO_2\,(\%) = \frac{\text{Oxygenated Hgb}}{\text{Total Hgb available}} \times 100 = \frac{[\text{Oxygenated Hgb}]}{[\text{Oxygenated Hgb} + \text{reduced Hgb}]} \times 100$$

Generally, hypoxemia is defined as an SaO_2 level less than 95%, with severe hypoxia being an SaO_2 level less than 90% and a pO_2 level less than 70 mmHg. The oxygen concentration of the blood (CaO_2) is equal to the amount of oxygen bound to Hgb ($HgbO_2$) plus the oxygen dissolved in blood. Hgb can bind chemically 1.34 mL O_2/g. Given a normal Hgb of 15 (150 g/L), the O_2 capacity of Hgb is 200 mL O_2/L (1.34 mL O_2/g x 150 g/L Hgb). Because oxygen has a low solubility, the amount of dissolved oxygen under normal conditions is relatively small, less than 2%-3% of the oxygen present. At a pO_2 level of 100 mmHg, one liter of blood holds only 3 mL oxygen in solution. When breathing 100% FiO_2, the amount of dissolved oxygen is 18-20 mL. The CaO_2 level for systemic arterial blood under normal conditions is: CaO_2 = Percent SaO_2 x O_2 capacity of Hgb + dissolved O_2.

CaO_2 = [.97 x 200 + 0.03 x 100] mL O_2/L = 194 + 3 = 197 mL O_2/L.[49]

Oxyhemoglobin Disassociation Curve. The oxygen molecule combines loosely and reversibly with the heme portion of the Hgb molecule. When the PaO_2 level is high, oxygen combines with Hgb. Conversely, when the PaO_2 level is low, oxygen is released from Hgb, as occurs in the tissue capillaries. The oxyhemoglobin disassociation curve plots the $HgbO_2$ saturation level or SaO_2, which is the percentage of Hgb bound to oxygen, against the PaO_2 level.[50] The SaO_2 level is plotted on the Y-axis vs the partial pressure of oxygen (PaO_2) on the X-axis. (*See Figure 6.*) From the graph, the sigmoidal shape of the oxyhemoglobin ($HgbO_2$) disassociation curve demonstrates the nonlinear relationship between SaO_2 and PaO_2. In the upper flat part of the oxyhemoglobin disassociation curve, minor changes in the SaO_2 level result in larger changes in the PaO_2 level. In the steep middle part of the curve, large changes in the SaO_2 level result in smaller changes in PaO_2 levels. As the PaO_2 level increases, there is a progressive increase in the percentage of Hgb bound with oxygen.

Various factors can cause a change in the oxyhemoglobin dissociation curve by shifting the curve to the right or the left. (*See Table 3.*) When the curve is shifted to the right, there is a decrease in the affinity of oxygen for Hgb; therefore, more oxygen is released to the tissues. Variables that shift the curve to the right include metabolic acidosis (decreased pH), increased temperature (fever), increased pCO_2 (respiratory acidosis), and increased 2,3-DPG levels. The 2,3-DPG level is increased with chronic hypoxemia, chronic alkalosis, anemia, high altitude adaptation, and hyperthyroidism. A shift of the oxyhemoglobin disassociation curve to the left results in an increased affinity of oxygen to Hgb at a lower PaO_2 level, with less oxygen released to the tissues. Factors resulting in a shift to the left are alkalosis

Figure 4. Absorption Signal as it Passes through Pulsatile Tissue (e.g., arterial blood)

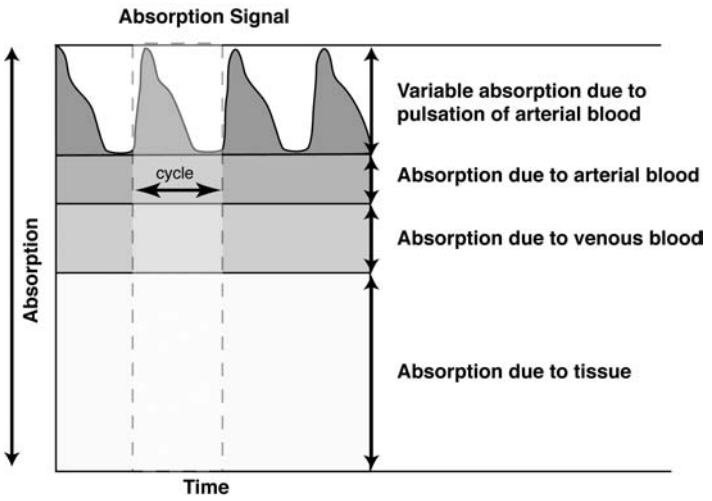


Figure 4. The venous blood and nonpulsatile arterial blood flow are fairly constant, and the tissue is constant. Arterial blood creates a pulsatile flow and increases the distance or path length through which the red and infrared wavelengths travel.

Adapted from McGough EK, Boysen PG. Benefits and limitatons of pulse oximetry in the ICU. J Crit Ill *1989;2:23-31.*

(increased pH), hypocarbia (decreased pCO_2), hypothermia (decreased temperature), and decreased 2,3-DPG level.[51] (*See Table 3 and Figure 6.)*

The clinical importance of the sigmoidal shape of the oxyhemoglobin disassociation curve is two-fold. First, in hypoxic patients, because their SaO_2 levels are on the steep part of the curve, large changes in SaO_2 levels correspond to small changes in PaO_2 levels. Secondly, in the high range of oxygenation (i.e., the plateau part of the curve), SaO_2 measurements are somewhat insensitive in detecting significant PaO_2 changes. (*See Figure 6.*)

Limitations of Pulse Oximetry

Limitations of pulse oximetry can be categorized into four categories: 1) those based on oxygen saturation level and the oxyhemoglobin disassociation curve, 2) those based on pulse oximetry's design, 3) technical aspects, and 4) patient factors. (*See Table 4.*)

Oxygen Saturation. Pulse oximetry measures oxygen saturation; therefore, unlike an arterial blood gas or end-tidal CO_2 monitoring, it yields no data on pH or $PaCO_2$ levels. Because of the characteristics of the oxyhemoglobin disassociation curve, the SaO_2 level is relatively insensitive to PaO_2 level changes in the upper flat part of the curve. In the middle steep part of the curve, small changes in PaO_2 levels result in large changes in SaO_2 levels. The SaO_2 level does not necessarily indicate the adequacy of ventilation. Hypercapnia due to hypoventilation may occur before a drop in the SaO_2 level is detected (one advantage of end-tidal CO_2 monitoring). However, pulse oxime-

Table 4. Limitations of Pulse Oximetry

PROBLEMS INHERENT WITH SAO_2 MEASUREMENTS

- Oxyhemoglobin dissociation curve
  - In middle steep part of curve, small changes in PaO_2 levels cause large changes in SaO_2 levels.
  - In upper flat part of curve, the SaO_2 level is relatively insensitive to PaO_2 changes.
- Does not measure ventilation
  - Does not measure pH, pCO_2 levels
  - Patients may have normal SaO_2 level until late deterioration.

DESIGN OF PULSE OXIMETRY:

- Uses spectrophotometry with two wavelengths to estimate SaO_2 level by comparison with levels in normal healthy adult volunteers
- Uses only 2 wavelengths, so measures *functional* saturation not *fractional* saturation (increase in presence of methemo globin or carboxyhemoglobin levels, increase in falsely high SaO_2 level)
- Accuracy affected by various dyes/pigments (e.g., methylene blue [absorbs light at 660 nm], fluorescein, indocyanine green, indigo carmine)
- Calibration curves obtained from data from small number of healthy adults
- Accuracy varies depending upon SaO_2 level; more accurate: SaO_2 measurement in 80%-100% range

INDIVIDUAL PATIENT VARIABLES

- Low perfusion states (e.g., shock, low cardiac output, decreased peripheral perfusion, increased/inadequate arterial blood pressure, increased/inadequate arterial pulsatile flow) may have inadequate signal
- Works best in middle ranges of heart rate, systolic blood pressure, and hematocrit level
- Decreased accuracy if low hemoglobin/hematocrit level (severe anemia) especially if low SaO_2 measurement
- Darkly pigmented skin
- High venous pressure (e.g., heart failure or superior vena cava syndrome) increases falsely low SaO_2 measurement

TECHNICAL VARIABLES

- Excessive motion causes motion artifact
- High ambient light
- Dark nail polish (if finger probe is used)
- Electrocautery
- Misplaced probe (penumbra effect)

FACTORS NOT AFFECTING PULSE OXIMETRY

- Fetal hemoglobin
- Sickle hemoglobin
- Hyperbilirubinemia
- Dialysis grafts (A-V fistulas)
- Polycythemia

Key: SaO_2 = oxygen saturation. PaO_2 = partial pressure of arterial oxygen. pCO_2 = partial pressure of carbon dioxide.

Figure 5. Reflectance Oximetry

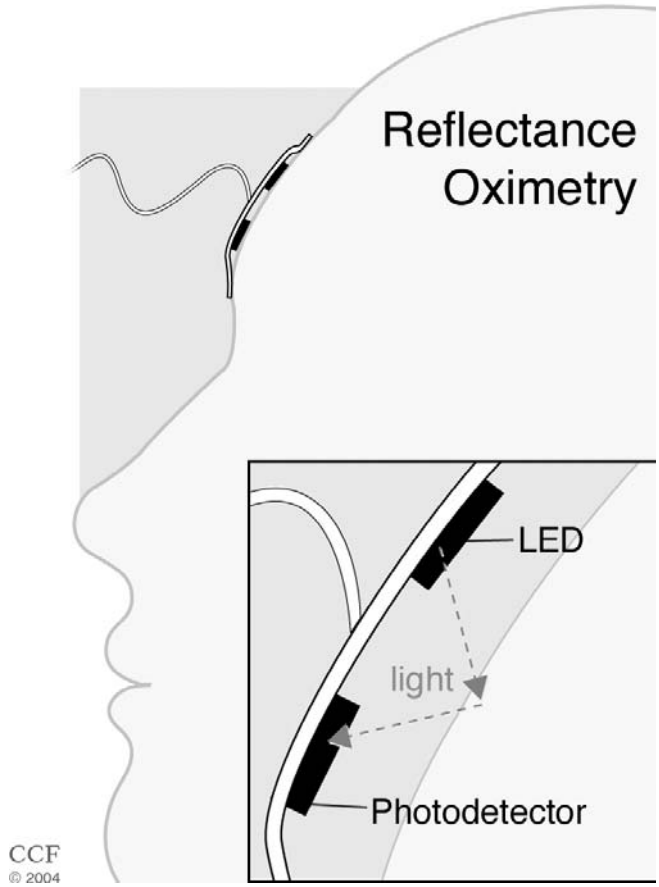


Figure 5. Reflectance oximetry is technically more difficult and has less accuracy than pulse oximetry, and therefore, is not used commonly.

Illustration courtesy of Cleveland Clinic Foundation, Department of Medical Ilustration.

Table 5. Factors Affecting Pulse Oximetry Readings

FALSELY HIGH SPO_2

- Presence of abnormal hemoglobin
  - Carboxyhemoglobin (COHgb)
  - COHgb light absorption is like $HgbO_2$ absorption in red spectra, so oximeter reads high SpO_2 level.
  - Methemoglobin absorbs light in both red and infrared wavelengths, so oximeter reads high SpO_2 level.
- Anemia (if Hgb <10)

FALSELY LOW SPO_2

- High venous pressure
  - Congestive heart failure
  - Superior vena cava syndrome
  - Traumatic venous obstruction
  - Application of tourniquet (proximal to pulse oximeter - e.g., in same arm)
  - Inflation of manometer cuff (proximal to pulse oximeter - e.g., in same arm)
- Diagnostic dyes
- Nail polish
- Skin color (deeply pigmented)

TECHNICAL FACTORS (POOR OR NO READING)

- Problems with sensing
  - Motion near sensor site mimics arterial pulsation
- Low flow states
  - Low blood flow leads to insufficient change in light emittance, resulting in no reading
- Ambient ligh
  - Increases light to photodetector
- Penumbra effect

try is an improvement over the clinical assessment of cyanosis because cyanosis is difficult to detect clinically[33,51] and is an unreliable clinical sign.[2]

Design Aspects of Pulse Oximetry. Some limitations of pulse oximetry are attributable to the design of pulse oximeters and its measurement. Pulse oximeters only measure two wavelengths, and thus, *functional* saturation, not *fractional* saturation.[7] Pulse oximeters measure the functional saturation, which equals the $HgbO_2$ level divided by the reduced Hgb level plus the $HgbO_2$ level times 100 to give a percentage.

$$\textit{Functional}\ SaO_2\ (\%) = \frac{HgbO_2}{HgbO_2 + \text{Reduced Hgb}} \times 100$$

$$\textit{Fractional}\ SaO_2\ (\%) = \frac{HgbO_2}{HgbO_2 + \text{Reduced Hgb} + \text{other Hgbs}} \times 100$$

Fractional saturation is the $HgbO_2$ level divided by the [$HgbO_2$ + reduced Hgb + other hemoglobins].[22] If other dyshemoglobinemias, (e.g., methemoglobin or carboxyhemoglobin) are present, then pulse oximetry (measurement of the functional SaO_2 level) gives a falsely high SaO_2 measurement.[52,53] Co-oximetry should be done if abnormal hemoglobins (e.g., methemoglobin or carboxyhemoglobin) are present because co-oximeters calculate the fractional SaO_2 level. However, because it uses only two wavelengths and two hemoglobins, pulse oximetry is less expensive, and the equipment is smaller and lighter and more portable than co-oximeters. Pulse oximeters do remain accurate if fetal hemoglobin is present;[54,55] co-oximeters inaccurately report elevated carboxyhemoglobin levels in the presence of fetal hemoglobin. Both fetal hemoglobin and as much as 5% carboxyhemoglobin may be present in the blood of newborns.

Pulse oximetry measurements are affected by various dyes/pigments.[56,57] For example, methylene blue—which is recommended therapy for methemoglobinemia—absorbs light at 660 nm, similar to the absorption rate of reduced hemoglobin. (*See Figure 2.)* The presence of methylene blue will cause falsely low pulse oximetry saturations.[56] Other intravenous dyes (e.g., fluorescein, indocyanine green, and indigo carmine) used for therapeutic or diagnostic purposes also will produce spuriously low pulse oximetry readings.[57] (*See Table 5.*)

Pulse oximeters have an onboard computer that uses empiric calibration curves derived from data from a small number of

Figure 6. Oxyhemoglobin Disassociation Curve

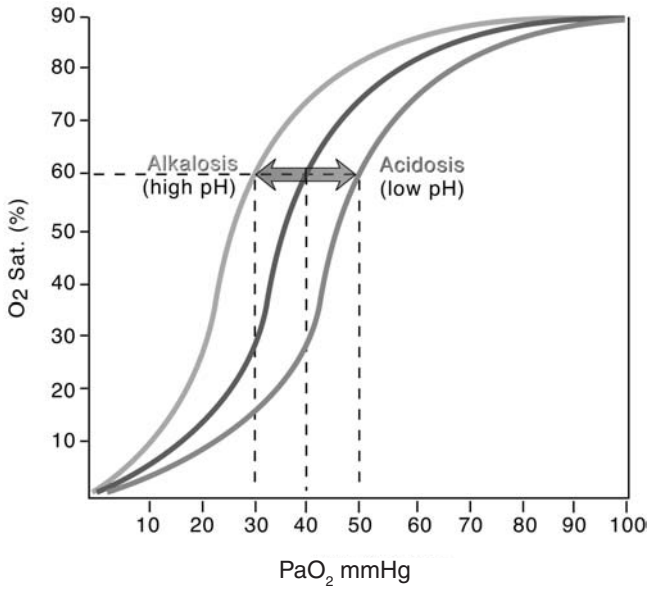


Figure 6. The sigmoidal shape of the oxyhemoglobin dissociation curve demonstrates the nonlinear relationship between SaO_2 and PaO_2 levels. Various factors, as shown in the graph, may shift the curve to the left or the right. When the curve shifts to the right, the Hgb has a decreaed affinity for oxygen, and therefore, more oxygen is released to the tissues. The reverse is true when the curve shifts to the left.

Illustration courtesy of Cleveland Clinic Foundation, Department of Medical Ilustration.

Table 6. Pearls and Pitfalls with Pulse Oximetry

- Measures oxygen saturation levels
- Does not measure ventilation
- Patients may have normal SaO_2 measurement until a late deterioration (e.g., upper airway obstruction).
- Trend is more important than an isolated reading.
- Patient readings are compared with reference data from healthy adult volunteers.
- Reference data may be affected by individual patient (volunteer) factors (e.g., skin thickness, skin color, Hgb concentration).
- Accuracy varies depending upon SaO_2 levels.
- More accurate at certain SaO_2 levels, less accurate at lower SaO_2 levels
- Response time—earlobe probes faster than finger probes.
- Sigmoidal shape of oxyhemoblobin disassociation curve

healthy adult volunteers.[22,55] The calibration curves programmed into the pulse oximeter's software differ among the various manufacturers, and may vary among pulse oximeters made by the same manufacturer. Whether the use of such calibration curves derived from healthy adults can be generalized for all ages (e.g., neonates, geriatric patients), all types of patients (e.g., male, female, ethnic groups), and all conditions/disease severity levels has not been evaluated extensively.

Again, the trend of pulse oximetry readings in a given patient and the presence or absence of hypoxia are more important than an isolated measurement of oxygen saturation.

Technical Aspects of Pulse Oximetry. Technical problems with pulse oximetry have been divided into two categories: 1) insufficient signal, as occurs with hypoperfusion or poor probe placement, and 2) too much signal with artifact/noise (excessive motion or ambient light).

Improper probe placement can cause a penumbra effect, whereby light from the LED is aimed directly at the photodetector instead of passing through the tissue (e.g., patient's finger), causing a mistakenly high pulse oximetry oxygen saturation measurement when the arterial blood oxygen saturation level is less than 85%, and a falsely low pulse oximetry oxygen saturation measurement when the arterial blood oxygen saturation level is greater than 85%.[2]

Excessive motion of the probe causes much artifact and unreadable or unreliable/inaccurate readings. Newer pulse oximeter models have programmed software that can measure and remove the noise components; newer pulse oximetry probes also can eliminate most of the motion artifact.[58]

The pulse oximeter's photodetector is nonspecific; therefore, high-intensity ambient light can result in interference, although a recent study documented no clinically or statistically significant variations in pulse oximetry from ambient light.[43] This problem is avoided by covering the probe with opaque material.

Interference from electrocautery can be decreased by placing the sensor or probe farther from the surgical site (e.g., increasing the distance from the operative field).[7]

The wavelengths of light transmitted by the pulse oximeters (660 nm and 940 nm) LEDs may vary by ± 30 nm, which may affect accuracy.[2]

Patient Variables Affecting Pulse Oximetry. Pulse oximeters correlate best with arterial blood gas saturation measurements when the oxygen saturation level is greater than 80% (with an accuracy of approximately ± 4%-5%).[64,65] Similarly, pulse oximeters work best in the middle ranges of heart rates (40 to 180 beats per minute), blood pressure measurements (systolic pressure in the 80-106 range), and hematocrit levels (20%–56%). Pulse oximetry has decreased accuracy when severe anemia is present (e.g., Hct < 10%).[64,65] The accuracy and precision of pulse oximeters is acceptable; in clinical practice, the presence or absence of hypoxia (and the trend) is more important than to define a specific level.

Pulse oximeters calculate the SaO_2 level by computing the ratio of $HgbO_2$ from the ratio of pulsatile to baseline tissue absorption, thus, the requirement for a pulsating vascular bed. When shock, severe hypotension, or vasoconstriction occurs (e.g., use of vasoconstricting drugs, such as epinephrine), the signal from the diminished arterial pulse can not be differentiat-

ed from background noise.[66,67] Most pulse oximeters will display a message that there is an inadequate pulse signal. Changing the sensor to another location with a higher perfusion, such as from an extremity (finger or toe) to the earlobe, may allow for a better pulse signal. With cold extremities, vasoconstriction, or low perfusion, warming the extremities may generate a measurable signal.

When venous pressure is high, falsely low pulse oxygen saturation measurements may result; the pulse oximeter construes any pulsatile measurement as arterial, including the increased venous pulsations.[7, 68] Situations in which there may be a high venous pressure include congestive heart failure, superior vena cava syndrome, traumatic venous obstruction (e.g., tension pneumothorax or pericardial tamponade), and transient iatrogenic causes (e.g., application of a tourniquet or inflation of a manometer cuff). *(See Table 5.)*

Deeply pigmented skin may decrease pulse oximetry's accuracy, although the effect is minor.[69,70] Using parts of the body with lighter pigmentation (e.g., the fifth finger or the earlobe) may minimize this variable. Fingernail polish has been cited as a variable affecting pulse oximetry readings when a finger or toe probe is used. A nursing recommendation is to remove fingernail polish or artificial nails if a finger or toe probe is used for pulse oximetry.[71] However, a recent study documented a small decrease (2%) in pulse oximetry readings occurring only with brown or black fingernail polish,[72] which could be eliminated by placing the finger probe side to side instead of top to bottom.[72,73] However, there are many variables that do not affect pulse oximetry readings (e.g., fetal hemoglobin,[55,74] sickle hemoglobin,[8,75] hyperbilirubinemia,[69,76] and dialysis grafts [A-V fistulas]).[7]

Summary

Pulse oximetry has wide usage throughout the hospital—including the ED and in prehospital care— because of its many advantages. It is a user friendly, inexpensive, safe, noninvasive point-of-care method for continuous monitoring of a patient's oxygenation status. It can serve as an early warning signal of impaired oxygenation in a given patient, while avoiding the pain and risks of arterial puncture. The clinician should be aware of the pathophysiology/background of pulse oximetry to use this technology appropriately. (*See Table 6.*)

In addition, the clinician should keep in mind scenarios where pulse oximetry readings may give either falsely high readings (e.g., carboxyhemoglobin or anemia) or falsely low readings (e.g., high venous pressures or diagnostic dyes) and use this information appropriately to manage the patient. Clinicians also should be aware of technical aspects of pulse oximetry, such as excessive probe motion and improper probe placement, that may result in inaccurate testing results. Continued improvement and advances in technology have resulted in valuable tools, such as pulse oximetry, that greatly enhance patient management in the acute care setting. Further advances will continue to contribute noninvasive, real-time information that facilitates the clinician's ability to diagnose and manage patients in the ED.

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Physician CME Questions

21. Which of the following characteristics apply to pulse oximetry?
    A. Noninvasive
    B. Allows continuous monitoring
    C. Point-of-care testing
    D. Requires little or no training to use
    E. All of the above

22. Which of the following statements is *incorrect* regarding pulse oximetry?
    A. It allows a quicker detection of hypoxic episodes.
    B. An improved patient outcome has been demonstrated with the use of pulse oximetry.
    C. It avoids the pain and risks of arterial puncture for blood gases.
    D. It is less expensive than an arterial blood gas measurement.
    E. It is considered the fifth vital sign.

23. Which of the following characteristics applies to pulse oximetry?
    A. Bulky, cumbersome, not portable
    B. Can be used in all ages and all types of patients
    C. Difficult to apply
    D. Heavy, awkward
    E. Not needed routinely

24. All of the following statements are correct regarding the mechanisms of pulse oximetry, *except*?
    A. Pulse oximetry is based upon the absorption of oxyhemoglobin and deoxyhemoglobin.
    B. It depends upon the ratio of pulsatile and nonpulsatile absorbances at the red and infrared wavelengths.

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge.

To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion. When your evaluation is received, a certificate will be mailed to you.

CME Objectives

The CME objectives for *Pediatric Emergency Medicine Reports* are to help physicians:

a.) Quickly recognize or increase index of suspicion for specific conditions;

b.) Understand the epidemiology, etiology, pathophysiology, historical and physical examination findings associated with the entity discussed;

c.) Be educated about how to correctly formulate a differential diagnosis and perform necessary diagnostic tests;

d.) Apply state-of-the-art therapeutic techniques (including the implications of pharmacologic therapy discussed) to patients with the particular medical problems discussed;

e.) Provide patients with any necessary discharge instructions.

C. The ratio of oxyhemoglobin and reduced hemoglobin is calculated from the absorbances.
D. The SaO_2 measurement is derived by plotting the ratio of the oxyhemoglobin to the reduced hemoglobin against a standardized curve.
E. The absorption of light is not affected by the distance through which the light has to travel or by the concentration of the solute through which the light passes.

25. Which of the following statements does *not* apply to the oxyhemoglobin disassociation curve?
A. Acidosis shifts the curve to the left.
B. A shift of the curve to the right results in a decreased affinity of oxygen for hemoglobin.
C. A shift of the curve to the right results in more oxygen being released to the tissues.
D. An increased 2,3 DPG level causes a shift of the curve to the right.
E. Alkalosis causes a shift of the curve to the left.

26. Which of the following statements is *incorrect* regarding the oxyhemoglobin disassociation curve?
A. In the upper high range of oxygenation (plateau part of the curve), the SaO_2 level is relatively insensitive in detecting significant pO_2 level changes.
B. In the upper flat part of the curve, minor changes in SaO_2 levels cause larger changes in pO_2 levels.
C. In the steep middle part of the curve, large changes in SaO_2 levels result in even larger changes in pO_2 levels.
D. As the pO_2 level increases, there is a progressive increase in the percentage of Hgb bound to oxygen.
E. The curve plots the SaO_2 level on the Y-axis against the pO_2 level on the X-axis.

27. Which of the following statements is true regarding the design of pulse oximeters?
A. It measures the fractional SaO_2 level.
B. It measures the oxyhemoglobin, reduced hemoglobin, reduced hemoglobin and all other hemoglobins levels.
C. It measures carboxyhemoglobin levels.
D. It measures the functional SaO_2 levels.
E. It measures methemoglobin levels.

28. Which of the following factors does *not* affect the pulse oximetry measurement?
A. Various dyes/pigments (e.g., methylene blue)
B. The calibration curve used by the manufacturer
C. The presence of fetal hemoglobin
D. The presence of carboxyhemoglobin
E. The presence of methemoglobin

29. Which of the following factors may affect the ability to obtain an accurate readable pulse oximetry measurement?
A. Improper probe placement causing a penumbra effect
B. Excessive motion of the probe
C. Interference from electrocautery
D. Interference from high intensity ambient light affecting the pulse oximeter's photodetector
E. All of the above

30. Which of the following statements is *incorrect* regarding patient variables that can affect pulse oximetry measurements?
A. Measurements may be affected by dark fingernail polish (if the finger probe is used) or by deeply pigmented skin.
B. Pulse oximetry works best in the lower range of heart rate, blood pressure, and hematocrit levels.
C. The pulse oximeter may have difficulty picking up a signal when vasoconstriction, shock, or severe hypotension is present.
D. The pulse oximeter calculates the ratio of pulsatile to baseline tissue absorption and therefore, requires a pulsatile vascular bed.
E. When the venous pressure is high, a falsely low pulse oxygen saturation measurement may occur.

Answer key:
21. E
22. B
23. B
24. E
25. A
26. C
27. D
28. C
29. E
30. B

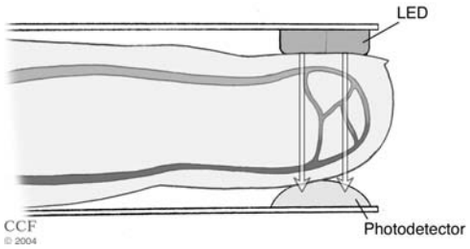
In Future Issues:

Hydrocephalus, ventriculoperitoneal (VP) shunts, and VP shunt complications

PEDIATRIC Emergency Medicine Reports
The Practical Journal of Pediatric Emergency Medicine

Pulse oximetry

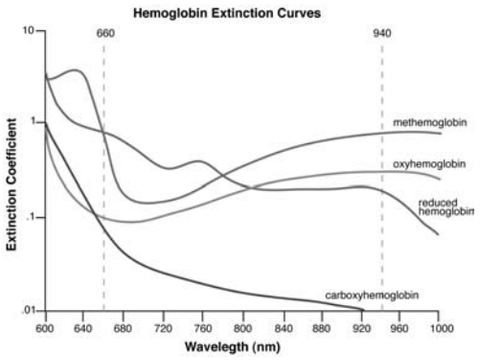
Pulse Oximetry with Light-Emitting Diodes and Photo Detector



The two wavelengths of light (red and infrared) are transmitted from two light-emitting diodes through a pulsatile vascular bed and strike a photodector.

Illustration courtesy of Cleveland Clinic Foundation, Department of Medical Ilustration.

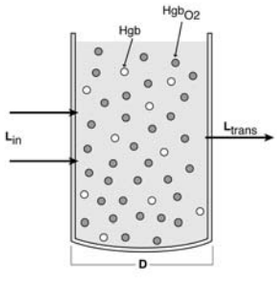
Absorbance Spectra Curves for Various Hemoglobins



The pulsatile component of red (660 nanometers) and infrated nanometers light absorbencies as they pass through tissue may be used to determine the ratio of $HbgO_2$ to reduced Hgb.

Illustration courtesy of Cleveland Clinic Foundation, Department of Medical Ilustration.

Schematic Diagram of Absorption Signal Passing through a Solution (e.g. blood)



$$\text{Absorbance} = \log\left(\frac{L_{in}}{L_{trans}}\right) = k \cdot C \cdot D$$

L_{in}	–	incident light
L_{trans}	–	light transmitted
D	–	distance light is transmitted
C	–	concentration of Hgb_{O2}
k	–	constant

The amount of light absorbed depends upon the concentration of solute, the distance or path through the solution, and the molar extinction coefficient of the solute.

Illustration courtesy of Cleveland Clinic Foundation, Department of Medical Ilustration.

Pearls and Pitfalls with Pulse Oximetry

- Measures oxygen saturation levels
- Does not measure ventilation
- Patients may have normal SaO_2 measurement until a late deterioration (e.g., upper airway obstruction).
- Trend is more important than an isolated reading.
- Patient readings are compared with reference data from healthy adult volunteers.
- Reference data may be affected by individual patient (volunteer) factors (e.g., skin thickness, skin color, Hgb concentration).
- Accuracy varies depending upon SaO_2 levels.
- More accurate at certain SaO_2 levels, less accurate at lower SaO_2 levels
- Response time—earlobe probes faster than finger probes.
- Sigmoidal shape of oxyhemoblobin disassociation curve

Clinical Uses of Pulse Oximetry

MONITORING DURING:

- Anesthesia
- Procedural sedation and analgesia (e.g., reduction of fractures)
- Lumbar puncture, other invasive procedures, especially in infants
- Transport (e.g., prehospital, within hospital)

EVALUATION OF CLINICAL STATUS

- Apnea monitoring (in infants)
- Periodic breathing
- Obstructive sleep apnea

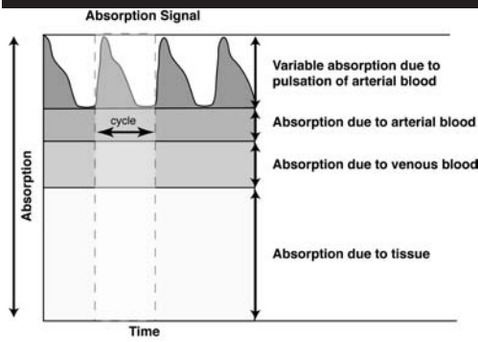
MONITORING OF THE AIRWAY:

- During mechanical ventilation
- During weaning from oxygen therapy
- During noninvasive positive pressure ventilation
- For preoxygenation prior to and during airway procedures (e.g., intubation)
- For evaluation of the need for increased FiO_2 and/or a definitive airway
- To detect problems with a definitive airway (e.g., misplaced/obstructed endotracheal tube)

MONITORING THE OXYGENATION IN:

- Unstable or critically ill patients (e.g., hypotension, shock, respiratory disease/failure, cardiopulmonary arrest)
- Patients with the potential for hypoxia, apnea, respiratory distress/failure, shock
- Patients with pulmonary disease (e.g., asthma, COPD, bronchiolitis, reactive airway disease, pneumonia, or airway obstruction)
- Patients with cardiac disease (e.g., heart failure, cyanotic congenital heart disease, acute coronary syndrome, or cardiomyopathy)

Absorption Signal as it Passes through Pulsatile Tissue (e.g., arterial blood)



The venous blood and nonpulsatile arterial blood flow are fairly constant, and the tissue is constant. Arterial blood creates a pulsatile flow and increases the distance or path length through which the red and infrared wavelengths travel.

Adapted from McGough EK, Boysen PG. Benefits and limitatons of pulse oximetry in the ICU. J Crit Ill *1989;2:23-31.*

Advantages of Pulse Oximetry

- Fifth vital sign
- Requires no special training to use
- Noninvasive
- Inexpensive
- Continuous real-time measurements (point-of-care testing)
- Avoids pain/risks of arterial puncture
- Standard of care during procedures requiring general anesthesia
- Standard of care during procedural sedation and analgesia
- Small, requires little space, portable
- Can be used almost anywhere
- Can be stand-alone equipment or incorporated into a multipurpose monitor
- Easy to apply and use
- Can be used in any age and any type of patient

Limitations of Pulse Oximetry

PROBLEMS INHERENT WITH SaO_2 MEASUREMENTS

- Oxyhemoglobin dissociation curve
  - In middle steep part of curve, small changes in PaO_2 levels cause large changes in SaO_2 levels.
  - In upper flat part of curve, the SaO_2 level is relatively insensitive to PaO_2 changes.
- Does not measure ventilation
  - Does not measure pH, pCO_2 levels
  - Patients may have normal SaO_2 level until late deterioration.

DESIGN OF PULSE OXIMETRY:

- Uses spectrophotometry with two wavelengths to estimate SaO_2 level by comparison with levels in normal healthy adult volunteers
- Uses only 2 wavelengths, so measures *functional* saturation not *fractional* saturation (increase in presence of methemoglobin or carboxyhemoglobin levels, increase in falsely high SaO_2 level)
- Accuracy affected by various dyes/pigments (e.g., methylene blue [absorbs light at 660 nm], fluorescein, indocyanine green, indigo carmine)
- Calibration curves obtained from data from small number of healthy adults
- Accuracy varies depending upon SaO_2 level; more accurate: SaO_2 measurement in 80%-100% range

INDIVIDUAL PATIENT VARIABLES

- Low perfusion states (e.g., shock, low cardiac output, decreased peripheral perfusion, increased/inadequate arterial blood pressure, increased/inadequate arterial pulsatile flow) may have inadequate signal
- Works best in middle ranges of heart rate, systolic blood pressure, and hematocrit level
- Decreased accuracy if low hemoglobin/hematocrit level (severe anemia) especially if low SaO_2 measurement
- Darkly pigmented skin
- High venous pressure (e.g., heart failure or superior vena cava syndrome) increases falsely low SaO_2 measurement

TECHNICAL VARIABLES

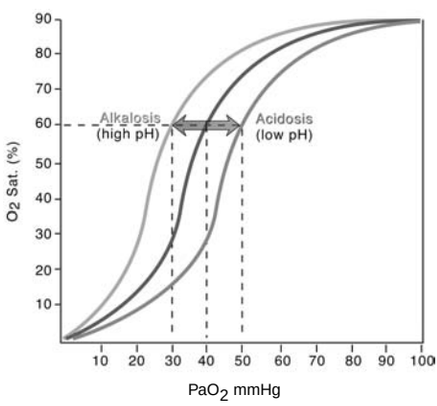
- Excessive motion causes motion artifact
- High ambient light
- Dark nail polish (if finger probe is used)
- Electrocautery
- Misplaced probe (penumbra effect)

FACTORS NOT AFFECTING PULSE OXIMETRY

- Fetal hemoglobin
- Sickle hemoglobin
- Hyperbilirubinemia
- Dialysis grafts (A-V fistulas)
- Polycythemia

Key: SaO_2 = oxygen saturation. PaO_2 = partial pressure of arterial oxygen. pCO_2 = partial pressure of carbon dioxide.

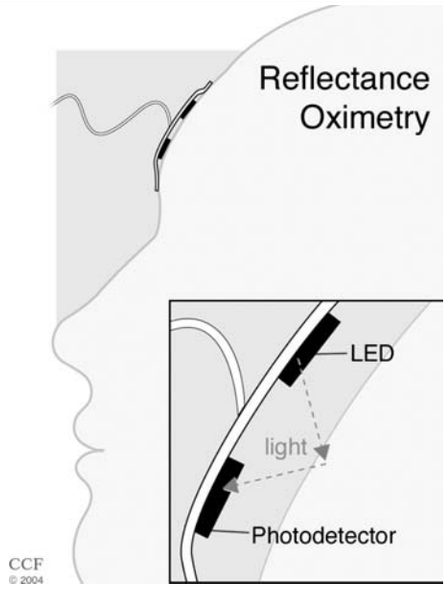
Oxyhemoglobin Disassociation Curve



The sigmoidal shape of the oxyhemoglobin dissociation curve demonstrates the nonlinear relationship between SaO_2 and PaO_2 levels. Various factors, as shown in the graph, may shift the curve to the left or the right. When the curve shifts to the right, the Hgb has a decreaed affinity for oxygen, and therefore, more oxygen is released to the tissues. The reverse is true when the curve shifts to the left.

Illustration courtesy of Cleveland Clinic Foundation, Department of Medical Illustration.

Reflectance Oximetry



Reflectance oximetry is technically more difficult and has less accuracy than pulse oximetry, and therefore, is not used commonly.

Illustration courtesy of Cleveland Clinic Foundation, Department of Medical Ilustration.

Factors Affecting Pulse Oximetry Readings

FALSELY HIGH SPO_2

- Presence of abnormal hemoglobin
  - Carboxyhemoglobin (COHgb)
  - COHgb light absorption is like $HgbO_2$ absorption in red spectra, so oximeter reads high SpO_2 level.
  - Methemoglobin absorbs light in both red and infrared wavelengths, so oximeter reads high SpO_2 level.
- Anemia (if Hgb <10)

FALSELY LOW SPO_2

- High venous pressure
  - Congestive heart failure
  - Superior vena cava syndrome
  - Traumatic venous obstruction
  - Application of tourniquet (proximal to pulse oximeter - e.g., in same arm)
  - Inflation of manometer cuff (proximal to pulse oximeter - e.g., in same arm)
- Diagnostic dyes
- Nail polish
- Skin color (deeply pigmented)

TECHNICAL FACTORS (POOR OR NO READING)

- Problems with sensing
  - Motion near sensor site mimics arterial pulsation
- Low flow states
  - Low blood flow leads to insufficient change in light emittance, resulting in no reading
- Ambient ligh
  - Increases light to photodetector
- Penumbra effect

Oxyhemoglobin Disassociation Curve

SHIFT TO LEFT	SHIFT TO RIGHT
• Increased affinity of oxygen to Hgb	• Decreased affinity for oxygen to Hgb
• Decreased availability of oxygen to tissues	• Increased availability of oxygen to tissues
• Factors causing shift to left - increased pH level (e.g., alkalosis) - decreased pCO_2 level (e.g., hypocarbia, respiratory alkalosis) - decreased 2,3 DPG level	• Factors causing shift to right - decreased pH level (e.g., acidosis) - increased pCO_2 level (e.g., hypercarbia, respiratory acidosis) - increased temperature (fever) - increased 2,3 DPG level - chronic hypoxia - chronic alkalosis - high altitude adaptation - hyperthyroidism - anemia

Supplement to *Pediatric Emergency Medicine Reports*, March 2005: "The Fifth Vital Sign: Pulse Oximetry in Noninvasive Respiratory Monitoring." *Author*: **Sharon E. Mace, MD, FACEP, FAAP**, Associate Professor, Ohio State University School of Medicine; Faculty, MetroHealth Medical Center/Emergency Medicine Residency; Clinical Director, Observation Unit; Director, Pediatric Education/Quality Improvement, Cleveland Clinic Foundation, Cleveland. *Peer Reviewer*: **David Kramer, MD, FACEP, FAAEM**, Program Director, Emergency Medicine Residency, and Vice-Chair, Department of Emergency Medicine, York Hospital, York, Pennsylvania

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Mar./Apr. 2005

The Burned Patient: Assessment, Diagnosis, and Management in the ED

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Burn injuries frequently present to the emergency department (ED). In the majority of cases, the burns are minor, yet, they require a careful assessment, cleaning, dressing, and careful follow-up. In the pediatric and geriatric populations, careful attention, to the history and physical examination, and an awareness of burn patterns associated with abuse, may protect the patient from further inflicted injury.

Patients with more severe burn injuries, especially those associated with house fires or explosions, should be assessed carefully for multiple trauma, and care should be taken to protect the spine until injury can be excluded clinically or radiographically. The airway of a burn patient may be particularly challenging; early aggressive intervention, when indicated, may make a potentially disastrous situation manageable. The authors review the diagnosis, classification, and management of patients who have sustained burns. — *The Editor*

Epidemiology

More than one million burn injuries occur in the United States each year; 700,000 of these injured individuals will seek care in an ED, and 45,000 will be hospitalized for their injuries.[1] In the United States, $2 billion are spent on burn care annually.[2]

Seventy percent of burned patients are male, and the average age of patients sustaining burn injury is 30 years. Infants account for 13% of cases, and adults older than 60 years for 11%. The extremes of age have been associated with an increased risk of death from burns and burn-related injuries.[3]

Burns can occur by several mechanisms. Scald burns arise from exposure to hot liquids or steam. Thermal burns are the result of contact with flames. Contact burns are caused by contact of the skin with hot or cold surfaces. Burns also occur from exposure to radiation, chemicals, and electricity.

Thermal, scald, and contact are the most common categories of burn injuries. In particular age groups, certain types of burns occur more commonly: scald and contact burns are prevalent from birth to 2 years of age, whereas thermal burns are common in the 5- to 20-year range.[3]

Most children dying as a result of burns sustained their injuries in house fires, and as many as 20% of pediatric burns are the result of abuse or neglect.[4] Low income, minority children are

three times more likely to die in a house fire than children in high-income categories.[5,6]

Likely causes of burn injury also vary among different ages. Mishaps with flammable liquids are common causes of burn injury in teenagers and young adults,[7] but for the elderly population, kitchen accidents and the resultant exposure to hot liquid or to an open flame are most prevalent.[8]

Burn injury also may occur in connection with industrial incidents as well as other major trauma. One-fifth to one-fourth of severe burns are work related, and 5% to 10% of burned patients sustain multisystem trauma concurrent with their burn injury.[9-11]

A study performed at Massachusetts General Hospital and Shriners Burns Institute in Boston found three distinct risk factors associated with higher mortality: age more than 60 years, burn injury greater than 40% of the total body surface area (TBSA), and the presence of concomitant inhalation injury. If none of these risk factors were present, the death rate associated with burn injury was 0.3%. The presence of one risk factor raised the mortality to 3%. Two risk factors correlated with a 33% mortality, and when all three risk factors were present, the risk of death jumped to 90%. These numbers apply only to adults younger than 90 years. If a burned patient survives the first week after the incident, the chance of death drops to 2.5%. Only 1.1% of burn injured patients surviving after 2 weeks will die from their injuries.[2]

A recent study from the Shriners Burn Institute at the University of Texas sheds some light on pediatric burn mortality numbers. On average, the investigators found that burns of 85% of the TBSA were 30% lethal. Concomitant inhalation injury increases the risk of death. The mortality rate for patients with burns affecting 50% of the TBSA and concomitant inhalation injury was 10%. In comparison, patients with burns covering 73% of the TBSA without inhalation injury had the same 10% mortality. This study also found that males younger than 3 years had a higher risk of death from their injuries than males of other ages. Lastly, Hispanic teenagers had a higher mortality from burn injury than African-American or Caucasian teenagers of the same age.[5]

Pathophysiology

Burn wounds have three distinct zones of tissue damage: the coagulation zone, the stasis zone, and the hyperemic zone. The coagulation zone consists of the tissue that has been irreversibly destroyed from the primary injury and cannot recover. Surrounding this area is the stasis zone, where damaged tissue with decreased perfusion is located, but the potential for recovery still exists. Adjacent to the stasis zone is the hyperemic zone, the tissue that has sustained minimal damage and will recover spontaneously.[7,12,13] The tissue in the zones of stasis and hyperemia, while still viable, is at risk of destruction from poor perfusion, edema, and inflammation.[13]

Blisters form when damaged capillaries, with increased vascular permeability, leak plasma into the interstitial space. The damaged epidermis separates from the underlying dermis. The fluid of the blister contains inflammatory mediators, such as arachidonic acid metabolites, thromboxane, and calmodulin, as well as plasma proteins and cellular debris. The high osmolarity of this fluid can cause additional water absorption from underlying tissue into the blister, which increases local wound tissue pressure and may cause ischemia in the already compromised tissue.[12]

As the largest organ in the human body, the skin provides protection, immunologic defense, and acts as a barrier to fluid loss. When the skin is burned, these functions are lost, enhancing the victim's risk of systemic illness, sepsis, and multiple organ failure.

Loss of the barrier function of the skin leads to massive fluid losses from evaporation. This fluid loss impedes tissue perfusion and oxygenation. The resultant hypovolemia can result in relative hypoperfusion to distant organs.[7] Injured tissues release inflammatory mediators and vasoactive substances, causing interstitial edema and organ dysfunction. Multi-organ failure usually develops between the second and eighth week after injury and accounts for one-third of burn-related deaths.[2,14] Increased age, increased TBSA burned, male sex, and the presence of inhalation

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In order to reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, Dr. Dietrich (editor in chief) reports that she is the medical director for the Ohio Chapter of the American College of Emergency Physicians. Editorial board members Bowman, Diebel, Falcone, Finerty, Hanlon, Mahadevan, Perkin, Santanello, Savitsky, and Stafford report no relationships with companies related to the field of study covered by this CE/CME program. Ms. Neff reports that she is a stockholder in Biopure. Drs. Teague, Swencki, and Tang (authors), and Dr. Menckhoff (reviewer) report no relationships with companies related to the field of study covered by this CE/CME program.

Figure 1. Inflicted Immersion Burn

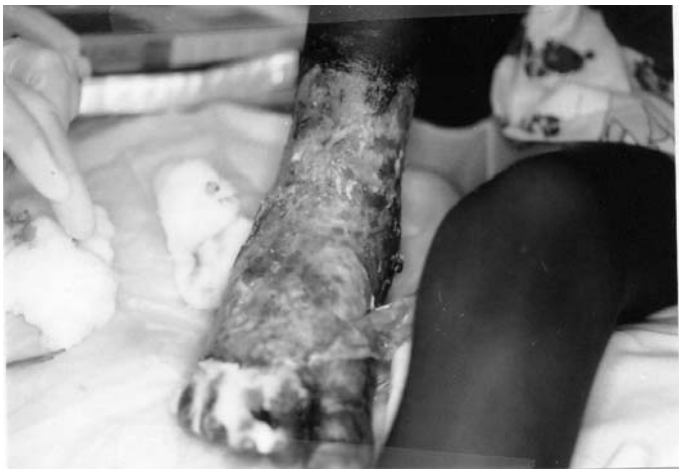


Figure 1. Photograph shows stocking distribution of an inflicted immersion burn. Note the clear demarcation between burned and spared skin. The child told medical staff of being forced to stand in hot water for several minutes.

Reprinted with permission from Bechtel, K. Identifying the subtle signs of pediatric physical abuse. Ped Emerg Med Rep *2001;6:61.*

injury all are associated with an increased likelihood of the development of multi-organ failure after burn injury.[14,15]

Loss of the skin's immunologic defenses leads to an increased susceptibility to infection. Systemic infection results from invasion of bacteria into the body through the burn eschar. Immediately following injury, the burned area is sterile, but then bacteria quickly colonize the wound. Rapidly reproducing in this avascular environment, the bacteria are able to gain access to the rest of the body.[8] Sepsis is common in burned patients and has been noted to precede the development of multi-organ failure. Advanced age and the presence of full-thickness burns are risk factors for the development of severe sepsis.[15]

In the first 1 to 3 hours after burn injury, edema develops and may increase up to 24 hours after injury.[16-18] The development of edema in the setting of burn injury is multifactorial. Vasodilation and increased transcapillary pressure in conjunction with increased extravascular osmotic activity of the burned tissue, increased microvascular permeability, and impaired cell membrane function with swelling of the cells all contribute to the development of edema.[19,20]

Cardiac Effects. Immediately after injury, myocardial function may be depressed; however, this typically improves within three days. This depression in myocardial function may be caused by circulating myocardial depressants or persisting hypovolemia, despite agressive fluid therapy and the lack of classic signs of hypoperfusion.[21] A hyperdynamic cardiovascular response then occurs, with an up-to-twofold increase in cardiac output.[8,22]

Metabolic Effects. Metabolic derangements (e.g., metabolic acidosis, respiratory alkalosis, and electrolyte disturbances) are common in patients with burn injury. Intracellular concentrations of sodium and calcium rise, while intravascular levels of potassium increase as the result of cell membrane alteration.[23]

In comparison with other critically ill patients, patients suffering from burn injuries have the highest metabolic rate.[24] The burned patient has increased energy expenditure, accelerated glycogen and protein breakdown, and lipolysis. This hypermetabolic state is the result of increased circulating catabolic hormones, catecholamines, cortisol, and glucagon.[22,24,25]

Catabolism begins by five days after injury.[24,26] Although it was once thought that catabolism resolved with wound closure, catabolism actually continues up to nine months after the initial injury.[27] The level of catabolism is increased with age, weight, and delay in surgical treatment. Catabolism intensifies with increases in TBSA burned, up to a TBSA of 40%. Sepsis, hyperglycemia, and decreased ambient air temperature also increase catabolism in these critically ill patients.[24,26,28]

Thermoregulation is altered after burn injury. In addition to the loss of the skin's protective function, which results in loss of body heat and hypothermia, the hypothalamic temperature regulation set point increases by 2º C above normal body temperature.[24,29]

The metabolic effects of burn injury can have serious sequelae for the patient. Patients experience loss of lean body mass and body weight, delayed wound healing, and immune depression as a result of their hypermetabolism. Fatty infiltration of the liver develops as a result of the increase in lipolysis.[30] Pediatric patients with severe burns have been found to have a delay in linear growth for two years after injury.[31]

Clinical Features

History. A thorough history is required from every burned patient and may require information from paramedics, family, or witnesses to the injury. Standard elements, such as medical history, surgical history, medications, allergies, and last tetanus immunization, are essential. Beyond those, the mechanism of injury is perhaps the most important information. Knowing how the burn was incurred will help direct the workup and physical examination and help delineate whether the burn injury occurred intentionally. The mechanism of burn injury also may indicate whether the inhalation of toxic gases, such as carbon monoxide or cyanide, may have occurred. Current use of alcohol or illicit drugs also is important to ascertain; it may have contributed to an alteration in mental status, the mechanism of injury, or comorbidities. In pediatric patients, it is essential to ascertain the circumstances surrounding the injury, maintaining a high index of suspicion for intentional acts.

Physical Examination. The physical examination of the burned patient begins with assessment of the ABCs (airway, breathing, circulation). After management of these crucial elements, a secondary survey focusing on recognizing concurrent traumatic injuries, if there is associated trauma, should be completed. Evaluate the patient's face and oropharynx for carbonaceous sputum, circumoral burns, and singed nasal hair, which could indicate the presence of an inhalation injury. During this survey, patterns of injury in pediatric patients that may indicate abuse, such as a stocking or glove-like appearance with sharp

Figure 2. Rule of Nines (Adult)

ADULT BODY PART	% OF TOTAL BSA
Arm	9%
Head	9%
Perineum	1%
Leg	18%
Anterior trunk	18%
Posterior trunk	18%

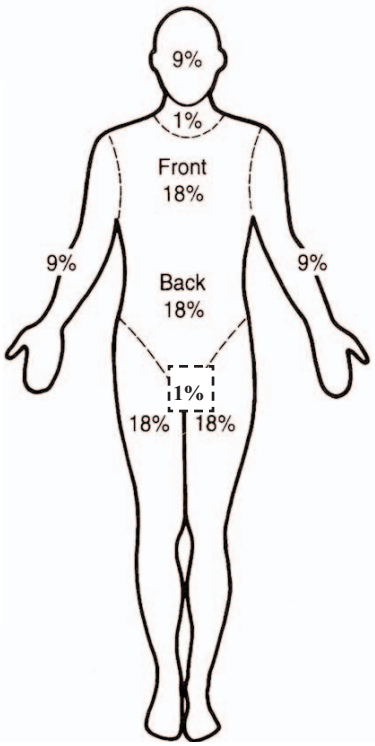
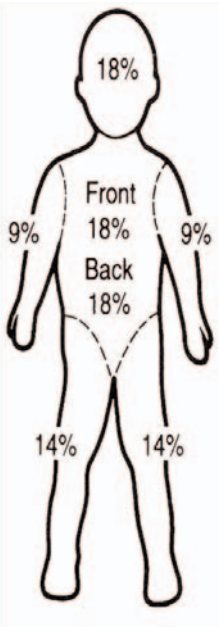


Figure 2. The Rule of Nines is designed to rapidly access the percentage of TBSA for a burn injury.

Illustration courtesy of the authors.

Figure 3. Rule of Nines (Child)



CHILD BODY PART	% OF TOTAL BSA
Arm	9%
Head and neck	18%
Leg	14%
Anterior trunk	18%
Posterior trunk	18%

Figure 3. Modification to the adult Rule of Nines to reflect the different proportions in the pediatric population.

Illustration courtesy of the authors.

margins, should be recognized. *(See Figure 1.)* The severity of the burn injury also should be appraised.

Severity of Burn Injury. To determine the severity of a burn injury, assess both the TBSA burned and the depth of the burn injury. The TBSA measurement is used to estimate fluid resuscitation requirements and to assess the risk of death. Burn depth is used to assess the burned patient's need for hospitalization, the need for surgical intervention, as well as the probability of scar development after the wound heals.[32]

There are three methods of estimating the total body surface area burned (First-degree or superficial burns are excluded from BSA calcuations). The Rule of Nines is the most commonly used system to estimate the extent of burn injury. It is much more accurate for adults than for children. The body is divided into areas that represent 9% of body surface area or multiples of 9%. The exception is the perineum, which is assigned the value of 1%. *(See Figure 2.)* For the pediatric patient, the Rule of Nines is altered by taking 4% from each leg and 1% from the perineum to add an additional 9% to the surface area of the head. *(See Figure 3.)* The values associated with affected areas are summed to estimate the TBSA burned.[3,33]

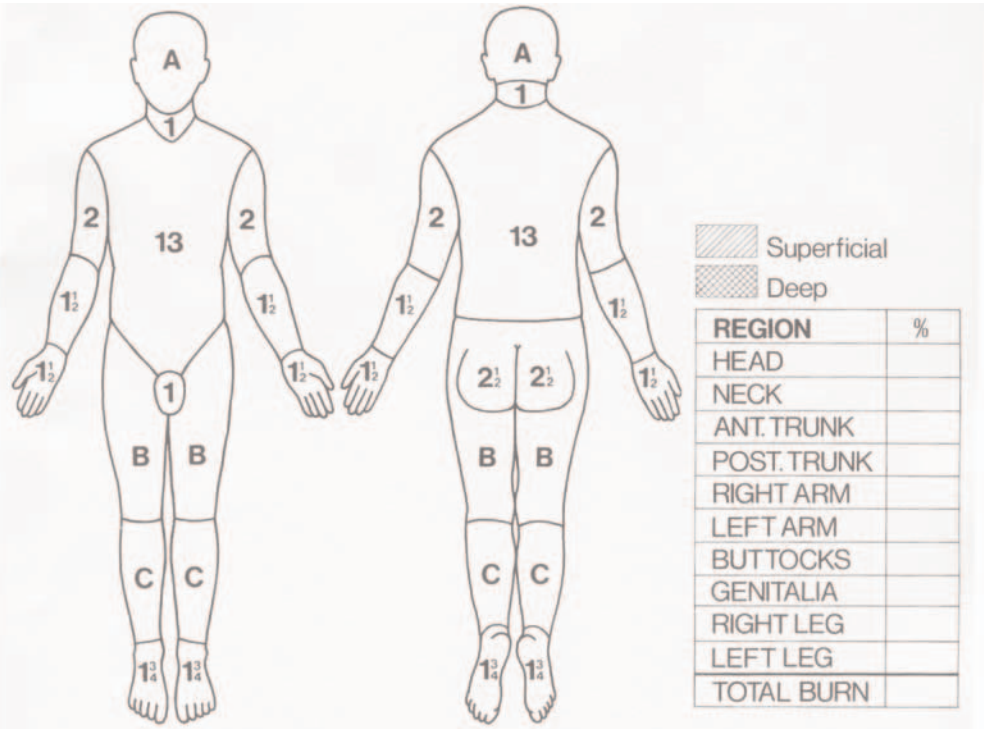
The Lund and Browder chart is considered a more accurate estimation tool to determine TBSA, especially for pediatric patients. This chart also divides the body into areas and assigns a percentage body surface area based on the patient's age. This chart accounts for the differences in proportionality between newborns, children, and adults.[3,34] *(See Figure 4.)*

Another frequently used technique for estimation of injury uses the surface area of the patient's palm, considered to represent 1% of the TBSA. This method is best used for patients with scattered small burns and is believed to be the least accurate of the three methods. Investigators at the University College London Medical School called this method into question. Their research found that the palmar surface of the hand represented only 0.4% body surface area of adults and 0.45% body surface area in children.[35]

Given the increasing incidence of obesity in our society, it is important to consider how estimation of TBSA in the obese patient differs from that process in nonobese patients. Underestimation of burned area on the trunk and legs becomes more common with increasing obesity. The trunk may constitute up to 50% of TBSA in the obese patient, while each leg may account for 20%. The head and arms of the obese patient account for a smaller body surface area than that assigned to them by the Rule of Nines.[36]

The depth of burn injury commonly has been referred to in terms of first-, second-, and third-degree burns. First-degree burns affect only the epidermis. They are warm and red, have no blisters, and generally are painful. In second-degree burns, both the dermis and epidermis are involved. Most burns of this type will be painful, red, and blistered, with moist bases. In third-degree burns, the entire dermis and epidermis are destroyed.

Figure 4. Lund and Browder Chart



REGION	%
HEAD	
NECK	
ANT. TRUNK	
POST. TRUNK	
RIGHT ARM	
LEFT ARM	
BUTTOCKS	
GENITALIA	
RIGHT LEG	
LEFT LEG	
TOTAL BURN	

RELATIVE PERCENTAGE OF BODY SURFACE AREA AFFECTED BY GROWTH

AREA	AGE 0	1	5	10	15	ADULT
A = ½ OF HEAD	9½	8½	6½	5½	4½	3½
B = ½ OF ONE THIGH	2¾	3¼	4	4½	4½	4¾
C = ½ OF ONE LEG	2½	2½	2¾	3	3¼	3½

Figure 4. The Lund and Browder Chart is the most accurate estimation tool to determine TBSA and accounts for differences in proportions between children and adults.

Illustration courtesy of the authors.

These burns appear leathery and dry and are typically tan in color. Third-degree burns usually are painless, secondary to destruction of pain receptors in the dermis.[37] In some instances, the term "fourth-degree burn" also is used. These are burns that involve underlying tissue such as muscle, bone, or fascia.

However, these categories have been replaced recently by the more accurate and less confusing terms of superficial, partial-thickness burns (which include both superficial and deep partial thickness), and full-thickness burns. This system of categories accounts for the anatomic structures affected by the burn (Table 1).[38]

Superficial burns affect only epidermis. Erythema is present, but no blisters form. The surface is usually dry and painful to touch. In certain areas, such as around the eyes, there also may be edema. These burns usually heal in 3 to 7 days and generally do not lead to scarring.

In superficial partial-thickness burns, injury extends through the epidermis into the superficial layers of the dermis. These burns are extremely painful. The burned area is erythematous, and the surface blanches readily, with brisk capillary refill. Blisters develop rapidly. Owing to the extensive vasculature of the epidermis, these are moist wounds and produce moderate edema.

Extending to the deeper layers of the dermis, deep partial-thickness burns may have a red and white waxy or mottled appearance. Although these wounds continue to blanch, capillary refill may be slow or entirely absent. Blisters usually are not present. The surface of the wound is moist, notable edema is present, and sensation is altered.[39] Most partial-thickness burns (both superficial and deep) heal spontaneously within 14 days. The extent of scarring resulting from these burns depends upon the depth of the burn. If located over a joint, these burns may require skin grafting.[38]

Full-thickness burns destroy epidermis and dermis and extend into the subcutaneous tissue. They have a white or charred appearance without any blistering. These burns are insensate secondary to destruction of sensory nerves; however, the area of a full-thickness burn will be bordered by an area of less severely burned tissue, which is painful. Subdermal burns extend into muscle, fascia, and bone. Burns of this magnitude require surgical intervention and have an associated risk of systemic disease. Fluid and protein shifts cause intense edema.[38,39] Skin grafting leads to extensive scarring, development of contractures, and impaired mobility.

Customarily, the determination of burn depth is made clinically; however, in serious burn injury, this assessment can be complicated. Wound biopsy has been used to histologically identify burn depth more accurately by examining the tissue for evidence of blocked and patent vasculature.[40] The less invasive technology of laser Doppler imaging (LDI) also is being used to assess microvascular blood flow in the dermis to determine burn depth.

LDI combines laser Doppler technology with scanning techniques to produce an image of tissue perfusion by tracking red blood cell movement.[41,42] This technology has been found to assess burn depth accurately in 97% of injuries compared with 60% to 80% by clinical assessment alone for adult patients.[41] In pediatric burned patients, this technology has been found to have a sensitivity of 90% and specificity of 96% for the detection of deep partial or full-thickness burns when used 36 to 72 hours after injury.[42] This technology has yet to become widespread in

Table 1. Burn Depth

CLASSIFICATION	FORMER CLASSIFICATION	STRUCTURES INVOLVED	COLOR	BLISTERS	PAIN
Superficial	First degree	Epidermis	Red, dry	No	Yes
Partial-thickness, superficial	Second degree	Epidermis into superficial dermis	Red, moist	Yes	Yes
Partical-thickness, deep	Second degree	Epidermis into deep dermis	Red/white, waxy	No	Altered
Full-thickness	Third degree	Epidermis, dermis, into subcutaneous tissue	White or charred	No	None
Subdermal	Fourth degree	Epidermis, dermis, into fascia, muscle, bone	White, charred, variable depending on involved structures	No	None

use, but it has the potential to become a useful adjunct in the assessment of burn injury depth in the future.

Diagnostic Studies

Burned patients should be placed on a cardiac monitor, and pulse oximetry should be assessed and monitored, if indicated. Basic laboratory studies should be obtained in patients with severe burns or concomitant trauma, including a complete blood count, type and crossmatch, chemistries, coagulation profiles, arterial blood gas measurement, and a pregnancy test, when appropriate. All patients with thermal burns should have arterial or venous blood sent for measurement of the carboxyhemoglobin level to evaluate for carbon monoxide poisoning.

An initial chest radiograph is warranted in all burned patients when an inhalation injury is possible. A normal study does not rule out pulmonary injury however, and serial chest radiographs may show delayed development of pulmonary edema or findings of pulmonary contusions. Computed tomography scans should be obtained as indicated in the patient with accompanying traumatic injuries or decreased mental status. In addition, the history and physical examination should guide radiologic examination of the extremities and cervical spine.

Initial Management

Stabilization of the ABCs is essential in managing any medical emergency. The initial approach to managing severe burn victims is no exception. Because severe burns often are associated with nonthermal injuries, seriously burned patients must be viewed as trauma patients and should be stabilized according to Advanced Trauma Life Support and the American College of Surgeons Committee on Trauma protocol.[8,43]

The first priority in stabilizing these patients is ensuring a patent airway, which can be challenging, secondary to oropharyngeal and laryngeal edema. Airway edema may progress rapidly in a burned patient who has inhaled heated gases or toxic products of combustion. Signs that indicate the patient may have had a significant inhalational injury include singed nasal hairs, facial burns, oral burns, sooty sputum, and stridor or wheezes. Fiberoptic laryngoscopy or bronchoscopy may be helpful in assessing the degree of airway trauma.

Once the airway is established, it is paramount to secure it; laryngeal edema, even more than oropharyngeal edema, makes intubation difficult in burn patients. If circumferential burns of the chest or an extremity are present, emergent escarotomy may be necessary. If the chest eschar compromises ventilatory motion incisions along the the costal margin, anterior axillary lines and across the top of the chest may be necessary to allow adequate chest movement. Circumferential eschar of an extremity may act as a tourniquet restricting adequate blood flow to an extremity. In this case, an escarotomy should be performed along the lateral aspect of the extremity, through the entire depth of the eschar, to allow the return of adequate blood flow. Obtaining intravenous access also is extremely important for adequate fluid resuscitation. If peripheral access is unobtainable, central access, or intraosseous access in children, generally is required.

Fluid Resuscitation

Fluid resuscitation in severe burn victims is controversial in many aspects. Despite many years of research in the area of burns, there does not seem to be standardization regarding the type of infusion fluid to use or definitive resuscitation endpoints.

The American Burn Association (ABA) suggests that patients with burns greater than 15% TBSA should undergo fluid replacement according to the Parkland Formula:[3]

4 mL (of IV fluid) x weight (kg) x % TBSA burn = Total amount of fluid for the first 24 hours

Table 2. Consensus Formulas for Fluid Resuscitation of Burned Patients after 24 Hours

TBSA BURNED (%)	FLUID ADMINISTRATION
0-30	None
30-50	0.3 mL/kg/%burn/24 hr
50-70	0.4 mL/kg/%burn/24 hr
70-100	0.5 mL/kg/% burn/24 hr

Key: TBSA = total body surface area.

Table 3. Resuscitation Endpoints

• Sensorium	Arousable and comfortable
• Digital temperature	Warm peripherally
• Systolic blood pressure	
- For infants –	60 mmHg systolic
- For older children –	70–90 mmHg + 2 x age in years
- For adults –	mean arterial pressure >60 mmHg
• Pulse	80–180/min (age dependent)
• Urine output	0.5–1 mL/kg/hr (glucose negative)
• Base deficit	<2 mEq/L

Adapted from Sheridan RL. Comprehensive treatment of burns. Curr Probl Surg *2001;38:641-756.*

This formula currently is the gold standard and applies only to adults. However, the Parkland formula is for replacement fluid administration and does not include approximation of maintenance fluids. Pediatric considerations will be discussed later in this article. Half of the calculated amount is given intravenously during the first 8 hours, and the rest is given during the remaining 16 hours. If initial fluid administration is delayed, half of the calculated volume is to be completed by the end of the eighth hour after injury. Interestingly, even though current teaching is to give fluids aggressively to burn victims, some studies have suggested that this type of fluid management increases oxygen delivery to ischemic tissue, triggering free radicals that can further damage tissue.[44]

The fluid of choice for initial resuscitation is an isotonic crystalloid fluid, such as lactated Ringer's solution. The lactate replaces the chloride in the solution, decreasing the likelihood of hyperchloremic acidosis.[8] Many studies have explored the use of hypertonic solution (i.e., 3% saline solution) as an alternative for burn victims.[22] Some studies have shown that use of a hypertonic solution may decrease the extent of edema.[45,46] However, the outcomes of studies that compared hypertonic solution versus crystalloid solution are inconclusive and suggest the use of such hypertonic solution does not improve, and may worsen, outcomes.

During the second 24 hours of resuscitation, fluid administration should decrease. This premise is based on the belief that after 18 to 24 hours, if resuscitation is successful, capillary integrity improves and therefore fluid requirements decrease.[8] The consensus formulas for fluid administration after the first 24 hours are listed in Table 2.

At this point in fluid management, the fluid of choice is a colloid formula, such as 5% albumin lactated Ringer's solution. In addition, electrolytes should be monitored closely.

The aforementioned formulas should be used in burn victims to achieve suggested endpoints of resuscitation. Infusion rates of fluids can be adjusted based on the resuscitation endpoints. *(See Table 3.)*[8] Resuscitation endpoints include stable vitals signs, normal mentation and sensorium. One important endpoint is maintaining adequate urine output, specifically 30-50 mL per hour in adults and 1mL/kg per hour in children.[47,48] `

Wound Care

Immediate wound care for burns is important for many reasons. Topical agents for less serious burns provide a means of pain control and decrease the rate of bacterial growth. Deeper burns may require surgical management or subsequent transfer to a burn center. How a burn wound is treated depends upon the depth of the wound. Overall, outpatient management of burns can be divided into the six Cs: clothing, cooling, cleaning, chemoprophylaxis, covering, and comforting.[32]

Clothing. Clear the patient's body of all materials that are hot or burned. In addition, clothing that appears to have come into contact with any chemicals also should be removed.

Cooling. Cold water has many purposes for burn wounds. Applying gauze soaked with cold water stops the burning process, relieves pain, and removes chemicals from direct contact with skin. Use caution with cooling methods for patients with burns greater than 10% TBSA; they may cause hypothermia, especially in children. Cold water should be applied to the burn area for at least 10 minutes and a maximum of 20 minutes.[49]

Cleaning. Cleaning a wound is essential to prevent infection; however, the procedures can cause a great deal of pain. Local, regional, or systemic anesthesia should be induced before cleaning a wound; topical anesthesia and injection of anesthetic agents directly into the wound should be avoided. There is increasing support for using mild soap and tap water to wash burns.[50-53] Disinfectants, such as povidone–iodine solution or chlorhexidine gluconate solution, should be avoided because the agents will hamper the healing process. If there are any residues adhering to the wound, such as tar or asphalt, they should be removed with the aid of large amounts of bacitracin ointment applied during a period of many days.

Blister formation commonly is associated with burns. Intact blisters can allow re-epithelialization 40% faster than blisters that are aspirated or deroofed.[13] However, studies have shown that blister fluid contains proteins that increase the likelihood of sepsis by decreasing normal lymphocytic and neutrophilic function. Deroofing minor blisters is controversial and needs further research. Some study results have suggested that small-to-moderate-sized blisters be covered with occlusive dressing for the first 72 hours after injury. Large blisters or blisters over a joint may be aspirated with a needle and a syringe leaving the roof of the blis-

ter intact. After 72 hours, the blistered skin may be excised using aseptic technique.[12]

Chemoprophylaxis. Burns are considered to be tetanus-prone wounds. Active immunization against tetanus should be given to burn patients when tetanus status is in doubt. For patients who are not immunized or who have incomplete tetanus status, passive tetanus immunization is recommended.[22]

Burn wounds are more vulnerable to infections and, ultimately, sepsis. The common pathogens that cause infection in burn victims are *Staphylococcus aureus, Pseudomonas aeruginosa, Streptococcus pyogenes*, and other coliform bacilli.[54]

Topical antibiotics are an essential element in burn wound management. Classically, the medication of choice is silver sulfadiazine. It is a good selection for most burns, especially for deep partial-thickness burns, because it may permit wound healing without the need for a skin graft. Silver sulfadiazine cream should not be used on the face or in patients who are pregnant, newborns, or nursing mothers with children younger than 2 months because of the risk of sulfonamide kernicterus. In addition, silver sulfadiazine is contraindicated in patients with sulfa allergies. A cerium nitrate silver sulfadiazine cream also is commercially available. Cerium is a lanthamide metal that interacts with calcium, which is an important element of epidermal cell growth. A few studies suggest that cerium, in conjunction with silver sulfadiazine, can decrease local inflammation and sepsis.[55]

Bacitracin also can be used as a topical antibiotic for wound management. The advantage of using bacitracin over silver sulfadiazine is its lower cost. However, studies have not compared the efficacies of one topical antibiotic with another.

Biologic dressings, such as xenograft and allograft, also may be used to prevent wound contamination and fluid loss. These dressings are associated with lower infections rates and faster healing compared with silver sulfadiazine.[39] Biologic dressings need to be applied within 6 hours after burn injury. These dressings allow skin epithelialization and eventually will peel off as the skin heals.

Nonbiologic dressings provide a moist wound environment and fast healing. These dressings require fewer changes and induce less pain compared with topical antibiotics.[39] However, nonbiologic dressings need a bulky dressing that must be changed daily.

Covering. Superficial burns generally do not need wound dressings. Patients with this type of burn should be instructed to see their physician if blisters form. Also, a skin lubricant, such as aloe vera, can be applied to the burn wound.

Partial and full-thickness burns should be covered with sterile dressings after the wound is cleansed and a topical antibiotic is applied. Patients should be instructed to change dressings with recommended frequencies of twice a day to once a week.[56] At each dressing change, the wound should be cleaned gently, a topical antibiotic should be applied, and the wound re-dressed.

Comforting. A burn injury can be extremely painful. Patients with small burns may be instructed to take nonsteroidal anti-inflammatory drugs and acetaminophen. Nonsteroidal anti-inflammatory medications also decrease inflammation and edema and increase blood flow. Opioids taken orally may be added for pain control. For more painful burns, adults should be given morphine intravenously or intramuscularly. Aggressive pain control should be pursued with some adults requiring large doses of intravenous morphine.

Table 4. Burn Center Referral Criteria

PATIENTS WHO MEET THE CRITERIA LISTED BELOW SHOULD BE REFERRED TO A BURN CENTER

- Partial-thickness and full-thickness burns greater than 10% of the total body surface area (TBSA) in patients younger than 10 years or older than 50 years
- Partial-thickness and full-thickness burns greater than 20% TBSA in other age groups
- Partial-thickness and full-thickness burns involving the face, eyes, ears, hands, feet, genitalia, or perineum, or those that involve skin overlying major joints
- Full-thickness burns greater than 5% BSA in any age group
- Electrical burns, including lightning injury. (Significant volumes of tissue beneath the surface may be injured and result in acute renal failure and other complications.)
- Significant chemical burns
- Inhalation injury
- Burn injury in patients with pre-existing illness that could complicate management, prolong recovery, or affect mortality
- Any burn patient in whom concomitant trauma poses an increased risk of morbidity or mortality may be treated initially in a trauma center until stable before transfer to a burn center.
- Children with burns seen in hospitals without qualified personnel or equipment for their care should be transferred to a burn center with these capabilities.
- Burn injury in patients who will require special social and emotional or long-term rehabilitative support, including cases involving suspected child abuse or neglect

Adapted from Committee on Trauma, American College of Surgeons. Guidelines for the operation of burn units. Resources for Optimal Care of the Injured Patient 1999. 1998:55.

Special Considerations

Pediatric Burns. In general, the management of pediatric burns is similar to that of adult burns. Children have smaller and shorter airways, which can make intubation difficult, especially if the child has fast-developing edema. Children require larger amounts of fluid during resuscitation because they have larger insensible fluid losses. Pediatric patients also have a larger surface-to-mass ratio, which makes temperature control difficult. As always, in managing any emergency, the ABCs in the pediatric burned patient must be secured during initial resuscitation. To make up for fluid loss, including insensible fluid loss, the following formula can be used:

(5,000 mL/M^2 BSA burned/24 hr) +
(2,000 mL/M^2 BSA nonburned/24 hr)[9]

Half the calculated fluid is given during the first eight hours, and the rest is given during the next 16 hours. As in adults, pain

Figure 5. Oral Commissure Burn

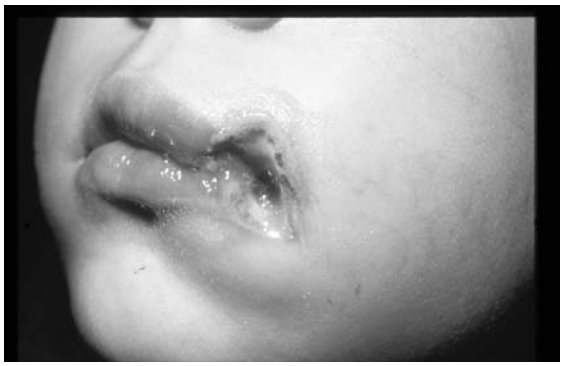


Figure 5. An oral commissure burn may occur when a child chews on a live electrical cord.

Reprinted with perrmission from Stewart C. Electrical injuries. Ped Emerg Med Rep 2001:1:4.

medication is fundamental in managing burns in children. A variety of pain medications can be used, such as nonsteroidal anti-inflammatory agents, opiates, benzodiazepines, neuroleptics, and dissociative drugs.[57] For example, a suggested starting dose of opiates (e.g., morphine) can be given in a dose of 0.1 to 0.2 mg/kg intravenously every 30 to 60 minutes as the patient's blood pressure and respiratory status tolerate. Some patients may require significantly higher doses.

Geriatric Patients. As the population ages, geriatric patients become increasingly common in EDs throughout the United States and other countries. Elderly females are more likely than elderly males to sustain a burn injury, and a lack of supervision is common.[58] The elderly are more likely to sustain a flame injury or a scald injury, and the majority of burn injuries have been reported to occur secondary to impaired judgment, mobility or both.[58] With increasing age, the survival decreases with reported survival rates in one series 86% in the 59-69 year old age group, 69% in the 70-79 year old age group, and 47% in patients older than 80 years.[58] In addition, the degree of concomitant chronic illness has a detrimental effect on survival.

Inhalation Injury. Inhalation injury is common in burn patients. Many people who sustain a burn injury were confined in a smoke-filled area and subsequently were exposed to large amounts of carbon monoxide. Usually 100% oxygen via face mask or intubation will reduce the half-life of carbon monoxide and is sufficient to treat an inhalational injury. However, hyperbaric oxygen also is a treatment option and usually is reserved for patients with carbon monoxide levels greater than 25%.[59] Other potential criteria for use of hyperbaric oxygen include patients with coma, transient loss of consciousness, ischemic electrocardiogram changes, focal neurologic deficits, and who are pregnant.[60]

Electrical Injuries. Electrical injuries also are considered a type of burn injury and often are managed at burn centers. Exposures to voltages of 200 to 1000 volts are considered moderate and are associated with local injury. Exposure to more than 1000 volts induces high-voltage injuries. They are associated with compartment syndromes, loss of consciousness, and myoglobinuria. Patients with these injuries must be monitored for cardiac arrhythmias for at least 24 to 72 hours.[8] In addition, delayed ophthalmologic and neurologic injuries can occur. Urine also must be monitored for myoglobinuria, and appropriate fluid must be administered.

Oral Electrical Burns in Children. Oral electrical burns in children usually are incurred by the sucking or chewing on the female end of a live extension cord or by biting through an electrical cord.[61] This type of injury is most common in children younger than 2 years, with a male predominance.[61] Electrolyte-rich saliva completes the circuit between the two electric poles. This results in an arc burn generating intense heat between 2500° and 3000° C that causes tissue necrosis.[62,63] The severity of the burn depends upon the length of contact, type of current, voltage, resistance of the tissue, and path of the electrical current through the body. [62,63] The low-voltage mechanism of this injury, usually resulting from household appliances consuming between 110 and 220 volts, leads to muscle contraction actually prolonging the length of exposure to the current.[63]

Oral electrical burn wounds have a central necrotic zone that appears gray or white. Surrounding this area, the tissue is edematous and raised. The most common area of involvement is the oral commissure. (*See Figure 5.*) The lower lip and cheek more likely are involved than the upper lip.[64] The heat of the electrical arc causes coagulation of tissues and thrombosis of blood vessels. As a result, bleeding is not very common in the acute phase of the injury. Edema of the surrounding tissues peaks within the first 24 hours.[65] Due to involvement of neural structures, the burns are usually painless and may result in deficits in sensory and motor function.[61,62]

In the acute care setting, these burn patients initially should be evaluated for systemic effects including arrhythmias and other injuries, which are unusual.[65,66] The wound should be irrigated, and topical antibiotics should be applied. Distinguishing between the viable and non-viable tissue is difficult in the acute phase, and debridement should not be attempted.[67] After an observation period in the ED, patients with isolated oral burn injury may be managed as outpatients with expeditious follow-up by a burn specialist and by a pediatric dentist.[64]

One of the most serious complications of oral electrical burns is delayed bleeding from the labial artery. This is most common 7 to 10 days post burn when swelling subsides and the eschar separates from the underlying tissue.[63,66] The patient's guardians should be warned of this possibility and should receive instruction on the proper application of pressure during transport back to the hospital should that occur. In the ED, the bleeding should be managed by applying direct pressure, if necessary, followed by applying epinephrine-soaked packing, and ultimately by suturing.[62]

Oral electric burns can be treated by different modalities. The use of oral splints to reduce wound contracture and to maintain position of the affected tissues during the healing process has been advocated.[62] The appliance is created by a pediatric dentist

and applied in the first few weeks post injury and commonly is worn for 6 to 12 months.[62] Surgical repair of tissue defects usually is delayed for at least 6 months after injury. At this time, the amount of scarring and the functional deficit can be better assessed.[2] The goals of reconstructive surgery are restoration of functionality and aesthetics.[67] These patients should be followed by a plastic surgeon well into their adolescence until fully grown.[7] Ultimately, most of these patients will have almost complete functional recovery of the mouth.[64]

Disposition

Most small superficial burns and some partial-thickness burns can be managed on an outpatient basis. Adequate patient education on dressing changes, topical medications, and appropriate follow-up is necessary. A patient who does not have follow-up resources should be encouraged to return to the ED for wound checks.

Some patients with partial-thickness burns need to be transferred to a burn center. The American Burn Association has published criteria for patients needing specialized care that are listed in Table 4.[68] Refer questions regarding specific patients to a burn center physician for consultation.

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CME/CE Questions

1. Burns can occur by several different mechanisms. What are the three most common types of burn injuries?
   A. chemical, scald, and electrical
   B. electrical, thermal, and scald
   C. scald, contact, and thermal
   D. radiation, chemical, and contact

CE/CME Objectives

Upon completing this program, the participants will be able to:

a) recognize or increase suspicion for traumatic injuries that present to the emergency department;
b) describe the various modalities used to identify different traumatic conditions covered in the newsletter;
c) describe how to correctly and quickly stabilize, and then to manage patients with the particular condition covered in the newsletter; and
d) identify both likely and rare complications that may occur with traumatic injuries.

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

2. Burn wounds consist of three distinct zones of tissue damage: the coagulation zone, the stasis zone, and the hyperemic zone. Which of the following is an accurate description of the stasis zone?
   A.. Tissue irreversibly destroyed by heat, which cannot recover
   B. Damaged tissue with decreased perfusion and the potential for recovery
   C. Intermediate damage to tissue, with no chance of recovery
   D. Minimal damage to tissue, with the chance of spontaneous recovery

3. The depth of burn injury commonly has been classified in terms of first, second, and third degree. However, these categories have been replaced recently by the terms superficial, partial thickness, and full thickness. Which statement below best describes partial-thickness burns?
   A. They only affect the epidermis, have no blister formation, heal in 3 to 7 days, and do not lead to scarring.
   B. They involve the underlying tissue, such as muscle, bone, or fascia; are extremely painful; and lead to systemic disease.
   C. They destroy the epidermis and dermis, extend into the subcutaneous tissue, and are insensate secondary to the destruction of nerves.
   D. They include the subcategories of superficial and deep and extend through the epidermis into the superficial and deeper layers of the dermis, respectively.

4. Which of the following statements accurately describes why it is important to assess the total body surface area (TBSA) of the burned patient?
   A. It is used to estimate fluid resuscitation requirements and to assess the risk of death.
   B. It is used to assess the patient's need for surgical intervention and stabilization.
   C. It is used to determine the probability of scar development after the wound heals.
   D. It uses the Rule of Nines, equally accurately in adults and children, to estimate the extent of burn injury.

5. To evaluate the burn patient for carbon monoxide poisoning, one must complete which of the following tests?
   A. serial chest radiographs and pulse oximetry
   B. measurement of arterial or venous blood carboxyhemoglobin level
   C. complete blood count, chemistries, and coagulation studies
   D. arterial blood gas measurements and liberal CT scans

6. The American Burn Association (ABA) recommends using which of the following for burns greater than 15% TBSA during the first 24 hours?
   A. The Resuscitation Consensus Formula
   B. Hypertonic solution (i.e., 3% saline solution)
   C. The Parkland Formula as the gold standard for adults
   D. A combination of colloid formula and isotonic crystalloid fluid

7. What is the essential reason to clean burn wounds?
   A. To ensure decreased blister formation
   B. To immediately remove adherents such as tar and asphalt
   C. To prevent the separation of the epidermis from the dermis
   D. To prevent future infection of the burn wound

8. Which are the most common pathogens that cause burn wound infections?
   A. *Staphylococcus aureus, Streptococcus pneumoniae, and Clostridium perfringens*
   B. *Pseudomonas aeruginosa, Streptococcus pyogenes, and Staphylococcus aureus*
   C. *Streptococcus pyogenes, Clostridium perfringens, and Staphylococcus epidermidis*
   D. *Staphylococcus epidermidis, Pseudomonas aeruginosa, and Streptococcus pneumoniae*

9. Topical antibiotics are an essential element in burn wound management. Classically, silver sulfadiazine is the medication of choice and can be used in which of the following patients?
   A. A pregnant woman with ≥ 15% TBSA burns
   B. A patient who has burn wounds to the face
   C. A patient with deep partial-thickness burns and no sulfa allergies
   D. A newborn with superficial extremity burn wounds only

10. Management of burns in children is similar to that of burns in adults with a few exceptions. Which of the following statements is true?
   A. Children require larger amounts of fluid during resuscitation because they have larger insensible losses.
   B. Pediatric patients have smaller surface-to-mass ratio, which makes their temperature easier to control.
   C. Children have a smaller and shorter airway, which makes intubation easier and right main stem intubation less likely.
   D. Pain medication is not as important in managing pediatric burns, and nonsteroidal anti-inflammatory agents usually are adequate.

Answer Key:

1. C
2. B
3. D
4. A
5. B
6. C
7. D
8. B
9. C
10. A

In Future Issues:

Maxillofacial trauma