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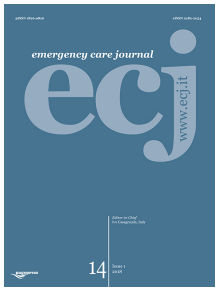

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SpO_2 monitoring in emergency and acute care: clinical utility, limitations, and interpretation pitfalls

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Key words: pulse oximetry; SpO_2; oxygen saturation; emergency care; acute care; clinical assessment; respiratory monitoring.

Abstract

Pulse oximetry provides a non-invasive numerical estimate of arterial oxygen saturation. It is simple to apply and interpret by clinicians at all levels, and its use has expanded substantially across prehospital care, emergency departments, acute care units, and household monitoring. Although pulse oximetry is portable, non-invasive, and cost-effective, its limitations are clinically important because it measures oxygen saturation rather than ventilation, oxygen delivery, or tissue oxygen utilization. Over-reliance on SpO_2 values for decisions related to oxygen administration, escalation of care, and clinical deterioration may conceal subtle but serious abnormalities. This review discusses the limitations of pulse oximetry in emergency and acute care settings, including sepsis, regional hypoperfusion, dyshaemoglobinemias, COVID-19, pulmonary embolism, hyperoxia, skin pigmentation-related bias, and other

relevant clinical contexts. The central message is that SpO_2 should be interpreted as one component of a broader assessment bundle and should not substitute for comprehensive clinical evaluation.

Introduction

Pulse oximetry is widely used in emergency and acute care. After the COVID-19 pandemic, it became increasingly available in both healthcare and household settings. It is portable, non-invasive, cost-effective, and capable of providing rapid continuous estimation of arterial oxygen saturation. These features make it a valuable monitoring tool for oxygenation in emergency and acute care settings. Consequently, it has transformed clinical assessment at triage, during prehospital care, in emergency departments, and in acute care units. In these settings, SpO_2 is frequently used to guide oxygen initiation and escalation of care. However, despite its clinical convenience, pulse oximetry measures only one dimension of respiratory physiology: the oxygen saturation of haemoglobin. Overreliance on this single parameter for assessment of breathing, oxygen delivery, or cellular respiration may create a dangerous blind spot. Therefore, SpO_2 monitoring is not a substitute for assessment of breathing and respiration. This review explores the limitations of SpO_2 monitoring and the vigilance required for its appropriate interpretation. Although the limitations of pulse oximetry have been extensively described in earlier literature,[1] many discussions remain focused on individual technical or physiological sources of error. The present review adds value by repositioning these known limitations within the practical realities of emergency and acute care, where decisions are rapid, thresholds are frequently protocol-driven, and monitor-derived numerical reassurance may override bedside clinical assessment. By organising the limitations into physiological, haemoglobin-related, technical, demographic, disease-specific, and cognitive/systems-level domains, this article aims to provide a clinically usable framework for recognising when a normal or acceptable SpO_2 value may conceal serious underlying pathology.

Pulse oximeters estimate oxygen saturation by measuring differential absorption of red light at 660 nm and infrared light at 940 nm by oxyhaemoglobin and deoxyhaemoglobin in pulsatile arterial blood.[2] The device estimates haemoglobin saturation in the pulsatile arterial component. It does not directly reflect actual oxygen content, oxygen delivery, ventilation, tissue perfusion, or tissue oxygen utilization. Accurate interpretation assumes adequate perfusion and absence of interfering haemoglobin species. In critically ill patients, these assumptions may frequently be violated and may mislead clinical decision-making.

Before discussing its limitations, it is important to acknowledge the clinical situations in which pulse oximetry remains useful. Pulse oximetry is not unreliable by default; rather, its reliability depends on the clinical context, signal quality, perfusion, haemoglobin status, and the question being asked at the bedside. Table 1 summarises common emergency and acute care conditions in which pulse oximetry can assist clinical assessment, while also highlighting its advantages and limitations.

Physiological limitations

This physiological limitation is evident when considering the sigmoid oxygen–haemoglobin dissociation curve. On its upper terminal plateau, significant declines in arterial oxygen tension may produce only minimal change in SpO_2.[3] Thus, patients may have significant impairment of gas exchange while the pulse oximeter continues to display apparently acceptable saturation values. Such clinical situations may occur in early acute lung injury, pulmonary embolism, or when patients are receiving supplemental oxygen. Supplemental oxygen can further mask deterioration by maintaining saturation despite worsening shunt fraction or ventilation–perfusion mismatch. Interpreting SpO_2 as a standalone indicator of oxygenation may therefore be misleading, as comparable saturation measurements can conceal widely divergent PaO_2 levels and respiratory physiology.[4] Right-to-left intracardiac shunts represent another important physiological limitation. In patients with patent foramen ovale, atrial septal defect, ventricular septal defect, pulmonary hypertension, Eisenmenger physiology, or cyanotic congenital heart disease, systemic desaturation may result from admixture of venous and arterial blood rather than primary pulmonary parenchymal disease.[5] A single peripheral SpO_2 value may confirm desaturation but cannot localise the mechanism,

quantify shunt fraction, or distinguish pulmonary ventilation–perfusion mismatch from anatomical shunting.

Patients may develop respiratory distress despite apparently normal SpO_2 values. Clinicians must identify and manage respiratory distress before it progresses to respiratory failure, as waiting for desaturation may delay intervention.

A normal SpO_2 also does not necessarily indicate adequate oxygen delivery. Oxygen delivery depends on haemoglobin concentration, cardiac output, and arterial oxygen content. Patients with severe anaemia may have normal SpO_2 values despite critically reduced oxygen-carrying capacity.[6] Similarly, in septic shock, normal-range saturation may occur despite impaired oxygen extraction and mitochondrial-level oxygen utilization. Tissue hypoxia in such contexts may be better reflected by lactate levels, venous oxygen saturation, or near-infrared spectroscopy rather than pulse oximetry alone.[7]

Pulse oximetry also provides limited insight into regional or microcirculatory oxygenation. SpO_2 may remain normal despite impaired regional perfusion. In trauma or hemorrhagic shock, peripheral vasoconstriction may preserve central saturation readings while distal tissues experience ischemia. Thus, the reassuring pulse oximeter display may conceal evolving end-organ hypoxia.

Haemoglobin-related interferences

Certain toxicological and haematological conditions further expose the limitations of pulse oximetry. In carbon monoxide poisoning, normal or elevated SpO_2 values are misleading because carboxyhaemoglobin may be misinterpreted as oxyhaemoglobin.[8] Methaemoglobinemia similarly disrupts accuracy, often driving readings toward approximately 85% irrespective of true oxygenation.[9] Sulfhaemoglobinemia, although rare, deserves specific recognition because it usually presents with persistent cyanosis, low SpO_2 values, and a normal or near-normal PaO_2, and unlike methaemoglobinemia it may not respond to methylene blue therapy.[10] This clinical context is important because the discrepancy between cyanosis, pulse oximetry, and arterial oxygen tension may otherwise be misinterpreted as pulmonary or circulatory failure.

These scenarios highlight the need for co-oximetry when dyshaemoglobinemia is suspected. Conventional two-wavelength oximeters fail to distinguish multiple haemoglobin species reliably.[11,12]

Sickle cell disease is another haemoglobin-related context in which SpO_2 should be interpreted cautiously. Patients with sickle cell disease may have baseline hypoxaemia, altered oxygen–haemoglobin dissociation, and discrepancies between pulse oximetry and arterial oxygen saturation, particularly during acute chest syndrome, vaso-occlusive crisis, infection, or other acute presentations.[13] Therefore, treatment decisions in such patients should not rely only on a single SpO_2 threshold, and arterial blood gas analysis or co-oximetry may be required when clinical findings and SpO_2 values are discordant.

Technical artifacts and signal limitations

In low-perfusion states, hypothermia, vasoconstriction, and shock, readings may be unreliable because of impaired signal quality.[14] In cardiac arrest or profound hypotension, pulse oximetry may fail to display a reliable value or may lag behind rapid desaturation. Technological improvements, including motion-resistant algorithms, have reduced artifacts, yet clinical vigilance remains essential.[15] In unstable patients, repeated attempts to optimise probe placement should not delay airway, breathing, circulation assessment or resuscitative interventions; values obtained under poor signal conditions should be interpreted cautiously. Nail polish and artificial nails may also interfere with pulse oximetry, particularly when dark colours, gel-based manicures, or acrylic nails alter light transmission through the nail bed. Dark-coloured nail polish has been shown to reduce the ability to obtain accurate readings in some devices, and removal of nail polish or use of an alternate site should be considered when readings are inconsistent with the clinical picture.[16] Ambient light is another practical source of error, especially in prehospital, transport, emergency bay, operating room, or high-intensity lighting environments. Although modern devices incorporate light-rejection algorithms, strong ambient light or optical interference may still distort readings; covering the probe or using an alternative site may improve reliability.[17]

Demographic variability, skin pigmentation, and equity concerns

Beyond physiological and technical constraints, skin pigmentation-related bias has emerged as one of the most clinically and regulatorily significant limitations of pulse oximetry. The term "racial bias" should be interpreted cautiously, because self-reported race and ethnicity are imperfect surrogates for objectively measured skin pigmentation. The proposed mechanisms include greater optical absorption and scattering by melanin at the probe site, altered signal-to-noise ratio at low saturations, differences in probe design and device-specific calibration algorithms, and historical underrepresentation of individuals with darker skin tones in validation datasets.[18,19] Therefore, this limitation is not simply a biological issue, but also a device-design, calibration, validation, and health-equity concern.

The magnitude of this bias is clinically meaningful. Large observational studies have shown that pulse oximeters may overestimate oxygen saturation in individuals with darker skin pigmentation, leading to higher rates of occult hypoxaemia, commonly defined as arterial oxygen saturation below the clinically relevant threshold despite apparently reassuring SpO_2 values.[20] In COVID-19 cohorts, pulse oximetry overestimated arterial oxygen saturation among Asian, Black, and Hispanic patients compared with White patients, and occult hypoxaemia occurred more frequently in these groups.[21] Importantly, this bias is most consequential near decision thresholds, where small differences in SpO_2 may influence triage category, oxygen escalation, hospital admission, intensive care referral, eligibility for disease-specific therapies, or discharge decisions.

The clinical consequences extend beyond numerical inaccuracy. Overestimated SpO_2 may delay recognition of hypoxaemia, create false reassurance, reduce the likelihood of arterial blood gas confirmation, delay oxygen therapy or escalation of respiratory support, and contribute to under-triage or delayed treatment eligibility.[19,21] These consequences are particularly relevant in emergency and acute care settings, where decisions are often protocol-driven and time-sensitive. Regulatory agencies have therefore emphasised the need for more representative clinical testing, objective assessment of skin tone, improved device validation across the spectrum of pigmentation, and clearer labelling of device performance.[22]

Until more equitable devices and validation standards are universally available, practical interim strategies are necessary. Clinicians should avoid relying on a single SpO_2 value in isolation, especially when readings are borderline, discordant with clinical findings, or being

used to make major treatment decisions. In patients with darker skin pigmentation or any patient with unexplained dyspnoea, tachypnoea, increased work of breathing, altered mentation, shock, rising lactate, or clinical deterioration, clinicians should use trends rather than isolated values, correlate SpO_2 with respiratory rate and work of breathing, and have a lower threshold for arterial blood gas analysis or co-oximetry. Race-based correction factors should not be applied simplistically; instead, the emphasis should be on clinical correlation, confirmatory testing when needed, and awareness that a "normal" SpO_2 may still conceal clinically important hypoxaemia.

Ventilation, hypercapnia, and oxygen toxicity

Pulse oximetry does not monitor ventilation. Ventilation reflects gaseous movement in and out of the lungs as well as the work of breathing. Patients with neuromuscular weakness or opioid/sedative-induced respiratory depression may develop hypercapnia while maintaining normal SpO_2. This problem is further accentuated when supplemental oxygen is administered. Such ventilation–oxygenation dissociation may be concealed by a normal SpO_2 value, leading to delayed recognition of respiratory failure. Studies comparing capnography and pulse oximetry during procedural sedation demonstrate that capnography detects hypoventilation significantly earlier than SpO_2 monitoring alone.[23] Therefore, oxygen saturation should never substitute for direct assessment of ventilation.

Another overlooked condition is iatrogenic oxygen toxicity and hyperoxia. This occurs because SpO_2 becomes insensitive once haemoglobin is almost fully saturated; therefore, a reading of 98–100% may conceal a wide range of PaO_2 values, including potentially harmful supraphysiological arterial oxygen tensions. The physiological basis of hyperoxic injury includes increased generation of reactive oxygen species, oxidative cellular injury, absorption atelectasis, pulmonary vasoconstriction or worsening ventilation–perfusion relationships, systemic and coronary vasoconstriction, reduced cardiac output, and cerebral vasoconstriction.[24] In patients at risk of hypercapnic respiratory failure, such as chronic obstructive pulmonary disease, obesity hypoventilation syndrome, severe chest wall disease, or neuromuscular weakness, excessive oxygen may also worsen carbon dioxide retention through ventilation–perfusion mismatch and the Haldane effect.[25]

High-risk clinical contexts include acute coronary syndromes, acute ischemic stroke, post-cardiac arrest care, chronic obstructive pulmonary disease or other hypercapnic respiratory failure states, mechanically ventilated critically ill patients, shock states after initial stabilization, and patients receiving prolonged high-flow or high-concentration oxygen.[24-27] Because SpO_2 cannot distinguish normoxia from hyperoxia on the upper plateau of the oxygen–haemoglobin dissociation curve, oxygen should be treated as a drug and titrated to a documented target range rather than administered liberally. A practical approach is to target SpO_2 94–98% in most acutely ill adults and 88–92% in patients at risk of hypercapnic respiratory failure, with arterial blood gas assessment when precise oxygenation, PaO_2, $PaCO_2$, or acid–base status is clinically important.[25]

Disease-specific and threshold-driven limitations

The COVID-19 pandemic expanded pulse oximeter availability beyond hospitals into households. However, COVID-19 also revealed the limitations of SpO_2-based triage and respiratory assessment. Patients with severe hypoxaemia and marked lung involvement on chest CT frequently presented with minimal dyspnoea and near-normal saturation values, a phenomenon termed “silent hypoxaemia”.[28] Proposed explanations include preserved lung compliance, altered hypoxic ventilatory drive, and impaired pulmonary vascular regulation. Evidence suggests that even modest declines in SpO_2 may be associated with increased mortality risk, highlighting the limitations of binary, threshold-driven clinical decision-making.[29]

Cognitive and systems-level risks

The prominent display of SpO_2 values on monitors may also introduce cognitive and systems-level risks. In emergency and acute care environments, clinicians may give excessive weight to a reassuring numerical value, particularly under time pressure. A normal oxygen saturation may contribute to erroneous triage or delayed evaluation in dyspneic patients. Potentially lethal conditions such as pulmonary embolism, early acute respiratory distress syndrome, occult hypoventilation, dyshaemoglobinemia, or evolving shock may be missed if SpO_2 is

interpreted in isolation. Integration of clinical context, respiratory rate, work of breathing, mental status, perfusion markers, arterial blood gas analysis, lactate, and other laboratory findings remains essential.

None of these limitations diminishes the value of pulse oximetry as a screening and monitoring tool. Rather, they define the boundaries within which SpO_2 should be interpreted. Pulse oximetry measures haemoglobin saturation in pulsatile arterial blood; it does not measure oxygen delivery, ventilation, dyshemoglobinaemia, microcirculatory function, tissue oxygen utilization, or the clinical trajectory of respiratory distress. To translate these limitations into bedside practice, SpO_2 should be interpreted through a tiered and context-specific assessment framework rather than as an isolated numerical threshold. **Table 2** provides a practical approach for integrating SpO_2 with respiratory rate, work of breathing, mental status, capnography, lactate, arterial blood gas analysis, and clinical context in emergency and acute care settings. The principal risk is not technological limitation alone, but the false reassurance that a normal value may create. SpO_2 is therefore best understood as part of comprehensive clinical assessment, not its surrogate.

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Table 1. Clinical usefulness, advantages, and limitations of pulse oximetry in emergency and acute care.

Clinical condition/context	How pulse oximetry is useful	Key advantages	Important limitations/cautions
Initial triage of acutely ill or dyspnoeic patients	Provides rapid screening for hypoxemia and helps identify patients needing oxygen or urgent evaluation	Non-invasive, rapid, continuous, simple to use, useful for prioritising care	Normal SpO_2 does not exclude respiratory distress, pulmonary embolism, early ARDS, sepsis, anemia, shock, or hypercapnia
Monitoring during oxygen therapy	Helps assess response to supplemental oxygen and guides oxygen escalation or de-escalation	Allows continuous monitoring and trend-based assessment	Supplemental oxygen may mask deterioration; SpO_2 cannot distinguish adequate oxygenation from hyperoxia at high values
Acute respiratory infections, pneumonia, COVID-19, and ARDS	Helps identify hypoxemia and monitor progression or response to treatment	Useful for serial monitoring and early recognition of falling saturation	Silent hypoxemia and threshold-driven decisions may delay escalation if work of breathing and respiratory rate are ignored
Asthma or COPD exacerbation	Helps detect hypoxemia and guide controlled	Useful for bedside monitoring during bronchodilator	In COPD or hypercapnic respiratory failure risk, excessive oxygen may worsen CO_2 retention;

	oxygen administration	therapy and transport	ABG/VBG may be required
Procedural sedation, opioid exposure, or neuromuscular weakness	Can detect oxygen desaturation during monitoring	Useful as part of procedural safety monitoring	Does not detect early hypoventilation or hypercapnia, especially when supplemental oxygen is used; capnography is preferred when available
Shock, sepsis, trauma, or hemorrhage	Can document arterial saturation and monitor trends during resuscitation	Easy continuous monitoring during resuscitation and transfer	May remain normal despite impaired oxygen delivery, poor perfusion, elevated lactate, anemia, or microcirculatory failure
Severe anemia or bleeding	Confirms haemoglobin saturation of available red cells	May remain technically normal despite severe anemia	Does not measure oxygen content or oxygen-carrying capacity; hemoglobin and perfusion status must be assessed
Carbon monoxide poisoning, methemoglobinemia, sulfhemoglobinemia, or suspected dyshemoglobinemia	May raise suspicion when SpO_2 is discordant with clinical appearance or ABG PaO_2	Useful as an initial clue when readings are unexpected	Conventional two-wavelength oximeters cannot reliably distinguish dyshemoglobins; co-oximetry is required
Post-cardiac arrest, acute coronary	Helps avoid hypoxemia and	Continuous monitoring is useful during	High SpO_2 values may conceal hyperoxia; oxygen should be titrated

syndrome, and acute stroke	monitor oxygen therapy	stabilization and transfer	to target range and ABG used when precise PaO_2 is needed
Prehospital care and patient transport	Allows rapid monitoring outside controlled hospital settings	Portable, low-cost, useful during ambulance transfer and field triage	Motion, vibration, ambient light, cold extremities, poor perfusion, nail polish, and probe displacement may produce unreliable readings
Pediatric, elderly, or non-communicative patients	Provides objective continuous oxygenation data when symptoms may be difficult to elicit	Helpful in patients unable to report dyspnoea reliably	Must still be interpreted with respiratory rate, work of breathing, mental status, perfusion, and clinical trajectory
Patients with darker skin pigmentation or borderline readings	Provides useful trend data when interpreted cautiously	Still valuable for serial monitoring when waveform and trends are assessed	May overestimate oxygen saturation and conceal occult hypoxemia; lower threshold for ABG/co-oximetry when discordant

Table 2. Context-based framework for interpreting SpO_2 in emergency and acute care.

Clinical context	**Why SpO_2 may mislead**	**Additional bedside/laboratory assessment**	**Practical action**
Stable patient with mild symptoms and reliable waveform	SpO_2 may appear reassuring despite early disease or supplemental oxygen use	Respiratory rate, work of breathing, mental status, perfusion, serial SpO_2 trend	Use SpO_2 as one vital sign; repeat assessment and document trend rather than relying on a single value
Dyspnoea with normal or near-normal SpO_2	Early respiratory distress, pulmonary embolism, early ARDS, silent hypoxemia, or compensated disease may be concealed	Respiratory rate, accessory muscle use, ability to speak, auscultation, chest imaging when indicated	Do not delay evaluation only because SpO_2 is normal; escalate assessment if clinical distress is present
Increased work of breathing or tachypnoea despite acceptable SpO_2	SpO_2 reflects oxygenation but not ventilatory effort or impending fatigue	Respiratory rate, work of breathing, mental status, serial examination, ABG/VBG when needed	Treat as possible respiratory distress; consider early senior review and escalation before desaturation occurs
Opioid/sedative exposure, procedural sedation, neuromuscular weakness, or obesity hypoventilation	Hypoventilation and hypercapnia may occur while SpO_2 remains normal, especially	Capnography, respiratory rate, depth of breathing, mental status, ABG/VBG for $PaCO_2$ and pH	Use capnography where available; do not use SpO_2 alone to exclude respiratory depression

	with supplemental oxygen		
COPD or other risk of hypercapnic respiratory failure	Excess oxygen may worsen CO_2 retention despite acceptable or high SpO_2	ABG/VBG, $PaCO_2$, pH, respiratory rate, mental status	Titrate oxygen to 88–92% unless otherwise indicated; obtain blood gas if drowsiness, acidosis, or deterioration occurs
Shock, sepsis, trauma, or regional hypoperfusion	SpO_2 may remain normal despite poor oxygen delivery, microcirculatory failure, or impaired oxygen extraction	Lactate, capillary refill, skin temperature, urine output, blood pressure, hemoglobin, venous saturation where available	Assess oxygen delivery and perfusion, not saturation alone; manage shock and repeat lactate/ABG as clinically indicated
Severe anemia or hemorrhage	SpO_2 may be normal despite critically reduced oxygen content	Hemoglobin, hemodynamics, perfusion markers, lactate, clinical bleeding assessment	Recognize that normal SpO_2 does not exclude inadequate oxygen delivery; correct the underlying oxygen-carrying deficit
Suspected carbon monoxide poisoning, methemoglobinemia, sulfhemoglobinemia, or sickle cell-related crisis	Conventional pulse oximeters may misread dyshemoglobins or fail to reflect true arterial oxygenation	Co-oximetry, ABG with co-oximetry, clinical history, cyanosis, exposure history	Do not rely on standard SpO_2; confirm with co-oximetry when dyshemoglobinemia is suspected

Borderline SpO_2 in patients with darker skin pigmentation or discordant clinical findings	Device bias may overestimate oxygen saturation and conceal occult hypoxemia	SpO_2 trend, respiratory assessment, ABG/co-oximetry when values are borderline or discordant	Maintain a lower threshold for confirmatory testing and escalation when clinical findings do not match SpO_2
Post-cardiac arrest, acute coronary syndrome, acute stroke, or prolonged high-concentration oxygen therapy	SpO_2 of 98–100% cannot distinguish normoxia from hyperoxia	ABG for PaO_2, oxygen delivery device settings, serial reassessment	Treat oxygen as a drug; titrate to a documented target range and avoid unnecessary hyperoxia

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