Physiological Monitoring for Procedural Sedation: The Routine and Beyond

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Introduction

Physiological monitoring of vital signs is essential for the safe practice of procedural sedation and analgesia. Oxygenation, ventilation, cortical activity, and hemodynamics can all be monitored noninvasively in spontaneously breathing patients. This chapter discusses the current guidelines and standards for patient monitoring and the essential monitoring modalities for procedural sedation and analgesia in children.

Current Guidelines and Standards

There are numerous procedural sedation and analgesia guidelines that have been created by specialty societies to standardize procedural sedation and analgesia practice in order to optimize patient safety (Table 5.1) [1]. The most widely disseminated guidelines are from the American Academy of Pediatrics [2], the American Society of Anesthesiologists [3], and the American College of Emergency Physicians [4]. In the early 1990, the Joint Commission took a special interest in procedural sedation and analgesia, and in 2001 released standards for pain management, sedation, and anesthesia care, with the central theme that sedation care should be comparable throughout a given hospital [5]. Patients sedated in settings outside the operating room should not receive a significantly different level of attention or monitoring than those sedated for a similar procedure in the operating room. To ensure this, the Joint Commission requires specific procedural sedation and analgesia protocols that apply consistently throughout each institution. These hospital-wide sedation policies vary from site to site based upon the specific needs and resources available within each institution.

At each hospital accreditation survey, the Joint Commission will evaluate whether clinicians practice procedural sedation and analgesia consistent with their hospital-wide sedation policy, and whether they provide sufficient documentation for such compliance. Physicians must be familiar with their hospital's sedation policies, and should work with their medical staff to ensure that such policies are suitably detailed. Most hospitals pattern their sedation policies after the Joint Commission standards and definitions.

The Joint Commission requires that practitioners who are permitted to administer deep sedation must be qualified to rescue patients from general anesthesia. Moderate sedation suffices for the majority of procedures in cooperative children, although it will not be adequate for extremely painful procedures, or in uncooperative patients. Deep sedation can facilitate these, but at greater risk of cardiorespiratory depression than moderate sedation [3, 5] (Table 5.2).

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Table 5.1 Specialty societies with published sedation guidelines

American Academy of Pediatrics
American Academy of Pediatric Dentistry
American Academy of Periodontology
American Association of Critical-Care Nurses
American College of Critical Care Medicine
American College of Emergency Physicians
American Nurses Association
American Society for Gastrointestinal Endoscopy
American Society of Anesthesiologists
American Society of Plastic and Reconstructive Surgeons
Association of Operating Room Nurses
Emergency Nurses Association
Joint Commission on Accreditation of Healthcare Organizations
National Institutes of Health
Society of Gastroenterology Nurses and Associates
Society of Nuclear Medicine

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Table 5.2 Levels of sedation

Minimal sedation (anxiolysis) [7]: A drug-induced state during which patients respond normally to verbal commands. Although cognitive function and coordination may be impaired, ventilatory and cardiovascular functions are unaffected.
Moderate sedation (formerly "conscious sedation") [7]: A drug-induced depression of consciousness during which patients respond purposefully to verbal commands, either alone or accompanied by light tactile stimulation. Reflex withdrawal from a painful stimulus is not considered a purposeful response. No interventions are required to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained.
Dissociative sedation [50, 51]: A trance-like cataleptic state induced by the dissociative agent ketamine characterized by profound analgesia and amnesia, with retention of protective airway reflexes, spontaneous respirations, and cardiopulmonary stability.
Deep sedation [7]: A drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully following repeated or painful stimulation. The ability to independently maintain ventilatory function may be impaired. Patients may require assistance in maintaining a patent airway and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained.
General anesthesia [7]: A drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation. The ability to independently maintain ventilatory function is often impaired. Patients often require assistance in maintaining a patent airway, and positive pressure ventilation may be required because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function. Cardiovascular function may be impaired.

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Observational Monitoring

Physiological monitoring has two components: observational monitoring by a designated clinician and electronic monitoring with mechanical monitoring devices. The most important element of procedural sedation and analgesia monitoring is close and continuous patient observation by an individual capable of recognizing adverse events. This person must be able to continuously observe the patient's face, mouth, and chest wall motion, and equipment or sterile drapes must not interfere with such visualization. This careful observation will allow prompt detection of adverse events such as respiratory depression, apnea, airway obstruction, emesis, and hypersalivation [6]. An individual with advanced life-support skills should be immediately available in all settings where deep sedation is performed.

During deep sedation, the individual dedicated to patient monitoring should be experienced with this depth of sedation and have no other responsibilities that would interfere with the required advanced level of monitoring and documentation. Individual hospital-wide sedation policies may have additional requirements for how and when deep sedation is administered based on their specific needs and available resources.

Vital signs should be measured at individualized intervals including at baseline, after drug administration, on completion of the procedure, during early recovery, and at completion of recovery. During deep sedation, vitals signs should be assessed every 5 min. In addition to recording vital signs at set intervals, clinicians must be especially vigilant during key phases of the sedation. Patients are usually at highest risk of complications 5–10 min following administration of IV medications and during the immediate post-procedure period when external stimuli are discontinued.

Electronic Monitoring

The use of electronic monitoring has greatly enhanced the safety of procedural sedation and analgesia. Continuous oxygenation (pulse oximetry with an audible signal), ventilation (capnography), and hemodynamics (blood pressure and electrocardiogram (ECG)) can all be monitored noninvasively in spontaneously breathing patients.

Oxygenation Monitoring

Pulse oximetry is the noninvasive measurement of the percent of hemoglobin bound to oxygen providing a continuous means of estimating in real-time the arterial oxygen saturation. The underlying principles of oximetry were developed in 1932 based on the Beer–Lambert law (the concentration of an unknown solute dissolved in a solvent can be determined by light absorption). Modern pulse oximetry technology, using optical plethysmography and spectrophotometry, was invented in 1974 and completed in 1980 with the addition of a probe and a miniaturized computer in the monitor [7]. The probe, consisting of red and infrared (IR) light sources and a photoelectric detector, is positioned across a pulsatile vascular bed such as the finger, the foot, or ear lobe [7, 8].

The most common type of oximetry (i.e., transmission oximetry) places the light sources on one side of the tissue bed and the photodetector on the opposite side. The pulsatile variation of the emitted red and IR light transmitted through the tissue bed is accessed by the oximeter which divides the signal into an arterial blood pulsatile component and a nonpulsatile component (venous and capillary blood). Data averaged over several arterial pulse cycles are represented as the oxygen saturation (SpO_2) [7–9]. There is a tight correlation between the arterial hemoglobin oxygen saturation (PaO_2) and the SpO_2 in a nonlinear fashion as described by the oxyhemoglobin dissociation curve (Fig. 5.1) [8–10]. The shape of the curve has important clinical implications. In the hypoxic patient, small changes in SpO_2 on the steep part of the curve result in large changes in the PaO_2, while SpO_2 values at high levels of

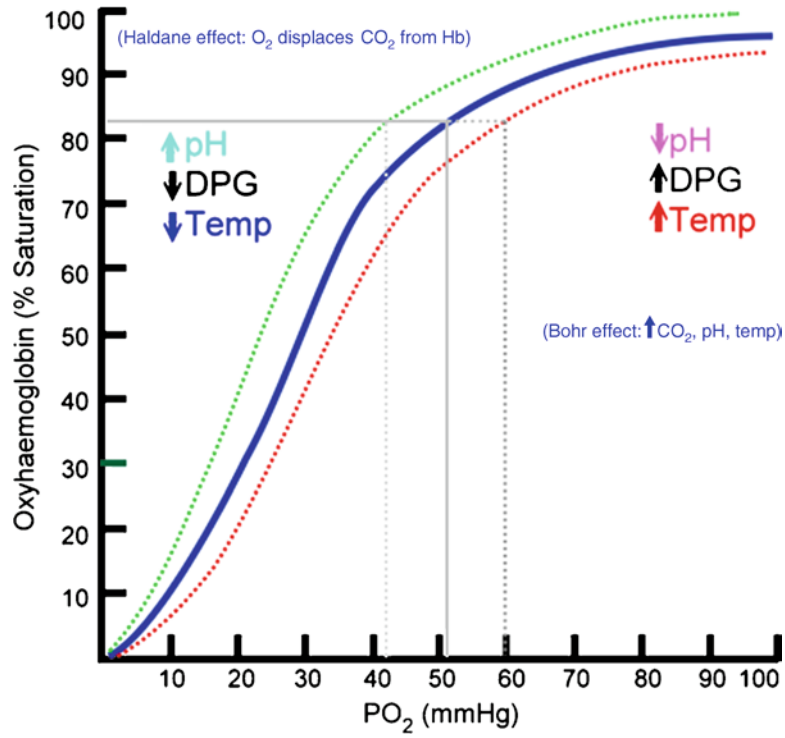


Fig. 5.1 Oxyhemoglobin dissociation curve

oxygenation (on the plateau of the curve) are relatively insensitive at detecting significant changes in PaO_2.

Patients with normal lung function and adequate gas exchange have an SpO_2 between 97 and 100%. Pulse oximeters are accurate for saturations >70% [10]. When SaO_2 falls below 95%, hypoxia may be present, although patients with obstructive lung disease may live in this range [8, 9]. Oxygen saturations below 90% represent significant hypoxia. At 75% saturation, oximetry bias is uniformly scattered (7% underestimation and 7% overestimation).

The finger is the most common probe site used for pulse oximetry. If the finger is inaccessible or unsuitable, other probe sites, such as the ear lobe or the bridge of the nose, may be used. In neonates and infants, probes sites include the great toe, the heel, the sole, and the lateral aspect of the foot.

There are a number of important limitations to the accuracy of pulse oximetry: poor perfusion secondary to severe vasoconstriction (e.g., low perfusion states, shock, hypothermia), artifact from excessive patient motion, severe anemia, high-intensity ambient light, abnormal hemoglobins, venous pulsations, synthetic fingernails and nail polish, or intravenous dyes [8, 10]. Recent advances in motion control technology have made pulse oximetry more reliable during patient motion. Carboxyhemoglobin (COHb) and methemoglobin (MetHb) contribute to light absorption and cause errors in saturation readings. The oximeter sees COHb as though it were mostly OxyHb and gives a false high reading. In the presence of high levels of MetHb, the SpO_2 is erroneously low when the arterial saturation is above 85% and erroneously high when the arterial saturation is below 85%. MetHb produces a large pulsatile absorbance signal at both the red and IR wavelengths. This forces the absorbance ratio toward unity, which corresponds to a SpO_2 of 85%. Further, in dark-skinned patients, false high readings and a higher incidence of failure of signal detection have been reported [8–10].

Pulse oximetry is not a substitute for ventilation monitoring, as there is a lag time, the extent of the lag depending on the age and physical status of the patient, between the onset of hypoventilation or apnea and a change in oxygen saturation. Therefore, during procedural sedation, ventilation monitoring should always accompany oxygenation monitoring. Hypoventilation and resultant hypercapnia may precede a decrease in hemoglobin O_2 saturation by minutes [11]. Further, supplemental O_2 may mask hypoventilation by delaying the eventual O_2 desaturation for which pulse oximetry monitoring is designed to recognize [12].

Ventilation Monitoring

Capnography is the noninvasive measurement of the partial pressure of carbon dioxide in exhaled breath represented as a numerical value (end-tidal CO_2) and a waveform. The CO_2 waveform or capnogram represents changes in the CO_2 concentration over the time of one respiratory cycle (Fig 5.2) [13]. Changes in the shape of the waveform are diagnostic of disease conditions, while changes in end-tidal CO_2 ($EtCO_2$ – the maximum CO_2 concentration at the end of each tidal breath) can be used to assess disease severity and response to treatment [14].

Modern capnography was developed in the 1940s and commercialized in the 1960s and 1970s with the development of mass spectroscopy. Capnography became a routine part of anesthesia practice in Europe in the 1970s and in the United States in the 1980s [13]. Most capnography technology is built on infrared (IR) radiation techniques and based on the fact that CO_2 molecules absorb IR radiation at a specific wavelength, with the amount of radiation absorbed having a close to exponential relation to the CO_2 concentration

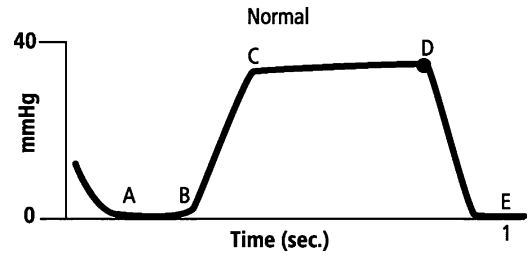


Fig. 5.2 Normal CO_2 waveform

present in the breath sample. Detecting changes in IR radiation levels with photodetectors allows for the calculation of the CO_2 concentration in the gas sample.

Carbon dioxide monitors measure gas concentration or partial pressure using one of two configurations: mainstream or sidestream. Mainstream devices measure CO_2 directly from the airway, with the sensor located on the endotracheal tube. Sidestream devices measure CO_2 by aspirating a small sample from the exhaled breath through tubing to a sensor located inside the monitor. Mainstream systems, as the sensor is located on the endotracheal tube, are configured for intubated patients. Sidestream systems, as the sensor is located inside the monitor, are configured for both intubated and non-intubated patients. The airway interface for intubated patients is an airway adapter placed on the hub of the endotracheal tube; and for spontaneously breathing patients, an nasal-oral cannula which allows concomitant CO_2 sampling and low-flow oxygen delivery.

Sidestream systems can be either high flow (with 150 cc/min as the amount of CO_2 in the breath sample required to obtain an accurate reading) or low flow (50 cc/min). Low-flow sidestream systems have a lower occlusion rate (from moisture or patient secretions) and are more accurate in patients with low tidal volumes (neonates, infants, and patients with hypoventilation and low tidal volume breathing) [15]. In high-flow systems, when the tidal volume of the patient drops below 150 cc (i.e., the flow rate of the system), the monitor will entrain room air to compensate, falsely diluting the $EtCO_2$ [16–18].

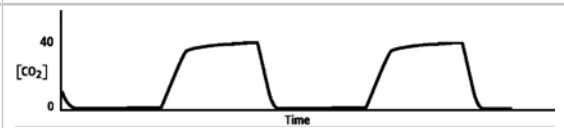
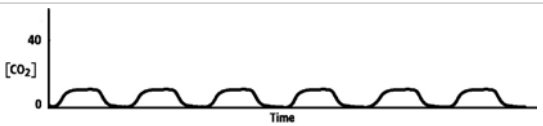
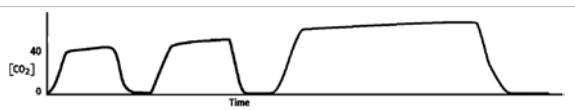
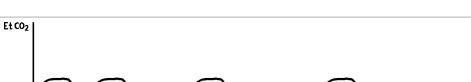
The CO_2 waveform, corresponding to a single breath, consists of four phases [2, 15]. Phase 1 (dead space ventilation, A–B) represents the beginning of exhalation where the dead space is cleared from the upper airway. Phase 2 (ascending phase, B–C) represents the rapid rise in CO_2 concentration in the breath stream as the CO_2 from the alveoli reaches the upper airway. Phase 3 (alveolar plateau, C–D) represents the CO_2 concentration reaching a uniform level in the entire breath stream and concludes with a point of maximum CO_2 concentration ($EtCO_2$). Phase 4 (D–E) represents the inspiratory cycle where the CO_2 concentration drops to zero as atmospheric air enters the airway (Fig. 5.2). A normal waveform is characterized by four distinct phases, a CO_2 concentration that starts at zero and returns to zero (i.e., there is no rebreathing of CO_2), and a maximum CO_2 concentration reached with each breath (i.e., $EtCO_2$).

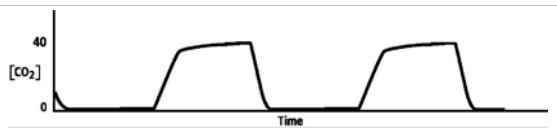
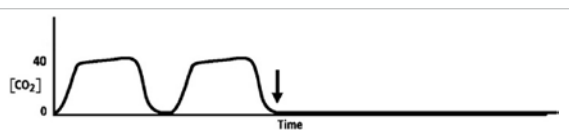
Patients with normal lung function have a characteristic rectangular shaped waveform and a narrow $EtCO_2$–pCO_2 gradient (0–5 mmHg), with the $EtCO_2$ accurately reflecting the $PaCO_2$ [14, 19]. Patients with obstructive lung disease will have a more rounded ascending phase and an upward slope in the alveolar plateau (Table 5.3) [20]. In patients with abnormal lung function secondary to ventilation–perfusion (V–Q) mismatch, the gradient will widen, depending on the severity of the lung disease [21–23].

The shape of the waveform is affected by the $EtCO_2$ and the expiratory time. The amplitude of the waveform is determined by the $EtCO_2$ value and the width is determined by the expiratory time. Hyperventilation (increased respiratory rate, decreased $EtCO_2$) results in a low amplitude and narrow waveform, while classical hypoventilation (decreased respiratory rate, increased $EtCO_2$) results in a high amplitude and wide waveform (Table 5.3). Acute bronchospasm results in a waveform with a curved ascending phase and upsloping alveolar plateau (Table 5.3). An $EtCO_2$ >70 mm Hg, in patients without chronic hypoventilation, indicates respiratory failure.

Capnography provides a continuous, breath-by-breath measure of respiratory rate and CO_2 exchange and can detect the common adverse airway and respiratory events associated with procedural sedation and analgesia [24]. Capnography is the earliest indicator of airway or respiratory compromise and will manifest an abnormally high or low $EtCO_2$ well before pulse oximetry detects a falling oxyhemoglobin saturation, especially in patients receiving supplemental oxygen. Early detection of respiratory compromise is especially important in infants and toddlers who have smaller functional residual capacity and greater oxygen consumption relative to older children and adults. Capnography provides a non-impedance respiratory rate directly

Table 5.3 Capnographic airway assessment for procedural sedation and analgesia

Diagnosis	Waveform	Features		Intervention
Normal		SpO_2 $EtCO_2$ Waveform RR	Normal Normal Normal Normal	No intervention required Continue sedation
Hyperventilation		SpO_2 $EtCO_2$ Waveform RR	Normal ↓ Decreased amplitude and width ↑	
Bradypneic hypoventilation (Type 1)		SpO_2 $EtCO_2$ Waveform RR	Normal ↑ Increased amplitude and width ↓↓↓	Reassess patient Continue sedation
		SpO_2 $EtCO_2$ Waveform RR	↓ ↑ Increased amplitude and width ↓↓↓	Reassess patient Assess for airway obstruction Supplemental oxygen Cease drug administration or reduce dosing
Hypopneic hypoventilation (Type 2)		SpO_2 $EtCO_2$ Waveform RR	Normal ↓ Decreased amplitude ↓	Reassess patient Continue sedation
		SpO_2 $EtCO_2$ Waveform RR	↓ ↓ Decreased amplitude ↓	Reassess patient Assess for airway obstruction Supplemental oxygen Cease drug administration or reduce dosing
Hypopneic hypoventilation with periodic breathing		SpO_2 $EtCO_2$ Waveform RR Other	Normal or ↓ ↓ Decreased Amplitude ↓ Apneic pauses	

Physiological variability		SpO_2 $EtCO_2$ Waveform RR	Normal Normal Varying[a] Normal	No intervention required Continue sedation	
Bronchospasm		SpO_2 $EtCO_2$ Waveform RR Other	Normal or ↓ Normal, ↑, or ↓[b] Curved Normal, ↑ or ↓[b] Wheezing	Reassess patient Bronchodilator therapy Cease drug administration	
Partial airway obstruction Partial laryngospasm		SpO_2 $EtCO_2$ Waveform RR Other	Normal or ↓ Normal Normal Variable Noisy breathing and/or inspiratory stridor	Full airway patency restored with airway alignment Noisy breathing and stridor resolve	Reassess patient Establish IV access Supplemental O_2 (as needed) Cease drug administration
				Airway not fully patent with airway alignment Noisy breathing and stridor persist	
Apnea		SpO_2 $EtCO_2$ Waveform RR Other	Normal or ↓[c] Zero Absent Zero No chest wall movement or breath sounds	Reassess patient Stimulation Bag mask ventilation Reversal agents (where appropriate) Cease drug administration	
Complete airway obstruction Complete laryngospasm		SpO_2 $EtCO_2$ Waveform RR Other	Normal or ↓[c] Zero Absent Zero Chest wall movement and breath sounds present	Airway patency restored with airway alignment Waveform present	
				Airway not patent with airway alignment No waveform	Positive pressure ventilation

Source: From Krauss and Hess [24]

[a]Varying waveform amplitude and width

[b]Depending on duration and severity of bronchospasm

[c]Depending on duration of episode

from the airway (via oral-nasal cannula) that is more accurate than impedance-based respiratory monitoring. In patients with obstructive apnea, impedance-based monitoring will interpret chest wall movement without ventilation as a valid breath.

Both central and obstructive apnea can be rapidly detected by capnography (Table 5.3). Loss of the waveform, in conjunction with no chest wall movement and no breath sounds confirms the diagnosis of central apnea. Obstructive apnea is characterized by loss of the waveform, chest wall movement, and absent breath sounds. The absence of the waveform in association with the presence or absence of chest wall movement distinguishes apnea from upper airway obstruction and laryngospasm. Response to airway alignment maneuvers can further distinguish upper airway obstruction from laryngospasm.

There are two types of drug-induced hypoventilation that occur during procedural sedation and analgesia (Table 5.3) [24]. Bradypneic hypoventilation, commonly seen with opioids, is characterized by an increased $EtCO_2$ and an increased $PaCO_2$. Respiratory rate is depressed proportionally greater than tidal volume resulting in bradypnea, an increase in expiratory time, and a rise in $EtCO_2$, graphically represented by a high amplitude and wide waveform (Table 5.3). Bradypneic hypoventilation follows a predictable course with $EtCO_2$ increasing progressively until respiratory failure and apnea occur. Although there is no absolute threshold at which apnea occurs, patients without chronic hypoventilation with $EtCO_2$ >70 mmHg are at significant risk.

Hypopneic hypoventilation, commonly seen with sedative-hypnotic drugs, is characterized by a normal or decreased $EtCO_2$ and an increased $PaCO_2$ as airway dead space remains constant and tidal volume is decreasing (Table 5.3). Tidal volume is depressed proportionally greater than respiratory rate, resulting in low tidal volume breathing that leads to an increase in airway dead space fraction (dead space volume/tidal volume). As tidal volume decreases, airway dead space fraction increases which in turn results in an increase in the $PaCO_2$–$EtCO_2$ gradient. Even though $PaCO_2$ is increasing, $EtCO_2$ may remain normal or be decreasing, graphically represented by a low amplitude waveform (Table 5.3). Hypopneic hypoventilation follows a variable course and may remain stable with low tidal volume breathing resolving over time as CNS drug levels decrease and redistribution to the periphery occurs, progress to periodic breathing with intermittent apneic pauses (which may resolve spontaneously or progress to central apnea), or progresses directly to central apnea.

The low tidal volume breathing that characterizes hypopneic hypoventilation increases dead space ventilation when normal compensatory mechanisms are inhibited by drug effects. Minute ventilation, which normally increases to compensate for an increase in dead space, does not change or may decrease [25]. As minute ventilation decreases, PaO_2 decreases. If minute ventilation decreases further, oxygenation is further impaired [26, 27]. However, $EtCO_2$ may initially be high (bradypneic hypoventilation) or low (hypopneic hypoventilation) without significant changes in oxygenation, particularly if supplemental oxygen is given. Therefore, a drug-induced increase or decrease in $EtCO_2$ does not necessarily lead to oxygen desaturation and may not require intervention.

Technical problems with capnography have limited its effectiveness and restricted its clinical applications. These problems include: interference with the sensor by condensed water and patient secretions, cross sensitivity with anesthetic gases in conventional CO_2 sensors, lack of ruggedness for intra- and interhospital transport, and power consumption issues related to portable battery operation time. These issues have been resolved in the newer generation capnography monitors. Early capnography airway interfaces (i.e., nasal cannula) had difficulty providing consistent measurements in mouth breathing patients and patients who alternated between mouth and nose breathing. The newer oral-nasal interfaces do not have these problems.

Hemodynamic Monitoring

Noninvasive blood pressure (NIBP) measurement is an automated method of repetitively determining blood pressure that is accurate in

both adults and children. Blood pressure can be obtained manually (only when the operator pushes a button) or automatically cycled at preset intervals with the cuff inflated to specific levels. NIBP provides a display of the heart rate, systolic, diastolic, and mean blood pressures by electronically determining the pulse amplitude. During deflation, the cuff determines the amplitude of the pulsations transmitted by movement of arterial wall under the cuff. A sudden rise in the magnitude of the pulsations accompanies the artery opening and represents the systolic pressure. The magnitude of the pulsations increases to a peak and then falls rapidly. The diastolic pressure is determined at the point where there are no further alterations in the magnitude of the pulsations. The accuracy of NIBP depends on utilizing the correct cuff size (especially important in children and obese patients) and on minimizing patient motion during measurement.

Continuous ECG monitoring is useful for the rapid detection of rhythm disturbances or ischemia. Continuous ECG monitoring for procedural sedation and analgesia is neither mandatory nor standard of care in patients without a cardiovascular disease. However, such monitoring is simple, inexpensive, and readily available and is frequently used during procedural sedation and analgesia in children.

Depth of Sedation Monitoring

Monitoring modalities that measure the brain's response to anesthetic agents have recently been studied for use in procedural sedation and analgesia [28–30]. Although these technologies have been used to monitor depth of sedation/anesthesia in the operating room, in 2006 the American Society of Anesthesiologists concluded that the clinical applicability in the operating room "has not been established" [31]. Further, the predictive value of this type of monitoring for the moderate and deep sedation outside the operating room remains unclear.

The most studied of these technologies is the bispectral index (BIS), that uses a processed electroencephalogram (EEG) signal to quantify sedation depth. A BIS value of 100 is considered complete alertness, a range of 40–60 consistent with general anesthesia and zero is no cortical activity [32].

Several studies have shown a reasonable correlation between BIS and standard observational sedation score in children older than 6 months (i.e., University of Michigan Sedation Scale (UMSS), Observer's Assessment of Alertness/Sedation (OAA/S), Ramsey Score) for commonly used sedatives such as midazolam, pentobarbital, chloral hydrate, and propofol. However, other studies have failed to consistently validate a tight correlation between BIS values and specific levels of sedation as measured by standard observational sedation scores.

A 2007 study of 248 children (1 month to 18 years), using pooled raw data from four independently conducted studies, found a moderate correlation between BIS and UMSS with the use of chloral hydrate, pentobarbital, propofol, and midazolam, but poor correlation with ketamine and with opioids. Bispectral index values were significantly lower for a same observed level of sedation with propofol and pentobarbital when compared to midazolam and chloral hydrate, making BIS an unreliable method of reaching a desired level of sedation [33]. The poor correlation observed with opioids is thought to be secondary to opioids providing sedation without hypnosis [33, 34]. Hence, it has been argued that BIS reflects cortical activity rather than level of consciousness [35].

Overly et al, in a study of 47 patients treated either with ketamine/midazolam, methohexital, propofol, or midazolam and a narcotic found a good correlation between BIS and OAA/S scale for non-dissociative agents, but not with ketamine [36]. Ketamine sedation, in multiple studies, has shown an unreliable correlation between BIS and standard sedation scoring, with persistence of high BIS or even an increase in BIS despite achieving deeper levels of sedation [33, 34, 36].

Dexmedetomidine, a selective alpha-2 adrenergic agonist that provides sedation without respiratory depression, has shown to correlate well with standard observational sedation scores. In a study of 11 mechanically ventilated children in an intensive care unit setting sedated with dexmedetomidine, significant correlations between

Richmond agitation sedation scale and BIS values were found [37].

A 2009 crossover study of nine adult volunteers receiving propofol or dexmedetomidine followed by the alternate drug 7 days later also showed good correlation between BIS and OAA/S. However, for a same OAA/S score, BIS values were significantly lower in patients sedated with dexmedetomidine suggesting that the BIS score is drug-specific with different scores signifying different levels of sedation for different sedation agents [38].

Bispectral index scores in infants less than 6 months of age have been noted to be unreliable during general anesthesia and procedural sedation, likely secondary to the fact that the BIS algorithm was developed using adult EEG data [34, 39].

In summary, procedural sedation studies using BIS monitoring have found unacceptably wide ranges of BIS values at various depths of sedation that did not correlate with standard sedation scores (e.g., Ramsey Score) [28–30]. As BIS does not reliably gauge depth in individual patients, it cannot currently be recommended for use in procedural sedation and analgesia.

Cerebral Oximetry

Another new technology with potential application to procedural sedation is cerebral oximetry. Through near-infrared spectroscopy, cerebral tissue oxygenation (i.e., regional oxygen saturation, rSO_2) is measured by monitoring the nonpulsatile signal component reflecting tissue circulation of arterioles, capillaries, and venules. Unlike conventional pulse oximetry, which monitors the pulsatile signal component reflecting arterial circulation, cerebral oximetry is reliable in low perfusion states, shock, and cardiac arrest. Cerebral oximetry represents a "weighted average" of the tissue circulation and reflects a potentially more accurate measurement of oxygen consumption, similar to and correlating with mixed venous saturations [40, 41].

Cerebral oximetry has been primarily studied in the operating room, except for a recent ED procedural sedation study, which demonstrated poor correlation between cerebral oximetry, pulse oximetry, and capnography [42]. In this study, 100 children of ages 9 months to 18 years were sedated with various agents (ketamine, fentanyl, pentobarbital, dexmedetomidine, or propofol). Changes in rSO_2 occurred in 2.1% of patients and were associated with changes in SpO_2 23% of the time and changes in end-tidal CO_2 29% of the time. Only a minority of hypoxic episodes resulted in a decrease in rSO_2, while the majority of changes in rSO_2 occurred in the absence of changes in cardiorespiratory parameters.

Although rSO_2 appears to be a more sensitive measure of cerebral oxygenation than pulse oximetry, isolated decreases in rSO_2 do not appear to correlate well with short or long-term neurological outcome, as illustrated in a small study of adult patients undergoing carotid endarterectomy. Importantly, there is no clear rSO_2 threshold under which clinically significant brain hypoxia occurs [43].

Noninvasive Cardiovascular Monitoring

Methods for advanced noninvasive cardiovascular monitoring continue to be refined. Through thoracic electrical bioimpedance, and similar to impedance cardiography, electrical cardiometry (or electrical velocimetry) enables the measurement of various cardiac parameters including cardiac output, cardiac index, stroke volume, systemic vascular resistance, and index of contractility. Such methods rely on the interpretation of a signal from sensors placed on the neck and chest, which quantify changes in conductivity of the blood in the aorta during the cardiac cycle [44–46].

Electrical velocimetry measurements have been shown to correlate with measurements derived from the Fick principle applied to blood sampled invasively in pediatric patients with congenital heart disease undergoing left heart catheterization [47], and to transesophageal echocardiography in ventilated children following cardiac surgery – although electrical velocimetry appeared to underestimate cardiac output

in terms of absolute values [48]. Impedance cardiography has shown good correlation with standard pulmonary artery thermodilution methods during cardiac surgery [49]. At present, the applicability and clinical relevance of advanced noninvasive cardiovascular monitoring to pediatric procedural sedation remains unclear.

Summary

There have been significant advances in noninvasive physiological monitoring of ventilation, oxygenation, and hemodynamics for procedural sedation in children with the advent of improved motion control for pulse oximetry, low-flow capnography systems, the potential of regional cerebral oximetry, and entropy depth of sedation monitoring. These systems bring enhanced safety and efficiency to pediatric procedural sedation.

Future directions in pediatric procedural sedation will include easier methods to integrate the expanding physiological monitoring data now available to the clinician (sophisticated methods for data display, interpretive algorithms, composite indices based on integration of physiological parameters), and new noninvasive technology to monitor blood pressure, vascular tone, cardiac output, cerebral activity, and oxygenation.

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