

Pediatric Sedation Pearls

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The practice of procedural sedation and analgesia in pediatrics is evolving rapidly. Some of our practices are backed by substantial supporting data, whereas for others, the evidence base is less well developed, leading to substantial differences in provider preferences. In addition, the multidisciplinary nature of providing sedation and differences in local custom and regulatory requirements all contribute to a certain amount of practice variability: the art of sedation. To provide a forum for exploring this, we invited experts in pediatric procedural sedation to share their practices and pearls, combining anecdotal experience with evidence from the literature.

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Pre-sedation Airway Assessment

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Procedural sedation and analgesia (PSA) has become common practice in pediatric emergency departments. However, some studies have shown that up to 17% of pediatric procedural sedations have some type of complication [1]. Most of these are respiratory events, and 1 in 200 require interventions to maintain a patent airway and appropriate ventilation of the patient [2]. Inadequate pre-sedation evaluation has been shown to be a common contributing factor to adverse events [3]. Therefore, a systematic approach to assessing a child before PSA is essential.

One key component of evaluation before PSA involves a focused assessment of the airway. This serves 2 purposes. The first is to help identify patients at higher risk for sedation-related complications (eg, a patient witnessed to have obstructive symptoms when lying supine before sedation is likely to become more obstructed with further airway relaxation). The second is to assess for potential difficulties that may arise with more definitive airway management, were the need to arise (eg, in a patient with pronounced micrognathia or macroglossia).

The Mallampati classification system from adult anesthesia literature can be used to identify patients at risk for having a difficult airway. Here, the adequacy of visualization of the posterior oropharynx has been shown to correlate with glottic exposure

during direct laryngoscopy. The original description required patients to sit upright with their head in neutral position and protrude their tongue fully. This may not be feasible before PSA in infants and young children, where underlying illness/injury or developmental stage may prevent patients from sitting upright or cooperating with the oropharyngeal examination.

In pediatric patients, a nearly complete airway assessment can be performed using a modification of Mallampati's original technique. Rather than sitting upright, the patient can be placed in a supine position while the neck is extended slightly (when not precluded by cervical spine immobilization). Recent data suggest that slight craniocervical extension improves the specificity of the Mallampati airway evaluation, without compromising sensitivity [4]. Then, while standing above the patient, the examiner can visualize the posterior oropharynx either by asking the patient to voluntarily protrude his/her tongue or by using a tongue depressor in younger or less cooperative patients. In addition to providing some risk stratification based on the Mallampati score, observation during these 2 maneuvers allows for assessment of an array of anatomical and clinical features including (1) any baseline airway obstruction while lying supine, (2) size of the mandible and chin, (3) adequacy of mouth opening, (4) presence and size of dentition, (5) size of the tongue relative to the oropharynx, (6) presence and degree of tonsillar enlargement, (7) anatomical abnormalities of the neck or airway, and (8) neck mobility. In addition, this active or "assisted" patient participation with neck extension and tongue

depression offers insight into the degree of subsequent cooperation. It may also help identify which factors contribute most in less cooperative patients, including discomfort, anxiety, or normal developmental response in younger children. This information can then be used to help gauge the degree of pharmacologic assistance likely to be required, in terms of which sedation regimen would be most effective and what depth of sedation might be required for a given procedure.

When such an airway evaluation identifies a potentially higher-risk patient, alternatives and modifications to PSA may need to be considered. Using local or regional analgesia, using lower levels of sedation, selecting reversible agents, identifying additional or more skilled personnel to assist, or opting for complete control of the airway with general anesthesia outside the emergency department, are potential alternatives.

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Laryngospasm Complicating Procedural Sedation And Analgesia In Children

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Laryngospasm is an infrequent (occurring in 0.4% of emergency department pediatric sedation cases) but potential life-threatening complication of procedural sedation and anesthesia (PSA) in children [1]. Cote et al published a critical incident review of pediatric sedation-related events resulting in death or severe neurologic injury. Most of these incidents were associated with failure to provide timely and effective airway/ventilatory support [2]. Since this report in 2000, many national organizations have published guidelines addressing pre-sedation evaluation, medications, monitoring, equipment, personnel, and recovery recommendations [3-6]. Implementing these recommendations has likely prevented additional critical incidents. A recent publication by the Pediatric Sedation Research Consortium on 30,037 sedation/anesthesia encounters outside the operating room from 26 participating institutions reported 0.24% unexpected apnea and 0.43% stridor and laryngospasm events but no deaths [7]. Despite these best practice and patient safety guidelines, the infrequent occurrence of laryngospasm can make it difficult for the emergency physician to gain experience and competency in managing laryngospasm events associated with sedation. Therefore the purpose of this report is to review the definition, risk factors and management of laryngospasm in children undergoing PSA.

Definition

Laryngospasm is thought to be precipitated by closure of the true vocal cords, causing complete or partial upper airway obstruction [8]. Laryngospasm with partial obstruction manifests as high-pitched inspiratory stridor unresponsive to airway alignment maneuvers such as chin lift or jaw thrust. A comprehensive definition of laryngospasm with complete obstruction is suggested by Cohen and Krauss [9] and includes 5 clinical findings listed in Table 1. In addition to laryngospasm, other airway complications associated with PSA include other forms of upper airway obstruction, apnea, bronchospasm, and aspiration [7]. To help in recognition of laryngospasm, and in differentiating it from these other entities, the following definition is suggested: laryngospasm is absence of air movement with chest wall movement and/or bag-mask ventilation despite airway alignment maneuvers.

Risk Factors

Risk factors for laryngospasm include factors related to the patient, medications, and the procedures performed. In children undergoing general anesthesia, the incidence of laryngospasm is 1.7% to 2.3% [10-12]. Patient-related risk factors for laryngospasm associated with general anesthesia include being younger than 3 months, upper respiratory tract infection, asthma, active gastroesophageal reflux disease, and exposure to environmental tobacco smoke [10-14].

Table 1 Five clinical findings defining laryngospasm.

1. Chest wall movement with no breath sounds on auscultation
2. No stridor or airway sounds
3. No response to airway alignment maneuvers
4. Sudden loss of carbon dioxide waveform
5. Inability to manually ventilate with bag-mask ventilation

The primary medication associated with laryngospasm in non-operating room anesthesia is ketamine [15]. Ketamine is known to not only preserve but enhance protective airway reflexes such as coughing and gagging. Procedure-related risk factors for laryngospasm include those procedures that result in stimulation of the posterior pharynx. For instance, laryngospasm has been reported to occur in as many as 20% of children after tonsillectomy and adenoidectomy after general anesthesia [16]. These risk factors may be additive. Propofol, for example, is only associated with laryngospasm in patients having procedures that result in stimulation of the posterior pharynx [17]. In one study on 483 children sedated with propofol for upper endoscopy, 10 (2.1%) had laryngospasm [18]. In contrast to these findings, laryngospasm occurred in 52 (9.5%) of 458 children sedated with ketamine for upper endoscopy, with the incidence significantly higher in children younger than 7 years (13.9 vs 3.6%) [19]. In a study of 1021 emergency department intramuscular ketamine sedations in patients younger than 15 years, 4 patients had laryngospasm, including 1 patient who had hypersalivation and developed laryngospasm during suctioning of the pharynx [20].

Management

The first step in the management of laryngospasm related to sedation is to take steps to reduce the risk. Because ketamine is commonly used in pediatric emergency medicine, and laryngospasm is a well-known adverse effect of ketamine, familiarity with this drug's contraindications is important (Table 2) [20]. Alternative medications and/or the operating room must be considered in patients for whom ketamine is contraindicated. Because increased salivation and airway secretions, other known side effects of ketamine, may necessitate airway suctioning, which in turn can precipitate laryngospasm, coadministration of an antisialogogue such as atropine or glycopyrrolate is common. However, in the series by Green et al [21], all 17 patients with hypersalivation, including 7 in need of airway suctioning, had received atropine, whereas hypersalivation was not documented in the 14 patients who did not receive atropine. These data as well as a study by Brown et al [22] that found hypersalivation scores to be similar in children receiving and those not receiving atropine with ketamine questions the effectiveness of atropine in reducing the adverse effect of hypersalivation from ketamine.

Monitoring devices, airway equipment, and personnel must be in place to prevent a "failure to rescue" event. Continuous capnography is strongly recommended because it will detect airway obstruction from laryngospasm (sudden loss of carbon dioxide waveform) much earlier than continuous pulse oximetry (hypoxia) or cardiac monitoring (bradycardia) [9]. With continuous capnography, the concern of continuous oxygen (O₂) administration delaying a decrease in O₂ saturation from unrecognized respiratory failure is no longer a factor. Providing continuous O₂ to sedation patients will create a hyperoxygen state, which may prevent hypoxia during the initial treatment of airway obstruction (laryngospasm) [23]. The time of onset of laryngospasm events has been reported to be 12 to 25 minutes after the administration of ketamine [1,9]. Therefore, it is strongly recommended to maintain continuous capnography monitoring until the patient has fully recovered.

Reports of laryngospasm complicating sedation describe these events as transient, almost always resolving with basic airway maneuvers. One should first establish a patent airway with chin lift or jaw thrust. Next, it is appropriate to suction the airway as needed, but not excessively, because stimulation of the posterior pharynx may prolong the laryngospasm. If there is no air movement after these steps, one should initiate positive pressure ventilation via bag-mask using 100% O₂. Occluding the pop-off valve of the bag may be needed to generate pressures great enough to open the true vocal cords. If there is still no air movement, one can administer a rapidly acting and short-duration neuromuscular paralytic agent in preparation for endotracheal intubation. Intravenous succinylcholine is recommended, if not medically contraindicated. As a temporizing measure while preparing to administer succinylcholine, consider applying pressure to the "laryngospasm notch" [24]. The notch is behind the lobule of each auricle (outer ear). It is bordered anteriorly by the condyle of the mandible, posteriorly by the mastoid process, and superiorly by the base of the skull. This can be performed while holding the mask to the face with the thumb and index finger of each hand and with the middle fingers applying firm pressure in an anterior direction to the head of each condyle. The degree of pressure needs to be great enough to surpass the pain threshold. This is similar to the jaw-thrust maneuver, but pressure is applied to the

Table 2 Contraindications for ketamine in PSA in children.

Absolute

Being younger than 3 mo (higher risk of airway complications)
Known or suspected psychosis, even if currently stable and controlled with medications

Relative

Age of 3-12 mo (higher risk of airway complications)
Procedures involving stimulation of posterior pharynx (higher risk of laryngospasm)
History of airway instability, tracheal surgery, or tracheal stenosis (presumed higher risk for airway complications)
Active pulmonary infection or asthma (higher risk of laryngospasm)
Known or suspected cardiovascular disease, including angina, heart failure, or hypertension
Head injury associated with loss of consciousness, altered mental status, or emesis (elevated intracranial pressure with ketamine)
Central nervous system masses, abnormalities, or hydrocephalus (elevated intracranial pressure with ketamine)
Glaucoma or acute globe injury (elevated intraocular pressure with ketamine)
Porphyria, thyroid disorder, or thyroid medications (enhanced sympathomimetic effect)

head of the condyles and not to the angle of the mandibles. The exact physiology of how this maneuver relaxes the vocal cords is unknown, but the technique is reportedly common knowledge within the field of anesthesiology.

Summary

Laryngospasm is a rare complication of PSA. Identifying high-risk factors related to the patient, medications, and procedures is the first step in preventing and/or anticipating this airway complication. Management involves basic airway skills that should be familiar to emergency physicians. However, the infrequent but inevitable occurrence of this life-threatening event lends itself to the rapidly developing field of high-fidelity simulation training [25]. This training has the potential to identify not only provider but also system errors that when addressed can lead to best/safe practice of PSA in children.

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A “Route” Less Traveled: Intramuscular Ketamine

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An 18-month-old boy presents to the emergency department with a chief complaint of a large, red, swollen area on his right

buttock. You determine that this lesion is an abscess that requires incision and drainage. The toddler weighs 17 kg (>95% percentile). You decide that this patient requires procedural sedation and analgesia (PSA) to effectively perform the procedure. In discussion with the parents, you agree that intravenous (IV) ketamine would be a good choice.

During the fifth unsuccessful attempt at IV catheter insertion, the mother comes to speak to you about her unhappiness over the continued IV catheter attempts and prefers to just have him held down for the procedure. “If he is going to get stuck, just stick him in the abscess,” she remarks. How do you respond to the mother? What are your options?

This scenario is quite common. This toddler is a difficult patient in terms of IV access, and the procedure planned is a painful one requiring PSA. Although there are oral alternatives for anxiolysis and pain management, the preferred method for many providers would be parenteral. A parenteral option often overlooked is the use of intramuscular (IM) ketamine. Two recent publications have demonstrated that whereas ketamine is a commonly used agent via the IV route, IM ketamine is not frequently mentioned [1,2].

Why is there reluctance to give ketamine IM? Do we need an IV in place during PSA in case we need to give rescue medications or perform a rapid sequence intubation? What is the appropriate dose? Is the adverse event profile different for IM ketamine vs IV ketamine? Are the parents going to find this acceptable?

The safety and efficacy of IM ketamine has been well demonstrated [3]. A recent randomized controlled trial comparing IV ketamine to IM ketamine found them to be equally efficacious and safe [4]. This study did find that IM ketamine was associated with significantly higher rates of vomiting. This may have been a spurious finding, as has been postulated [5]. However, this finding has been reported in another study that used IM ketamine at the same dose [6].

Various doses of IM ketamine have not been studied to compare adverse event frequencies. Although significant respiratory adverse events are uncommon, they do occur. With any PSA event, one must be prepared to recognize and address any complications that arise. Most respiratory complications with ketamine can be addressed with supplemental oxygen, airway positioning, and bag-valve-mask ventilation. The optimal IM dose is still somewhat controversial. The dose studied in the mentioned trials was 4 mg/kg and was associated with a prolonged recovery time as compared with a smaller dose (1 mg/kg) of IV ketamine. In choosing a dose, it is therefore important to take into consideration the expected length of the procedure. Likewise, it is also important to inform parents about the duration of the recovery period. Parental satisfaction is also an important factor to consider, and this has been shown to be equivalent between the IM and IV routes of administration. Although not studied, parental preference may be to have their child receive 1 IM injection as opposed to the potential of multiple IV attempts.

Let us return to our patient. He has been given a 4 mg/kg IM dose of ketamine, and the incision and drainage procedure is performed successfully. The parents ask you, “Why didn't you just do that in the first place?” This is a good question.

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Dream Preparation with Ketamine

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As we all know, ketamine uniquely stimulates hallucinatory manifestations during recovery. Although these so-called emergence reactions are rarely unpleasant in pediatric patients [1,2], most of us have periodically encountered children or adolescents who describe distressing visual imagery or who otherwise exhibit more than minimal agitation during recovery. Prophylactic benzodiazepines do not prevent such dysphoria [1,2]; however, it is my anecdotal impression that another intervention does—dream preparation.

My first exposure to ketamine dream preparation was in “Papua, New Guinea”, where I traveled as a member of a Loma Linda University team sent to provide relief for overworked local physicians at a missionary bush hospital in the remote highlands [3]. At our orientation, we learned that intravenous ketamine was the modality of choice for essentially all operating room procedures that did not require opening the abdomen, including major orthopedic surgeries such as amputations. I volunteered to play anesthetist and administered ketamine to scores of patients during my stay.

At first, I was quite apprehensive regarding emergence reactions. I anticipated their frequency to be high, given that we were treating adults as well as children and that many of our patients had substantial emotional distress, for example, painful suppurative infections, debilitating diseases, and injuries from tribal violence. To my surprise, however, it became quickly apparent that almost uniformly, ketamine recoveries were calm. Midway through the first week, I clued in that the “sisters”—as the Papuan nurses were called—were whispering something into the patients' ears in the local Enga dialect just before my pushing ketamine.

It took some persistence to get these shy missionary nurses to admit what they were up to. “We are telling the patients that while asleep they will dream about heaven and that they will talk to angels.” Sure enough, many of these patients were waking up praying or singing hymns, and most had distant contented looks in their eyes! Although the ethical basis for using ketamine as an adjunct for proselytism is dubious, I could not help but be impressed by the apparent mitigation of dysphoria.

On my return, I wasted no time in trying out this technique. An 8-year-old girl with a forehead laceration was agitated and tearful. I told her that the sleepy medicine would give her wonderful dreams, but that she needed to choose in advance something very special to dream about. Her mother quickly suggested the prior week's trip to Disneyland, and as I got the girl to start chatting about her favorite experiences there, her spirits brightened. At a particularly animated moment, in went the ketamine. Thirty minutes later, her first recovery words were “Mickey Mouse,” and I was hooked!

Although ketamine dream preparation was first described more than a quarter century ago [4], it has never been subjected to controlled research. Despite this, it makes intuitive sense, is easy to do, and gives the child and parents something to think about during those tense pre-sedation moments. Although admittedly anecdotal and unproven, dream preparation has been my routine ever since, whenever feasible.

Naturally, all good things sometimes backfire. One of my colleagues treated a 17-year-old adolescent boy who gave a twisted smirk when encouraged to plan a pleasurable dream before his fracture reduction. During recovery, he began loudly detailing an explicit fantasy too lurid for both his parents and the children in the adjacent curtained treatment areas. Generous doses of IV midazolam gratefully silenced the salacious tale.

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Nitrous Oxide: Laughter May Be the Best Medicine

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For more than a century, nitrous oxide (N₂O) has been used to reduce procedural distress in children. Although used extensively by dentists and anesthesiologists, there are few reports of its use in children in the emergency department. However, inhaled N₂O blended with oxygen (O₂) has many benefits for pediatric patients undergoing brief, painful procedures in this setting. The gas is well tolerated, but best of all, it does not require intravenous access and has rapid onset of sedation, anxiolysis, analgesia, and amnesia, and recovery in less than 5 minutes without adverse cardiopulmonary effects in healthy patients. When combined with local anesthesia, if possible, it is suitable for many emergency procedures, including suturing, venipuncture, lumbar puncture, burn debridement, nursemaids' elbow reduction, digital block

administration, and, when combined with oxycodone, for fracture reduction and abscess drainage.

The use of distraction, story-telling, and imagery (eg, imagining flying) help prepare children for the clinical effects of the gas and significantly enhance efficacy. We involve a parent and the patient as much as possible during this process and encourage children to hold the mask. While breathing N₂O, children are able to follow commands, describe sensations of floating, frequently laugh, and occasionally chew or lick masks that have been scented with flavored lip balm (eg, bubble gum) to enhance acceptance of the mask. Furthermore, we are learning that many children prefer not being “put to sleep” for these procedures when pain can be primarily managed by local anesthesia.

Emesis is the most common adverse event reported and varies from 3% to 25%, likely depending on the procedure, coadministration of narcotics, concentration of N₂O, and duration of administration. When 50% N₂O is used alone, protective airway reflexes are preserved, and length of fasting does not seem to impact the occurrence of vomiting. In general, we require a 2-hour nil per os status before administration when other analgesic/sedative agents will be coadministered with N₂O.

Despite the favorable clinical and safety profile, N₂O is seldom used in pediatric emergency patients. A major issue in the United States is that there is no dedicated N₂O delivery apparatus commercially available for use with a full face mask. The demand valve–equipped fixed N₂O delivery apparatus commonly available in emergency departments is difficult for children to activate, but patients of all ages easily use continuous-circuit devices. Dental machines allow continuous flow of gas but are designed for nose cone administration and therefore must be adapted. We like the full face mask because it allows more control of what the patient is inhaling and minimizes the likelihood that mouth breathers will be inadequately sedated. The N₂O machine we developed in collaboration with our anesthesiologists and biomedical engineer is a continuous circuit that allows normal breathing by all ages. The machine was constructed with readily available anesthesia parts and includes 3 essential components: a gas source, a blender, and a scavenger. It allows titration of N₂O so as not to frighten the child with the “rush” described with immediate administration of 50% N₂O. We usually titrate to 50% over 3 to 4 minutes while we are talking with the child about flying, swimming, and other activities, to give them a context for their sensations. At a minimum, an O₂ analyzer is required to confirm that at least 30% O₂ is being delivered to the patient. It is critical that individuals administering N₂O have a full understanding of the mechanics and alarms of the apparatus, how they might fail, and how to perform a machine and monitor check before each use. Although initially time- and resource-intensive, the investment in education, collaboration, and equipment has been well rewarded.

Suggested Readings

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Pediatric Procedural Sedation in the Community Emergency Department

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The practice of emergency medicine is different in a community hospital emergency department (ED). The principles of patient care and the specific treatments are similar to those in any tertiary care center, but the logistics of their delivery can vary dramatically between the 2 types of institutions. Pediatric procedural sedation and analgesia (PSA) is a prime example of this variability.

In most community EDs, a time exists in which only a single emergency physician is caring for the entire department with little or no in-hospital support, particularly for pediatric patients. Such practice environments are not obstacles to pediatric PSA, and in fact, reports from the Procedural Sedation in the Community Emergency Department registry indicate that the physicians in such practices do quite well during these times [1,2]. However, effective pediatric PSA can be challenging in the community hospital, and some “tricks” can be used to minimize its impact on a busy department regardless of the physician coverage. The keys to successful pediatric PSA in the community hospital ED are as follows.

1. Access to the right medications. Because time is of the essence, community emergency physicians require access to reliable medications. With limited availability of anesthesia personnel in the hospital, emergency physicians will need to be credentialed to use, at a minimum, propofol, ketamine, and a narcotic such as fentanyl or remifentanyl. Other drugs such as pentobarbital, droperidol, and nitrous oxide may have niche applications in PSA. The results of the Procedural Sedation in the Community Emergency Department registry and other publications strongly support the safe use of these agents by emergency physicians regardless of the setting.
2. Give the correct dose the first time. Administering timid doses of a PSA agent in a small child for fear of respiratory depression will likely lead to inadequate sedation, repeated dosing, and a much longer procedure. Propofol, barbiturates, ketamine, and the narcotics all have very well-defined dose-response curves. In children, the recommended dose (mg/kg) will consistently produce the intended clinical response with minimal adverse effects, so give an adequate dose the first time and get on with the procedure.
3. Avoid midazolam as a single agent. In contrast to the drugs previously listed, the benzodiazepines, and midazolam in particular, have very variable dose-response activity in pediatric patients. The same dose (mg/kg) of midazolam can produce everything from deep sedation to disinhibition across a wide age range of children. For

those who have not experienced it, nothing can disrupt a community ED more than an inconsolable screaming 3-year-old child. The notion that midazolam is somehow a safer agent because it can be reversed is more than offset by the high percentage of children who fail sedation with this agent. In head to head comparisons, virtually every other procedural sedation agent outperforms midazolam.

4. Do not awaken children given ketamine. Children given ketamine should be permitted to recover undisturbed. Repeated shaking and calling of a child's name when emerging from ketamine sedation can lead to agitation because they are very suggestive at this point in time. Children should be appropriately monitored and simply allowed to come around on their own with a minimum of stimulation until completely recovered.
5. Know variable routes of delivery. Vascular access can be problematic in a child in any ED. Knowing variable routes of delivery can make pediatric procedural sedation more appealing. Ketamine, pentobarbital, and diphenhydramine can all be given intramuscularly; fentanyl can be given intranasally or transmucosally; and methohexital can be given rectally. Nitrous oxide is a pure inhalation agent, but the logistics of its use can be challenging. Do not hesitate to use an alternate delivery route as an alternative to an intravenous line in a difficult-access patient.
6. Get comfortable with PSA. From cardiac surgery to tumor resections, experience makes a difference. The more often a group of clinicians performs a procedure, the better their outcomes. Although outcomes do not seem to vary with the number of cases performed in pediatric PSA, the more frequently such cases are performed, the less intimidating they will become. The argument then comes full circle in that the more comfortable the staff become with pediatric procedural sedation, the more frequently they will use it.
7. Most important, do not get cocky. Pediatric PSA is a safe and effective ED activity and is generally performed with excellent results. Confidence and comfort in the sedation of a patient of any age is essential, although a certain degree of apprehension and adherence to PSA clinical guidelines will keep the entire team honest. An overconfident emergency physician is either inexperienced or not smart enough to recognize his or her own mistakes.

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The Use of Propofol for Pediatric Procedural Sedation in the Emergency Department

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Physicians caring for children in the emergency department (ED) setting must frequently perform medical procedures that

cause patients to experience pain, anxiety, and fear. Practitioners of pediatric emergency medicine rely on an ever-expanding armamentarium of agents to treat these symptoms. One useful agent recently introduced to EDs for pediatric procedural sedation and analgesia (PSA) is propofol (Diprivan). The rapid onset of propofol's unusually potent effects requires that it be administered differently than other sedative agents in common use.

Propofol is a potent, ultra-short-duration sedative-hypnotic introduced in 1989. It produces no analgesia but has powerful sedating, amnesic, and anticonvulsant properties. The drug is chemically unrelated to barbiturates or benzodiazepines and has the appearance of an opaque white liquid due to its formulation as a lipid emulsion (hence its nickname, "milk of amnesia"). Because of the nonactive components of its formulation, patients allergic to eggs or soy should not receive propofol. The rapidity and strength of its sedating effect make it an appropriate agent to use for sedating children for short, painful procedures. These include orthopedic reduction, cardioversion, intubation, hernia reduction, and burn debridement.

Propofol was originally described in the anesthesia literature for intraoperative use. It was subsequently used in pediatric intensive care unit settings, often as a continuous infusion. Prolonged use of propofol infusions are associated with negative patient outcomes; however, this does not affect its utility for short ED procedures. To prove to ourselves (and others) that propofol was safe and effective for use in our practice setting, physicians in the ED at Primary Children's Medical Center in Salt Lake City, Utah, performed several studies during the initial years of propofol's use in our ED [1,2,3]. Other reports have appeared in the medical literature since that time, which support these results [4].

Method of Use

Before sedating a patient with propofol, it is important to assemble the personnel and supplies needed to maximize patient safety and the success of the procedure. At our institution, 4 providers must be present at the bedside before sedation can proceed: the patient's nurse, who assists with medication administration and records vital signs and medication data; the physician administering the propofol; the physician performing the medical procedure (most commonly, an orthopedic surgeon); and an ED technician or nurse at the head of the patient's bed whose sole responsibilities are to maintain the patient's airway, administer blow-by oxygen from a bag-valve-mask device, and provide ventilatory support as needed.

The presence of 4 caregivers at the bedside does constitute a significant investment of ED personnel time and effort and would not be practical for procedures requiring long periods of sedation. The reduction and splinting of the typical forearm fracture takes about 10 minutes, which can be accommodated within the limits of our ED staffing. Only physicians experienced in propofol administration may give the drug to the patient. At our facility, this includes pediatric emergency medicine attendings and fellows.

Patients receiving propofol are placed on monitors for pulse oximetry, heart rate, and blood pressure. I require that all patients receiving propofol be placed on blow-by oxygen via face mask and bag device. Because few patients ever require bagged breaths, this additional step is probably indicative of my personal anxiety level

more than of any true patient need. My rationale for doing this is that if patients have a short period of apnea when receiving initial doses of propofol, they are at less risk for hypoxemia if they start out with an oxygen saturation of 100%. Our ED does not routinely use capnometry when sedating patients with propofol, although this may change over time. To insure a patent airway, I also routinely ask that a towel roll be placed under the patient's shoulder blades, to place their head in a "sniffing" position.

If propofol is being administered for a painful procedure, a standard dose of an analgesic such as fentanyl or morphine should first be ordered. After several minutes, propofol is then given in an initial dose of 1 mg/kg IV (with a maximum of 40 mg), followed by additional doses of 0.5 mg/kg. Propofol often stings veins, although this may be minimized by rapid saline flushes between doses. My goal is not to produce a completely motionless or unresponsive patient. Instead, I aim to lower the patient into a state of sleep from which they can be aroused by significant pain, such as an orthopedic reduction. In the case of an orthopedic reduction, I know that the patient is ready for the procedure when I can hit the patient's arm and get no patient response.

When a painful procedure such as an orthopedic reduction is being performed, I expect the patient to move around or produce unintelligible vocalizations. If family members remain present in the room, it is important to mention that this response is both expected and desired. During the period when a patient's fracture is being manipulated, I am at the bedside watching the patient's response, giving additional 0.5 mg/kg doses of propofol as needed. The typical total dose of propofol received by a patient undergoing a forearm fracture reduction is usually in the range of 3 to 4 mg/kg. When doses of propofol higher than this are received by the patient, it usually indicates that a procedure took longer than usual to perform and is not due to any difficulties in achieving an adequately sedated state.

Many of our ED physicians, including myself, ask that propofol be brought to the patient's bedside in separate syringes containing 1 mg/kg of the drug (with a maximum of 40 mg). I feel that this makes the accidental administration of excess drug less likely and makes the physician pushing the drug acutely aware of how much the patient is getting. The need to change syringes containing propofol does slow its administration somewhat, which I feel adds a useful margin of patient safety as well. A 3-way stopcock allows alternating administration of active medication and saline flush.

Recovery from propofol sedation is usually fast and pleasant, usually occurring approximately 15 minutes after the last dose has been given. Even after sedating hundreds of patients with this agent, I have yet to have a patient vomit or experience an emergence reaction during recovery. Patients usually have complete amnesia for their medical procedure; those with forearm fractures often ask when we are going to fix their arm.

Summary

Propofol has proven to be a safe and effective tool for sedation of children who require painful medical procedures in the ED in which I practice. The combination of rapid effect, "titrateability," amnesia, antiemetic properties, and rapid recovery, is approached only by etomidate. Parents, patients, and medical and nursing staff all express high levels of satisfaction, and it has replaced many other methods of PSA (eg, Bier block, ketamine) used formerly.

Drawbacks to the use of propofol sedation include its therapeutic window, which is narrower than other commonly used sedatives such as midazolam. A lowered blood pressure may be noted, which does not appear to harm the patient, who remains pink and well perfused. Political battles are sometimes encountered between emergency physicians who wish to provide their patients with the benefits of this method of sedation and hospital committees or anesthesiologists wishing to restrict its use.

The advantages of sedation with propofol for short, painful medical procedures far outweigh the drawbacks. For this reason, it has become the standard method of achieving short periods of moderate-to-deep sedation in our patient population.

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Alfentanil and Remifentanil During Procedural Sedation With Propofol

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The optimal method to treat procedural pain during sedation with propofol, and the degree to which it should be relieved, has not been determined. It is generally accepted that propofol does not provide analgesia. It does consistently provide amnesia [1], but the effect of unrecalled pain is currently not well understood. At the very least, acute pain results in stimulation of patients, increasing their sedative requirements. It also decreases their respiratory drive, increasing their tendency to develop respiratory depression. There are a variety of preprocedural approaches, and it is likely that they result in different degrees of pain relief, influencing both the level of sedation needed to perform the procedure and the amount of sedative used. It is also likely that patients who receive more preprocedural analgesia are more prone to respiratory depression during the sedation.

Supplemental opioids have been described for dosing before, after, or concurrent with propofol. Typically, the opioid used proximal to propofol sedation is fentanyl. However, the relative half-lives of the 2 drugs differ by an order of magnitude, and combining them affects such things as the duration of the sedation and the depth of sedation, which vary greatly because of very different pharmacokinetics of the 2 medications.

In a perfect model of sedation with propofol, patients would have their pain completely controlled, as their respiratory status would tolerate, using a fairly long-acting opioid such as

morphine or hydromorphone. Then the patient could be sedated using propofol at a dose adequate for amnesia only, reducing the risk of side effects and providing the patient with comfort and amnesia. Most procedures that require sedation are painful, and pain relief achieved at pain levels when the patient is at rest before the procedure may not persist after the procedure has begun. It therefore stands to reason that giving extra pain medications during the sedation will be helpful and to the patient's benefit. In the absence of knowledge about the effect of pain that cannot later be recalled, it will at least reduce the stimulation patients experience from the procedure, reducing their sedative requirement. Most procedures in the emergency department (ED) are very short, however, and using a pain medication that lasts longer than the procedure will result in a patient who is overtreated for pain once the painful stimulation of the procedure is over, increasing the duration of sedation compared with using propofol alone.

This brings the focus to opioids, which have a duration of action more similar to the duration of a typical ED procedural sedation with propofol: remifentanyl and alfentanil [2-4]. Remifentanyl is metabolized by esterases present throughout plasma and tissues and is cleared very quickly. It also has a very low pK_a , which allows it to cross the blood-brain barrier very quickly. It has a half-life of 3.2 minutes after prolonged (even after very long periods of) infusion. The drug is typically described as dosed between 0.5 and 1 $\mu\text{g}/\text{kg}$ followed by an infusion of 0.15 $\mu\text{g}/\text{kg}/\text{min}$. The short half-life and quick onset makes it an excellent choice for concurrent use with propofol. The downside is that the benefit of adding it to propofol for a brief ED procedure is not clear [4] and that setting up an infusion by a pump can be a cumbersome addition if the sedation was otherwise going to be done with boluses of medications.

Alfentanil has generally been shown to be inferior to remifentanyl for sedation when used as an infusion, but its properties give it a benefit over remifentanyl for procedural sedation and analgesia (PSA) in the ED. Alfentanil is cleared hepatically like fentanyl, but after a single bolus, its duration of effect is limited mostly by redistribution, lasting about 8 minutes. This increases with subsequent dosing and infusions, peaking 1 hour into an infusion. For the purposes of ED PSA, the 8-minute duration of a single bolus makes its effect close to the duration of many short ED procedures, allowing its use without initiating an infusion. It has been described for use with propofol at doses of 10 to 15 $\mu\text{g}/\text{kg}$ as a single bolus, and if needed, followed by an infusion of 15 $\mu\text{g}/\text{kg}/\text{hr}$. If a short-acting narcotic is going to be added to procedural sedation with propofol, a single bolus of alfentanil is an excellent choice for brief procedures. Remifentanyl is probably better for procedures long enough to warrant an infusion, although ED-specific studies of both agents are warranted.

An added benefit of either drug is that both have exhibited excellent efficacy in preventing the pain associated with propofol infusion. The alfentanil doses of 15 and 20 $\mu\text{g}/\text{kg}$ and the remifentanyl dose of 0.5 $\mu\text{g}/\text{kg}$ given immediately before the administration of propofol have been shown to prevent pain from propofol administration [5,6]. Many other techniques to control pain from propofol infusions have been described, but none have been superior to the descriptions of these 2 agents. These findings are a little more concrete than the theoretical benefit of adding narcotics to treat pain that likely will not be later recalled and, pending further evidence, may be adequate to indicate their addition to usual PSA with propofol.

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The Adolescent Patient and Procedural Sedation

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Adolescent patients frequently require procedural sedation and analgesia (PSA) in the emergency department (ED) to alleviate pain and anxiety associated with acute injury, painful medical conditions, and diagnostic or therapeutic studies. Although pain and anxiety are equally important considerations in adolescents as in infants and school-aged children, it is important that the medical practitioner providing PSA for adolescent patients recognize that unique features of these patients make their care different from that of younger patients. These include cognitive and developmental factors as well as physiologic and pharmacokinetic differences. I became interested in this topic after an experience I had in the ED. A 13-year-old adolescent boy was referred from an outside ED for reduction of a radius/ulna fracture resulting from a fall from an all-terrain vehicle. He was given morphine for pain control at the outside ED and arrived with his arm splinted and complaining of minimal pain. His medical history was unremarkable. He denied alcohol, tobacco, or drug use. After discussing the PSA options with the patient and his parents, he was given atropine, midazolam, and ketamine. The fracture reduction was completed, and the patient was splinted. As he began to wake up from sedation, he began laughing very loudly. His mother asked him why he was so happy, to which he remarked, "Oh, cool, look at all of the colors and stars, they are on the wall." This continued for approximately 5 minutes, after which he returned to baseline mental status. He then questioned me about what drug I had given him and "how can I get more?"

That evening, as I recounted the day's events in my head, I began to ponder: in the future, if I had the same patient, which PSA agent would I select? Could I have selected a "better" PSA agent for this patient? I selected ketamine because it provides potent sedation, analgesia, and amnesia, while preserving spontaneous respirations and protective airway reflexes [1-5]. It has a very good safety profile, and multiple studies have shown high parental and provider satisfaction [1,2,6-10]. Its use in adolescent and adult patients has been limited because of the concern for recovery reactions such as emergence anxiety,

nightmares, and hallucinations [11-15]. The true incidence of such reactions, however, is not known. Several studies suggest that it is not a major problem in older adolescents [15,16]. Midazolam is often used in combination with ketamine to decrease these side effects, although existing studies have failed to support this practice [9,17].

A drug combination frequently used for PSA in adolescent patients is that of fentanyl and midazolam. Fentanyl is a potent opiate that produces analgesia within 2 to 3 minutes of administration [18]. Midazolam is a short-acting benzodiazepine that produces sedation, anxiolysis, and anterograde amnesia within 2 to 3 minutes of IV administration [19]. Studies indicate that the efficacy of this combination is high, but there appears to be an increase in the risk of respiratory depression [1,20-24]. One study showed that older children and adolescents may be at decreased risk of adverse respiratory events with this combination [25]. My personal experience with this combination is that some patients achieve satisfactory sedation, whereas in other patients, it is harder to achieve the level of sedation needed to complete the procedure without developing respiratory depression and hypoxia. In addition, I have found that parental and patient satisfaction with this combination is not comparable with that of ketamine. One of my patients who received this combination for PSA during a fracture reduction remarked, "I remember and felt the entire procedure".

At that time, I decided that I would have to find another option for this group of patients. I designed and conducted a study comparing ketamine/midazolam with propofol/morphine in patients older than 8 years undergoing fracture reduction. I learned very quickly that this may have been an overzealous undertaking. It was very difficult to recruit patients for this study because there was physician and nursing discomfort with the use of propofol for PSA. Since that time, there has been an extensive body of literature published about the safety and efficacy of propofol for PSA for children and adults in the ED [26-34]. Propofol's track record of use in adult patients should be reassuring with respect to its role for adolescents. Nonetheless, propofol's use by nonanesthesia personnel remains restricted in many institutions.

There are also a number of adjunctive measures that I use to decrease pain and anxiety for adolescent patients. Although not often considered for adolescents, child-life specialists play an integral role by providing distraction with imagery, music, movies, or video games. They are also able to provide detailed explanations of the procedures in terms that are easily understandable to the patient. Lidocaine and L-M-X (Ferndale Laboratories, Ferndale, MI) are used in our ED for intravenous placement, venipuncture, implantable catheter access, and lumbar puncture. Lidocaine, epinephrine, and tetracaine (LET) are applied at triage for appropriate lacerations. In adolescent patients, who are able to comprehend what is being done, such measures to alleviate pain may be all that are required for these minor procedures that could provoke great anxiety in younger children.

Currently, there is no "perfect" PSA agent for the adolescent patient. There are multiple agents available, and each has its own set of risks and benefits. Despite the concern for emergence reactions, such as the one illustrated by the patient in this vignette, I continue to use ketamine for PSA on an individual basis. During the presedation assessment, I try to elicit from both the patient and the parent risk factors associated with hallucina-

tions such as frequent dreaming or a history of personality disorders. In addition, I am vigilant about elucidating any history of alcohol or drug use. In those patients at risk for adverse events with ketamine administration, I use the fentanyl/midazolam combination as my PSA agent of choice. Propofol is currently not approved for use as a PSA agent in my ED; however, it appears to be an agent of choice for the adolescent patient requiring sedation for a brief, painful procedure.

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