CLINICAL PRACTICE

Pulmonary Aspiration Risk during Emergency Department Procedural Sedation—An Examination of the Role of Fasting and Sedation Depth

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Abstract. The assessment of pre-procedure fasting and control of sedation depth are prominent elements of widely disseminated procedural sedation guidelines and of the Joint Commission on Accreditation of Healthcare Organizations' standards. Both exist primarily to minimize the risk of pulmonary aspiration of gastric contents. This paper critically examines the literature on pre-procedure fasting and controlling sedation depth in association with pulmonary aspiration, and interprets this evidence in the context of modern emergency medicine practice. The article reviews the pathophysiology of aspiration and changing concepts regarding aspiration risk over the last decade. After reviewing studies on aspiration risk during general anesthesia, the paper reviews the risk of aspiration during labor and delivery as a more ap-propriate comparison group for aspiration risk during emergency department procedural sedation and analgesia (ED PSA). It is noted that aspiration during ED PSA has not been reported in the medical literature and that aspiration during general anesthesia and labor and delivery is uncommon. The literature provides no compelling evidence to support specific fasting periods for either liquids or solids prior to PSA, and existing guidelines for elective patients are of necessity arbitrary and based upon consensus opinion. The article discusses the implications in the areas of training and preparedness, monitoring, and research for the emergency physician practicing PSA. **Key words:** aspiration; pneumonia; sedation; fasting; guidelines. ACADEMIC EMERGENCY MEDICINE 2002; 9:35–42

PROCEDURAL sedation and analgesia (PSA) is a daily technique in most emergency departments (EDs) and a requisite skill for emergency physicians (EPs). Two essential elements of current PSA practice—assessment of pre-procedure fasting and limiting sedation depth—exist largely to minimize the risk of pulmonary aspiration of gastric contents. Both items are a central focus of established PSA guidelines[1–3] and the Joint Commission on Accreditation of Healthcare Organizations' (JCAHO's) standards.[4] Each presents a special challenge in the ED setting, as patients are rarely fasting prior to their ED presentations, and the depth of sedation necessary to successfully complete many ED procedures (especially in children) is deeper than that required in most other PSA settings.

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Individual hospitals are required by the JCAHO to standardize sedation practices, and most impose specific restrictions on their practitioners regarding fasting and depth of sedation. Such policies have substantial impact on ED clinical practice and present barriers to the adoption of potent agents such as propofol and etomidate for PSA.[5,6] Accordingly, we critically examine the literature on pre-procedure fasting and limiting sedation depth in association with pulmonary aspiration, and interpret this evidence in the context of modern emergency medicine practice.

ASPIRATION PATHOPHYSIOLOGY

Pulmonary aspiration can be defined as "inhalation of oropharyngeal or gastric contents into the larynx and lower respiratory tract."[7] Such aspiration may be entirely asymptomatic (e.g., "silent aspiration" during surgery), or may manifest as a clinical syndrome characterized by any combination of bronchospasm, hypoxia, cough, dyspnea, or auscultatory abnormalities.[8–10] Aspiration pneumonitis is defined as "acute lung injury after the inhalation of regurgitated gastric contents."[7] Although complete recovery following even pro-

TABLE 1. Aspiration Risk during General Anesthesia (Studies* 1980 or Later)

Study	Population	Total Subjects	Aspiration Overall	Aspiration during Emergency Surgery	Aspiration Mortality
Hovi-Viander[15] 1980	All	338,934	Not stated	Not stated	1:67,787
Cohen et al.[52] 1986	All	112,721	1:1,566	Not stated	Not stated
Olsson et al.[12] 1986	All	185,358	1:2,131	Not stated	1:46,340
Tiret et al.[16] 1986	All	198,103	1:7,337	Not stated	1:49,526
Kallar[53] 1988	All	529,150	1:5,879	Not stated	None
Leigh and Tytler[13] 1990	All	84,835	1:14,139	Not stated	1:84,835
Warner et al.[8] 1993	Adults	215,488	1:3,216	1:895	1:71,829
Mellin-Olsen et al.[14] 1996	All	85,594	1:3,424	1:809	None
Borland et al.[54] 1998	Children	50,880	1:978	Not stated	None
Warner et al.[9] 1999	Children	63,180	1:2,632	1:373	None

*For complete reference citations, see the reference list.

nounced aspiration pneumonitis is frequent,[8,9,11–14] the condition can be fatal.[8,11–16]

Aspiration of gastric contents was first reported as a complication of general anesthesia in 1862,[17,18] although it was not until 1946 that the pathophysiology was identified.[11] Mendelson instilled gastric aspirate directly into the tracheas of experimental rabbits, and subsequently noted histologic changes in the lung consistent with chemical pneumonitis.[11] He concluded that regurgitated gastric acid was responsible for the pulmonary damage observed with aspiration syndrome, and as a result advocated the then-unusual concept of fasting prior to anesthesia.[11,19]

Following implication of gastric acidity in the pathophysiology of aspiration, subsequent studies focused on determining the minimum volume and pH of gastric contents necessary to produce aspiration pneumonitis. Although initial animal research indicated that a minimum of 0.4 mL/kg of gastric acid with a pH <2.5 was required to produce lung changes consistent with aspiration,[20] later more-reliable studies suggest that this minimum volume is 0.8 mL/kg.[21] Although most animal models of aspiration instilled various fluids directly into the trachea, the minimum regurgitated gastric volume needed for aspiration pneumonitis to occur has been shown to be considerably more than 0.8 mL/kg.[22,23] This is consistent with the clinical observation that not all regurgitated fluid is aspirated. Finally, several prerequisite abnormal physiologic conditions are necessary for aspiration to occur, which include reduced barrier pressure (relative incompetence of the lower esophageal sphincter) and loss of protective airway reflexes (cough, gag).[7,22,24]

Based upon this work, it has been historically taught that the extent of pulmonary injury from aspiration is directly related to the volume of gastric contents and to their degree of acidity. Accordingly, preventive measures to reduce the risk of aspiration were incorporated into anesthesia practice, including preoperative fasting and adminis-

tration of drugs to alter gastric pH (antacids, H_2 receptor blockers, proton pump inhibitors) and enhance lower esophageal sphincter tone and gastric motility (prokinetic agents).[25]

CHANGING CONCEPTS REGARDING ASPIRATION RISK

Major advances in the past half-century have fundamentally changed the way aspiration risk in general anesthesia is viewed. First, it has been recognized that the normal glottis is far from a perfect protective barrier, and the act of introducing acidic or bacteria-laden fluids into the bronchial tree in and of itself does not by necessity lead to clinical pathology. Silent aspiration (i.e., aspiration that does not lead to a clinically apparent aspiration syndrome) of nasopharyngeal secretions occurs regularly during normal sleep in healthy individuals.[7,26–30] Occult or silent aspiration of gastric contents also occurs during general anesthesia.[31–36] In one study,[31] six of 734 patients who swallowed dye preoperatively had this colored marker suctioned from their tracheas upon conclusion of their surgery, and all were asymptomatic. Four of these six had cuffed endotracheal tubes in place, which failed to prevent the aspiration. Similarly, Culver and colleagues noted that 8.3% of 300 patients experienced silent aspiration of dyed gastric contents during general anesthesia using uncuffed endotracheal tubes.[32]

The second critical development was the finding that administration of clear liquids up to two hours before surgery does not adversely affect gastric volume or pH. Numerous investigators have noted that fasting more than two hours after ingesting clear liquids does not significantly change gastric volume or pH when compared with a simple two-hour fast,[37–42] and that gastric volumes and pH at the time of induction vary greatly, especially in children.[37,42–45] Additionally, a substantial number of patients regardless of fasting had gastric volumes and pH at levels originally considered to rep-

resent high aspiration risk.[22,37,41,44] These reports suggest that moderate increases in gastric volume above the traditional threshold (0.4 to 0.8 mL/kg and pH < 2.5) may not be a risk factor for aspiration[46,47] and call into question the premise behind routinely attempting to reduce stomach acid quantity with drug therapy and prolonged fasting from clear liquids. Furthermore, there are no data to support improved outcomes with antacids and other pharmacologic agents, and these therapies are no longer routinely recommended.[25,48,49]

Based on the above landmark research and widespread practice diversity,[50] the American Society of Anesthesiologists (ASA) in 1999 shortened their fasting recommendations for clear liquids in healthy patients undergoing elective procedures.[25,47] According to the ASA, for clear liquids "published evidence is silent on the relationship between fasting times, gastric volume, or gastric acidity and the risk of emesis/reflux or pulmonary aspiration in humans." For solid food "there is insufficient published evidence to address the safety of any preoperative fasting period."[25] Given this lack of relevant evidence upon which to base a guideline, a consensus decision was made to recommend for all ages two hours fasting for clear liquids, and six hours for solids and other liquids.[25] Although the optimal duration of fasting from milk and solids is unclear, what is certain is that aspiration pneumonitis in healthy patients from clear, nonparticulate fluids is generally a self-limited disease without serious sequelae, while aspiration of acidic particulate fluid tends to result in greater pulmonary damage. Although adopted by the ASA in 1999, surveys show that liberalized NPO (nothing by mouth) policies in regard to clear liquids have been in widespread use over the last decade, and an associated increase in the incidence of aspiration has not been observed.[47,50,51]

ASPIRATION RISK DURING GENERAL ANESTHESIA

Although aspiration is a widely feared complication of general anesthesia, clinically apparent aspiration in modern anesthesia practice is exceptionally rare (Table 1), and in healthy patients the overall morbidity and mortality is low.[8,9,12–16,52–54] When existing studies in Table 1 are pooled, the overall incidence of aspiration is 1:3,420, with aspiration mortality in 1:125,109. Litigation regarding aspiration comprised less than 2% of the 1,541 claims in an ASA review of closed malpractice actions.[55]

Reported risk factors for aspiration are shown in Table 2. Other variables anecdotally implicated in this regard[10,49]—pregnancy, meal within three hours, opioid therapy, obesity, and diabetes—do

TABLE 2. Reported Risk Factors for Pulmonary Aspiration*

Airway difficulties (e.g., difficult intubation, laryngospasm)[8,12]
Emergency surgery[8,9,12,15,16]
Advanced age (e.g., >70 years)[12,16]
Higher ASA† physical status classification[8,12,16]
Conditions predisposing to gastroesophageal reflux (e.g., esophageal disease, hiatal hernia, peptic ulcer disease, gastritis, bowel obstruction, ileus, elevated intracranial pressure)[8,9,12]

*For complete reference citations, see the reference list.
†ASA = American Society of Anesthesiologists.

not appear to be independent predictors of aspiration.[8] Children are probably at no higher risk than adults,[9] although evidence is conflicting.[12,16,54] Aspiration can also unpredictably occur in patients without risk factors.[8,12,16] When significant risk factors are identified and there is an urgent need for the procedure, rapid-sequence induction of anesthesia using cricoid pressure is widely recommended. However, aspiration may still occur despite this precaution.[48]

ASPIRATION RISK DURING LABOR AND DELIVERY

The parallel and more extensively studied experience of aspiration risk in early obstetrics may be a more useful comparison group for PSA than that of general anesthesia. Prior to the widespread introduction of epidural anesthesia, women in labor commonly received anesthetics by mask without intubation, frequently to substantial depths of sedation and anesthesia. Like ED patients selected for PSA, women in labor are generally healthy, present at all hours, and are rarely fasting. Pregnancy is associated with decreased gastric emptying, so obstetric patients are classically all assumed to have "full stomachs."[20] Historically women in labor have been kept NPO for fear of aspiration; however, in recent years this philosophy has been reversed.[48,56] In a 1991 review on this topic, Elkington concludes "for otherwise uncomplicated parturients, a nonparticulate diet should be allowed as desired" throughout labor and that "there are no data demonstrating the medical relevance of any particular policy from a risk-reduction perspective."[56]

Multiple large obstetric series establish that the risk of clinically apparent aspiration in sedated, nonfasted parturients is remote. Mendelson's original report of aspiration[11] consisted of 44,016 nonfasted obstetric patients between 1932 and 1945, of whom more than half received "operative intervention" with ether by mask without endotracheal intubation. He described aspiration in 66 cases (1:667). Although several of the patients were critically ill from their aspirations, "recovery

was usually complete" within 24 to 36 hours and only two died from this complication (1:22,008). More recently, Krantz and Edwards[57] describe 37,282 vaginal deliveries of which 85% were performed with general anesthesia by mask and without intubation; 65 to 75% had ingested liquids or solid food within four hours of onset of labor. They noted five mild cases of aspiration (1:7,456) with no sequelae. Ezri and colleagues noted one occurrence of "mild aspiration" without adverse outcome in 1,870 women undergoing nonintubated peripartum surgery with intravenous ketamine, benzodiazepines, barbiturates, and/or fentanyl.[58] Soreide and colleagues observed four episodes of aspiration each during 36,800 deliveries and 3,600 cesarean sections, respectively, with no mortality.[10]

Based on the above data, the majority of hospitals permit free intake of clear liquids during labor.[59]

Aspiration Risk during Procedural Sedation and Analgesia

The risk of aspiration during ED PSA has not been studied. Although there are no cases of aspiration during ED PSA reported in the medical literature, it is possible in the current medicolegal climate that such events are underreported. Furthermore, the current literature may or may not reflect all the events that actually occur given its limitations (retrospective not prospective studies and reporting predominantly from university settings). Although several series have validated the safety of current PSA practice in the ED,[60,61] large studies would be required to accurately define a reliable incidence for rare adverse events in this setting. In a recent compilation of 95 anecdotal adverse events during PSA by non-anesthesiologists, no occurrences of aspiration were reported.[62]

Emergency physicians are by nature of their training skilled at PSA, resuscitation, vascular access, and advanced airway management, permitting them to effectively recognize and manage the potential complications associated with PSA. In the compilation of PSA adverse events by non-anesthesiologists discussed above, the most common clinical errors were delayed recognition of respiratory depression and arrest, inadequate monitoring, and inadequate resuscitation[62]—mistakes that are unlikely for trained EPs. Indeed, only four of these 96 described adverse events involved EPs, and no adverse outcomes are reported for this subset.

Several factors suggest that the relative risk of aspiration during ED PSA may be substantially lower than during general anesthesia:

1. Approximately two-thirds of aspiration during general anesthesia occurs during manipulations of the airway (endotracheal tube placement and removal)[8,12,16]—high-risk events that do not occur during PSA. Although most patients undergoing general anesthesia have cuffed endotracheal tubes in place, aspiration can occur despite such protection.[31–34,36] Furthermore, the majority of "emergency" patients identified as at higher risk during general anesthesia[8,9,12,15,16] aspirated during intubation and extubation.

2. Unconsciousness without response to painful stimuli is not a targeted sedation endpoint in ED PSA. Accordingly, protective airway reflexes should be substantially or fully retained in most cases. In contrast, complete or near-complete loss of protective airway reflexes is typical during general anesthesia.[2]

3. The majority of PSA is performed on patients who are healthy (i.e., ASA physical status classes I or II) in spite of their acute injury or illness.[60,61] In contrast, approximately 40% of patients undergoing general anesthesia have substantial underlying or critical illness (i.e., ASA class III, IV, and V),[8] including those judged unsuitable for PSA and referred for general anesthesia. Patients with poorer physical status are well known to represent higher aspiration risk,[8,12,16] and Olsson and colleagues in a series of 185,358 cases[12] observed aspiration mortality only in patients who "were in poor physical condition before the operation."

4. The majority of PSA is performed upon young adults and children, rather than elder patients who are known to be at higher risk of aspiration during anesthesia.[12]

5. Inhalational anesthetics are emetogenic, with nausea and vomiting common (25–60% incidence[63–65]) during postanesthesia recovery. With the exception of low-dose nitrous oxide, EPs do not administer inhalational agents, and the emesis rate with typical PSA agents is substantially lower (0.25–6.7%).[60,61]

6. A considerable number of ED procedures in children are accomplished with ketamine as the sole agent,[60,61,66] a drug that preserves protective airway reflexes.[61,67,68] Despite 30 years of widespread ketamine use, there are no documented cases of clinically significant ketamine-associated aspiration in a patient lacking established contraindications to this drug.[61,68,69]

7. Some have argued that acutely injured or ill patients should be considered nonfasted regardless of time of last oral intake, in the supposition that gastric emptying is delayed by acute stress or anxiety.[22,24,49,70] However, there is no compelling evidence to support this assertion.[24,70] Several studies have failed to show delays in gastric emptying associated with anxiety in both adults[71–73] and children.[43]

PRE-PROCEDURE FASTING FOR PSA

It has been stated that fasting guidelines prior to PSA "should be the same as those for general anesthesia,"[49] and such a recommendation is reflected in recent ASA fasting guidelines.[25] Although this conservative stance might be considered warranted for safety reasons, it must be recognized that this position reflects a best-guess effort to provide some sort of standard in the absence of firm evidence. As experience in PSA grows and evidence continues to point to a lower aspiration risk, it is only natural that the need for equally strict fasting guidelines will be challenged. Even the ASA has acknowledged that "the literature provides insufficient data to test the hypothesis that pre-procedure fasting results in a decreased incidence of adverse outcomes in patients undergoing sedation/analgesia (as distinct from patients undergoing general anesthesia)."[3] The available data, as discussed in the previous section, suggest that aspiration risk during ED PSA is lower than for general anesthesia, which is already exceptionally low. In spite of this fact, EPs should maintain the safety systems already in place to minimize aspiration risk. What can be questioned in light of the data is whether it is appropriate to apply the same NPO guidelines to ED PSA that are used for general anesthesia in the operating room.

The concept of pre-procedure fasting is logistically difficult for EPs, who have no control over patients' oral intake prior to ED presentation. In actual practice, EPs routinely perform PSA on patients noncompliant with the ASA elective procedure fasting guidelines. Procedures can sometimes be delayed for a number of hours in the ED; however, this must be balanced with prolongation of pain and anxiety for the patient, inconvenience for the patient and his or her family, and expenditure of room space and other finite ED resources. Additionally, many ED procedures require urgent if not immediate attention, e.g., debridement and repair of animal bite wounds, acute burn management, arthrocentesis for suspected septic arthritis, reductions of joint dislocations, lumbar puncture in the uncooperative septic patient, hernia reduction, eye irrigation for ocular trauma or chemical burns, and emergent cardioversion. The ASA and American Academy of Pediatrics' (AAP's) PSA guidelines[2,3] were not crafted with ED patients in mind and fail to provide any specific fasting recommendations applicable to the unique environment of the ED. According to the ASA, "in urgent, emergent, or other situations when gastric emptying is impaired, the potential for pulmonary aspiration of gastric contents must be considered in determining the timing of the intervention and the degree of sedation/analgesia."[3] According to the AAP, "when

proper fasting has not been assured, the increased risks of sedation must be carefully weighted against its benefits, and the lightest effective sedation should be used."[2] Even PSA guidelines from the American College of Emergency Physicians (ACEP) do not provide specific directives for risk-stratifying PSA candidates, stating "recent food intake is not a contraindication for administering PSA, but should be considered in choosing the depth and target level of sedation."[1]

SEDATION DEPTH CONTROL

The progression from mild sedation or analgesia to general anesthesia represents a continuum not easily divided into discrete stages.[67] Low doses of opioids or sedative/hypnotics induce mild analgesia or sedation with little danger of adverse events. Administering additional medication achieves higher central nervous system levels and results in a progressive decrease in consciousness with a proportionately increased risk of cardiorespiratory depression and loss of protective airway reflexes. The point at which clinically important impairment of protective airway reflexes occurs is unclear. Such reflexes are reliably maintained during moderate sedation and reliably lost during general anesthesia.[2,67] The critical question is whether deep sedation is associated with impairment of protective reflexes, or whether such danger is only encountered when "pushing" deep sedation to a point at which it approaches or reaches general anesthesia. Unfortunately, not only are there no research data to answer this question but also there is no safe and practical way to assess the status of protective airway reflexes, especially once patients are sedated to the level of not being able to follow verbal commands. This is especially true for all levels of sedation in young children who do not understand or are unreliable in following verbal commands.

In order to arbitrarily stratify the sedation continuum to permit hospital policymaking, the JCAHO has adopted the four levels of sedation and anesthesia defined by the ASA (Table 3).[4,74] Of the PSA agents used in the ED, only the ketamine dissociative state is inconsistent with definitions for all of these defined levels, and therefore must be considered separately from this discussion of the sedation continuum.[75] Since individual patients have differing responses, the JCAHO expects that practitioners permitted by hospitals to administer moderate sedation are qualified to rescue patients from inadvertent deep sedation, and that practitioners permitted to administer deep sedation are qualified to rescue patients from inadvertent general anesthesia.[4] By nature of their training, EPs are competent to rescue patients from general an-

TABLE 3. Definitions of American Society of Anesthesiologists/Joint Commission on Accreditation of Healthcare Organizations Levels of Sedation

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Minimal sedation (anxiolysis): 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/analgesia (formerly "conscious sedation"): 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.

Deep sedation/analgesia: 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: 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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esthesia, and essentially all hospitals permit EPs to administer deep as well as moderate sedation. Indeed, deep sedation is a practical necessity for many emergency procedures in frightened children.[76]

IMPLICATIONS FOR EMERGENCY PHYSICIANS

The implications of the aspiration literature for EPs practicing PSA fall into three areas: training and preparedness, monitoring, and research.

1. Training and Preparedness.

• Although the literature shows that aspiration is a highly unlikely event during PSA, prophylactic safety measures should continue for all patients receiving PSA in the ED. These safety measures include but are not restricted to training of ED personnel, attentiveness to interactive monitoring, presedation identification of high-risk patients, and promptness and quality of resuscitation efforts when adverse events occur. Therefore, EPs must consistently maintain a high level of vigilance throughout PSA.

• There is no literature basis for any specific fasting period for either liquids or solids prior to PSA, and existing guidelines are of necessity arbitrary and based upon consensus opinion. The fact that the ASA guidelines are opinion-based and not data-driven does not diminish the importance of essential safety measures (as described above).

• Emergency physicians should enhance preparedness for PSA adverse events through ongoing self-study. Structured PSA training should be an integral part of emergency medicine residency and pediatric emergency medicine fellowship curricula. Practice in managing rare adverse events is important to maximize the quality of resuscitations during PSA.

• During their presedation evaluations, EPs should routinely and thoroughly screen for aspiration risk factors (Table 2) and potentially difficult airways (e.g., short neck, small mandible, large tongue). When such patients are identified and the level of required sedation will approach or reach deep sedation, EPs should carefully consider referring such patients for general anesthesia in the operating room if time permits.

• When the nature of the procedure is such that it requires both immediate attention and a deep level of sedation to be successfully accomplished, EPs can capably perform deep or dissociative sedation using standard[2–4,67,77,78] precautions and monitoring. Care must be taken to avoid pushing deep sedation to levels approaching general anesthesia, as it can be difficult to distinguish between deep sedation and general anesthesia.

• Emergency psysicians should assess the timing and nature of oral intake prior to PSA, and balance the remote potential for aspiration with the timing and urgency of the procedure at hand and the anticipated sedation depth. Although all ED patients will be assumed to have full stomachs, it is acknowledged that at many times moderate, deep, or dissociative sedation will remain indicated in spite of this status. In these circumstances, ketamine may be preferred over other PSA agents in children due to its unique ability to preserve protective airway reflexes.[61,67,68,79]

2. Monitoring.

• Emergency physicians should maximize the use of available monitoring technologies in order to reduce potential PSA risks. Although pulse oximetry is a reliable measure of oxygenation, it does not directly measure ventilation. Capnography provides a continuous, breath-by-breath measure of ventilatory function and the earliest detection of respiratory abnormalities with apnea resulting in an almost instantaneous loss of the carbon dioxide waveform.[80–83]

3. Research.

• Agents new to ED PSA, such as propofol and etomidate, should undergo careful investigation prior to widespread use. To avoid undue risk of aspiration, investigators must establish that PSA can be performed with these agents in a manner that consistently avoids pushing the limits of deep sedation, and objective, detailed information about maximal sedation depth should be a mandatory reported element in such studies. The reporting of continuous capnography in such research is also highly desirable in order to quantify the degree of associated hypoventilation and respiratory depression.

• If clinically significant aspiration during ED PSA occurs, the involved EP should ensure that the complication and its circumstances are promptly reported in the literature so that others can learn from this experience. If concerns regarding anonymity prevent such action, the EP in question should refer the occurrence to other EPs (e.g., established sedation researchers) for confidential reporting.

CONCLUSIONS

The risk of aspiration during ED PSA appears extremely low, and that the literature provides no compelling evidence to support specific presedation fasting periods for either liquids or solids. Existing fasting guidelines for elective patients are of necessity arbitrary and based upon consensus opinion. Despite this, EPs should maintain the safety systems already in place and control sedation depth to minimize aspiration risk. Noncompliance with the ASA/AAP elective procedure fasting guidelines is not a contraindication to PSA in the ED. Emergency physicians should continue to assess the timing and nature of oral intake prior to PSA, and balance the remote potential for aspiration with the timing and urgency of the procedure at hand and the anticipated sedation depth.

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