



Procedural Sedation and Analgesia

K E E P I N G O U R
P A T I E N T S S A F E

Reference Manual for *The Safe and Effective PSA Course*

M I C H A E L D A R E

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Overview of PSA

The use of procedural sedation and analgesia (PSA) has expanded across multiple disciplines in the last two decades. The accumulation of evidence showing that PSA can be safely and effectively delivered in a variety of locations and by a variety of professions is growing. The pressures driving this change are multifaceted. As health care costs continue to rise, new ways of more economically managing patients have evolved. Many procedures and short surgeries previously requiring overnight admission into hospital can now be done in a short time with a quick recovery and discharge, sometimes in a matter of hours. Another driving force is the evolution of patient care to better manage pain and anxiety during procedures, the goal being minimal physiological and psychological stress and enhanced patient comfort.

Statement of Need

With the tremendous growth of procedures being performed under PSA along with the fact that PSA is being undertaken in an increasing variety of out-of-hospital locations, it is essential for all health care providers involved with this type of care to understand all aspects of PSA and how to respond to adverse patient events when they occur. Today, a variety of healthcare professionals are involved in sedation: nurses, paramedics, physician assistants, non-anesthesia physicians, dentists, and respiratory therapists. The need for regular training has been established within multiple international guidelines and standards. The College of Physicians and Surgeons of BC states that qualified and regulated health care professionals other than anesthesiologists who administer IV sedation in non-hospital medical and surgical facilities must hold current ACLS training, participate in emergency mock drills at least every six months, and that they complete an airway management and/or procedural sedation management course⁽¹⁾. In-hospital staff follows similar standards in most institutions, though provincial guidelines are lacking. Guidelines at present vary from one health authority to another and from one hospital to another. Regardless, the need to maintain core competencies to care for PSA patients in in-hospital settings is required by staff working in a variety of areas such as ER, ICU, CCU, daycare surgery, and radiology and is set forth as a core principle in multiple guidelines developed worldwide, all with the goal of safely and effectively delivering PSA to patients.

Learning Objectives for the Safe and Effective PSA Course

This manual is a reference manual for the *Safe and Effective PSA Course* which is presented in a 1-3 day format depending on the audience that is taking it. The goals of the course are as follows:

- Understand definitions relating to PSA.
- Understand the pharmacology of common PSA agents.
- Understand the process of patient evaluation, safe PSA delivery, and post procedure recovery.
- Understand the anatomy, physiology, and pharmacology that lead to adverse patient events in PSA.
- Understand how to avoid adverse events and how to respond to PSA emergencies.

- Understand special considerations for unique patient populations such as the elderly, pediatrics, and patients with specific medical issues.
- Understand the parameters of patient monitoring used in PSA and demonstrate that understanding during hands on case based scenarios.
- Demonstrate in case based simulations the following key knowledge and skills:
 - ECG interpretation of key life threatening arrhythmias.
 - The ability to interpret vital sign parameters, including pulse oximetry and exhaled carbon dioxide and respond appropriately.
 - Vascular access alternatives such as intraosseous access.
 - Basic and advanced airway management appropriate to participant's level of training (RT, MD, RN, LPN, dentist).
 - Resuscitation skills such as CPR, defibrillation, cardioversion, and pacing.

To assist the learner to reach these goals the course is delivered in a very interactive manner with short didactic talks, group discussions, and hands on simulations. The course provides a low stress, fun and interactive environment that foster learning, positive team building, and critical thinking skills.

Section 1: PSA Delivery

Chapter 1: Understanding Procedural Sedation and Analgesia

Definition of Sedation

Procedural sedation refers to a technique of administering sedatives or dissociative agents with or without analgesics to induce a state that allows the patient to tolerate unpleasant procedures while maintaining cardiorespiratory function⁽²⁾. The objective is that the patient experiences a depressed level of consciousness but still maintains oxygenation and airway control independently.

Levels of Sedation

The American Society of Anesthesiologists (ASA) defines the levels of sedation as follows⁽³⁾:

1. **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.
2. **Moderate sedation/analgesia** (conscious sedation) – a drug-induced depression of consciousness during which patients respond purposefully to verbal commands, either alone or accompanied by light tactile stimulation. No interventions are required to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained.
3. **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.
4. **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.
5. **Dissociative Sedation.** The American College of Emergency Physicians also defines a level of sedation called dissociative sedation which is a trancelike cataleptic state characterized by profound analgesia and amnesia with retention of protective airway reflexes, spontaneous respirations and cardiopulmonary stability. Ketamine is the only approved dissociative agent⁽⁴⁾.

Table 2.1. Continuum of depth of sedation.

	Minimal Sedation (Anxiolysis)	Moderate Sedation or Analgesia (Conscious Sedation)	Deep Sedation or Analgesia	General Anesthesia
Responsiveness	Normal Response to Verbal Stimulation	Purposeful* response to verbal or tactile stimulation	Purposeful response after repeated or painful stimulation	Unarousable, even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous Ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular Function	Unaffected	Usually maintained	Usually maintained	May be impaired

Source: American Society of Anesthesiologists Task Force on Sedation and Analgesia by Non-Anesthesiologists ⁽³⁾

Background History of Sedation

The practice of sedation has evolved significantly over the last several decades. Once under the direct control of anesthesiology with legal responsibility for all sedation falling upon this specialty, expertise in procedural sedation is now part of the core competencies of several physician specialties (emergency medicine and gastroenterology for example) and several other professions (physician assistants, dentistry, nurse practitioners, and nurse anesthetists). As studies demonstrate the safety of performing various levels of sedation in both hospital and non-hospital settings, the scope of practice of providers has expanded and the demand for different procedures to now include sedation has grown.

Recent technological and pharmacological advances have drastically changed the practice of sedation. Pulse oximetry was developed during World War II but did not evolve into a common tool used in medicine until the early 1980's ⁽⁵⁾. It revolutionized the safety of procedural sedation. By measuring the hemoglobin oxygen saturation, a wealth of information was provided to the clinician. Later capnography, the continuous monitoring of exhaled CO₂, became the stand of care for all patients undergoing general anesthesia. ASA established it as a standard of basic anesthetic monitoring in 1999. In 2010 ASA amended standards for basic anesthetic monitoring and included a statement relating to monitoring exhaled CO₂ during sedation. "During moderate or deep sedation the adequacy of ventilation shall be evaluated by continual observation of qualitative clinical signs and monitoring for the presence of exhaled carbon dioxide unless precluded or invalidated by the nature of the patient, procedure, or equipment."⁽⁶⁾ Capnography and pulse oximetry together could have helped in the prevention of 93% of avoidable anesthesia mishaps according to a closed claim study done by ASA.

Pharmacological advances have led to several milestones in the evolution of sedative medications. Early drugs generally had a slower onset of action and a prolonged duration of action along with having many undesirable side effects. Their use exposed patients to increased risks in comparison to modern agents, for example long exposure to sedative action and associated risks such as nausea, vomiting and airway compromise. One of the most common modern sedative medications is midazolam⁽⁷⁾. It has a quick onset of action, a short duration of action, and a very favorable safety profile. Although it lacks analgesic properties it provides very good amnesia when dosed correctly. Currently the opioid of choice for many practitioners to provide analgesia is fentanyl. It also has a short duration of action and a rapid onset of action^(7, 8, 9). Propofol can be used to induce general anesthesia, but subhypnotic doses have benefits of titratable sedation with rapid onset/offset. Its antiemetic properties are especially beneficial in the ambulatory setting, but it should be noted that it lacks analgesic effects^(7, 8).

In summary the practice of sedation has evolved over the last 30 years. At one time it was only the task of anesthesia personnel to provide sedation, but now multiple professions have developed their own practice guidelines and standards. They have been able to provide PSA to patients who need it in a variety of settings in a safe and effective manner. Controversies do exist between professions tasked with delivering sedation. Current ASA guidelines state that anesthesiology should oversee the credentialing of physicians for sedation privileges within institutions⁽³⁾. The American College of Emergency Physicians in turn published guidelines for PSA in 2005 stating that their profession would credential their own physicians, as airway management and procedural sedation were core competencies within their practice⁽⁴⁾.

The Sedation Continuum

Although the intent is to maintain the patient in a state of self-maintained cardiopulmonary function, the medications used and the patient's response to them may result in deeper sedation than intended. This places the patient at risk for airway and cardiopulmonary compromise. The important concept to remember here is that sedation is a continuum, and that patient responses may be unpredictable despite careful titration and monitoring. ASA states that if a patient's level of sedation progresses to a stage that is deeper than originally planned, the practitioner should be able to rescue the patient from the deeper level of sedation⁽³⁾. For example, individuals who administer moderate sedation/analgesia (formerly known as "conscious sedation") should be able to rescue patients who enter a state of deep sedation/analgesia, and those administering deep sedation/analgesia should be able to rescue patients who enter a state of general anesthesia. Practitioners must be able to respond with the appropriate knowledge and skills to handle these emergencies.

Indications for PSA

A procedure doesn't have to be painful to induce fear and anxiety in our patients. Previous experience with a noxious procedure or fear of the unknown can all contribute to how well the patient is able to tolerate our interventions.

Goals for IV Procedural Sedation

- Provide the patient with a safe environment where a painful or unpleasant procedure is required.
- Alleviate patient anxiety.
- Minimize physical discomfort.
- Maximize amnesia.
- Control motor behavior and movement if necessary so as to perform painful/unpleasant procedures such as a lumbar puncture or fracture reduction.
- Minimize the risk of the procedure, and ensure safe discharge of the patient.

Potential Indications for Procedural Sedation and Analgesia

- Colonoscopy
- Upper GI endoscopy
- Esophageal Dilatation
- Savory -Gilliard Dilators
- Foreign Body Removal
- Sclerotherapy
- Variceal banding
- Injection gold probe
- Peg tube insertion
- Polypectomy
- Bronchoscopy
- Trans-esophageal Echocardiogram (TEE)
- Cardioversion
- Cataracts
- Therapeutic Dilatation and Curettage
- Dilatation and Curettage
- Hysteroscopy
- Arteriogram
- Chest tube insertion
- Limb manipulations
- Pacemaker generator change
- Endo-bronchial Ultrasound (EBUS)
- ERCP Endoscopic Retrograde Cholangiopancreatography (ERCP)
- Wound care management
- Angioplasty
- Embolization
- Fallopian tube recannulization
- Tunneled line placement
- Drainage tube placement
- Dental procedures
- Insertion of venous access devices
- Manual removal of placenta
- Cystoscopy
- Colonic dilation
- Flexible sigmoidoscopy
- Intraocular lens insertion
- Blepharoplasty
- Ptosis

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1. IV Procedural Sedation and Analgesia for Adults Guideline (2009). Non-Hospital Medical and Surgical Facilities Program Committee. *College of Physicians and Surgeons of British Columbia*
2. American College of Emergency Physicians. Clinical policy for procedural sedation and analgesia in the emergency department. *Ann Emerg Med*. 1998;31:663-677.
3. American Society of Anesthesiologists Task Force on Sedation and Analgesia by Non-Anesthesiologists. Practice guidelines for sedation and analgesia by non-anesthesiologists. *Anesthesiology* 2002; 96: 1004-17.
4. American College of Emergency Physicians. Clinical policy on procedural sedation and analgesia in the emergency department. *Ann Emerg Med*. 2005;45:177-196.
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7. Watson DS, Odom-Forren J. Practical Guide to Moderate Sedation/Analgesia, 2nd edn. New York, NY: Mosby, 2005.
8. American Association of Nurses. Policy Statement on Conscious Sedation. Washington, DC: AAN, 1991.
9. Cravero JP, Blike GT. Review of pediatric sedation. *Anesth Analg* 2004; 99: 1355-64.

Chapter 2: PSA Guidelines, Policies, and Recommendations across Specialties

PSA is delivered through a wide variety of sedation providers and medical subspecialties. Because of this there has been no consensus on a universally accepted set of guidelines to guide practice. While there are differences in opinion and descriptions when discussing the specifics of PSA care, the common elements and considerations largely outweigh the differences. These documents range from those that contain broad descriptions of appropriate monitoring and treatment to those that offer specific guidelines on the use of particular drugs or NPO intervals. The methodologies used to produce these guidelines vary. The most recent guidelines from the ASA and the American College of Emergency Physicians (ACEP) are an evidence-based review of current sedation literature. Standards across Canada mirror the commonalities of most of the major American standards.

This manual is based on the most current PSA standards at the time of writing. Health care professionals should regularly review standards and their governing body's regulations to stay current. The following is a list of some of the available guidelines and standards that have been published relating to PSA.

Key American Guidelines and Policy, Position Statements

ASA Policies and Recommendations

The ASA has produced more statements and guidelines that relate to nonanesthesia PSA providers than any other organization. They include the following:

- Standards for basic anesthetic monitoring (2011). Available online at www.asahq.org/For-Healthcare-Professionals/Standards-Guidelines-and-Statements.aspx.
- Statement on anesthetic care during interventional pain procedures for adults (2010). Available online at www.asahq.org/For-Healthcare-Professionals/Standards-Guidelines-and-Statements.aspx.
- Statement on granting privileges for deep sedation to non-anesthesiologist sedation practitioners (2010). Further information from ASA (www.asahq.org).
- Continuum of depth of sedation: definition of general anesthesia and levels of sedation/analgesia (2009). Available online at www.asahq.org/For-Healthcare-Professionals/Standards-Guidelines-and-Statements.aspx.
- Distinguishing monitored anesthesia care ("MAC") from moderate sedation/analgesia (conscious sedation) (2009). Available online at www.asahq.org/For-Healthcare-Professionals/Standards-Guidelines-and-Statements.aspx.
- Guidelines for office-based anesthesia (2009). Available online at www.asahq.org/For-Healthcare-Professionals/Standards-Guidelines-and-Statements.aspx.

- Standards for postanesthesia care (2009). Available online at www.asahq.org/For-Healthcare-Professionals/Standards-Guidelines-and-Statements.aspx.
- Statement on qualifications of anesthesia providers in the office-based setting (2009). Available online at www.asahq.org/For-Healthcare-Professionals/Standards-Guidelines-and-Statements.aspx.
- Statement on safe use of propofol (2009). Available online at www.asahq.org/For-Healthcare-Professionals/Standards-Guidelines-and-Statements.aspx.
- Statement on nonoperating room anesthetizing locations (2008). Available online at www.asahq.org/For-Healthcare-Professionals/Standards-Guidelines-and-Statements.aspx.
- Guidelines for ambulatory anesthesia and surgery (2008). Available online at www.asahq.org/For-Healthcare-Professionals/Standards-Guidelines-and-Statements.aspx.
- Statement on granting privileges for administration of moderate sedation to practitioners who are not anesthesia professionals (2006). Available online at www.asahq.org/For-Healthcare-Professionals/Standards-Guidelines-and-Statements.aspx.
- Statement on granting privileges to nonanesthesiologist practitioners for personally administering deep sedation or supervising deep sedation by individuals who are not anesthesia professionals (2006). Available online at www.asahq.org/For-Healthcare-Professionals/Standards-Guidelines-and-Statements.aspx.
- Practice guidelines for sedation and analgesia by non-anesthesiologists (2002). Anesthesiology 2002; 96: 1004–17. Available online at www.asahq.org/For-Healthcare-Professionals/Education-and-Events/Guidelines-for-Sedation-and-Analgesia-by-non-anesthesiologists.aspx.
- Practice advisory for preanesthesia evaluation (2002). Anesthesiology 2002; 96: 485–96.

The Sedation Practice Guidelines for Practitioners who are not Anesthesiologists is probably the most widely quoted document concerning sedation that the ASA has produced. The latest version of this document was published in 2002 as a revision of the original 1995 guideline. The purpose of this guideline was to “allow clinicians to provide their patients with the benefits of sedation/ analgesia while minimizing the associated risks.”⁽¹⁾ The guideline describes levels of sedation that have been universally adopted. They require PSA providers to be able to rescue a patient to one level deeper than the intended level of sedation. They give recommendations for NPO times for elective PSA: 2 hours for clear fluids, 4 hours for breast milk, 6 hours for light meals and formula, and 8 hours for full meals.

ASA provides guidelines for patient monitoring during sedation. ASA recommends ECG, blood pressure, and pulse oximetry for all deep sedation patients. Continual monitoring of sedation depth through stimulation-response analysis is recommended. Until 2011, the ASA emphasized but did not require capnography, stating that capnography should be considered, but is not required, for all patients receiving deep sedation and for patients whose ventilation cannot be directly observed during moderate sedation. The ASA updated in July, 2011 the Standards for Basic Anesthetic Monitoring making for the first time end-tidal carbon dioxide monitoring a standard of care for moderate as well as deep sedation.

In 2005 the ASA produced the "Statement on granting privileges for administration of moderate sedation to practitioners who are not anesthesia professionals." ⁽²⁾ It defines the different groups and qualifications of PSA providers: anesthesia professionals (anesthesiologist, certified registered nurse anesthetist, anesthesiologist assistant), non-anesthesiologist practitioner (other physicians, dentists, podiatrists), and the supervised sedation professional (licensed registered nurse, advanced practice nurse, etc.). This grouping has raised some controversy, as the term "nonanesthesiologist" can represent physicians of various levels of skill, training, and experience.

The ASA defines the rescue capabilities that are required for sedation providers at each level of sedation. For deep sedation it recommends that the nonanesthesiologist be able to bag-valve-mask ventilate, insert an oral pharyngeal airway, a laryngeal mask airway, and perform an endotracheal intubation. Practitioners should be familiar with the use and interpretation of capnography. Deep sedation of children and adults requires PALS and ACLS certification as well as separate education training and credentialing ⁽²⁾.

The ASA "Statement on the Safe Use of Propofol" first published in 2004 and amended in 2009, advises that "the involvement of an anesthesiologist in the care of every patient undergoing anesthesia is optimal. However, when this is not possible, non-anesthesia personnel who administer propofol should be qualified to rescue patients whose level of sedation becomes deeper than initially intended and who enter, if briefly, a state of general anesthesia."⁽³⁾

The Joint Commission on Accreditation of Health Care organizations (JCAHO)

Current accreditation for all hospitals and other healthcare organizations in the United States is handled through a body called the Joint Commission. Their handbook, The JCAHO 2007 Comprehensive Accreditation Manual for Hospitals was intended to set the standards for sedation and anesthesia care for patients in any setting. There are recommendations for the training that must be provided to nonanesthesia sedation providers: "Individuals administering moderate or deep sedation and anesthesia are qualified and have the appropriate credentials to manage patients at whatever level of sedation or anesthesia is achieved, either intentionally or unintentionally." Referring specifically to deep PSA it states, "Individuals must be qualified to rescue patients from general anesthesia and are competent to manage an unstable cardiovascular system as well as a compromised airway and inadequate oxygenation and ventilation." It goes on to specify "Each organization is free to define how it will determine that the individuals are able to perform the required types of rescue. Acceptable examples include, but are not limited to, ACLS certification, a satisfactory score on a written examination developed in concert with the department of anesthesiology, a mock rescue exercise evaluated by an anesthesiologist." ⁽⁴⁾

Key Joint Commission Publications:

- Accreditation handbook for office-based surgery: what you need to know about obtaining accreditation (2011). Available online at www.jointcommission.org/assets/1/18/2011_OBS_Hdbk.pdf.
- Comprehensive accreditation manual for hospitals (CAMH): the official handbook (2011 update). Available online at www.jcrinc.com/Joint-Commission-Requirements/Hospitals.

American College of Emergency Physicians (ACEP)

ACEP has put forward several policy statements relating to PSA. Several statements were of a "push back" nature where the ACEP exerted their own authority over self-regulation in response to positions of the ASA. "Emergency physicians and nurses under their supervision are qualified to provide procedural sedation/analgesia in the emergency department, and ACEP is the authoritative body for the establishment of guidelines for procedural sedation and analgesia (PSA) by emergency physicians."⁽⁵⁾ Their clinical policy, "Procedural sedation and analgesia in the emergency department" revised in 2005 attempted to "remove the bias from recommendations for procedural sedation by creating a document that is, to the degree possible, evidence based. There remains a relative lack of high-quality data in some areas of procedural sedation."⁽⁵⁾ One major deviation from the ASA guidelines relates to the ASA NPO guideline. In the last 10 years, there have been several studies in the emergency medicine literature that have reported very low rates of aspiration or pulmonary complications in patients who were sedated without meeting the NPO recommendations from the ASA ^(6,7). Previous publications from the ACEP have concluded that there is insufficient evidence to conclude that fasting actually changes outcome for sedation ⁽⁸⁾.

Key ACEP Publications:

- ACEP policy statement: Sedation in the emergency department (2011 revision). Available online at www.acep.org/policystatements.
- Clinical policy: Procedural sedation and analgesia in the emergency department (2005). Ann Emerg Med 2005; 45: 177-96. Available online at www.acep.org/clinicalpolicies.
- Policy statement: Delivery of agents for procedural sedation and analgesia by emergency nurses (2005). Ann Emerg Med 2005; 46: 368. Available online at www.acep.org/policystatements.

Key Canadian and BC Guidelines and Policy, Position Statements

The Canadian Anesthesiologists Society

- Guidelines to the Practice of Anesthesia (Revised Edition 2013). Available online at http://cas.ca/English/Page/Files/97_Guidelines_2013.pdf
- Position Paper on Procedural Sedation: An Official Position Paper of the CAS (2013). Available at http://cas.ca/English/Page/Files/97_Appendix%206.pdf

College of Physicians and Surgeons of British Columbia (CPSBC)

The CPSBC published a concise guideline on PSA for non-hospital medical and surgical facilities in 2009 that gives guidance based largely on ASA guidelines. It gives information on required practitioner qualifications, training, staffing requirements, and practice guidelines for delivery of PSA. It closely mirrors ASA guidelines.

- IV Procedural Sedation and Analgesia for Adults Guideline (2009). Available online at www.cpsbc.ca/files/u6/NHMSFP-IV-Sedation-for-Adults-Guideline.pdf

The College of Dental Surgeons of British Columbia

- Deep Sedation in Dentistry (Non-Hospital Facilities) (2008). Available online at <http://www.cdsbc.org/~ASSETS/DOCUMENT/Deep%20Sedation%20Services%20in%20Dentistry%20Aug2008.pdf>
- Minimal and Moderate Sedation Services in Dentistry (Non-Hospital Facilities) (2008). Available online at <http://www.cdsbc.org/~ASSETS/DOCUMENT/Minimal%20and%20Moderate%20Sedation%20Services%20in%20Dentistry%20Aug2008.pdf>

The College of Registered Nurses of British Columbia (CRNBC)

- Scope of Practice for Registered Nurses, Standards, Limits, Conditions (2013). Available online at www.crnbc.ca/Standards/Lists/StandardResources/433ScopeforRegisteredNurses.pdf

Child Health BC, A BC Children's Hospital Initiative

- Guidelines for Procedural Sedation and Analgesia (2011) Available online at <http://www.childhealthbc.ca/guidelines/category/14-procedural-sedation>

Other Guidelines and Policy, Position Statements

American Academy of Pediatrics (AAP) and American Academy of Pediatric Dentistry (AAPD)

Guidelines for monitoring and management of pediatric patients during and after sedation for diagnostic and therapeutic procedures: an update (2006). Pediatrics 2006; 118: 2587–602.

Available online at www.aapd.org/media/Policies_Guidelines/G_Sedation.pdf.

American Association of Critical-Care Nurses (AACN)

Position statement on the role of the RN in the management of patients receiving IV moderate sedation for short-term therapeutic, diagnostic, or surgical procedures (2002).

Available online at www.aacn.org/WD/Practice/Docs/Sedation.doc.

American Association of Nurse Anesthetists (AANA)

Position statement on the qualified providers of sedation and analgesia: considerations for policy guidelines for registered nurses engaged in the administration of sedation and analgesia (2003). Available from AANA (www.aana.com).

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2. Statement on Granting Privileges for Administration of Moderate Sedation to Practitioners who are not Anesthesia Professionals. (Approved by the ASA House of Delegates on October 25, 2005, and amended on October 18, 2006).
3. Statement on the Safe Use of Propofol. (Approved by the ASA House of Delegates on October 27, 2004, and amended on October 21, 2009).
4. Joint Commission for Accreditation of Hospitals (JCAHO). Operative or other high-risk procedures and/ or the administration of moderate or deep sedation or anesthesia. The Comprehensive accreditation manual for Hospitals: official handbook. Oakbrook Terrace, IL: JCAHO; 2007. p. PC-41.
5. American College of Emergency Physicians. Clinical policy on procedural sedation and analgesia in the emergency department. *Ann Emerg Med.* 2005;45:177-196.
6. Roback MG, Bajaj L, Wathen JE, Bothner J. Preprocedural fasting and adverse events in procedural sedation and analgesia in a pediatric emergency department: are they related? *Ann Emerg Med.* 2004;44(5): 454– 9.
7. Agrawal D, Manzi SF, Gupta R, Krauss B. Preprocedural fasting state and adverse events in children undergoing procedural sedation and analgesia in a pediatric emergency department. *Ann Emerg Med.* 2003;42(5): 636– 46.
8. Mace SE, Brown LA, Francis L, et al. Clinical policy: critical issues in the sedation of pediatric patients in the emergency department. *Ann Emerg Med.* 2008;51(4): 378– 399, 399 e1– 57.

Chapter 3: Pharmacology of PSA

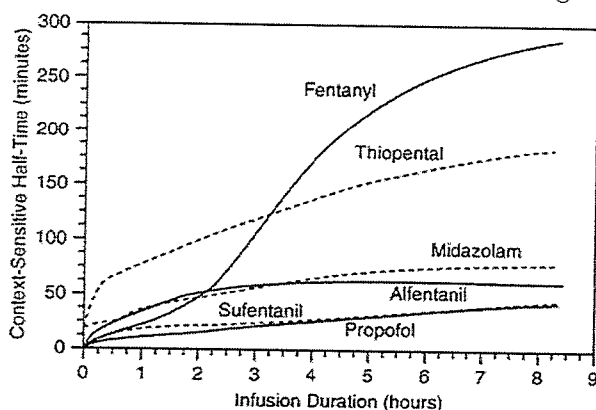
Introduction

The numbers of procedures requiring PSA has increased dramatically. Qualified non-anesthesia providers are initiating PSA to patients for a variety of diagnostic, therapeutic, and surgical procedures. The settings in which PSA is being administered are also diverse. With the goal of providing patients with the benefits of sedation and analgesia while minimizing the risks associated with PSA practitioners should understand the pharmacology of the agents used in PSA. They should understand the role and use of pharmacologic antagonists along with the use of sedative/analgesic combinations administered as appropriate for the patient's condition and the procedure being performed. This chapter focuses on the pharmacology of the medications used for moderate and deep sedation.

Pharmacology Basics

- **Agonist:** a drug that interacts and binds with a receptor site and activates that receptor.
- **Antagonist:** a drug that binds with a receptor site without activating the receptor while simultaneously blocking an agonist from activating the receptor.
- **Synergism:** the effect of two drugs given together is greater than the sum effect of the two drugs. This is often seen when opioids and benzodiazepines when they are used in combination.
- **Pharmacokinetics:** refers to the properties such as onset of action and duration of action of a drug. It describes the absorption, distribution, metabolism and elimination of a drug. This is affected by the route of administration.
- **Pharmacodynamics:** refers to the responsiveness of receptors to a medication and the mechanism through which these effects occur.
- **Volume distribution:** the distribution of a medication between plasma and the rest of the body when a dose is administered. It is influenced by the characteristics of the drug such as lipid solubility, binding to plasma proteins, ionization rate, and molecular size.
- **Elimination half-life:** the time it takes for plasma concentration of a drug to reach 50% of its original concentration. Because elimination half-life is directly proportional to volume distribution and inversely proportional to its clearance, renal and hepatic disease greatly influences elimination half-life and the duration of action a medication may have for a patient.
- **Context Sensitive half-life:** Elimination half-life does not always explain how quickly the action of a drug will wear off especially when multiple boluses are given or infusions are used.

Figure 3.1. Context sensitive half-life of drugs used in sedation



Source: Principles and Practice of Hospital Medicine (2012). McGraw-Hill Medical

Figure 3.2. Drug classes commonly used in sedation and their effects

Drug Class	Sedation Effect	Anxiolysis Effect	Pain Control	Cardiovascular Depression	Respiratory Depression
Benzodiazepines	++	++	-	+	-
Opioids	+	+	++	+	++
Propofol	++	+	-	++	++
Ketamine	++	+	+	-	-

The ideal pharmacological agent or agents used in combination for moderate and deep procedural sedation would be able to produce optimal sedation and analgesia quickly, have a short duration of action to facilitate a quick recovery without recollection of the procedure, and would not have any adverse side effects such as respiratory depression. No drug or drug combination presently in use has this perfect profile.

Common Agents Used in PSA

Midazolam

This drug is a short acting, water-soluble benzodiazepine possessing the properties of sedation, amnesia, and anxiolysis. It is also an anticonvulsant and muscle relaxant. It has replaced all other benzodiazepines for the most part that were used in moderate and deep sedation^(1,2). It is metabolized by the liver and excreted by the kidneys.

- Adult dosing:
 - 0.02-0.1 mg/kg initial dose. Max single dose 2.5 mg, 1.5 mg in elderly.
 - May repeat 25% of initial dose every 3 minutes to a maximum of 5.0 mg cumulative dose, 3.5 mg in the elderly.
 - Given by direct IV push over 2 minutes.
- Pediatric dosing:
 - 0.05-0.1 mg/kg IV every 3 minutes.
 - Do not exceed cumulative dose of 0.4 mg/kg or 6.0 mg.
 - Oral dose 0.5-0.75 mg/kg.

- Onset of action: 1.5-5 min, peak within 10-15 minutes.
- Duration of action: 30-60 minutes.
- Adverse effects: respiratory depression and apnea at higher doses, hypotension, diminished reflexes, and impaired coordination.
- Special considerations and pearls:
 - Wait a minimum of two minutes after a dose to assess the patient's response.
 - Midazolam has no analgesic properties.
 - If pre-medicated with an opioid dose should be reduced by 25%.
 - When given with other CNS depressants may increase sedation and risk of respiratory depression.
 - When used in combination with fentanyl, respiratory depression may occur in up to 25% of patients.

Fentanyl

This drug is a very potent synthetic opioid that is 100 times stronger than morphine. It has favorable characteristics for use in PSA such as a rapid onset of action, short duration of action, and less of a negative impact on the cardiovascular system than other opioids. It is the analgesic of choice in PSA when used in combination with sedatives such as midazolam, propofol, or etomidate. It produces no active metabolites and causes much less of a histamine release compared to morphine, hence less hypotension from vascular vasodilation.

- Adult/Pediatric dosing:
 - 1-1.5 mcg/kg initial dose.
 - Subsequent doses of 1.0 mcg/kg every 3 minutes.
 - Given by slow IV push over 15 seconds.
- Onset of action: 1-2 minutes.
- Duration of action: 30-60 minutes.
- Contraindications:
 - Severe respiratory depression or distress.
 - Increased ICP.
- Adverse effects: respiratory depression and apnea, bradycardia, hypotension, nausea and vomiting, pruritus, muscle and glottis rigidity, and euphoria.
- Special considerations and pearls:
 - Rapid IV administration may cause muscle rigidity, severe respiratory depression, or cardiovascular collapse.
 - Respiratory compromise can be fully reversed with naloxone but hypotension may not be fully reversed due to histamine release.
 - Pruritus can be managed with antihistamines.

Propofol

Propofol is a very short acting sedative hypnotic agent. It has sedative, amnestic, anticonvulsant, and antiemetic properties. The exact mechanism of propofol is unknown though one theory is that it increases the binding of GABA to its receptor sites. Much of the controversy over which specialty (does each specialty self-govern their use of PSA or is it under the control of anesthesiology) oversees PSA within a hospital relates to this agent in that it is used for the induction and maintenance of general anesthesia when given at higher doses. It should be noted that propofol has no analgesic properties so in PSA it is often used in combination with fentanyl ⁽³⁾. Some of the benefits of propofol use are its rapid onset of action and ultra-short duration of action which results in a rapid recovery of patients, faster than with agents such as midazolam ⁽⁴⁾. However due to its

potency there is the risk of obtaining much deeper levels of sedation than desired including inducing full general anesthesia accidentally.

- Adult/Pediatric dosing:
 - 0.5-1 mg/kg bolus IV over 3-5 minutes.
 - May repeat at 0.5 mg/kg IV every 3-5 minutes.
- Onset of action: Less than 1 minute.
- Duration of action: 5-10 minutes, full recovery within 10-15 minutes.
- Pharmacokinetics: The drug is rapidly redistributed to muscle and fat tissue and achieves therapeutic concentrations in the CNS very quickly.
- Adverse effects:
 - Hypotension can occur in up to 30% of patients.
 - Respiratory depression or apnea can occur in up to 25% of patients.
 - Bradycardia.
 - Pain at the site of injection (can be alleviated by mixing with small amounts of lidocaine- 0.25 mg/kg).
- Special considerations and pearls:
 - Strict aseptic practice is necessary when using propofol as the lipid emulsifier can support bacterial growth.
 - Does not have any analgesic properties.
 - Level of sedation can change quickly to deeper levels so careful monitoring is necessary.
 - If hepatic failure, patient may require lower dosing.
 - Contains soybean oil and egg yolk lecithin. Avoid in patients with allergies to those compounds.

Etomidate

This medication is an ultra-short acting sedative hypnotic with both amnesic and anesthetic properties. It has a very rapid onset of action and short recovery time similar to propofol. It also has minimal effect on the respiratory system, and due to the fact that it does not promote histamine release it also has minimal effect on blood pressure. The main use of etomidate is as an induction agent. It is often used in emergency settings for rapid sequence intubation. It has an interesting profile that makes it a good agent when patients are hemodynamically unstable and it is one of only a few anesthetic agents that lowers intracranial pressure while maintaining normal arterial pressures⁽⁶⁾. It is gaining in popularity in PSA for short procedures such as orthopedic reductions and cardioversion^(6, 7).

- Adult/Pediatric dosing:
 - 0.1-0.2 mg/kg IV bolus over 30-60 seconds.
 - 0.05 mg/kg IV for subsequent doses if needed.
- Onset of action: Less than 1 minute.
- Duration of action: 3-5 minutes, full recovery within 5-15 minutes.
- Adverse effects:
 - Pain at site of injection.
 - Transient myoclonus, uncontrolled eye movements. Push very slowly for rapid sequence intubation or trismus may occur
 - Temporary reduction in adrenal cortisol production for 4-8 hours.
 - Transient decrease in cerebral blood flow.
- Special considerations and pearls:
 - Consider administration of lidocaine for vein irritation.
 - Avoid use in patients with septic shock.

- Monitor for signs of adrenal insufficiency such as hyperkalemia and hypotension ⁽⁸⁾.
- Premedication with midazolam or fentanyl can decrease incidence of myoclonus.
- There is a 30-fold difference between the effective and the lethal dose of etomidate which makes it a very safe agent.

Ketamine

Ketamine produces a dissociative state and is a rapid acting anesthetic with very strong analgesic properties. This drug is not titratable for varying levels of action but instead a full dissociative state occurs when a certain dosage threshold is reached. This occurs usually at 1.0-1.5 mg/kg. Ketamine allows for full preservation of airway reflexes and it is one of few agents that increase heart rate, blood pressure, and cardiac output ⁽⁹⁾. It is also a bronchodilator. These characteristics make it useful when dealing with patients in shock states, when fasting status is in question, or for patients with severe reactive airway disease.

- Adult/Pediatric dosing: 1-2 mg/kg.
- Onset of action: 1 minute.
- Duration of action: 5-10 minutes.
- Contraindications: age < 3 months, cardiovascular disease, globe injuries, glaucoma, psychosis, thyroid problems, active pulmonary infection.
- Adverse effects: hypertension and tachycardia if rapidly administered, emergence reactions (approx. 12% in adults), excessive salivation, hypotension if catecholamine depleted, transient laryngospasm, and nausea and vomiting (6-7%).
- Special considerations and pearls:
 - Pretreatment with a benzodiazepine can potentially reduce emergence reactions by 50%. The validity of this treatment has recently being challenged. Serious emergence reactions are actually not common.
 - After initial dose the patient will most often have an approximately 20 second apneic episode. This will resolve and normal breathing will resume. Pre-oxygenate prior to administration.
 - Excessive salivation may be treated with atropine or glycopyrrolate.
 - Used for short procedures that do not require skeletal muscle relaxation.
 - Avoid if patient predisposed to nightmares or has history of psychosis.
 - Concomitant use of CYP3A4 inhibitors (azole antifungals, erythromycin, clarithromycin, verapamil, propofol) may increase levels of ketamine as will concomitant use of CYP2C9 inhibitors such as NSAIDs

Common Agent Combinations Used in PSA

The practice of combining agents to get the best PSA profile possible is a very common practice. The following special considerations should be reviewed whenever combination therapy is being used:

- The risk of adverse events is increased when combination drug therapy is used.
- The agent that poses the greatest risk of respiratory depression should always be given first.
- The longer acting of the two agents should be administered first.
- Allow for enough time to pass after administering the first agent to evaluate its effect before giving the second agent.

Fentanyl and Midazolam

The combination of opioids and benzodiazepines was the first combination therapy used in PSA. Midazolam and fentanyl have been used in such areas as the ER and ambulatory daycare for several decades and have a proven safety record. They combine the sedative, amnestic, and anxiolytic properties of benzodiazepines with the analgesic properties of opioids. Fentanyl should be administered first with sufficient time given to evaluate for respiratory depression before the midazolam is given. The risk of cardiac and respiratory depression increases when these agents are used together and respiratory depression can occur in up to 25% of patients. This combination is a good choice for moderate sedation, but if used for deep sedation there is a risk that as soon as the stimulus of the procedure itself is over the patient will then suffer from possible respiratory depression or even apnea until the medications wear off.

Dosing example: Give fentanyl slow IV over 15 seconds at a dose of 1 mcg/kg then wait one minute while evaluating respiratory status. Next dose the midazolam at 0.02 mg/kg slowly over 2 minutes. Repeat boluses of midazolam every 3 minutes as needed.

Propofol and Fentanyl

This combination is very popular when doing painful short procedures as they are often done in locations such as the emergency department. While propofol has potent sedative and amnesic properties it lacks any analgesic properties. When fentanyl is combined with propofol instead of midazolam the interval from the start of procedure to discharge is shortened significantly ^(10, 11, 12). Riphaus et al found that the mean recovery time as well as the quality of the recovery was superior using this combination (14 min versus 25 min) ⁽¹³⁾.

Dosing example:

- Give initial dose of fentanyl 1-1.5 mcg/kg over 15 seconds; wait 1 minute to assess respiratory depression.
- Next give propofol 0.5-1 mg/kg over 30-60 seconds.
- If needed give additional boluses of propofol of 0.5 mg/kg if patient is too light.
- You can use 20-40 mg of lidocaine to avoid injection site pain.

Ketamine and Propofol (Ketofol)

The first article describing the combination of ketamine and propofol for use outside the OR, particularly in the ER, was published in 2007. The authors from the University of British Columbia in Vancouver coined the term ketofol and mixed 0.5 mg/kg of both ketamine and propofol in one syringe reporting on PSA with ketofol of 117 patients ⁽¹⁴⁾. Their reported success rate was about 97% with a short time to recovery. Three patients developed hypoxia, and 3 developed an emergence reaction. They concluded that ketofol provides potent sedation, analgesia, and amnesia with a short duration of action, supporting hemodynamic and respiratory stability when used in the emergency room. Propofol provides excellent sedation but no analgesia. Ketamine provides both good sedation and analgesia but many practitioners are hesitant to use it outside of the pediatric population due to a higher incidence of emergence reactions reported in the adult population ⁽¹⁵⁾. The addition of ketamine to propofol provides for an analgesic effect while permitting a lower dose of each agent. Moreover, the two agents are complementary in their adverse effect profiles – propofol lowers blood pressure and pulse whereas ketamine raises both, propofol is an antiemetic while ketamine can cause

nausea and vomiting. The end result in theory is a potent sedative-analgesic-amnesic drug combination with a neutral hemodynamic profile and a very low rate of respiratory depression or airway compromise.

David and Sharp did a trial published in 2011 comparing propofol alone to propofol and ketamine given one after the other in separate syringes. Their patients were given a pre-dose of 0.5 mg/kg ketamine followed by 0.5mg/kg propofol titrated to sedation. This trial provided the most compelling evidence to date that the sedative effects of propofol and sub-dissociative ketamine are indeed synergistic, with ketamine bridging the gaps of sedation with propofol to provide a more consistent sedation depth ⁽¹⁶⁾.

Mixing- one syringe method:

- Drawl up 10ml of propofol in a 20cc syringe.
 - Propofol comes 10mg/1ml.
- Discard 2cc from a 10cc saline flush syringe. Draw up 2cc of ketamine.
 - Ketamine 50mg/ml (adjust the dose if a different concentration id used).
 - You now have 10mg/ml.
- Inject the Ketamine syringe into a 20cc syringe of propofol.
- There is now 20 ml of ketofol at a concentration of 5mg/ml- each ml having 5 mg ketamine and 5mg of propofol.

Dosing example:

- 0.5-0.75 mg/kg of propofol and ketamine (ketofol) as an initial bolus.
- This test dose shows how the patient is responding, within 30-45 seconds repeat at 0.5mg/kg if sedation is inadequate. Continue boluses at 1-1.5 minute intervals as needed at 0.5mg/kg.

Reversal Agents

Reversal agents should not be used to expedite recovery times and their routine use should be avoided. The duration and action of the following reversal agents is often shorter than the opioids and benzodiazepines that are used. This can lead to a patient relapsing into a deep state of sedation. Most guidelines state that a patient must be monitored for a minimum of 2 hours after the last dose of a reversal agent is given. A preferred method is to undertake a partial reversal with the reversal agent administered in small doses titrated to desired effect such as an increased LOC and an increased respiratory rate without conducting a full reversal. This often will avoid adverse events such as signs of acute withdrawal to opioids or benzodiazepines.

Naloxone

This agent is an opioid antagonist which is used to reverse respiratory depression, apnea, chest wall rigidity, pruritus, and hypotension from opioid use.

- Dose: 0.2mg IV may repeat every 3 minutes.
- Onset of action: Less than 1 minute.
- Duration of action: 15-30 minutes.
- Adverse effects: narcotic withdrawal, analgesic cessation.

Flumazenil

Flumazenil is used to reverse serious respiratory depression related to benzodiazepine use. It may precipitate seizures in some patients and panic attacks in those with an underlying panic disorder.

- Dose:
 - 0.2 mg IV slowly over 15 seconds.
 - May repeat at 1-minute interval.
 - Maximum cumulative dose is 1 mg.
- Onset: <1 minute.
- Duration of action: 30–45 minutes.
- Contraindications:
 - Hypersensitivity to flumazenil.
 - Use of benzodiazepines to control seizures or increased ICP.
 - Use with caution in patients who may be dependent on benzodiazepines or alcohol.
- Adverse effects:
 - Seizures.
 - Nausea/vomiting.
 - Hyperventilation.
 - Emotional lability, anxiety.
 - Sweating.

Summary

The safe and effective administration of PSA by non-anesthesia providers is occurring with increasing frequency in a variety of locations within and outside of the hospital setting. Providing PSA for the variety of patients and procedures requires knowledge of the commonly used drugs. An understanding of the pharmacology of the benzodiazepines and opioids as well as their reversal agents is important to assure patient safety. Other drugs used in sedation, such as propofol, ketamine, and etomidate warrant special attention. Furthermore, techniques to obtain the desired end point with drug administration without undesirable side effects should be kept in mind. Slow titration of PSA agents while monitoring the patient's response (level of consciousness, respiratory rate, blood pressure, etc.) dictates the dosage given. Drug choice should be based on the procedure and the patient pre-procedure assessment. Painful procedures require analgesia and possibly sedative-hypnotics, whereas non-painful procedures may only require sedation. Dosing of agents varies based on patient characteristics such as height, weight, and age. Comorbidities such as hepatic and/or renal failure also frequently affect dose requirements. Decreased doses are required when combining benzodiazepines and/or opioids with other CNS-depressive drugs. Decreased doses should also be used in the elderly and the debilitated.

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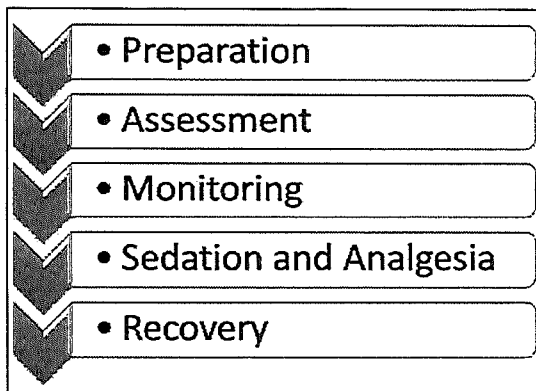
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Chapter 4: The Phases of PSA

Much of the structure relating to the process of delivering PSA revolves around the key concepts of delivering 1) effective PSA and 2) safe PSA. Section 2 focuses on understanding the safety issues involved, the human factors, and the risks for adverse outcomes. In this chapter we will look at a common model for the stages involved in delivering PSA. See Figure 4.1 for an overview.

Figure 4.1. Phases of PSA



Preparation

Before any PSA can be undertaken the structure of the PSA team, the physical location for PSA delivery, and equipment requirements must be addressed. For safe and effective PSA the following is needed:

- A proper location considering the type of procedures being undertaken and the patient population using the service.
- Adequate time schedule to perform the procedures and deliver PSA safely.
- Adequate staff with sufficient training to deliver PSA safely.
- Proper equipment and medications.
- An adequate emergency response protocol in place should an adverse event occur.

Patient Assessment

Patient selection for PSA has been mirrored on long established anesthesiology guidelines from organizations such as the ASA. Staff needs to obtain a history and perform a physical examination to identify medical illnesses, medications, allergies, and anatomical features that may affect safety such as if there is a need to provide emergency airway management. A formal screening questionnaire can be employed in more elective cases. Patient screening may start from the first phone contact by asking simple screening questions about snoring/sleep apnea, age, height and weight, the presence of common comorbid diseases such as diabetes, heart and lung disease, and routine use of narcotics or sedatives.

In certain situations and locations where PSA is being undertaken in a more emergent fashion pre-screening assessments are not possible. In these situations the first evaluation is done by the physician overseeing the sedation or one of the procedural RNs assisting. Information gathered should be standardized and include:

- Medication use.
- Exercise tolerance.
- Medical history.
- Surgical history.
- Allergies.
- NPO status.
- Drug intolerance.
- Social habits.
- Previous experiences with anesthesia, sedation, and other procedures.

It is important to uncover intolerances to medications, positioning issues, neck range of motion, sleep apnea/snoring, difficult intubation or any history of unexpected events during procedures or surgeries. The patient assessment should include a baseline measurement of a consciousness-sedation score (Fig. 4.2), which should also be monitored and documented during PSA and through recovery. In addition, it should include a baseline modified Aldrete score or the equivalent, so that the patient can be compared against baseline during PSA and while being recovered in order to determine criteria for discharge from post-sedation monitoring (Fig. 4.3).

Figure 4.2. Richmond Agitation-Sedation Scale (RASS)

Score	Description
+4	Combative, violent, a danger to staff
+3	Aggressive, pulls or removes tubes or catheters
+2	Frequent non-purposeful movements, fights ventilator
+1	Anxious, apprehensive, but not aggressive
0	Alert and calm
-1	Awakens to voice (eye opening/contact) for > 10 seconds
-2	Light sedation, briefly awakens to voice (eye opening/contact) for < 10 seconds
-3	Moderate sedation, movement or eye opening; no eye contact
-4	Deep sedation, no response to voice, but movement or eye opening to physical stimulation
-5	Unarousable, no response to voice or physical stimulation

Figure 4.3. The Modified Adult Aldrete Score

Criteria	Ability	Score
Activity	Able to move voluntarily or on command:	
	4 extremities	2
	2 extremities	1
	0 extremities	0
Respiration	Able to breath and cough freely	2
	Dyspnea; shallow or limited breathing, tachypnea	1
	Apneic	0
Circulation	BP +/- 20 mmHg of presedation level	2
	BP +/- 21-49 mmHg of presedation level	1
	BP +/- 50 mmHg of presedation level	0
Consciousness	Fully awake	2
	Arousable or calling	1
	Not responding	0
O ₂ Saturation	Able to maintain saturation > 92% on room air	2
	Needs oxygen to maintain saturation > 90%	1
	Oxygen saturation < 90% even with supplemental oxygen	0

Source: Based on Aldrete JA, Kroulik D. A postanesthetic recovery score. *Anesth Analg* 1970; 49: 924-34. A minimum score of 10 (or baseline), with no score less than 1 in any category is required for discharge from sedation monitoring.

Screening and assessment also involves classification of the patient by means of the ASA physical status classification. In hospital PSA by nonanesthesiologists is usually limited to ASA levels I-III. For non-hospital medical and surgical facilities CPSBC states that only ASA levels I and II patients should undergo PSA with some exceptions. "Only patients at categories I and II risk level as defined by the American Society of Anesthesiologists should normally be accepted in the facility. However, risk level Category III patients may be treated there if the patient's disease is not expected to be affected by the anesthetic."⁽¹⁾

pain 0-2
Nausea 0-2
Total:
14

Figure 4.4. ASA Patient Classification Status

Score	Classification	Examples
ASA-I	A normal healthy patient	No significant medical history
ASA-II	A patient with mild to moderate systemic disease.	Controlled hypertension, controlled diabetes, upper respiratory infection, smoking, thyroid tumor that does not threaten the airway. Pregnancy and extremes of age are sometimes included in this category.
ASA-III	A patient with severe systemic disease that limits normal daily activity.	chronic obstructive pulmonary disease, chronic stable angina, obesity (which is a multisystem disease), lung tumor that decreases pulmonary function
ASA-IV	A patient with incapacitating disease that is in constant threat to life.	Congestive heart failure, unstable angina, severe pulmonary or hepatic dysfunction, major trauma, prematurity with respiratory distress and necrotizing enterocolitis
ASA-V	A moribund patient not expected to survive 24 hours with or without surgery.	Ruptured aneurysm, major trauma, massive intracerebral injury.

Another key aspect of patient assessment is to evaluate the patient's airway anatomy to estimate the difficulty of airway management should an emergency airway crisis develop. This includes the completion of a Mallampati score and a C-L airway exam (Fig. 4.6) ^(2,3). Parts of these scores are incorporated into a commonly used mnemonic LEMON (Fig. 4.5) ⁽⁴⁾. LEMON was developed by Walls and Murphy in their Manual of Difficult Airway Management and has been externally validated.

Figure 4.5. Lemon Mnemonic

Letter	Refers to
L	Look externally- If it looks like a difficult airway it probably is. Bleeding, trauma, small jaw, large tongue, short neck, large teeth, obesity, agitation state, beard.
E	Evaluate the 3-3-2 rule: (Fig. 4.7) • Interior incisor distance – 3 finger breadths (less is worse) • Hyoid-mental distance – 3 finger breadths (less is worse) • Thyroid to floor of mouth – 2 finger breadths (more or less=bad)
M	Mallampati score (Classes 3 and 4 are difficult airway views)
O	Obstruction/obesity. Obstruction: Muffled voice, difficulty swallowing, stridor, sensation of dyspnea.
N	Neck Mobility - cervical spine immobilization, intrinsic spine immobility such as ankylosing spondylitis or rheumatoid arthritis can make intubation extremely difficult.

Figure 4.6. (A) Mallampati scoring classes during an airway examination (1–4); (B) Cormack and Lehane C-L Visualization grades during direct laryngoscopy (1–4)

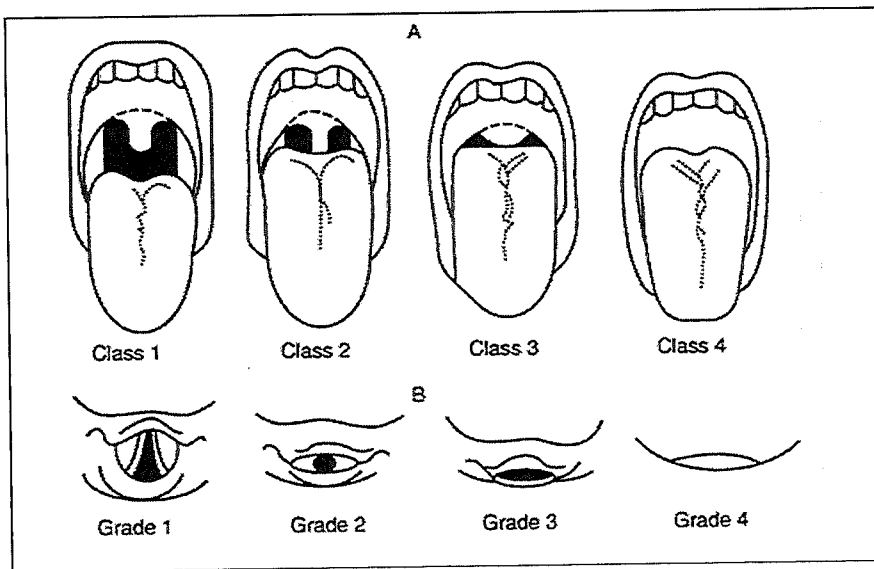
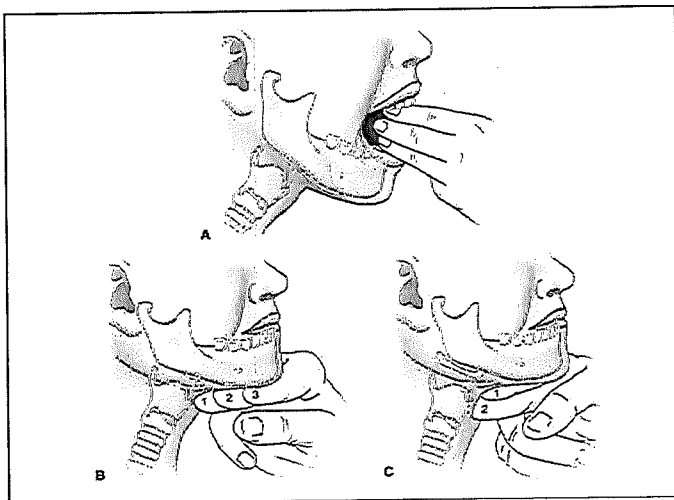


Figure 4.7. 3-3-2 Rule



The LEMON mnemonic helps the PSA team predict the difficulty of airway management if there is an emergency airway situation such as a planned deep sedation becoming one of general anesthesia. Another mnemonic, possibly even more important than LEMON is MOANS. The MOANS mnemonic lists the validated indicators of difficult bag-valve-mask (BVM) use.

Figure 4.8. MOANS mnemonic predicting difficulty of BVM use

Letter	Refers to
M	Mask seal/male face/Mallampati class 3-4. Trauma to the face, blood and debris, beards all can make getting a BVM seal difficult.
O	Obesity/Obstruction. BMI > 26 kg/m ² indicates likely difficulty using BVM.
A	Age > 55 yo, perhaps because of a loss of muscle and tissue tone in the upper airway.
N	No teeth. Leave dentures in situ if possible.
S	Stiff/Snoring - patients with reactive airway disease such as COPD, asthma, ARDS, and pulmonary edema where high pressures are needed to ventilate and get chest rise. History of snoring or sleep apnea.

One last assessment measurement is usually calculated for patients having PSA and that is Body Mass Index (BMI).

Figure 4.9. Body Mass Index Scale

WEIGHT lbs kgs	100 45.5	105 47.7	110 50.0	115 52.3	120 54.5	125 56.8	130 59.1	135 61.4	140 63.6	145 65.9	150 68.2	155 70.5	160 72.7	165 75.0	170 77.3	175 79.5	180 81.8	185 84.1	190 86.4	195 88.6	200 90.9	205 93.2	210 95.5	215 97.7	
HEIGHT in/cm																									
5'0" - 152.4	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	
5'1" - 154.9	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	
5'2" - 157.4	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	
5'3" - 160.0	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	
5'4" - 162.5	17	18	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	31	32	33	34	35	36	37	
5'5" - 165.1	16	17	18	19	20	20	21	22	23	24	25	25	26	27	28	29	30	30	31	32	33	34	35	35	
5'6" - 167.6	16	17	17	18	19	20	21	21	22	23	24	25	26	27	28	29	30	30	31	32	33	34	35	35	
5'7" - 170.1	16	16	17	18	18	19	20	21	22	22	23	24	25	26	27	28	29	29	30	31	32	33	34	34	
5'8" - 172.7	15	16	16	17	18	19	19	20	21	22	22	23	24	25	26	27	28	29	30	31	32	33	34	34	
5'9" - 175.2	14	15	16	17	17	18	19	20	21	22	22	23	24	25	26	27	28	29	30	31	32	33	34	34	
5'10" - 177.8	14	15	15	16	17	18	18	19	20	20	21	22	23	23	24	25	25	26	27	28	29	30	31	31	
5'11" - 180.3	14	14	15	16	16	17	18	18	19	20	21	22	23	23	24	25	25	26	27	28	29	30	31	31	
6'0" - 182.8	13	14	14	15	16	17	17	18	19	19	20	21	21	22	23	23	24	25	26	27	28	29	30	30	
6'1" - 185.4	13	13	14	15	15	16	17	17	18	19	19	20	21	21	22	23	24	25	26	27	27	28	29	29	
6'2" - 187.9	12	13	14	14	15	16	16	17	18	18	19	19	20	21	21	22	23	24	25	26	27	27	28	28	
6'3" - 190.5	12	13	13	14	15	15	16	16	17	18	18	19	20	21	21	22	23	23	24	25	26	27	27	27	
6'4" - 193.0	12	12	13	14	14	15	15	16	17	17	18	18	19	20	20	21	22	23	23	24	25	26	26	26	

BASED ON CLINICAL GUIDELINES ON THE IDENTIFICATION, EVALUATION, AND TREATMENT OF OVERWEIGHT AND OBESITY IN ADULTS. NHLBI OBESITY EDUCATION INITIATIVE. NATIONAL INSTITUTES OF HEALTH, 1998.

 Underweight (BMI less than 18.5)
 Healthy weight (BMI 18.5 to 24.9)
 Overweight (BMI 25 to 29.9)
 Obese (BMI 30 to 39.9)
 Extremely obese (BMI 40 and above)

Before proceeding with PSA the NPO status of the patient should be known or verified. Managing NPO status for elective PSA situations is not difficult. Patients undergoing PSA for elective procedures should be NPO for a sufficient period of time to allow for gastric emptying (Fig. 4.10).

Figure 4.10. Summary of ASA pre-procedure fasting guidelines

Ingested Material	Minimal Fasting Period
Clear liquids	2 hours
Breast milk	4 hours
Infant formula	6 hours
Nonhuman milk	6 hours
Light meal	6 hours

Managing the patient who is having a more urgent-emergent PSA undertaken is different. Recent literature reviews looking at whether pre-procedural fasting was necessary before initiating PSA found that there was no evidence to support the ASA guidelines⁽⁵⁾. The combination of vomiting and the loss of airway reflexes have been shown to be an extremely rare occurrence⁽⁶⁻¹²⁾. Much of the data on aspiration risks has been extrapolated from general anesthesia literature. In these circumstances the risk of aspiration is increased with the manipulation of the airway during intubation and extubation. ACEP's clinical policy on PSA states the following: "Although recent food intake is not a contraindication for administering procedural sedation and analgesia, the emergency physician must weigh the risk of pulmonary aspiration and the benefits of providing procedural sedation and analgesia in accordance with the needs of each individual patient."⁽¹²⁾ Higher risk for aspiration has been identified in patients with age > 75, higher ASA classifications, potential for prolonged or difficult ventilation, and conditions predisposing to GERD.

Figure 4.11. Fasting Guidelines from the ACEP 2007⁽¹³⁾

Oral intake in the prior 3 hours		Procedural Urgency ^a				Procedural sedation and analgesia targeted depth and duration ^c
	<i>Emergent Procedure</i>	<i>Urgent Procedure</i>	<i>Semi-Urgent</i>	<i>Non-Urgent</i>		
<i>Nothing</i>	All levels of sedation	All levels of sedation	All levels of sedation	All levels of sedation	↑ Increasing potential aspiration risk ↓ Minimal sedation only Dissociative sedation: brief or intermediate-length moderate sedation Extended moderate sedation Brief deep sedation Intermediate or extended-length deep sedation	
<i>Clear liquids only</i>	All levels of sedation	All levels of sedation	Up to and including brief deep sedation	Up to and including extended moderate sedation		
<i>Light snack</i>	All levels of sedation	Up to and including brief deep sedation	Up to and including dissociative sedation: non-extended moderate sedation	Minimal sedation only		
<i>Heavier snack or meal</i>	All levels of sedation	Up to and including extended moderate sedation	Minimal sedation only	Minimal sedation only		
Higher-risk patient^d						
Oral intake in the prior 3 hours		Procedural Urgency ^a				
	<i>Emergent Procedure</i>	<i>Urgent Procedure</i>	<i>Semi-Urgent</i>	<i>Non-Urgent</i>		
<i>Nothing</i>	All levels of sedation	All levels of sedation	All levels of sedation	All levels of sedation		
<i>Clear liquids only</i>	All levels of sedation	Up to and including brief deep sedation	Up to and including extended moderate sedation	Minimal sedation only		
<i>Light snack</i>	All levels of sedation	Up to and including dissociative sedation: non-extended moderate sedation	Minimal sedation only	Minimal sedation only		
<i>Heavier snack or meal</i>	All levels of sedation	Up to and including dissociative sedation: non-extended moderate sedation	Minimal sedation only	Minimal sedation only		

Brief: <10 minutes
Intermediate: 10-20 minutes
Extended: >20 minutes

Lastly a discussion with the patient or guardian for consent is required in most situations. Discuss all interventions that will be provided, including risks, benefits, potential side effects and alternative treatment options. Obtain consent for the PSA separate from the consent for procedure.

Monitoring of Patient

Detailed discussion of monitoring parameters occurs in Section 2. Listed here are some of the general consensus rules for levels of monitoring for moderate and deep PSA. It has been clearly demonstrated that many of the medications used in PSA can lead to patient hypoxemia and respiratory depression so the monitoring of breathing and respiratory status is a cornerstone of safe PSA delivery.

Minimal moderate and deep sedation monitoring during PSA:

- Level of consciousness (ability to maintain own airway, follow commands, response to pain, GCS or AVPU scale).
- Respiratory rate and depth.
- Blood pressure.
- Heart rate.
- Pulse oximetry.
- Skin color.

CPSBC and ASA recommend initial baseline vitals, then vitals every 5 minutes x 3 after sedation starts and after last dose of PSA agents then every 15 minutes thereafter.

The use of ECG monitoring and quantitative capnography depends on the level of sedation and patient history.

Consider ECG monitoring for:

- ASA score > 2.
- Previous cardiac disease and/or dysrhythmias.
- Patient complaints and or observations of cardiopulmonary signs.
- Patient taking medications with cardiac stimulant or depressing actions.

Consider End-Tidal CO₂ monitoring for all deep sedations or for PSA where the patient cannot be assessed observationally for respiratory status. ERCP in a prone position is an example of such a procedure.

Delivering PSA

Staffing

For both moderate and deep PSA an individual dedicated to monitoring the patient should be present other than the practitioner performing the procedure. For deep sedation that individual should have no other responsibilities. During moderate sedation that person can assist with minor tasks that are interruptible after the PSA level has been stabilized along with the patient's vital signs as long as sufficient monitoring of the patient's sedation level can be maintained ⁽⁵⁾.

Equipment and Supplies

- Non-invasive BP machine.
- Pulse oximetry with audible signal.
- ECG monitor with audible signal.
- Oxygen and oxygen supplies.
- BVM.
- Stethoscope.
- Thermometer.
- IV supplies, catheters, fluids, syringes, needles.
- PSA agents and reversal medications.
- Emergency medications.
- Suction.
- Capnography.
- Close at hand advanced life support crash cart with several advanced airway choices (intubation supplies, LMAs, King Airways).

Delivery of Agents

The most common method of delivering PSA medications is through a parenteral route though buccal, nasal, and IM routes are used with certain agents especially with pediatric patients. Medication doses must be calculated, drawn up and labeled prior to commencement of the procedure. Drugs should be given slowly and in small incremental doses that are titrated to the desired end point of sedation and analgesia. Enough time between doses must be allowed for the effect of the drug to take place. A thorough knowledge of each individual drug's usual dose, its mechanism of action, and its onset and duration of action is imperative. The treatment of respiratory complications can usually be relieved by stimulating the patient, delivering oxygen, repositioning the airway with chin tilt or jaw-thrust, suctioning any upper airway secretions, and withholding further medications until the issue is resolved. If necessary, temporarily assist the patient's breathing with a bag-valve mask device. Rapid administration is more likely to produce hypotension and respiratory depression. Generally analgesic agents are given first before hypnotic-sedatives so that their action on respiratory drive can be assessed. The choice of agent(s) depends on:

- Type of procedure.
- Length of procedure.
- Desired depth of sedation.
- Patient factors.
- Physician comfort with particular agent(s).

Have antagonists ready if reversal of agents is needed in an emergency situation.

Recovery

Most medications used in PSA are not immediately metabolized so post procedure monitoring until discharge is vital for patient safety. Decreased stimulation, delayed drug absorption, and slow elimination place patients at risk during the recovery period. Staff needs to monitor for adverse events such as hypoxemia, apnea, obstruction of the airway, emesis, and cardiovascular events. The duration and frequency of monitoring is often standardized, but it can be individualized and depends on the level of sedation used, the duration of action of the agents used, the type of procedure undertaken, and

the individual pre-existing medical conditions of the patient. All patients should be monitored until they return to their baseline physiological status (mental, hemodynamics). The use of the Richmond Agitation-Sedation Scale and the modified adult Aldrete score or similar scale/scoring tools should be used.

Guidelines for discharge:

- The patient is alert and oriented to their baseline presedation level.
- Stable vital signs.
- Tolerating fluids.
- Patient is ambulatory.
- Airway is patent, with protective reflexes intact.
- Sufficient time post-administration of IV medications.
- Discharge patients accompanied by a responsible adult.
- Instructions given to avoid any activity that requires coordination or judgment.
- Written instruction on when to return to an ED for any complications.

Summary

Careful consideration to all phases of PSA needs to be reviewed for patient safety and satisfaction to be optimal. This chapter covered the processes involved in delivering PSA safely and effectively. Strict adherence to pre-sedation assessment, adequate monitoring, and post-sedation discharge criteria can decrease risk of complications.

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Chapter 5: Age Related Issues in PSA

Introduction

The pediatric population presents several unique challenges to the PSA team. Also PSA objectives and the specifics of safety management vary depending on where PSA services are being delivered. Common locations for delivering PSA include:

- Radiology suite.
- Endoscopy suite.
- Critical care areas such as the Emergency Department, ICU, PACU, and CCU.
- Interventional cardiology suite.
- Physician office/outpatient setting.
- Dentistry office.
- Reproductive technologies office.
- Prehospital setting by paramedic services.

In this chapter issues relating to delivering PSA to pediatric patients are discussed along with delivering PSA to the elderly patient.

Pediatric PSA

In this chapter a comprehensive review of pediatric sedation is not possible. With the rapid increase in the indications for pediatric sedation, there has been a corresponding increase in the guidelines and policies intended to reduce the risk of complications and adverse patient events. Numerous agencies worldwide have developed, implemented, and validated specific guidelines for the training of sedation personnel. In the United States, the American Academy of Pediatrics, the American Society of Anesthesiologists, the American Academy of Pediatric Dentistry, and the American Academy of Emergency Medicine have published such guidelines ⁽¹⁻⁴⁾. The American Academy of Pediatrics Guidelines for monitoring and management of pediatric patients are very applicable ⁽⁴⁾.

Adverse Events in Pediatric Sedation

The multicenter Pediatric Sedation Research Consortium calculated in 2006 that the overall incidence of an adverse event in pediatric PSA was 3.4% ⁽⁵⁾. An adverse event being any clinical situation which prompted an intervention aimed at limiting a perceived threat to the patient's wellbeing. Oxygen desaturation was the most frequently reported event. No deaths occurred though one cardiac arrest was reported in the study of a child with significant underlying disease. Overall, there were 1/400 respiratory-related events and 1/200 patients required some form of ventilation support. This data underscores the critical nature of competent airway management skills as a foundation for all safe pediatric sedation programs.

The following factors have been associated with and increased pediatric adverse event rate:

- Age < 6 months.
- Deeper levels of sedation.
- Severe pre-existing disease.
- Length of PSA.
- Complexity of procedure.
- Multiple doses of drugs needed.
- The use of more than one agent.

Unique Aspects of Pediatric Anatomy and Physiology

The following is a list of some of the pediatric characteristics that need consideration:

- Young infants have less oxygen reserve compared to adults. Hypoxemia can develop rapidly.
- Pediatric patients range from approximately 2.5-100 kg, so pediatric crash carts are larger and should have all necessary size-age-appropriate equipment.
- Small children and infants have small diameter airways and since resistance to airflow is inversely proportional to the 4th power of the radius, edema and constriction of pediatric airways can lead to profound airflow resistance.
- Infant larynx more superior in neck.
- Epiglottis shorter, angled and more over the glottis.
- Larynx cone-shaped and narrowest part of upper airway at the cricoid ring.
- Tongue relatively large.
- Head naturally flexed and occludes airway when patient in a supine position due to large occiput.
- Caution when intubated: extension of head may result in tracheal extubation, while flexion may lead to main stem intubation.
- Gastroesophageal reflux is common in infants. Watch out for vomiting post sedation.
- Children have more reactive airways; they are more likely to exhibit laryngospasm, or suffer more pronounced arterial desaturation should laryngospasm or bronchospasm occur.
- Small child has higher vagal tone, leading to a bradycardia response with autonomic stimulation.
- Small infants rely on heart rate to increase cardiac output during times of stress; vagal discharge during airway manipulation can lead to decreased blood pressure or abnormal heart rhythms such as transient heart blocks and junctional bradycardia.
- Have reduced functional residual capacity, leading to more rapid oxygen depletion should apnea occur.
- Oxygen consumption is higher on a proportional basis compared with adults but tidal volumes are relatively similar.

Key Point: pediatric patients are more prone to airway obstruction, apnea episodes, rapid desaturation and hypoxemia.

Behavioral Factors

Assessing the level of sedation in pediatric patients presents a challenge. Pediatric patients can move easily from one sedation state to a deeper level with minimal increases in sedation dosing. Pediatric patients of all ages may present for PSA and the team must take into consideration the patient's developmental age and produce a plan accordingly. Infants may easily be lulled into cooperation by a parent; however toddlers may exhibit a wide range of behaviors. They may refuse to take medications orally; they may resist the insertion of an IV catheter. Other routes such as SC, IM, buccal, and rectal may be considered for certain agents. Increased anxiety and agitation going into the procedure may lead to higher levels of PSA being used with unwanted over sedation when procedural stimulation is completed.

Drug Selection and Administration

Overall the sedation methods used for pediatrics are similar to adults. A good approach is to use opioid analgesics in combination with a sedative, an approach that has been proven to be safe and effective in the pediatric population ⁽⁶⁻¹¹⁾. Pena and Kraus report no increase in respiratory related adverse events compared to other agents when fentanyl and midazolam were used ⁽¹²⁾. Graff and colleagues reported only 11% of children undergoing sedation with fentanyl and midazolam had a respiratory event and that all were minor requiring no assisted ventilations ⁽¹³⁾. With the use of the popular adult combination therapy of fentanyl and midazolam in pediatrics the need for respiratory support is rare and the occurrence of life threatening events in an appropriately monitored child is near zero ⁽¹⁴⁾.

Dissociative PSA with ketamine may be the easiest means of providing moderate or deep PSA, and may be an excellent alternative to the multidrug cocktail approach. The patient does not need to be premedicated with midazolam to prevent emergence delirium ⁽¹⁵⁾. The use of medications such as atropine or glycopyrrolate is not needed for the sedation of older children and those not having oropharyngeal procedures ⁽¹⁶⁾. Ketamine causes a dissociation of the central nervous system to all outside stimuli. This causes a trance like cataleptic state with potent analgesia, sedation, and amnesia. Ketamine has positive effects on heart rate and blood pressure and causes no loss of airway reflexes or respiratory depression. The optimal dose is 1.5 mg/kg IV or 4-5 mg/kg IM. Both routes are considered equally safe ^(17, 18). Repeated doses for longer procedures does not change the dissociative state causing the patient to lose airway reflexes like it does with opioid and hypnotic sedative agents but instead just prolongs it.

Other choices in pediatric PSA include the use of etomidate or propofol in combination with an analgesic such as fentanyl. There are several choices for sedation without the need for analgesia as is often needed for children to tolerate unpleasant procedures or those that require the child to not move. These include benzodiazepines, diphenhydramine, and chloral hydrate.

One interesting route for drug administration in pediatrics (increasing use in adults also) is the intranasal (IN) route. It is supported by extensive literature, is safe, and is reflected by a high degree of patient and caregiver satisfaction ^(19, 20). Midazolam, ketamine, dexmedetomidate, and sufentanil are the most common sedative medications used by the IN route ^(19, 20). Midazolam burns by the IN route so it is recommended to give

lidocaine 2% or 4% - 0.2 ml per nostril 5 minutes prior to the midazolam to stop the burning.

Figure 5.1. Drugs, routes, and onset of action (20, 21)

Drug	Route	Time of onset (min)	Duration (min)
Fentanyl	IV	1-3	20-30
Midazolam	IV	1-3	20-30
	IM	10-20	60-120
	IN	10-15	60
	PO	10-30	60-90
	PR	5-10	30-60
Propofol	IV	1-2	5-10
Ketamine	IM	5	20-30
	IV	1	5-10
	IN	5-10	30-40
Etomidate	IV	2-3	20
Diphenhydramine	IV	5	2-6
	PO	60	4-6 hours
Chloral Hydrate	PO and PR	15-30	60-120
Flumazenil	IV	1	20-60
	IN	3-4*	*
Narcan	IV	1	15-30
	IM	10-15	60-90
	IN	3-4*	*

IN- intranasal, PR- per rectum.

*-Limited study information available.

Optimal volumes for IN instillation per nostril are 0.2-0.3 ml with a maximum volume of 1 ml. To get the best absorption an atomizer should be used if available (Fig. 5.3). To optimize the bioavailability of IN drugs the caregiver should:

- Minimize volume; maximize concentration- most concentrated IV form of drug should be used.
- Maximize total mucosal absorption area- use both nostrils.
- Use a delivery system that maximizes mucosal coverage- atomizer.

In summary the management of pediatric patients undergoing PSA presents some interesting challenges. Trained personnel in pediatric sedation, resuscitation, and airway management need to be part of any pediatric PSA team. Accurate weight measurement, drug dose calculations, and a protocol for having two persons sign off on drugs are essential. Age appropriate equipment to monitor and manage adverse effects of PSA needs to be readily available at the bedside.

Figure 5.3. Mucosal Atomization Device (MAD)

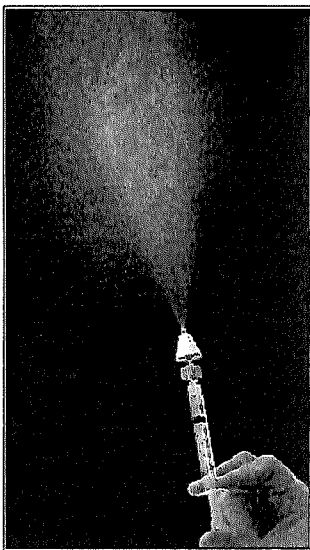


Figure 5.4. Intranasal drugs, uses, dosages, considerations ⁽²⁰⁾

Scenario	Drug and dose	Important considerations
Pain Control	- Fentanyl: 2 mcg/kg - Sufentanil: 0.5 mcg/kg - Ketamine 1 mg/kg	- Titration is possible - Sufentanil – use pulse oximetry - Half up each nostril
Sedation	- Midazolam: 0.5 mg/kg (combination w/ pain agents) - Ketamine 5-10 mg/kg - Sufentanil 1-1.5 mcg/kg Combination formulas: - Midazolam plus sufentanil: 0.2 to 0.3 mg/kg of midazolam plus 0.75 to 1 mcg/kg of sufentanil - Midazolam plus ketamine: 0.2 to 0.3 mg/kg of midazolam plus 5 mg/kg of ketamine	- Use lidocaine to prevent burning for midazolam - Use concentrated formulas - Sufentanil at doses > 1.5 mcg/kg associated with respiratory depression and desaturation.
Seizures	- Midazolam: 0.2 mg/kg - Lorazepam 0.1 mg/kg	- Support breathing while waiting - Use concentrated formula
Opiate Overdose	Naloxone: 2 mg	- Support breathing while waiting - Use concentrated formula
Epistaxis	Oxymetazoline or Phenylephrine + Lidocaine	Support breathing while awaiting onset

The Elderly

The elderly population of age greater than 65 is a rapidly growing segment of our society. Although those considered elderly are at a higher risk for complications, a better indicator of risk may be the physiological age of the patient. Figure 5.5 lists the major considerations for sedation in the elderly that require consideration.

Figure 5.5. Physiological differences and sedation considerations in the elderly
Adapted from www.sedationfacts.org (accessed March, 2013)

System	Physiological differences	Sedation considerations
Cardiovascular	- Reduced tissue elasticity in arteries and veins - Ventricular hypertrophy - Dec. cardiac output - Reduced arterial oxygenation - Inc. risk of arrhythmias	- Inc. oxygen consumption - Slow reaction to hemodynamic changes - Slower cardiorespiratory response to hypercarbia and hypoxia
Body composition	- Higher body fat proportions - Less intracellular fluid	- Expanded distribution volume for drugs - Higher risk of over sedation with water soluble meds - Slower recovery with lipid soluble drugs
Pulmonary	- Dec. respiratory drive - Less lung capacity - Diminished response to hypoxemia and hypercarbia - Increased work of breathing with decreasing lung compliance	- Decreased respiratory reserve and ability to respond to respiratory depression - Higher apnea rates
Neurological	- Loss of neuro-density - Less neurotransmitters	- Increased sensitivity to CNS meds - More delirium and confusion
Renal	Decreased function	Longer duration of action of some drugs
Hepatic	Decreased function	- Increased duration of action for lipid soluble drugs - Altered drug metabolism
Airway	- Decreased gag reflex - Chronic microaspirations - Loss of teeth and denture use - Arthritis of neck	- Increased risk aspiration - Difficult BVM use - Difficulty in performing head tilt-modified jaw thrust - Difficulty in intubation

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Section 2: PSA Safety

Chapter 6: Patient Monitoring and Equipment for PSA

Introduction

The monitoring of patients during PSA is one of the most important aspects of patient safety. The most indispensable monitor is the qualified staff member who observes the patient for skin and mucosal color, visual signs of pain and lightening sedation, respiratory rate and depth, and audible adventitious respiratory signs. Monitoring equipment is there to supplement the observational skills of the staff. Team members must be able to operate and interpret the information these adjuncts provide. They must also understand and be able to act on the information gathered appropriately to keep patients safe. In a clinical policy written by ACEP in 2005 the following was stated; "During procedural sedation and analgesia, there must be an individual available who is capable of recognizing and managing respiratory and hemodynamic emergencies."⁽¹⁾

In this chapter some of the key monitoring tools will be reviewed. Also the necessary common emergency equipment needed for the safe delivery of moderate and deep sedation is discussed.

Monitoring Requirements

Monitoring requirements for various levels of PSA change depending on the location of services, the institutional guidelines, regional guidelines, and guidelines of the specialty overseeing delivery (dentistry, ER, gastroenterology etc.) Presented here are the CPSBC guidelines for IV PSA in non-hospital settings⁽²⁾.

Monitoring Parameters:

- ECG*
- Capnography*
- Noninvasive BP
- Respirations
- Heart rate
- Pulse oximetry
- LOC
- Temperature

* The CPSBC states the following in relation to ECG and capnography.
ECG: "Initiate, monitor and interpret ECG rhythms as indicated i.e. ASA score >2 previous cardiac disease and/or dysrhythmias, patient complaints and/or observations of cardiorespiratory symptoms e.g. SOB, irregular pulse, decreased

SpO₂), patient presently taking medications with stimulant and/or depressant effects."⁽¹⁾

Capnography: "Establish End-Tidal CO₂ during monitored anesthesia care (MAC) as determined by the anesthesiologist present when:

- Deep sedation is a probable or planned outcome.
- Moderate or dissociative sedation is a probable or planned outcome in any patient with ASA 3 classification.
- The respiratory rate assessment may be difficult due to procedural draping or positioning. "⁽²⁾

The ASA has established the following as required monitoring parameters ⁽²⁾:

- LOC
- ECG: Must be used for all patients undergoing deep sedation
- E_TCO₂ : Quantitative capnography required for all moderate and deep PSA as of July, 2011
- Pulse oximetry
- Respiration evaluation by auscultation and observation
- Noninvasive BP
- Heart rate

Much controversy has accompanied the guideline for the use of E_TCO₂ monitoring put forward by the ASA in 2011. A variety of governing bodies and specialty associations have challenged the guideline as not being evidence based. They also state that hospital areas caring for patients of much higher risk do not have a mandate to use capnography on deeply sedated patient and patients still deep from general anesthesia such as PACUs.

For both moderate and deep PSA an individual dedicated to monitoring the patient should be present other than the practitioner performing the procedure. For deep sedation that individual should have no other responsibilities. During moderate sedation that person can assist with minor tasks that are interruptible after the PSA level has been stabilized along with the patient's vital signs as long as sufficient monitoring of the patient's sedation level can be maintained ⁽³⁾.

In a prospectively collected data base of 1367 pediatric patients undergoing PSA the highest risk of adverse events was found to occur within 25 minutes of receiving the last dose of PSA agent ⁽⁴⁾. In another study looking at fentanyl and midazolam used in combination, all cases of apnea occurred within 5 minutes of medication administration ⁽⁵⁾.

Monitoring for Oxygenation and Ventilation

The most likely adverse event of serious threat to patient morbidity and mortality is one of a respiratory nature. The monitoring of a patient's respiratory status has two components: ventilation and oxygenation. There seems to be much confusion about the use of pulse oximetry and whether or not it is an effective tool to monitor ventilation and detect apneic episodes.

Pulse Oximetry

Hemoglobin saturation (SaO_2) is the amount of oxygen reversibly bound to hemoglobin in arterial blood. Pulse oximetry allows for a noninvasive continuous measurement of this parameter at the bedside. It is now commonly referred to as the fifth vital sign. Oximeters are made up of a light source (LED), a photo detector and a microprocessor. It relies on a process called spectral analysis which uses light absorption characteristics that are unique in all matter types to determine the physiochemical properties of a substance. Limitations are listed in Fig. 6.1.

Figure 6.1. Etiology and examples of unreliable pulse oximetry ⁽⁶⁾

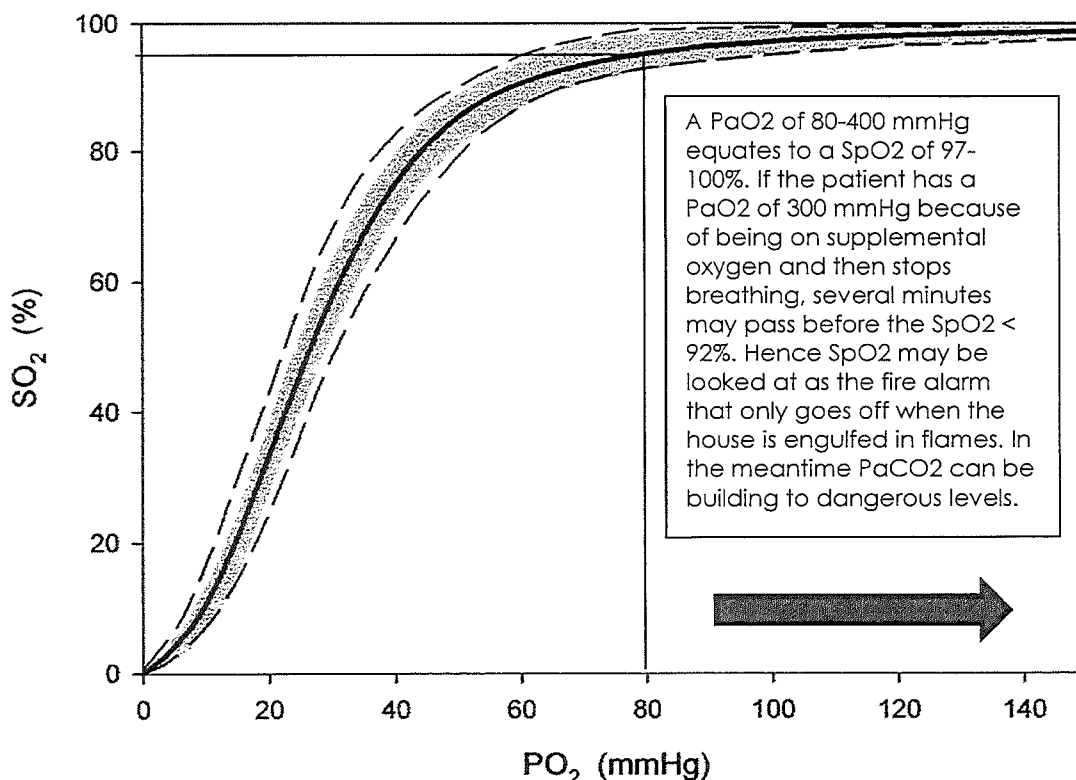
Etiology	Examples
Sensor Location	• Poor blood flow to extremities in critical illnesses. Forehead reflective oximetry best. • Extraneous light sources
Motion artifact	• Exercise • CPR • Shivering • Seizures • Tremor
Signal degradation	• Poor peripheral perfusion • Hypotension • Hypoperfusion • Vasoconstriction • Nail polish
Physiologic range	• Inaccurate if systolic BP < 80 mmHg • Increasingly inaccurate at SaO_2 < 75%
Dyshemoglobinemia	• Met-Hgb • CO-Hgb
Intravenous dye	• Methylene blue • Indocyanine green

The response time of the oximetry readings lags behind the patient's physiologic status from 4-20 seconds in most monitors. Centrally located probes tend to respond faster.

The use of oximetry is to give information on arterial blood oxygen content. One issue when pulse oximetry is used to detect hypoventilation and apnea is that there can be a significant delay between an apnea/hypoventilation episode starting and that event then being reflected by decreasing oxygen saturation levels. The addition of supplemental oxygen to the equation can even further delay apnea/hypoventilation detection. "The oxyhemoglobin dissociation curve describes the relationship of oxygen partial pressure (PaO_2) and saturation (SaO_2). Its sigmoidal shape hinges on varying hemoglobin affinity with successive oxygen binding. It is important to note that SpO_2 provides poor correlation with PaO_2 in the normal range. **Normal SaO_2 is associated with a wide range of PaO_2 (80 to 400 mm Hg)**, which includes two extremes of oxygen reserve.

^{“(6)}

Figure 6.2. Oxyhemoglobin dissociation curve



In a study of 634 patients receiving patient controlled analgesia clinicians determined that capnography was more effective than pulse oximetry in providing earlier detection of respiratory depression. The time lag noted between the two modalities was as high as 4 minutes ⁽⁷⁾. Relying on pulse oximetry is a late determinate of ventilation and respiratory compromise ⁽⁸⁾. Lightdale states, "Capnography allowed early detection of arterial oxygen desaturation because of alveolar hypoventilation in the presence of supplemental oxygen. The current standard of care for monitoring all patients receiving sedation relies overtly on pulse oximetry, which does not measure ventilation." ⁽⁹⁾

How then should practitioners use SpO_2 when using potent agents such as etomidate, propofol, or ketamine for moderate and deep sedation? One approach is that if using SpO_2 alone without capnography the patient breaths room air. "When breathing room air, the patient's oxygen saturation will progressively decline as alveolar ventilation decreases and CO_2 rises. This will rapidly result in a decreased SpO_2 , which allows ample time for correction by repositioning or giving additional stimuli. **If the patient is placed on even a small amount of supplemental oxygen, he will continue to saturate well even as alveolar ventilation is almost nil and CO_2 rises to dangerous levels.**" ⁽¹⁰⁾

Another approach, since ASA guidelines recommend considering oxygen for moderate and deep sedations, is to get the benefits of preoxygenation and accurate ventilation monitoring by using quantitative capnography.

End-Tidal CO₂ Monitoring

CO₂ monitors measure the partial pressure of CO₂ in mmHg in expired gas. When measured at the end of expiration it is referred to as end-tidal CO₂ (ETCO₂) which approximates alveolar CO₂. Wave form ETCO₂ machines give the most data on metabolism, perfusion, and ventilation.

Figure 6.3. Colorimetric CO₂ detector used to confirm intubation of the trachea

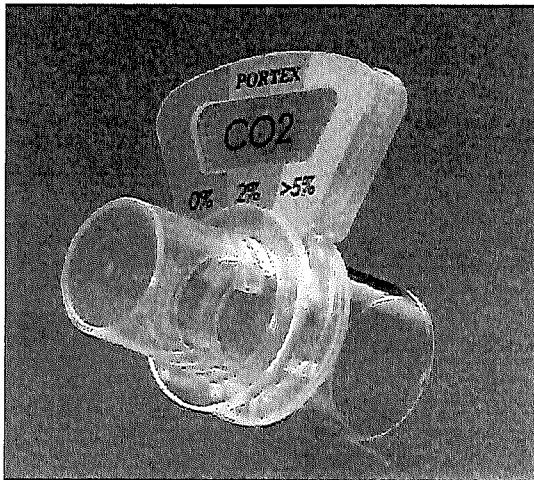
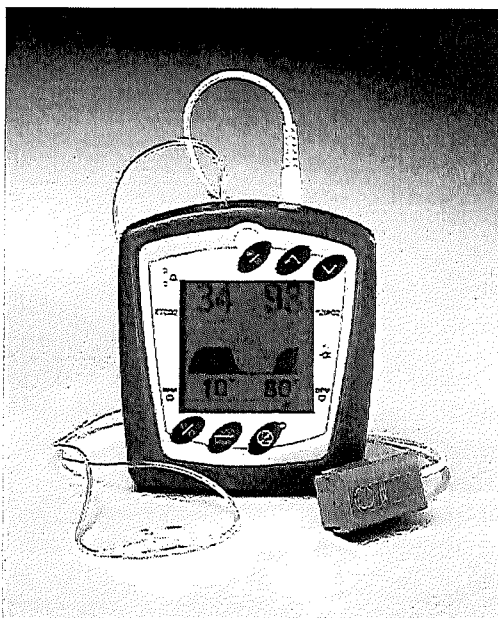
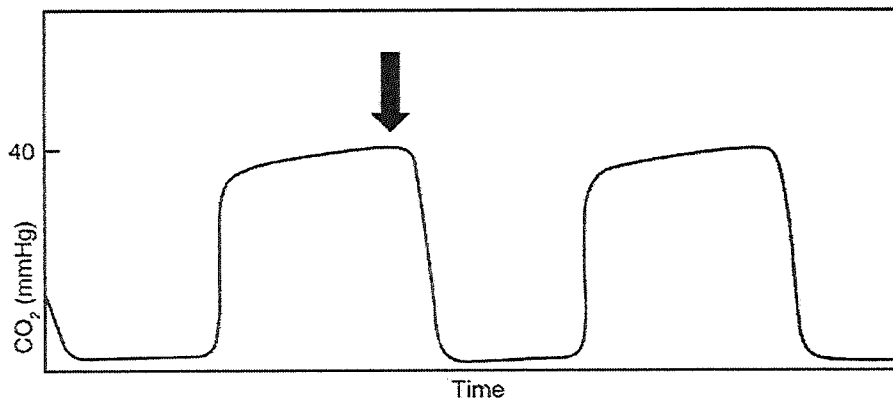


Figure 6.4. Quantitative CO₂ Detector (Continuous waveform End-Tidal CO₂ Monitor)



Most of these devices use an infrared sensor, which measures the amount of infrared light absorbed by the passing CO₂ gas. Nonintubated patients undergoing sedation are usually monitored with a sidestream capnograph through a nasal cannula. A normal capnogram has a typical rectangular shaped waveform that should start from 0 mmHg and end at 0 mmHg. If the baseline maintains itself at above zero CO₂ rebreathing is occurring or hypoventilation. The plateau has a gentle positive slope with ETCO₂ been measured at the end of exhalation (black arrow, fig. 6.5). With the beginning of inspiration there is a rapid drop back to baseline.

Figure 6.5. A Normal Capnogram



In the setting of PSA the use of capnography is the earliest and most sensitive indicator of apnea or hypoventilation. "Several studies show that patients undergoing procedural sedation have a high rate of acute respiratory events including hypoventilation and apnea, and clinical assessment of chest rise is not sensitive for detecting these events. Oxygen desaturation is a late finding in hypoventilation, especially in patients receiving supplemental oxygen. Addition of capnography to standard monitoring provides advanced warning and reduces hypoxic events."⁽⁶⁾

When using capnography to assess for respiratory depression it must be understood that both increases and decreases in expired CO₂ can mean a patient is experiencing hypoventilation issues.

Capnographic evidence of hypoventilation can include:

- ETCO₂ > 50 mmHg.
- A change of 10% or more from baseline.
- An absolute change of 10 mmHg or more.
- Loss of waveform.

Classic hypoventilation results in a waveform with a wide base and increased amplitude (Fig. 6.6). Shallow ineffective breathing will give a low amplitude waveform as CO₂ levels are diluted by dead space gases (Fig. 6.7).

Figure 6.6. Classic hypoventilation waveform capnogram

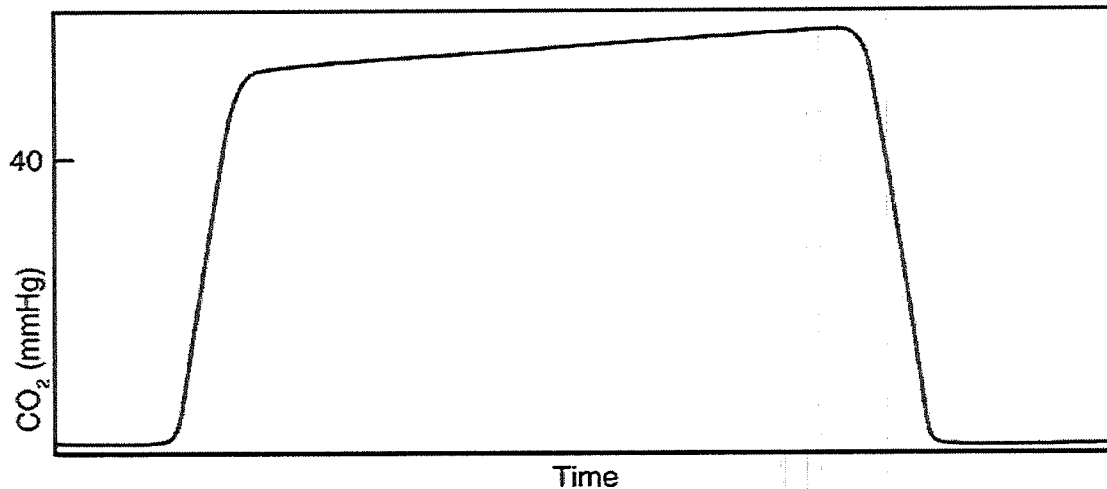
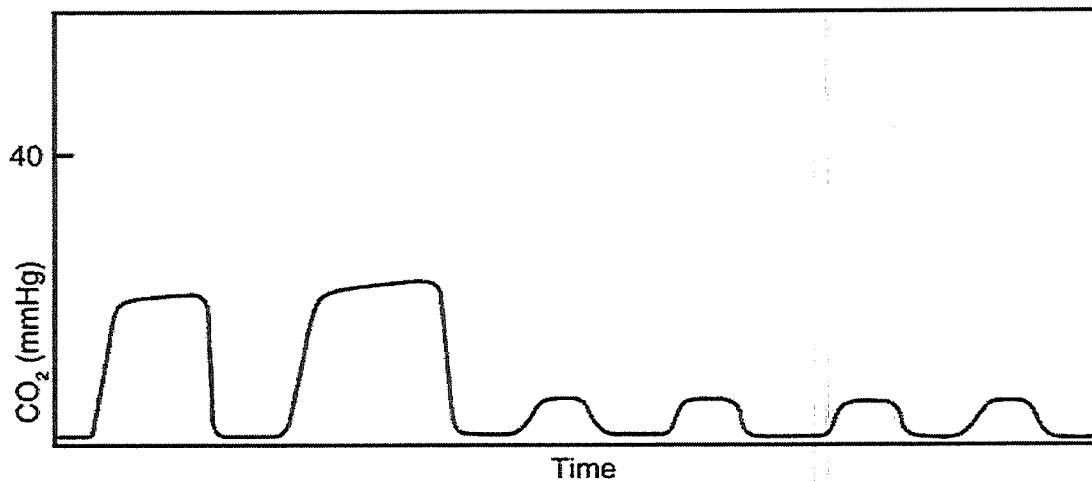


Figure 6.7. Shallow ineffective breathing capnogram



Emergency Equipment Needs

Emergency equipment needs depends on the type of location that PSA is being delivered in along with the depth of sedation being undertaken. During moderate and deep sedation the ASA recommends that a defibrillator should be immediately available for all patients. Pharmacological agents used to reverse both opioids and benzodiazepines need to be available along with the resuscitation medications common to a full crash cart. Appropriately sized airway equipment for delivering oxygen, suction, positive pressure ventilation, and the establishment of a patent airway need also be available.

Example of emergency equipment needed for PSA: ⁽³⁾

- Intravenous equipment
- Gloves
- Tourniquets
- Alcohol wipes
- Sterile gauze pads
- Intravenous catheters [24-22-gauge]
- Intravenous tubing [pediatric "microdrip" (60 drops/ml)]
- Intravenous fluid
- Assorted needles for drug aspiration, intramuscular injection
- [intraosseous bone marrow needle]
- Appropriately sized syringes [1-ml syringes]
- Tape
- Basic airway management equipment
- Source of compressed oxygen (tank with regulator or pipeline supply with flowmeter)
- Source of suction
- Suction catheters [pediatric suction catheters]
- Yankauer-type suction
- Face masks [infant/child]
- Self-inflating breathing bag-valve set [pediatric]
- Oral and nasal airways [infant/child-sized]
- Lubricant
- Advanced airway management equipment (for practitioners with intubation skills)
- Laryngeal mask airways [pediatric]
- Laryngoscope handles (tested)
- Laryngoscope blades [pediatric]
- Endotracheal tubes
- Cuffed 6.0, 7.0, 8.0 mm ID
- [Uncuffed 2.5, 3.0, 3.5, 4.0, 4.5, 5.0, 5.5, 6.0 mm ID]
- Stylet (appropriately sized for endotracheal tubes)
- Pharmacologic Antagonists
 - Naloxone
 - Flumazenil
- Emergency medications
 - Epinephrine
 - Ephedrine
 - Vasopressin
 - Atropine
 - Nitroglycerin (tablets or spray)
 - Amiodarone
 - Lidocaine
 - Glucose, 50% [10 or 25%]
 - Diphenhydramine
 - Hydrocortisone, methylprednisolone, or dexamethasone
 - Diazepam or midazolam

Summary

Patient monitoring by observation skills, but also though the use of monitoring devices such as ECG, pulse oximetry and capnography is essential for safe PSA delivery. Monitoring for sedation procedures primarily involves the observation of blood pressure, oxygenation, respiratory function, ECG, and capnography tracings. The use of capnography for PSA is controversial but should be considered when available especially for all deep sedations. Having the patient on room air for sedation without capnography has some validity in that the detection of respiratory depression will not be masked by the accumulation of oxygen in the blood when using supplemental oxygen. ECG monitoring is optional and should be used when indicated by the situation or the history of the patient.

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Chapter 7: Adverse Events in PSA

Introduction

"In health care, the premium placed on practitioner autonomy, the drive for productivity, and the economics of the system may lead to severe safety constraints and adverse medical events."⁽¹⁾ This chapter describes the factors that lead to adverse events, what those adverse events are, and the likelihood of those events occurring. The human factor involved in safety issues in health care is also discussed.

Safety- Human and Organizational Factors

Delivering PSA safely is a multistage process that starts before even meeting the patient. There needs to be a safe and reliable sedation service design which allows for the free flow of communication, a service which adopts a culture of safety. Simply being hospitalized carries a 200-fold greater risk of dying from the care process than driving in traffic, and a 2,000-fold greater risk of dying than while flying on a commercial flight ^(2, 3).

A clinical microsystem (CM) is a group of staff and clinicians working together to provide care to a population of patients with a shared clinical purpose. A PSA team is an example of such a microsystem. CM provides a conceptual framework for approaching organizational learning and the delivery of safe PSA. To improve safety it is necessary to study the components that make up the system which are humans, technologies and their complex interactions ⁽¹⁾.

There are three reasons for adverse events during PSA ⁽¹⁾:

- All human beings, regardless of their skills, abilities, and specialist training, make fallible decisions and commit unsafe acts. This human propensity for committing errors and violating safety procedures during sedation can be moderated by selection, training, well-designed equipment, and good management, but it can never be entirely eliminated.
- No matter how well designed, constructed, operated, and maintained they may be, all man-made systems possess latent failures to some degree. These unseen failures are analogous to resident pathogens in the human body that combine with local triggering factors (i.e., life stress, toxic chemicals, etc.) to overcome the immune system and produce disease.
- All human endeavors involve some measure of risk.

Figure 6.1. Characteristics of a high performing sedation team

The role of clinical leaders is to balance setting and reaching collective goals, and to empower individual provider autonomy and accountability, respectful action, and reflection

The hospital looks for ways to support the work of the sedation service and helps to coordinate the handoffs between other clinical microsystems (i.e., PACU, ICU, etc.)

There is selective hiring of the right kind of people. The orientation process is designed to fully integrate new staff into a safety culture

The interaction of staff is characterized by trust, collaboration, willingness to help each other, appreciation of complementary roles, respect, and recognition that contribute to a shared purpose of safer and high-quality pediatric care

All clinical microsystems have responsibility for the ongoing education and training of staff including simulation and team training

An atmosphere for learning and redesign is supported by the continuous monitoring of care, use of trigger tools and benchmarking, and staff who are empowered to innovate

The primary concern is to meet all patient needs – caring, listening, educating, and responding to special requests, innovating to meet patient needs, and service flow

Performance focuses on patient outcomes, avoidable costs, streamlining delivery, using data feedback, reducing variation, and encouraging frank discussions about performance

Source: Adapted from Barach and Johnson ⁽⁴⁾.

Human error and performance limitations have been extensively studied in several industries such as aviation, road and rail travel, and nuclear power for several decades. Theories of error and accident causation have been developed for other human activities although they have just started to be applied to the field of medicine. One common realization from this type of research is that errors are rarely isolated to one individual's actions. Adverse event analysis may often reveal deep-rooted unsafe features of an organization. "These developments have led to a much broader understanding of accident causation, with less focus on the individual who makes an error and more on preexisting organizational and system factors that provide the context that enables errors to occur. ⁽¹⁾"

Adverse events that occur with systems/organizations which have a wide number of technical and procedural safeguards have been termed organizational accidents ⁽⁵⁾. These events do not arise from a single error but instead by the accumulation of errors that started well before the event in question. One of the basic principles of error management is that the transitory mental states associated with error production – momentary inattention, distraction, preoccupation, forgetting – are the least manageable links in the error chain because they are both unintended and largely unpredictable ⁽⁶⁾.

The Design of a Safe PSA Team

The delivery of safe PSA relies on coordinated teamwork, good communication skills, a culture of learning not a culture of blame, and trust between team members. Effective teamwork has been long accepted in multiple industries as being the key to safety.

Pitfalls in PSA teamwork (adapted from Barach et al.)⁽⁷⁾:

- Poor communication between team members.
- Poor training of the team leader in team dynamics and leadership.
- Not having enough dedicated team members.
- Reluctance to question the leader or other senior team members.
- Failure to establish and maintain consistent supportive organizational infrastructure.
- Absence of experienced team members.
- Conflicting occupational cultures.
- Failure to prioritize task demands.
- Failure of members to function as part of a team.
- Failure to establish and maintain consistent supportive organizational infrastructure.

"Acquiring a safety culture is a process of collective learning and mindfulness that recognizes the inevitability of error and proactively seeks to identify latent threats. Characteristics of a strong safety culture include a commitment of the leadership to discuss and learn from errors, communications founded on mutual trust and respect, shared perceptions of the importance of safety, encouraging and practicing teamwork, and *incorporating non-punitive systems for reporting and analyzing near miss and adverse events.* ⁽¹⁾" Clinicians must be empowered by organization leadership to be honest and reflective of their practice. Each team member must be able to: (1) anticipate the needs of the others; (2) adjust to each other's actions and to the changing environment; (3) monitor each other's activities and distribute workload dynamically; and, (4) have a shared understanding of accepted processes and how events and actions should proceed ⁽⁸⁾.

One application of processes established in anesthesiology is a modification of the "time out" team concept of preoperative verification of the correct patient, procedure, site, and implants. There is an active discussion among all team members and the procedure is not started until all questions and concerns are resolved. A similar "time out" approach for PSA teams might include:

- Correct patient.
- Correct procedure.
- Patient appropriate for PSA.
- Correct drug(s) for type and length of procedure.
- Correct drug dosages checked by two team members.
- Correct safety equipment operable and ready.
- Correct location for the patient and the type of procedure.
- Adequate staffing for the procedure and PSA.

All team members understand that they are all safety "gatekeepers" and that all concerns and questions will be answered before starting. The adoption of checklists as seen in many industries is further improving safety in healthcare. The initiative of the WHO Safe Surgery Saves Lives Program in 2009 led to the adoption of the Surgical Safety

Checklist ⁽⁹⁾. PSA organizations worldwide should consider a similar global initiative to create checklists to standardize care and improve patient outcomes.

Adverse Events That Occur with PSA

Respiratory System Complications

The vast majority of adverse events in PSA are of a respiratory nature. They can usually be attributed to over sedation or an unexpected response to the agents by the patient. The signs and symptoms include respiratory depression, hypoventilation, apnea, hypoxemia, hypercapnia, laryngospasm, bronchospasm and complete airway obstruction ⁽¹⁰⁾.

Hypoventilation, Hypoxemia and Hypercapnia

Hypoventilation and the resulting issues of decreased blood oxygen levels and rising blood CO₂ content are a potentially serious adverse event of PSA. Hypoventilation can be attributed to relaxed laryngeal tissues that increase airflow resistance and obstruction, along with a decreased CNS respiratory drive caused by the agents used. Patients who are ill and/or have chronic disease processes may not metabolize medications in a predictable manner. The complex pharmacokinetics and pharmacodynamics of agents with these patients can lead to cardiopulmonary compromise at doses not normally attributed to causing those types of events. Benzodiazepines and opioids are two PSA drug categories that are often linked to respiratory adverse events. When used in combination their effects are synergistic on the respiratory system. Oversedation can be prevented by:

- Slow titration of agents over a period of time. Avoid rapid boluses.
- Avoid giving multiple agents simultaneously. Administer the agent with the most depressing action on the respiratory system first and then after a period of time the second agent is given again using small slowly administered boluses.
- Take into consideration the physiological status of the patient (renal and hepatic issues, BMI, dehydration, poor cardiac function) and adjust drug choices and dosing accordingly.

Airway Obstruction

Obstruction of a patient's airway can be caused by relaxed muscle tone of the submandibular muscle and surrounding tissues leading to upper airway obstruction from the tongue or a closed epiglottis, laryngospasm, bronchospasm, secretions or foreign body obstruction (FBO). **Laryngospasm** is a form of airway obstruction caused by a tonic contraction of the glottis muscles including the true and false cords. It is much less common with PSA than general anesthesia. Factors associated with causing laryngospasm are:

- Airway manipulation including with the use of oral or nasal pharyngeal airways
- Excessive secretions
- Vomitus
- Suctioning
- Preexisting respiratory infection

Laryngospasm can usually be managed with positive pressure ventilation with a BVM but on occasions medications are used and the patient is paralyzed and intubated.

Bronchospasm occurs as a lower airway obstruction due to increased bronchial smooth muscle tone. The signs and symptoms the patient presents with depend on the degree of bronchospasm. Mild signs may be a slight wheeze only detectable by stethoscope. More severe bronchospasm may cause audible wheezing, chest tightness, use of accessory muscles, indrawing, and severe dyspnea. Risk factors for bronchospasm development include having reactive airway disease, release of histamines, upper respiratory infection, aspiration, excessive secretions, and bronchial irritation from suctioning. Propofol has been linked to causing bronchospasm ⁽¹¹⁾. **Negative pressure pulmonary edema (NPPE)** is a pulmonary complication that can occur rarely in patients when a patient breaths against an acute upper airway obstruction creating high negative intrathoracic pressures. This leads to a shift of fluids from pulmonary capillary beds into alveoli. Patients at risk of developing NPPE include those with a history of sleep apnea, laryngospasm, FBO, obesity, and opioid use.

Pulmonary Aspiration

The incidence of pulmonary aspiration in PSA is most likely very low ⁽¹²⁻¹⁷⁾. The one location where patients are most likely not NPO as per ASA guidelines is in the ER. Aspiration during PSA in the ED has never been reported in medical literature ⁽¹⁸⁾. If it does occur pulmonary aspiration can cause a variety of sequelae depending on the type of aspirate. Signs and symptoms include rales and rhonchi, dyspnea, wheezes, tachycardia and tachypnea, and oxygen desaturation. Many drugs used for PSA can induce nausea and vomiting while obtunding protective reflexes. Patients at risk for aspiration are those with increased intra-abdominal pressure, poor gastric emptying, or GERD. Increases in intra-abdominal pressure can be found in pregnant patients and in patients with ascites for example.

Cardiovascular System Complications

Hypotension

Hypotension is the most common cardiovascular adverse event found with PSA. The mechanism for hypotension is a decreased sympathetic tone which leads to vasodilation of vascular beds. This in turn leads to peripheral pooling of blood and decreased preload. The other mechanism for some PSA agents is that they have a direct negative inotropic effect on the heart. The physiologic status of the patient can contribute to the degree of hypotension also- preexisting heart disease, dehydration, blood loss, sepsis, and vasovagal response.

Hypertension

Hypertension can occur during PSA. It may be an indication that the patient has had inadequate PSA analgesia for a given procedure. The stress and pain then leads to an autonomic sympathetic release. Epinephrine containing local anesthetics can also lead to hypertension when larger quantities are used. In patients with preexisting hypertension this can precipitate a hypertensive crisis.

Dysrhythmias

The most common dysrhythmia seen in PSA is a sinus tachycardia. Like with hypertension the most common cause is inadequate levels of PSA. Other more ominous differentials need to be ruled out such as hypotension and hypoxemia. Appropriate treatments for these underlying causes should be instituted. Patients with preexisting cardiac risks/history can have dysrhythmias especially in the presence of high anxiety, pain, hypotension, or hypoxia.

Syncope

Syncope is one of the most common cardiac complications during sedation ⁽¹⁹⁾. It may be associated with increased parasympathetic tone, hypotension, hypoxemia, or rarely cardiac dysrhythmias.

Gastroenterological Complications

Nausea and Vomiting

The incidence of vomiting in patients who have undergone PSA is unclear in the literature. The rate of events leading to harm is very low as noted with the pulmonary aspiration discussion. Opioids used in PSA along with some other agents such as etomidate and ketamine can cause vomiting. Patients at risk for vomiting include:

- Young age
- Female gender
- Previous history of vomiting with anesthesia or sedation
- Obesity
- Motion sickness history
- Patients with high anxiety

Other Complications

Other rare complications of PSA which are not life threatening include delirium, urinary retention, and paradoxical sedative excitement (PSE). PSE can occur when medications such as propofol or benzodiazepines are used but is a very rare reaction occurring in less than 1 % of cases using these medications.

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Chapter 8: Responding to Adverse Events and Emergencies

Introduction

Most adverse events in PSA are not necessarily of any clinical significance especially when staff intervenes early. In this chapter there is a review of how to respond to the adverse events discussed in Chapter 7. Also discussed is the management of serious emergencies related to PSA. This chapter does not go into depth for topics such as the full resuscitation of the pulseless patient. The reader should refer to current ACLS and PALS guidelines for in-depth resuscitation information.

Being Prepared

Wherever PSA is undertaken, there must be emergency protocols in place which delineate the actions necessary in the event of an emergency, what the individual staff responsibilities are, what emergency equipment is required and where it is located, and what training program is needed to maintain emergency response competencies. In an out of hospital setting such as a dental office these protocols would look vastly different to those of a large hospital emergency department. Who calls 911 if there is an emergency? Who goes down to guide the paramedics up to the office. Who brings the crash cart into the sedation room? All of these issues must be dealt with and an emergency plan put into place. Staff should be trained appropriately for their roles- CPR, use of an AED or manual defibrillator, use of a BVM. Mock simulations on dealing with PSA emergencies should be regularly undertaken with all staff that provide PSA or make up the emergency response team.

Responding to Respiratory Events

The most common adverse event in PSA involves the unwanted actions of the agent or agents used on the respiratory system. A slowing down of respiratory rate, a decreasing respiratory depth, or even apnea is commonly associated with moderate and deep sedation. The level of response by staff is usually tempered by the degree of compromise the patient is experiencing.

Often indications of hypoventilation or apnea can be dealt with by verbal and tactile stimulus applied to the patient. Saying something like "John, take a couple of deep breaths" may be all that is needed. A gentle shaking of the patient or application of painful stimuli can be added if necessary. When patients are more deeply sedated these actions may not be enough if respiratory depression is severe. This can be the case when deep sedation is applied but the patient reaches a level of general anesthesia (GA), perhaps when the drugs actions exceed the time the procedure takes. When the procedure ends the deep sedation action shifts to the level of having the patient enter GA. In such a case the team must be qualified to manage the cardiovascular and

respiratory functions of the patient until the situation is resolved. This includes all aspects advanced airway and hemodynamic management.

Basic Airway Management

Dealing with an anatomical functional blockage of the airway from relaxation of patient tissues and their tongue is a common situation staff must be able to respond to. The first steps if signs of a partial or complete airway obstruction occur are to reposition the patients head, jaw, and neck. The head tilt/chin lift is the initial maneuver used to open the airway in these situations (Fig. 8.1). If the patient is not supine then a modified jaw thrust with some head tilt can be applied. If necessary the patient may need to be quickly brought into a supine position. The modified jaw thrust refers to a jaw thrust with the addition of head tilt. The jaw thrust with or without head tilt is more effective in displacing the tongue anteriorly with the mandible and hyoid than is the head tilt/chin lift. The effectiveness of these airway maneuvers has been substantiated in multiple studies.

Figure 8.1. Head tilt-chin lift

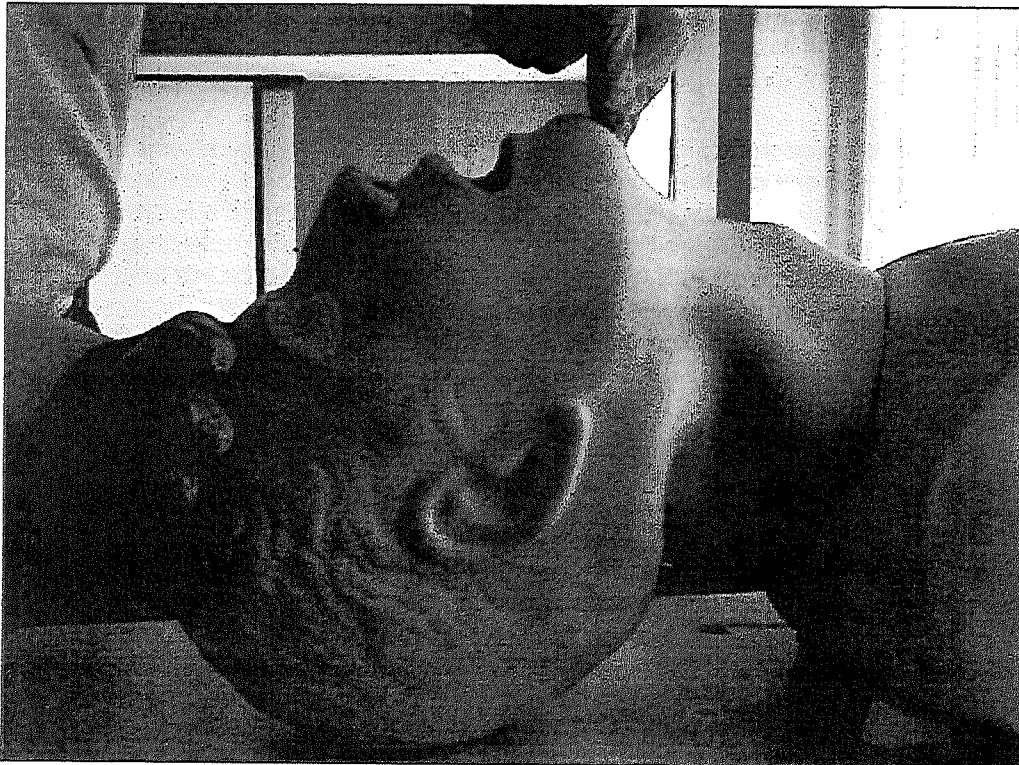
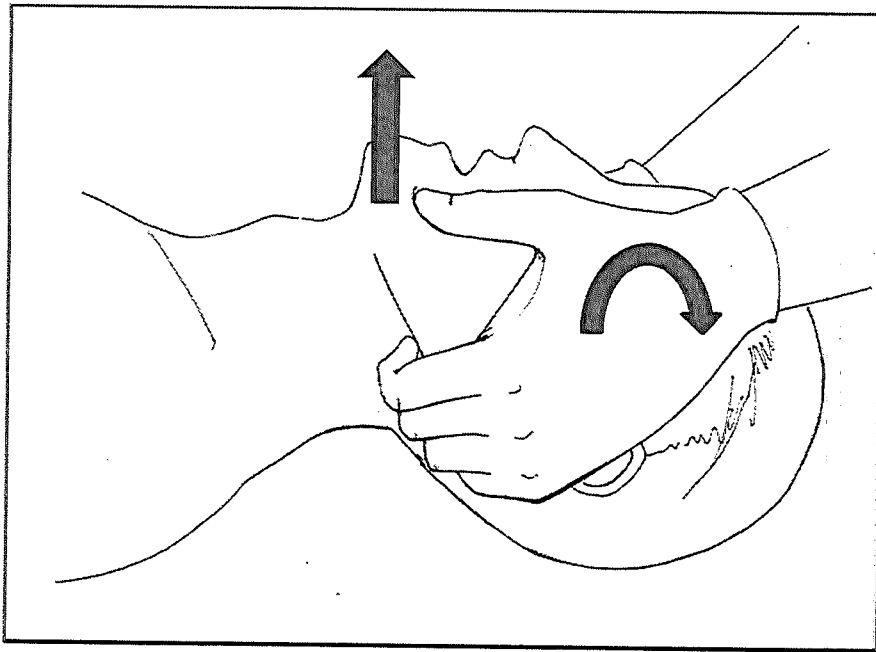
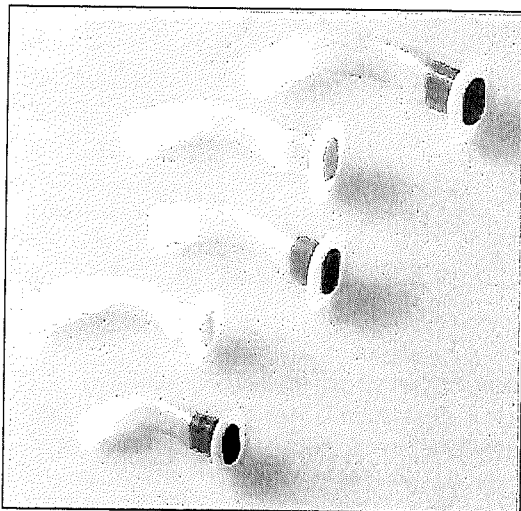


Figure 8.2 Modified jaw thrust



If the situation dictates that the patient will have a loss of protective reflexes for a prolonged period of time then the use of oral or nasal pharyngeal airway should be considered. Both prevent the tongue from occluding the airway and provide an opening for spontaneous breathing or positive pressure ventilation with a BVM.

Figure 8.3. Oral pharyngeal airways (OPA)



Oral airways are placed in adults upside down then rotated into position as the person inserting feels the tip of the airway hitting the soft palate (Fig. 8.4). In infants and toddlers they are placed in with the same orientation as they will sit in (without the upside down rotation) to minimize the risk of soft palate trauma. Sizing is from the lips to the angle of the jaw (Fig. 8.5).

Figure 8.4. Placing an oral pharyngeal airway

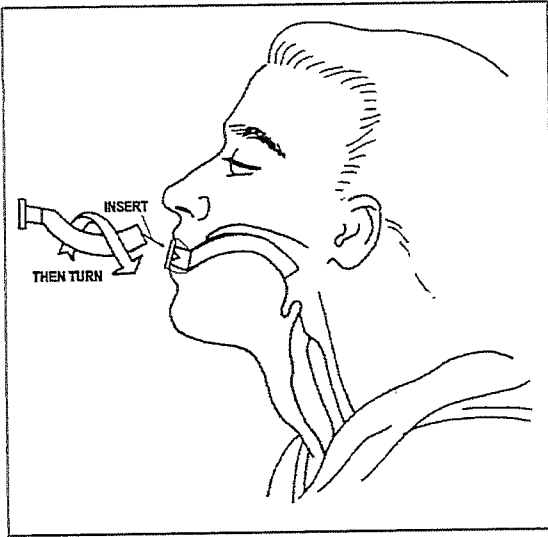


Figure 8.5: Sizing an oral pharyngeal airway



Once a patent airway is established the patient may begin spontaneous breathing adequately. As these airway procedures are being undertaken other staff must decide to use reversal agents if opioids or benzodiazepines were used. Refer to Chapter 3 for information on reversal medications.

If it is determined that the patient is apneic or hypoventilating then staff will have to ventilate the patient with a bag-valve-mask (BVM). This device is not easy to use for many individuals. The flow of oxygen from the outlet should be sufficient so that the

reservoir bag stays filled or partially filled at all times. Flows of 12-15 LPM will insure 100% oxygenation in adult patients. The use of two people to ventilate will allow for successful delivery of respirations in most patients. The person squeezing the bag should deliver each breath over 1-2 seconds using enough volume to just see the chest rise. Excessive inspiratory pressures can lead to insufflation of air down the esophagus and subsequent regurgitation. Ventilate at 10-12 breaths per minute or to pulse oximetry. Avoid hyperventilation. The second person holds the mask forming a seal with two hands (Fig. 8.8).

Figure 8.6. Bag-valve-mask

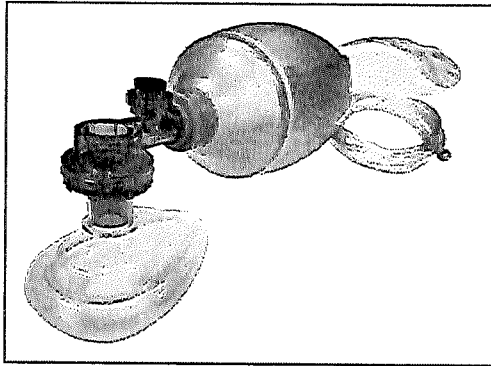


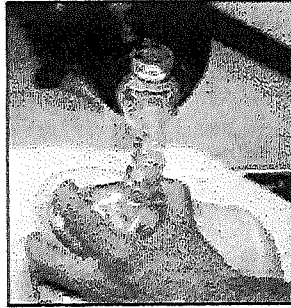
Figure 8.7. MOANS mnemonic predicting difficulty of BVM use

Letter	Refers to
M	Mask seal/male face/Mallampati class 3-4. Trauma to the face, blood and debris, beards all can make getting a BVM seal difficult.
O	Obesity/Obstruction. BMI > 26 kg/m ² indicates likely difficulty using BVM.
A	Age > 55 yo, perhaps because of a loss of muscle and tissue tone in the upper airway.
N	No teeth. Leave dentures in situ if possible.
S	Stiff/Snoring - patients with reactive airway disease such as COPD, asthma, ARDS, and pulmonary edema where high pressures are needed to ventilate and get chest rise. History of snoring or sleep apnea.

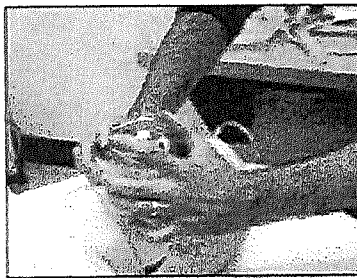
Figure 8.8. BVM techniques



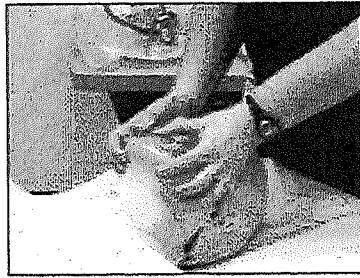
1) Ideal BVM positioning is obtained by aligning the patient's external auditory meatus with the sternal notch.



2) Traditionally, the 'EC' hand position is utilized to obtain a seal with the mask.



3) With the two-provider technique, one person should hold the mask with both hands, while the other bags the patient.



4) An alternative method is for the mask holder to apply pressure to the mask while using four fingers to apply jaw lift.

Advanced Airway Management

In Chapter 4 the issues that relate to difficulty in using a BVM were discussed along with the MOANS mnemonic (Fig. 8.7). When staff is failing it ventilate a patient in respiratory arrest a decision has to be made to place an advanced airway. An advanced airway is a device that isolates the trachea away from the esophagus and directly allows for ventilation of only the lungs. It is fitted with a universal adapter on the end to attach to a bagging unit without a mask on the face (failure to get a seal with the mask is the leading reason for BVM failure). Traditionally the advanced airway was a endotracheal tube placed by intubation but now several easier to place devices are available such as the laryngeal mask airway (LMA) and the King Airway. See Figures 8.9 and 8.10. These devices do not go past the vocal cords and into the trachea. Such devices are called supraglottic airways.

Figure 8.9. King Airway

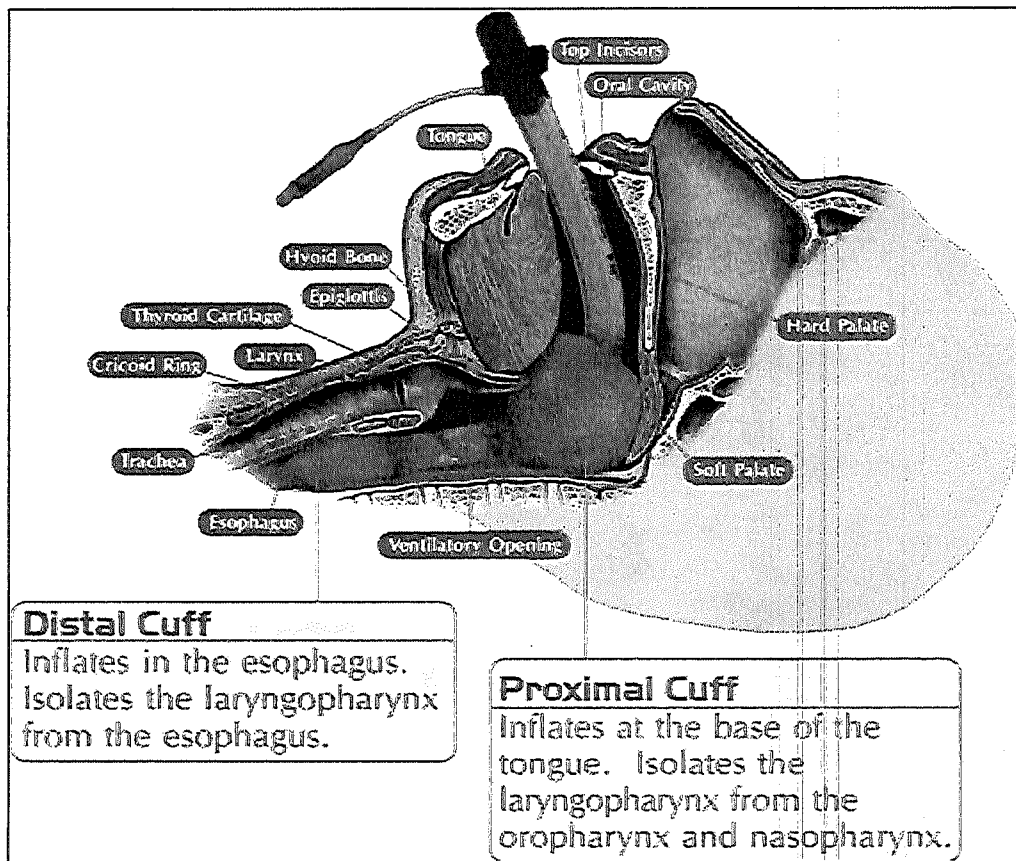


Figure 8.10. Laryngeal mask airway (LMA)

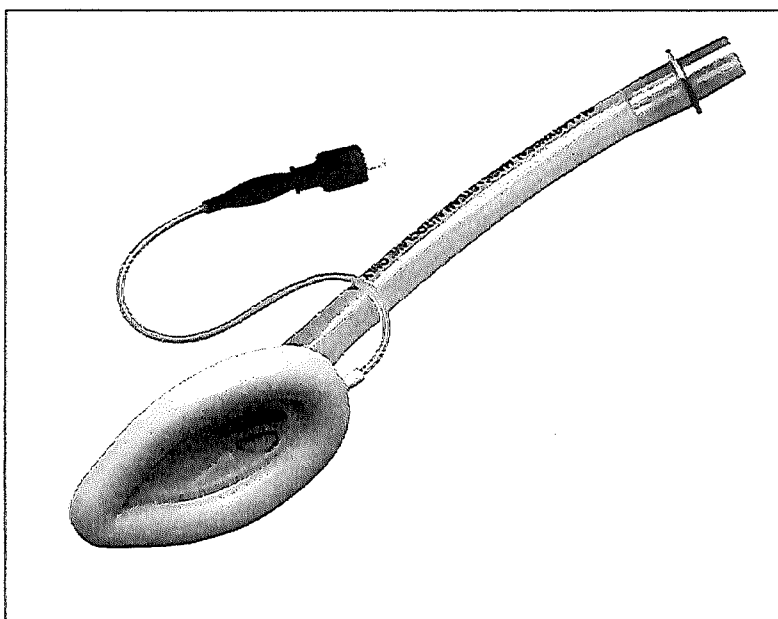
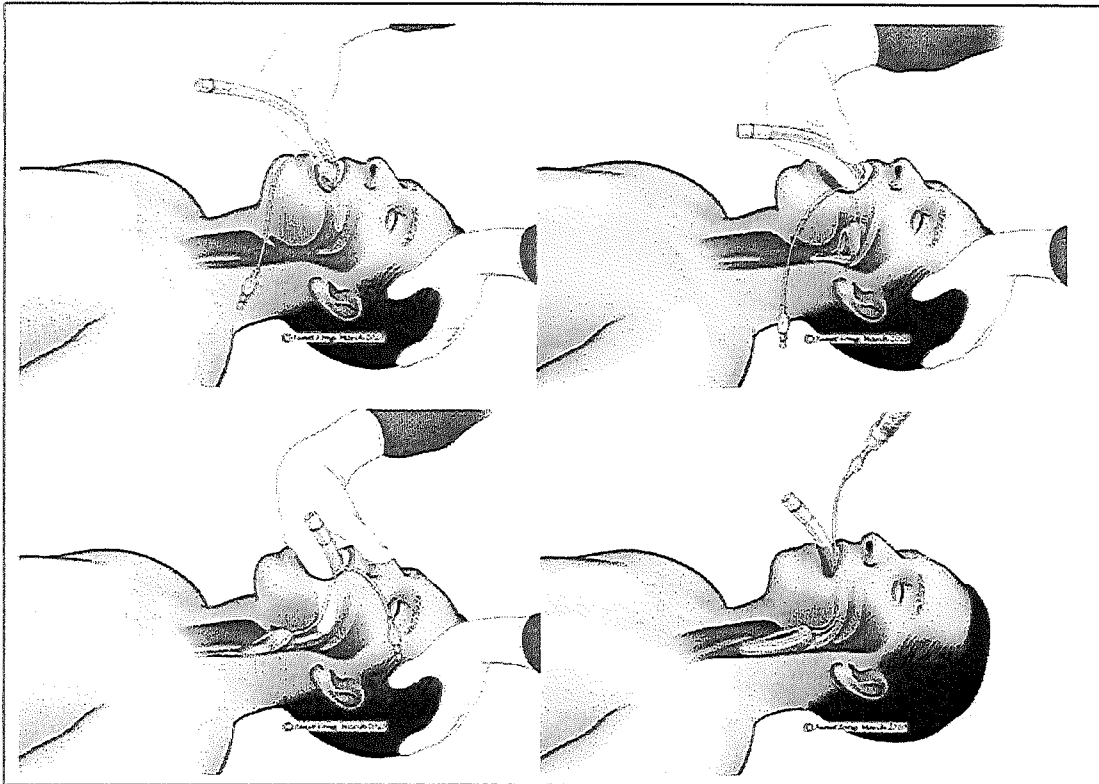
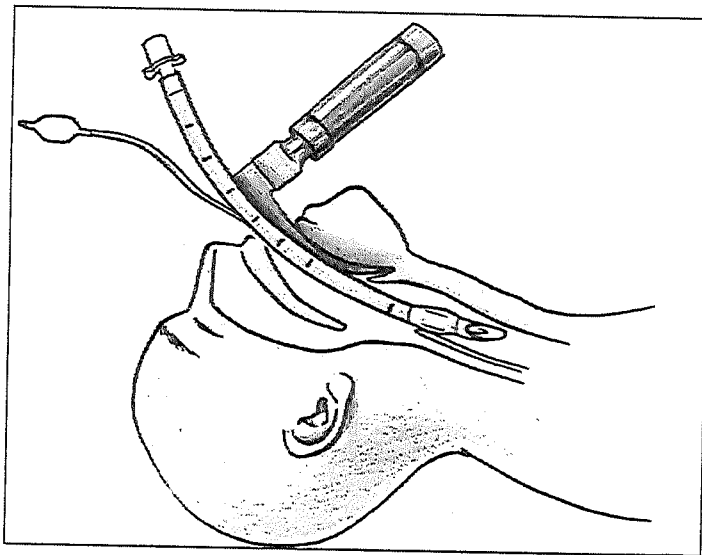


Figure 8.11. Placement of an LMA



The issues with intubation, the “golden” standard for advanced airway management, are that many doctors and other healthcare providers who are allowed to intubate cannot easily undertake a direct laryngoscopy intubation as they do not do the skill frequently enough to maintain it. The LMA and King Airway are easier to place requiring no direct visualization of the glottal opening. These newer types of alternative airways should be readily available in the emergency equipment area of all PSA locations where moderate or deep sedation is undertaken.

Figure 8.12. Intubation with a laryngoscope



Laryngospasm

Most papers looking at the incidence of laryngospasm discuss its occurrence in the setting of patients under GA. The incidence in the setting of PSA is unknown but thought to be very low. In GA the incidence is 1.7% in children up to 9 years of age which is double that of adults. In pediatric patients with upper respiratory infection the incidence rises to 9.6% (1). Refer to figure 8.13 for treatment. The Laryngospasm notch is a pressure point. If bilaterally applied pressure is placed it can break severe laryngospasm (mechanism unknown). See Figure 8.13.

Figure 8.13. Location of laryngospasm notch

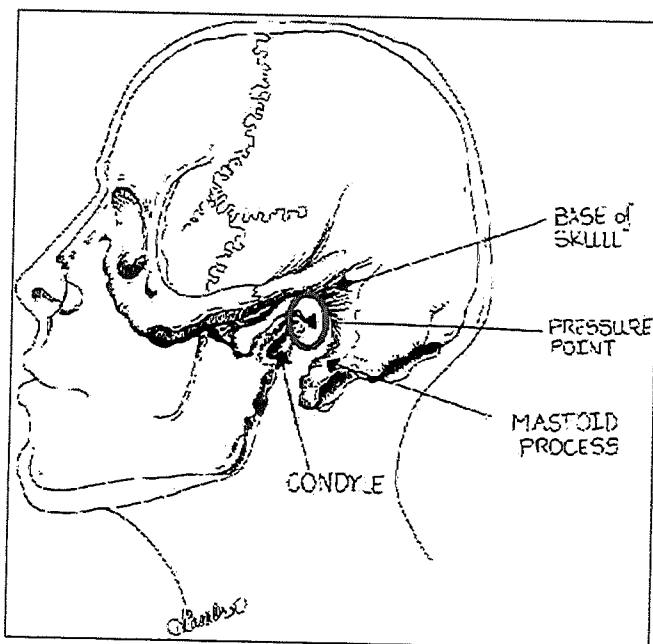
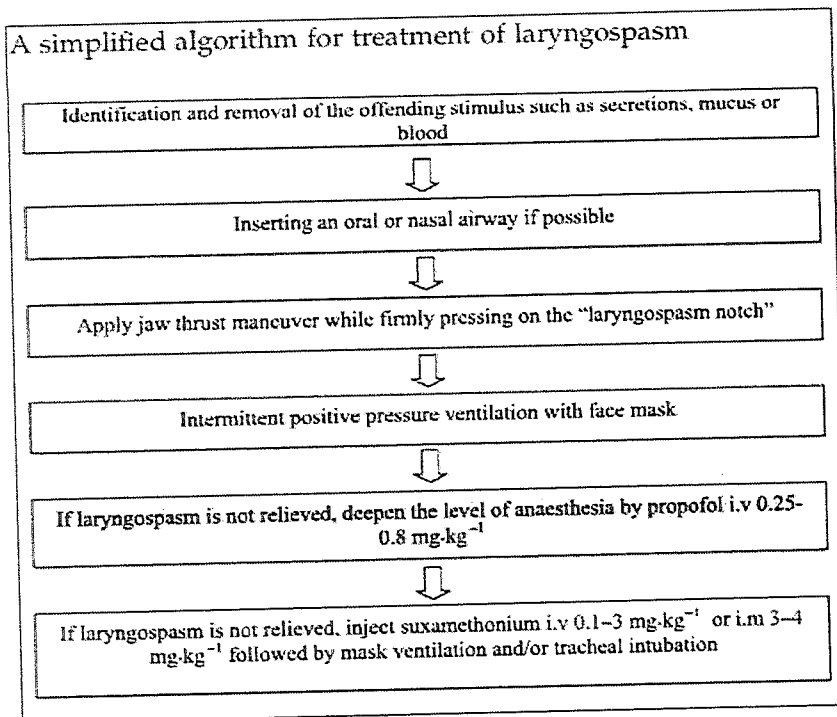


Figure 8.14. Laryngospasm Algorithm



Bronchospasm

Bronchospasm is a lower airway obstruction due to contraction or spasm of bronchial smooth muscle. It may result from an anaphylactic allergic reaction or an anaphylactoid reaction, independently or in combination with laryngeal edema. More common it is a consequence of the hyperreactive airway typical in patients with asthma. Regardless of the cause for bronchospasm, the patient will exhibit dyspnea and wheezing attributed to obstruction in the chest, not the throat or mouth. Bronchial smooth muscle is under autonomic nervous control and requires beta-2 sympathomimetics for relaxation such as ventolin. Following primary assessment, including oxygen supplementation, a selective beta-2 agonist should be administered by a metered inhaler or by nebulization. Other treatments such as steroids can wait until later as their onset of action occurs hours later.

Management of Cardiovascular System Complications

Hypotension

A common complication of PSA is hypotension. It most often occurs as a result of decreased vascular tone and the resulting vasodilation. Sedatives can also have a direct myocardial depressant effect (negative inotropic effect) on the heart. Hypotension can also be procedural, arising from dehydration, vasovagal response, hemorrhage, sepsis, and anaphylaxis (2). Treatment if pressures drop below 90 mmHg or 15-20 mmHg from baseline is to provide IV fluid boluses of 250-500 ml of normal saline rapidly along with positioning the patient supine or in a modified trendelenburg position. These boluses may

need to be repeated. In patients with a limited cardiac reserve (heart failure for example) care must be taken when giving fluid boluses. On occasion vasopressors also need to be given in small doses. Agents such as ephedrine can be used to increase vascular tone.

Anaphylaxis

Anaphylaxis is likely when all of the following 3 criteria are met:

- Sudden onset/rapid progression of symptoms.
- Life-threatening Airway and/or Breathing and/or Circulation problems.
- Skin and/or mucosal changes such as flushing, urticaria, angioedema are present

Remember:

- Skin or mucosal changes alone are not a sign of an anaphylactic reaction.
- Skin and mucosal changes can be subtle or absent in up to 20% of reactions. (some patients can have only a decrease in blood pressure, i.e., a Circulation problem)
- There can also be gastrointestinal symptoms (e.g. vomiting, abdominal pain, incontinence).

See Figure 8.14 for treatment guidelines.

Vaso-Vagal Reactions and Syncope

Syncope is one of the most common cardiovascular complications of PSA ⁽⁴⁾. It can be associated with hypotension, hypoxemia, or even on occasion cardiac dysrhythmias. Figure 8.15 shows the most current classification system for syncope presented in *The World Journal of Cardiology* in 2010. ⁽⁵⁾ Most syncope related to PSA is probably a combination or syncope due to drug induced orthostatic hypotension and reflex syncope from a vasovagal source. The vasovagal reflex syncope is induced mainly from a vagal tone induced bradycardia and resulting low cardiac output. This may be exacerbated by drug induced or volume depletion hypotension leading to decreased venous return. Treatment includes:

- Maintain A,B,C's.
- Place patient in a supine position.
- Consider modified trendelenburg.
- Consider fluid boluses as per hypotension treatment guidelines.
- Immediately stop any triggering activity.
- For persistent bradycardia consider atropine.
- Vitals, cardiac monitoring.
- If other dysrhythmias detected treat as per PALS/ACLS guidelines and consider necessity of calling a "code blue".

Figure 8.14. Anaphylaxis Treatment Algorithm ⁽³⁾

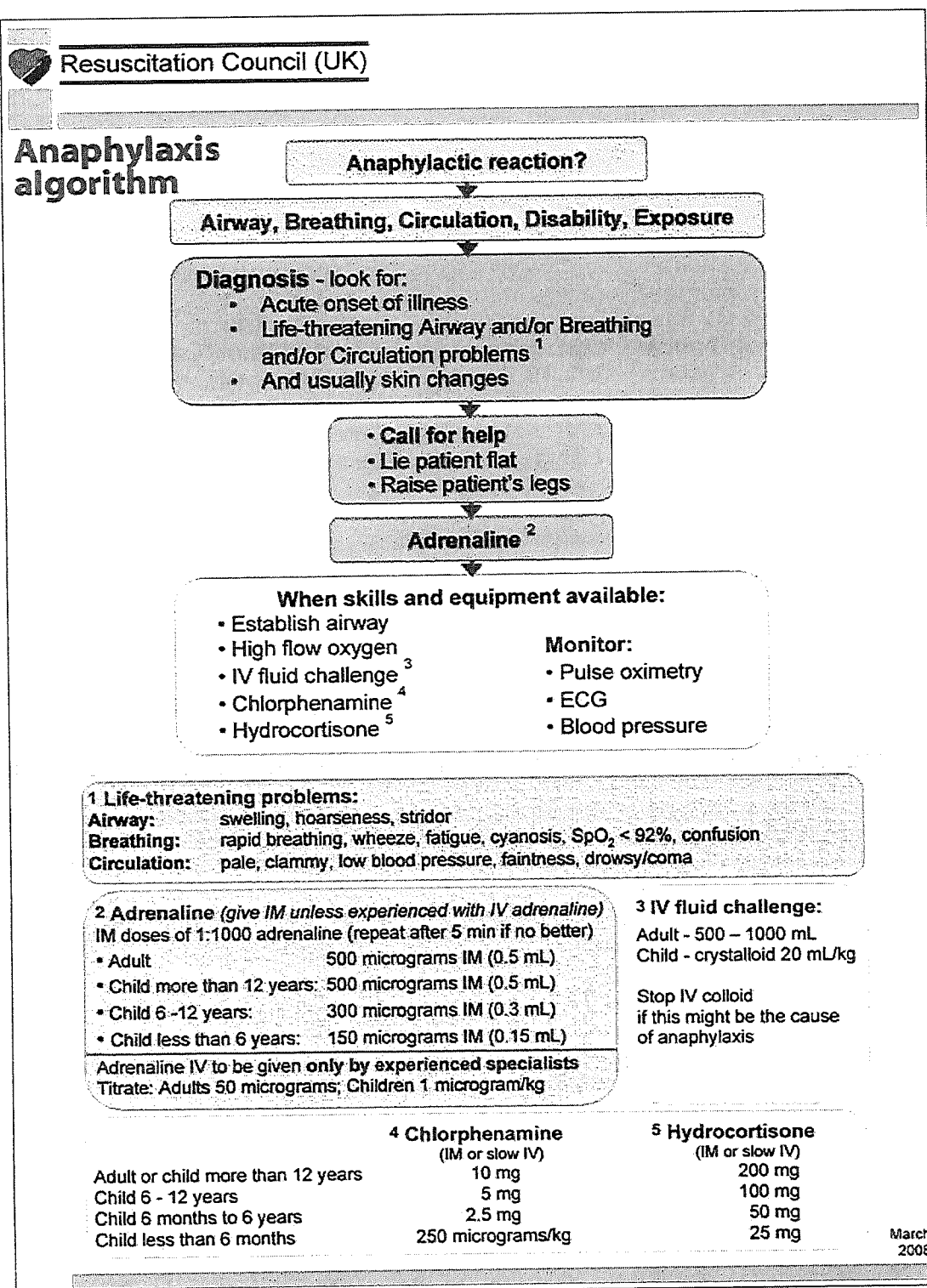
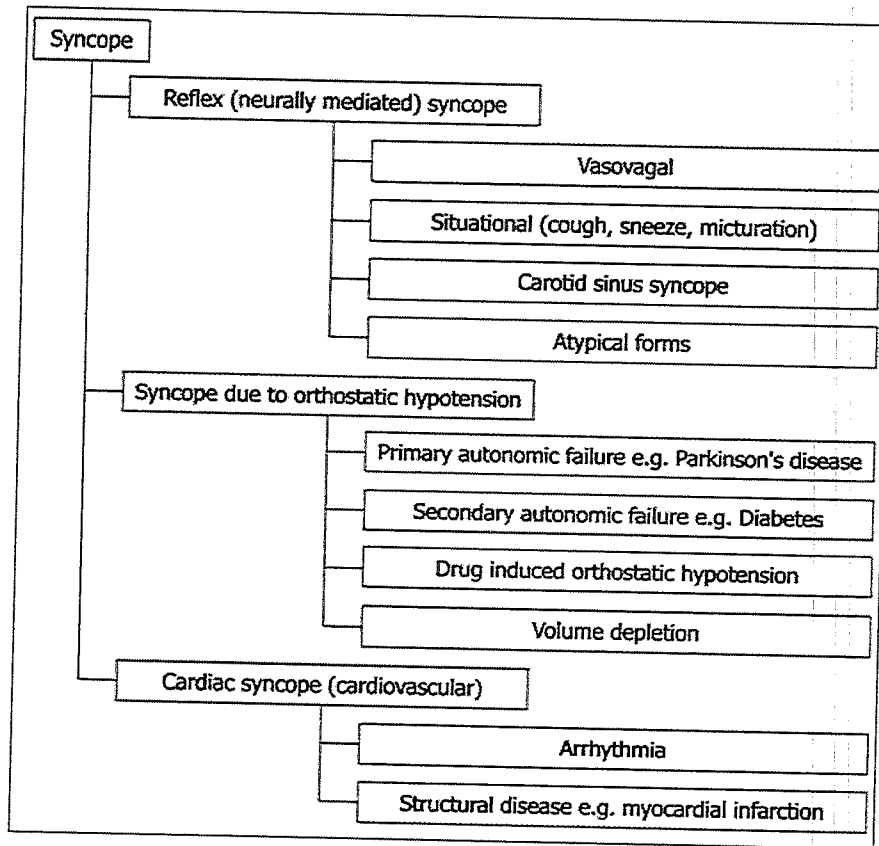


Figure 8.15. Classification of syncope (6)



Hypertensive Crisis

In patients with preexisting hypertension, some may develop a hypertensive crisis during PSA. It may be triggered by stress and pain or by the use of drugs such as epinephrine. during the procedure. If severe, immediate and controlled reduction of blood pressure. Drugs commonly used include nicardipine, nitroglycerin, nitropruside, and beta blockers.

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