Facial Plastic and Reconstructive Surgery

A Comprehensive Study Guide

Brian J.-F. Wong
Michelle G. Arnold
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Editors

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Illustrated by Bryan Lemieux
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Contents

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Introduction

This review book was undertaken as a project designed to address a need in the specialty of Facial Plastic and Reconstructive Surgery. There are many wonderful textbooks available in print and hard copy that are exhaustive and detailed on all aspects of this specialty. However, as a resource for review and a guide for study, we found no comprehensive text. In North America and now globally, certifying board examinations in the specialty of Facial Plastic and Reconstructive Surgery are gaining broad acceptance as a metric of certification, professional excellence, and achievement. Hence, an aim of this book is to aid those who wish to pursue these standardized examinations.

Admittedly, part of our motivation was selfish. Each of us will have to take Maintenance of Certification Examinations in the near future, and we recognized a need for a concise study guidebook for Facial Plastic and Reconstructive Surgery. A study guide is softcover and something that lives in your backpack. It is designed to be annotated with notes and scribbled on. It needs to be light and easily carried. And there is something ethereal about paper that remains transcendent at least for the current rising generation of Facial Plastic Surgeons. Otolaryngology—Head and Neck Surgery has such guides with K.J. Lee's Essential Otolaryngology or Reza Pasha's Clinical Reference Guide serving as excellent examples. Hence, we felt there was a need and thus addressed it. Our approach toward developing this review book and study guide is rather novel, and we took our inspiration from "crowdsourcing" and reached out to others for content. Naturally, we focused on those who had a vested interest in producing a practical review book, the actually examinees. For the most part, with this first edition, we identified fellows in training, who would soon take a board certification examination in Facial Plastic and Reconstructive Surgery, and asked them to write the chapters. These are individuals who, at this point in their careers, are focused and most directed at understanding the subtlety as well as esoterica that permeates this field. We feel this multitude of voices and perspectives, though it does lend to some variability in content and organization, provides a richer, more constructive and informed read.

This is the first edition, and with the support of our managing editors at Springer, the first of what we hope to be many yearly revisions. As such, each chapter was written de novo, and each author had a unique view with respect to identifying, structuring, and presenting material pertinent for the advanced

reader. This was not designed as a textbook or introductory volume for beginners. Significant base knowledge and understanding is critical and important. This book was conceived as concise resource that would allow someone to review quickly relevant information in this specialty.

Each year, we will recruit a new set of authors, who will edit and revise each and every chapter. Over time, this iterative approach hopefully will result in an exhaustive and succinct survey of the specialty, and evolve into the ideal preparatory text for the various board examinations in North America and abroad. While we feel it will take one or two more iterations before this edition hits its stride, we believe we have a solid foundation. To that end, in this inaugural edition, we are very fortunate to have as contributing authors three AAFPRS Anderson Prize winners, and their chapters are elegantly written, concise, and to the point.

This project has been our labor of love and the product of thousands of emails, innumerable late night phone calls, and brainstorming sessions. We also worked closely with two artists who illustrated this book and generated over 90 % of the original artwork contained herein. Brian and Aaron Lemieux are identical twin brothers, and in addition to being first-rate medical illustrators, they also happen to be brilliant medical students. We feel this is yet another reason why our book stands apart.

We believe this review book to be living body of work, as each year it will be updated and reviewed by a new set of 15–20 surgeons pursuing advanced training in Facial Plastic and Reconstructive Surgery. We are grateful and appreciative of the efforts of Daniel Dominguez and Rebecca Amos at Springer, who have shepherded us through this process and provided guidance along every step of the way.

Lastly, we dedicate this book to our spouses and our mentors.

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Part I

Basic Principles, Perioperative Management, and Miscellaneous Topics

Analgesia and Conscious Sedation

1

Christian P. Conderman

Sedation and Analgesia

- **Sedation is a Continuum** of states from minimal sedation (anxiolysis) to general anesthesia; depth of sedation can be fluid and a patient's clinical status can quickly go from a state of lighter to deeper sedation and vice versa; sedative-analgesics can be given in combination with local and regional anesthesia for greater effect and a reduction in the overall amount of sedative-analgesic medication that may be necessary.
- **Purpose**: (1) allows patients to tolerate unpleasant procedures by relieving anxiety, discomfort, or pain (2) may expedite conduct of procedures in children and uncooperative adults that are not particularly uncomfortable, but that require the patient not to move.
- Sedation can never compensate for an inadequate local anesthetic block; if the regional or local block is deemed inadequate it should be repeated prior to administration of further sedative-analgesic medication.
- Sedation practices may result in **cardiac or respiratory depression** resulting in hypoxemia and must be appropriately recognized and treated to avoid the risk of hypoxic brain injury, cardiac arrest, and/or death.
- **Primary causes of morbidity** associated with sedation-analgesia are drug-induced respiratory depression and airway obstruction.
- Sedatives and analgesics tend to **impair airway reflexes in proportion to the degree** of sedation-analgesia achieved.
- Practitioners must be able to "**rescue**" patient from a deeper state of sedation than anticipated, i.e., for moderate sedation may include managing a compromised airway or hypoventilation and for deep sedation may include need to manage respiratory or cardiovascular instability with appropriate medications or interventions.
- Four variables are used to define the level of sedation: (1) level of responsiveness, (2) airway function, (3) spontaneous ventilation, and (4) cardiovascular function (Fig. 1.1).
- Reflexive withdrawal from stimulus is **not a purposeful response** and indicates a state of **deep sedation or general anesthesia.**

Minimal Sedation—drug-induced state facilitating performance of a procedure that maintains normal responsiveness and doesn't impair airway, ventilation, or cardiovascular function. Cognition and coordination may be impaired. For example, single oral sedative or analgesic or application of <50 % nitrous oxide with no other sedative or analgesic.

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Continuum of Depth of Sedation: Definition of General Anesthesia and Levels of Sedation/Analgesia

	Minimal Sedation (Anxiolysis)	Moderate Sedation/Analgesia (Conscious Sedation)	Deep Sedation/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

Fig. 1.1 Depth of sedation from minimal to a state of general anesthesia is dependent on four variables (figure taken from ASA practice guidelines for sedation/analgesia by non-anesthesiologists). Used with permission

ASA Physical Status Classification

ASA PS Classification	Definition	Examples including, but not limited to:
ASA I	A normal healthy patient	Healthy, non-smoking, no or minimal alcohol use
ASA II	A patient with mild systemic disease	Mild diseases only without substantive functional limitations. Examples include (but not limited to): current smoker, social alcohol drinker, pregnancy, obesity (30 < BM < 40), well-controlled DM/HTN, mild lung disease
ASA III	A patient with severe systemic disease	Substantive functional limitations; One or more moderate to severe diseases. Examples include (but not limited to): poorly controlled DM or HTN, COPD, morbid obesity (BMI^{3}40), active hepatitis, alcohol dependence or abuse, implanted pacemaker, moderate reduction of ejection fraction, ESRD undergoing regularly scheduled dialysis, premature infant PCA < 60 weeks, history (>3 months) of MI, CVA, TIA, or CAD/stents.
ASA IV	A patient with severe systemic disease that is a constant threat to life	Examples include (but not limited to): recent (< 3 months) MI, CVA, TIA, or CAD/stents, ongoing cardiac ischemia or severe valve dysfunction, severe reduction of ejection fraction, sepsis, DIC, ARD or ESRD not undergoing regularly scheduled dialysis
ASA V	A moribund patient who is not expected to survive without the operation	Examples include (but not limited to): reptured abdominal/thoracic aneurysm, massive trauma, intracranial bleed with mass effect, ischemic bowel in the face of significant cardiac pathology or multiple organ/system dysfunction
ASA VI	A declared brain-dead patient whose organs are being removed for donor purposes	

Fig. 1.2 ASA guidelines. Used with permission

Moderate (Conscious) Sedation—depressed state of consciousness with patients remaining purposefully responsive to verbal commands alone or accompanied by light tactile stimulation. **No intervention required to maintain a patent airway.** Ventilation and cardiovascular (CV) function adequate/maintained.

- Example of moderate sedation regimen: initial IV doses of 2 mg versed and 50 mcg fentanyl with titration doses of 0.5–1 mg for versed (up to 1 mg/kg) and incremental doses of 25 mcg of fentanyl until suitable level of sedation is reached.

Deep Sedation—Depressed state of consciousness during which patients not easily aroused, even by (repeated) painful stimulation**.** Purposeful response may be elicited with repeated or painful stimulation, however. **Ability to maintain ventilatory function and a patent airway may be impaired.** CV function usually maintained.

General Anesthesia—state in which patients are not arousable, even by painful stimulation. 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 (CV) function may be impaired**.

ASA Classification Chart

The ASA (American Society of Anesthesiologists) guidelines are used to measure a patient's overall health and tolerance for procedures (Fig. 1.2). In general, patients with more severe systemic disease (III and higher) should have a preoperative anesthesia evaluation. Consideration should be given to performing the procedure under general anesthesia as they may be at higher risk of complication(s) during sedation.

Summary of Task Force Recommendations for Sedation-Analgesia by Non-anesthesiologists

Patient Evaluation—pre-procedure H&P increases likelihood of satisfactory sedation and decreases likelihood of adverse outcomes; components of H&P assessment of major organ system abnormalities, previous adverse experiences with sedation-analgesia, allergies, current meds and potential drug interactions, time and nature of last oral intake (see table below), history of tobacco, alcohol, or substance abuse. Exam should include vital signs (VS), auscultation of heart and lungs, and evaluation of airway, head, and neck (see below).

Assessment of Airway—Anatomic features related to difficult tracheal intubation predisposed to upper airway obstruction: Hx of previous difficult intubation, stridor, snoring or sleep apnea, advanced rheumatoid arthritis or cervical abnormality, chromosomal and other developmental abnormalities; **OSA and obesity increase the risk of airway obstruction during sedation.**

- **Exam findings portending difficult airway**: significant obesity, short neck, limited neck extension, decreased hyoid-mental distance (<3 cm in adults), neck masses, cervical spine disease, tracheal deviation, facial dysmorphia
- Oral evaluation: small opening/trismus, edentulous, protruding incisors, loose or capped teeth, dental appliances, high-arched palate, macroglossia, tonsillar hypertrophy, non-visible uvula (**Mallampati classification**), micrognathia, retrognathia, malocclusion.
- In general, obstruction of the airway during sedation is the result of lost muscle tone, especially at the level of the velopharynx; sedation produces loss of wakefulness and cortical influence on the maintenance of airway tone; sedatives can also preferentially depress airway neuromuscular function.

Airway Management—Acute airway obstruction or excessive ventilatory depression mandates emergent management—(1) determination that airway obstruction or ventilatory depression exists: presence of hypoventilation, significant decrease in SpO_2, or lack of patient responsiveness (2) use of verbal and tactile stimuli to prompt patient to breathe and reassess degree of airway obstruction or ventilatory depression (3) if obstruction or ventilatory depression is present, give supplemental O_2, give further verbal and tactile prompting to breathe, jaw-thrust, manual bag mask ventilation, pharmacologic antagonists, calling for help (4) readily available equipment (see below), seal btw mask and face, gently ventilate, and insert oral airway.

Pre-procedure Preparation—Labwork should be guided by a patient's medical condition and should only be considered if results would affect management of sedation-analgesia; counsel and consent patient before moderate/deep sedation; fasting decreases risks during sedation (see below) (Fig. 1.3). In emergency situations (fasting not practical)—consider potential for pulmonary aspiration to determine target level of sedation (less sedated) and consider delaying procedure or protecting trachea with intubation.

Monitoring—Appropriate and effective monitoring is key to safe sedation and monitoring based on four physiologic variables used to define the level of sedation. Parameters below should be recorded during sedation **at minimum** (1) before beginning the procedure, (2) after administration of sedative-analgesic, (3) regular intervals during procedure (q 5 min once stable), (4) during initial recovery, and (5) immediately

Fasting Protocols for Sedation and Analgesia for Elective Procedures

	Solids and Nonclear Liquids*	Clear Liquids
Adults	6-8 hours or NPO after midnight	2-3 hours
Children older than 36 months	6-8 hours	2-3 hours
Children aged 6-36 months	6 hours	2-3 hours
Children younger than 6 months	4-6 hours	2 hours

* Includes Milk, formula, and breast milk (high fat content may delay gastric emptying)

Fig. 1.3 Fasting guidelines for sedation/analgesia in the ambulatory setting (From Sedation and Analgesia in Ambulatory Settings). Used with permission

prior to discharge. Importance of respiratory function highlighted by the fact that two of the four characteristics used to define sedation are respiratory in nature and experts agree that **ventilation and oxygenation** are related, albeit different processes, and **should be monitored separately.**

- **Level of responsiveness**: A patient's response serves as a guide to their level of consciousness and should be routine during all procedures. Many published scales exist and most rely on two variables—a stimulus (e.g., verbal or painful stimulation) and the patient's response to determine the level of sedation. Patients who can communicate verbally likely have an adequate airway and sufficient ventilatory drive. A thumbs-up or similar nonverbal communication can be used in lieu of a verbal response when not feasible (e.g., upper endoscopy) and indicates moderate sedation. Reflex withdrawal indicates deep sedation or general anesthesia.
- **Pulmonary ventilation**: Observation of ventilatory function (removal of CO_2) by auscultation or observation (i.e., watching chest rise) reduces the risk of adverse outcomes and is a means to monitor respiratory rate. This should be continually monitored and $ETCO_2$ monitoring should be considered in all patients undergoing deep sedation and those who cannot be monitored directly during moderate sedation; respiratory disturbances detected by capnography were found to **precede hypoxemia** and serve as an early warning for impending ventilatory compromise.
- **Oxygenation**: Pulse oximetry should be used to monitor **all patients** undergoing sedation-analgesia. Early detection of hypoxemia reduces the risk of adverse events during sedation-analgesia as signs of hypoxemia (e.g., cyanosis, and tachycardia) can be unreliable. SpO_2 in the 80s places a patient's oxyhemoglobin dissociation curve at a tenuous point and further declines can lead to a rapid decline in saturation levels resulting in low and dangerous oxygen levels. Monitoring oxygenation by pulse ox **is not a substitute for monitoring ventilation.**
- **Hemodynamics**: Sedation-analgesia can produce significant autonomic and hemodynamic reflexes or disturbances including hypo- and hypertension, tachy- and bradycardia, arrhythmias, and myocardial ischemia. BP and HR should be recorded prior to initiating a procedure and **VS** should **be monitored at 5-min intervals** once a stable level of sedation is established for both moderate and deep sedation. Electrographic monitoring can be used to detect more than 80 % of ischemia with proper use of modified V5 electrode-chest lead at fifth intercostal space, anterior axillary line. **Continuous EKG** should be provided for **all patients undergoing deep sedation** and those undergoing **moderate sedation with significant comorbidities** (significant CV disease or dysrhythmias) and/or procedures that can evoke autonomic reflexes or hemodynamic disturbances.

Availability of personnel—A designated individual, i.e., other than the practitioner performing the procedure, should be present to

monitor the patient throughout procedures performed with sedation-analgesia. During deep sedation, this individual should have no other responsibilities; however during moderate sedation, he/she may assist with minor interruptible tasks once the patient's level of sedation-analgesia and VS has stabilized.

Training of personnel—The individual responsible for monitoring the patient should be trained in recognition of complications associated with analgesia-sedation. As noted above, since sedation is a continuum, practitioners must be able to rescue a patient from a deeper state of sedation than intended, as outlined above. He/she should understand the pharmacology of the agents used as well as the role and indication for antagonists for opioids and benzodiazepines. At least one individual capable of establishing a patent airway and positive pressure ventilation should be present when sedation-analgesia is administered. An individual with **advanced life support skills** should be **immediately available (within 5 min) for moderate and in the procedure room for deep sedation.**

Emergency equipment—Pharmacologic antagonists and other emergency medications (e.g., epinephrine, ephedrine, nitroglycerin, lidocaine), defibrillators, and equipment for establishing an intravenous line and airway (including pediatric endotracheal tubes where appropriate) should be immediately available for all cases of sedation-analgesia.

Supplemental O_2—Supplemental O_2 should be considered for moderate sedation and should be administered **during all cases of deep sedation** to reduce the possibility, frequency, and duration of hypoxemic episodes. Its use has been shown to decrease the magnitude of desaturation and decrease the incidence of ST changes in patient with ischemic heart disease. O_2 at 2–3 L/min or an inspired concentration of approximately 30 % O_2 can help maintain normal oxyhemoglobin saturations even in the presence of significantly reduced minute ventilation. Supplemental O_2 can delay recognition of apnea and hypoxemia is not identical to a state of pulmonary hypoventilation.

Use of Sedative-Analgesics

- **Combination**—Sedatives and analgesics used in combination can provide adequate moderate and deep sedation; however**, combinations may increase likelihood of adverse outcomes, i.e., ventilatory depression and hypoxemia**. Each component should be given to achieve the desired effect, e.g., additional analgesic for pain relief and additional sedative for anxiolysis or reduction in awareness. Respiratory function must be **continually monitored** when given in combination and there should be an appropriate reduction in the dose of each component based on the patient's status.
- **Titration**—Incremental administration improves patient comfort and decreases risks for both moderate and deep sedation, i.e., sedative/analgesic agents should be given in small, incremental doses that are titrated to the desired end point and not given as one-time bolus. When administered via non-intravenous routes, allowance should be made for the time to bioavailability and potential for unpredictable effect (IM). **Repeated doses of PO meds to supplement sedation are not recommended.**
- **Induction agents for sedation-analgesia**—Propofol, methohexital, and ketamine can be used to achieve moderate and deep sedation; however propofol and methohexital can produce a **rapid, profound decrease in LOC and cardiorespiratory function** culminating in a state of general anesthesia. Ketamine, while associated with less cardiorespiratory depression, can still cause laryngospasm, airway obstruction, and pulmonary aspiration. Moreover, because of its dissociative properties, signs of depth of sedation may be obfuscated. When these meds are used for analgesia-sedation, practitioner should care for the patient **as if it were a case of deep sedation** and should be qualified to rescue the patient from general anesthesia.

Intravenous access—When analgesics-sedatives are given intravenously, IV access should be maintained until patient is no longer at risk for CV or respiratory depression (until ready for discharge). In cases where oral sedation is given, the need for access can be determined based on the potential need for additional medication and/or resuscitative drugs. In all cases, an individual with the skills to establish IV access should be immediately available.

Reversal agents—Acute reversal for opioids (**Naloxone** 0.4–2 mg initial dose may be repeated every 2–3 min; 0.1 mg/kg in pediatric pts) and benzodiazepines (**flumazenil** 0.2 mg dose, may repeat ×3; 0.01 mg/kg in pediatric pts with max of 3 mg/h) may result in pain, hypertension, tachycardia, and pulmonary edema. The literature supports the use of these agents to reverse opioid-induced sedation and respiratory depression as well as BZD-induced sedation and ventilatory suppression when given alone or in combination with an opioid. Nonetheless, the task force recommended that **respiratory depression be treated initially with encouragement or stimulation to breathe deeply, supplemental O_2 and, if necessary, positive pressure ventilation by mask**. Reversal agents should be immediately available and may be especially helpful where airway control and positive pressure ventilation are difficult. After reversal, patients need to be monitored to ensure that sedation and cardiorespiratory effects do not recur as **flumazenil and naloxone have shorter half-lives than the opioid and BZD they are meant to antagonize**. Routine reversal of sedative or analgesic medication is discouraged; that is, their use was discouraged to routinely awaken patients from a state of sedation at the conclusion of a procedure.

Recovery/discharge—Patients may continue to be at significant risk of adverse effects following procedures as stimulation is reduced, delayed drug absorption can occur and slow drug elimination may contribute to residual sedation and cardiorespiratory depression. Patients **should not be discharged until** they are **near their baseline LOC and are no longer at increased risk for cardiorespiratory depression. VS should be stable and the patient should be well hydrated prior to discharge. Sufficient time should have elapsed after the last administration of reversal agents (if given) to ensure that the patient is not at risk for re-sedation after the reversal has worn off.** An outpatient should be discharged in the presence of a responsible adult who will accompany them home and stay with him/her until the pt can function independently; written instructions should be provided; if a designated adult is unable to assume responsibilities, arrangements should be made to admit the pt to a hospital or aftercare facility.

Special situations—Patients with severe underlying medical conditions (extremes of age, severe cardiac, renal, pulmonary, hepatic dysfunction, pregnancy, etc.) should be seen by appropriate consultants prior to undergoing moderate or deep sedation. For deep sedation, immediate availability of an individual with postgraduate training in anesthesia will decrease the likelihood of adverse events, and the task force was equivocal in this regard for moderate sedation. An anesthesiologist should be consulted if it is likely that sedation to the point of unresponsiveness is anticipated or in the context of a severely compromised or medically unstable patient (e.g., anticipated difficult airway, severe COPD, CAD, or CHF).

Medications

Basics: Opioid analgesics produce potent, dose-dependent analgesia, but little sedation and sedatives alone do not result in optimal sedation for painful procedures. A synergistic effect exists when using opioids and sedatives in combination that can result in dose-dependent and potentially profound respiratory depression and apnea, and may require a dose reduction in both BZD and opioid when given in combination (although either class of medication can result in hypoventilation when given alone). Moderate-to-severe pain cannot be effectively treated with moderate IV sedation/analgesia and these procedures require general anesthesia if local anesthesia cannot be used. Clinicians who

embark upon moderate sedation with the notion that it can provide adequate conditions will inevitably end up with a level of sedation deeper than moderate sedation. Naloxone should be given before flumazenil if significant respiratory depression exists as respiratory depression is more likely due to the opioid's effect.

Opioids/narcotics: Group of naturally occurring, synthetic and semisynthetic medications that act on six opiate receptors (Mu, Kappa, and Sigma most important) in CNS, each having a unique activity profile based on affinity for given receptor and its lipid solubility; Narcotics enhance the effect of other sedatives and reduce the amount of local required.

- **Mu** receptor is responsible for **analgesia** and euphoria, Kappa receptor results in sedation and respiratory depression, and Sigma receptors cause dysphoria.
- **Naloxone**—Opioid antagonist at all six receptors with **plasma half-life that is less than that of morphine**, i.e., narcotic overdose may recur after having been reversed due to shorter length of action of antagonist. Reversal can lead to catecholamine release that may lead to CV compromise in pts with underlying cardiac disease. Similarly, it can precipitate withdrawal Sx in chronic narcotic users.
- Clinical findings associated with opioid use:
  - Nausea—due to increased tone at GI sphincters and decreased peristalsis
  - Hypotension—due to peripheral histamine release
  - Pinpoint pupil—characteristic of narcosis due to stimulation of Edinger–Westphal nucleus
- Can lower seizure threshold in pts with seizure disorders
- Can produce significant respiratory depression in a dose-dependent manner but usually only produce mild hemodynamic depression
- Should be used carefully in pts taking other CNS-depressing meds, i.e., phenothiazines (thorazine, compazine), antihistamines, sedatives, and in combination with other narcotics
- Full agonists (e.g., morphine, fentanyl, demerol) have no ceiling effect to analgesia and this effect continues in a linear fashion until effect or adverse effect is achieved
- **Morphine**: Prototypical narcotic
  - Long-lasting (half-life 2–3 h), slow onset of action (poorly lipid soluble), may cause respiratory depression
- **Demerol (meperidine)**:
  - Greater lipid solubility → hypotension and dysphoria that may be more profound than morphine
  - Respiratory depression equal with equivalent doses
  - **Can interact with MAOi's and SSRIs → serotonin syndrome: seizures, coma, hypertension, and pyrexia**
  - Intramuscular doses may be erratically absorbed
  - Tremors, myoclonus, and seizures can result from accumulation of normeperidine (active metabolite with longer half-life than parent compound that may prolong duration of action); normeperidine seizures are **not responsive to naloxone**
- **Fentanyl**—Most commonly administered opioid with nearly ideal characteristics when used for sedation
  - Highly lipid soluble with immediate onset (and peak effect in 5 min) and short duration when given in small analgesic doses
  - Quickly redistributed through fat and skeletal muscle leading to saturation with repeated doses over 4-h period; poor drug for long-term pain control; increased muscular activity can release medication in recovery room; more potent respiratory depression, less N/V and Hypotension
- **Remifentanil, alfentanil, sufentanil**—opioids related to fentanyl with similar or higher potency
  - Remifentanil is metabolized by a plasma enzyme and accounts for its ultra-short duration of action
  - Along with fentanyl, these compounds do not cause histamine release and may be preferred in pts with CV instability

- **Dilaudid (hydromorphone)**—congener of morphine that is 3–5 times more potent; onset 15–30 min; half-life 2–3 h

Benzodiazepines (BZDS): Class of sedative-hypnotic medications that act on thalamus, hypothalamus, and limbic system via potentiation of inhibitory GABA neuronal activity. This effect is mediated by BZD receptor (linked to GABA receptor) causing conformational change with a resultant increased affinity for GABA. This causes an increased influx of chloride ions and subsequent hyperpolarization of the neuron that leads to an inhibitory response preventing propagation of further action potentials. BZDs have **amnestic, anxiolytic, sedative-hypnotic, anticonvulsant, and muscle-relaxing effects**. In general BZDs have **little or no analgesic** properties. Three parenteral BZDs are available in the USA: midazolam (versed), diazepam (valium) (both valium and versed are more lipophilic than ativan), and lorazepam (ativan), which has a longer onset and overall effect.

- **Hepatic degradation** is the only route of excretion mandating dose reduction in patients with liver dysfunction for these medications; patients with hepatic encephalopathy are at increased risk of exacerbation of their Sx when BZDs are given.
  - Age and degree of liver dysfunction must be considered prior to administration of BZDs and may require reduction in dosage.
- Hypoventilation that may be seen is likely due to depressant effects of BZD on respiratory center in brain and BZDs usually do not cause CV compromise (except in states of deep sedation where peripheral vasodilation may occur, resulting in decreased cardiac output and peripheral resistance causing systemic hypotension).
- All BZDs are highly protein bound in plasma and patients with hypoalbuminemia may have more active form of medication in circulation.
- Diazepam and midazolam are also subject to high degree of first-pass metabolism.
- **Valium (diazepam)**
  - Long half-life (~30 h for parent compound, up to 80 h with metabolites)
  - Undergoes oxidative metabolism in liver via CYP450 and has active metabolites which may prolong medication effect
  - Respiratory depression is a major side effect
  - Generally considered safe during gestation, although its use should be tapered or stopped prior to delivery as neonatal toxicity and withdrawal can be seen in infants
  - Increased volume of distribution in elderly patients as compared to young, healthy adults
  - May cause **phlebitis** at injection site due to preservatives in solution
- **Versed (midazolam)**—sedative of choice for most clinicians in short, ambulatory procedures
  - Water soluble with pH-dependent structure. Relatively short-acting due to high lipophilicity with rapid onset; context-sensitive half-life predicts that 1–2 h is often required for recovery
  - ~5× more potent than valium
  - Potent respiratory effects—depresses airway function with significant increases in airway resistance
  - Produces anterograde amnesia more commonly than valium
  - Should be given in small incremental doses; bolus doses should only be given if intubation and ventilation are anticipated
  - Dose reduction by half should be considered in elderly patients; liver and kidney play roles in excretion with hepatic blood flow being a main determinant of metabolism and excretion
  - Patients receiving concomitant P450 inhibitors (e.g., azoles, phenytoin, diltiazem) and inducers (rifampin) may need dosing modifications
- **Ativan (lorazepam)**—not routinely used in procedures requiring sedation, as it is less lipophilic than versed and valium. This results in a longer onset of action (30–40 min) and

therapeutic concentrations that remain in the CNS for longer periods.
  - Overall duration of action can last for 6–8 h.

Propofol—alkylphenol that is used for induction and maintenance of anesthesia and sedation in minor procedures and sedation in ICU patients

- Possesses sedative, amnestic, and analgesic effects.
- Highly lipophilic with rapid distribution to tissues and CNS and rapid redistribution to blood resulting in a rapid metabolic clearance.
- Exerts its actions through GABA-mediated interactions, specifically at $GABA_A$ receptors:
  - Related but not identical to BZD mechanism of action
- May rapidly produce a state of general anesthesia. Short-acting with more rapid recovery than seen with midazolam and less amnesia at equal sedative doses. Leads to faster recovery times, and less post-op n/v.
- Decreases in cerebral blood flow and cerebral oxygen consumption can also be seen; can cause reduction in BP and decrease in cardiac output without an increase in HR.
  - Concomitant administration of fentanyl can increase hemodynamic effects of propofol and the combination of these two medications should be used with caution in patients with CAD.
- Respiratory depression can be significant and it is accompanied by depression of airway tone and reflexes. Apnea can occur, especially if given as bolus.
- Patient must be monitored as under deep sedation when using propofol; best titrated to effect by administration of continuous IV infusion based on weight-adjusted dose.
- Disadvantages: Cost, narrow-therapeutic window, cardiopulmonary complications, requires presence of specially trained personnel.

Barbiturates—classified by duration of action: ultra-short, short, intermediate, and long acting; primarily provide hypnosis and do not provide analgesia or muscle relaxation. Multiple sites of action in CNS including multiple voltage-regulated ion channels; most prevalent effect on GABA receptors; allosterically enhance binding of BZDs and GABA agonists and inhibit GABA receptor antagonists

- **Thiopental**: ultra-short duration; used in induction of anesthesia
- **Pentobarbital**: short-intermediate; used as sedative-hypnotic and antiepileptic

Ketamine—IV anesthetic, structurally related to phencyclidine and cyclohexamine; produces anesthesia with muscular rigidity and open eyes

- Patients may have purposeful movements unrelated to surgical or noxious stimuli.
- Produces prolonged analgesia, blocks pain signal transmission, and produces anesthesia by blocking sodium channels; causes dissociative state upon emergence that causes patients to hallucinate.
- Dose-related increase in HR and BP through direct stimulation of CNS and sympathetic nervous system (SNS).
- Can cause increases in intraocular pressure.
- **Contraindicated** in patients younger than 3 months of age and in those with histories of airway instability, tracheal abnormalities, active pulmonary disease, CV disease, head injury, central nervous system (CNS) masses, hydrocephalus, porphyria, and thyroid disease and in patients with h/o psychosis.
- Can be associated with high potential for laryngospasm when used during upper endoscopy.

Dexmedetomidine—alpha-2 agonist with similar mechanism to clonidine with much higher selectivity for its receptor.

- Centrally induces sedation, anxiolysis, analgesia, and hypnosis with some anesthetic effects; peripherally, it attenuates the

hyperdynamic response and improves hemodynamic stability by attenuating SNS activity, thereby lowering BP and preventing pain-induced hemodynamic fluctuations.
- Spares respiratory drive and decreases need for supplemental O_2.
- Properties lead to less opioid/BZD use intraoperatively and less post-op nausea.
- Can be effective means of sedation-analgesia in facial surgery and may reduce the dosage of opioid and BZD required to achieve sedation and analgesia.
- Stabilizes CV parameters in response to pain; therefore, infiltration of local does not cause wide swings in BP and HR.
- Primary adverse effects are bradycardia and hypotension.

Clonidine—alpha-2 agonist, albeit with less selectivity for its receptor (compared to dexmedetomidine)

- Provides analgesia and sedation while stabilizing hemodynamic parameters.
- Due to its unfavorable pharmacokinetics, it is not routinely used during procedures requiring sedation:
  - Delayed onset of action (30–60 min) when given IV
  - Long-lasting, making intraoperative titration and fine-tuning of sedation difficult
  - Propensity to cause long-term orthostatic hypotension, making it further unsuitable in this setting
- Primary use is an adjunct in rhytidectomy for perioperative blood pressure control.

Total intravenous anesthesia (TIVA)—eliminates paralytics and use of volatile gases using IV agents exclusively for patient sedation. The usual regimen includes deep sedation with propofol and varying amounts of ketamine, midazolam, or fentanyl.

- Should be administered by a provider with postgraduate training in anesthesia due to the use of propofol.

Local Anesthesia

Acts by preventing depolarization of the ionic electrical gradient across the cell membrane of peripheral nervous system, via **reversible blockade of channels which prevents rapid influx of sodium ions which stops propagation of AP** (Fig. 1.4).

- **Amides** (2 "I"s in generic name): amide link more stable, resists changes in pH and temperature, metabolized by hepatic degradation; no plasma metabolism; e.g., lidocaine

	Topical		Infiltrative		Onset	Duration	Notes
	Concentration	Dose	Concentration	Dose			
Epinephrine			1:1000 -1:200,000 (1 mg/mL - 1 mg/200 mL or 1000 mcg/ml - 5 mcg/ml)	10 mcg/kg			
ESTERS							
Cocaine	4-10% (40-100 mg/mL)	3 mg/kg Topical	Not used		Immediate	30-60 mins	
Procaine (Novocaine)			1-2%	14 mg/kg Adults 5 mg/kg Children	2-5 mins	1 hr	
Tetracaine (Pontocaine)	0.5-2.0% (5-20 mg/mL))	1mg/kg Topical	0.10 - 0.25 %	1-1.5 mg/kg Infiltrative	20-30 mins	4-5 hrs	
Benzocaine (Americaine)	20%	200 mg max dose	Not used				
AMIDES							
Lidocaine (Xylocaine)	2-4 % (20-40 mg/mL)		0.5-2.0 %	4-5 mg/kg(without epi) 7 mg/kg (with epi)	7-15 mins	1.5-2 hrs	
Mepivicaine (Carbocaine	Not Effective		1-2%	7 mg/kg			
Prilocaine (Citanest)	Not Effective		1-2 %	7 mg/kg	10-15 mins	1.5-2 hrs	Methemoglobinemia with dose >600 mg
Bupivicaine (Marcaine)	Not Effective		0.25-0.75%	2 mg/kg (without epi) 3 mg/kg (with epi)	30 mins	3 hrs	High degree of cardiotoxicity

Fig. 1.4 Summary of concentration and dosages of common local anesthetic agents (table adapted from Essential Otolaryngology and references tables in Eval of Conscious Sedation in FPS)

(xylocaine), bupivicaine (marcaine), prilocaine (citanest)
- **Esters**: cocaine (broken down by plasma pseudocholinesterase), procaine, tetracaine; ester link is heat labile making esters less stable overall; hydrolyzed by cholinesterases in plasma and liver
- **Advantages**: control of airway, smoother recovery period with better pain control, less stress on CV system, less nausea → early discharge, less cost, less bleeding, awake patient can assist with positioning
- **Disadvantages**: apprehension, not all surgeons equally adept at administering local or regional anesthesia, systemic toxicities of local anesthetics, larger nerves more difficult to block, small risk of permanent nerve damage with intraneural injection, local anesthetics may have decreased potency in acidic (e.g., infected) environment due to configurational changes in anesthetic medication, resuscitation equipment, and personnel should be on hand to manage toxicity

Local anesthetic toxicity and management—Toxicity is dose dependent and is reduced by careful calculation and titration of dosage, co-administration of vasoconstrictant, avoidance of intravascular injection (leads to immediate high blood levels that can cause toxicity with small doses), and BZD premedication for its anticonvulsant effect by elevation of seizure threshold. Absorption varies by mucosal surface and application to the tracheobronchial tree can lead to rapid rises to toxic levels if excessive doses are given. Progression from mild toxicity to death is common to all of the aforementioned agents. Supplemental O_2 should be administered at first sign of toxicity and an IV line is mandatory in all but the most minor procedures and especially if the use of powerful sedative-analgesics such as midazolam and fentanyl is anticipated in addition to local.

- **CNS excitation**—Generally **first** sign of toxicity → agitation, muscle twitching, and hypertension; additional Sx in lower blood levels (1–5 μg/mL of blood lidocaine levels)—lightheadedness, euphoria, tingling of the lips, tinnitus, bitter/metallic taste.
- **Seizures:** When blood levels reach moderate range (8–12 μg/mL), a local anesthetic seizure can occur; this can be preceded by a prodrome of slowed speech, jerky tremors, and hallucinations; seizures should be controlled with BZDs (e.g., 0.1 mg/kg of valium or 1–2 mg of versed to raise seizure threshold); once seizure has developed maintenance of oxygenation is of critical importance and supplemental O_2 should be administered immediately with control of airway → patient may require intubation, ventilation, and vasopressors with persistent seizure.
- **CNS depression**—Occurs with very high blood levels (20–25 μg/mL) and toxic effects on cardiorespiratory system are seen. Somnolence, coma, and bradycardia accompany shallow respirations and respiratory acidosis ensues. This is followed by apnea, hypotension, and cardiovascular collapse and arrest.
  - Treatment should be directed toward maintenance of respiration and circulation with intubation, positive pressure ventilation, and cardiovascular support with vasopressors and fluids to restore circulation.
- **Bupivicaine** in concentrations of 0.75 % (and lower) can produce **prolonged cardiac depression if injected intravenously.**
- True allergies are rare, although esters more commonly act as allergens and cross-reactivity with amides is not described; that is, amides can be given with a hx of allergy to ester anesthetic; oftentimes allergic reaction is to preservative in solution (e.g., methylparaben, or metabisulfite).
- **Prilocaine** has **unique dose-related side effect of causing methemoglobinemia** in doses of 500 mg or more - > Tx: 1–2 mg/kg of IV methylene blue (both application of methylene blue and methemoglobinemia and can cause abnormal Sp02 reading).

Cocaine

- Naturally occurring ester that has both vasoconstrictive and topical anesthetic properties with a narrow therapeutic range.
- Mechanism—blocks norepinephrine reuptake at presynaptic terminal and blocks sodium channels for local anesthetic effect.
- Maximal dose: **3 mg/kg** and the use of more than 200–300 mg should not be necessary (e.g., 200 mg = 5 mL of 4 % solution).
- Metabolized by plasma pseudocholinesterase and should not be used in patients with **pseudocholinesterase deficiency** (dx suspected with prolonged apnea after administration of succinylcholine); patients on physostigmine or neostigmine may be prone to the toxic effects of cocaine as these agents inhibit pseudocholinesterase.
- Contraindicated in patients with h/o cardiac arrhythmias or ischemic myocardial disease, pts with h/o epilepsy or seizures, caution is urged in patients with HTN.
- **Toxicity**—primarily CNS in nature—excitability, followed by HA, N/V, tachycardia with rapid progression to delirium, Cheyne-Stokes respiration (cyclical periods of hyperpnea followed by apnea) and convulsions: Thought to be due to overwhelming sympathetic stimulation of CNS and cardiovascular and respiratory systems. If reaction is severe and therapeutic measures are not instituted rapidly, it can progress to respiratory arrest and death.
  - Hypertension and tachycardia can result prior to CNS excitation as well as sensitizing target organs to the effects of SNS stimulation-vasoconstriction, tachycardia, mydriasis, and elevated body temperature.
  - Cardiac arrhythmias (especially in pts on MAOis and TCAs)—ventricular ectopy (bigeminy is cardinal sign of myocardial hypoxia); antiarrhythmics may be necessary, e.g., lidocaine or propranolol.
- **Tx** = Provides adequate oxygenation, provides supportive measures, i.e., maintenance of patent airway, ventilation, IV administration of appropriate medications.
- Combination of ketamine and cocaine is best avoided as catecholamine release/effect by cocaine can be enhanced with this combination; halothane also sensitizes myocardium to the effects of catecholamines.
- In acute overdose, victim may report confusion, dry throat, and dizziness. Hyperreflexia may be present and tonic–clonic convulsions may occur.
  - Propranolol (Inderal) has been shown to be effective in acute setting of cocaine overdose.

Regional Anesthesia

Knowledge of cervicofacial neural anatomy guides infiltration of anesthetic medications to enhance patient comfort. Blockade of trigeminal and cervical branches achieves desired effect; volume necessary to produce anesthesia is directly proportional to the size of the nerve and larger nerves can take up to 30 min for full anesthetic effect to be seen.

V1—**Supra-orbital** and **supratrochlear** nerves can be blocked by injecting 2 cc at superior orbital rim **using superior orbital notch** as landmark and is done by lateral to medial insertion toward medial eyebrow and ending with ~1 cc over nasal bone (in some cases the lateral/deep branch of the supra-orbital nerve may need to be blocked 1 cm above the superior orbital rim at the ZF suture line in a sub-frontalis plane). **Nasociliary** nerve can be injected with 2 cc injected 2 cm deep to medial brow along medial orbital wall. The **external branch of the anterior ethmoid nerv**e can be blocked at the lower border of the nasal bone **6–10 mm** off the midline (this can reduce painful injections of the nasal tip).

V2—**Maxillary nerve** can be blocked at **foramen rotundum** by pre-auricular approach through sigmoid notch using spinal needle aiming slightly superiorly until the lateral pterygoid plate is reached and redirecting the needle anteriorly and superiorly for ~1.5 cm; 5–10 cc is usually required to block this nerve (paresthesias may be elicited to identify correct location and care must be used to avoid intravascular injection

in the infratemporal fossa). **Sphenopalatine ganglion** can be blocked via greater palatine foramen. A 4 cm 27 gauge needle is used and the **midline and buccal edge of second maxillary molar** serve as landmarks with foramen lying half way between these two points; needle is used to locate foramen and is inserted approximately 1.5 cm and 5 cc injected after withdrawal to avoid intravascular injection. **Infraorbital nerve** can be blocked where it exits infraorbital foramen **5–8 mm below infraorbital rim** (at line dropped from medial limbus on primary gaze); foramen opens downward and medial; 1–1.5 cc is usually sufficient for this block. **Zygomaticotemporal** block can be performed along posterolateral orbital rim with foramen approximately 1 cm below the lateral canthal attachment (needle inserted 10–12 mm posterior to ZF suture line aiming toward posterior concave surface of lateral orbital rim). **Zygomatico-facial** nerve emerges through foramen on anterior surface of zygoma with multiple branches and exits on anterolateral surface of malar eminence—can be inserted by injecting dime-sized area lateral to junction of inferior and lateral orbital rims; lower mid-face can be a difficult area to block during laser procedures (unless block done at foramen rotundum) and often requires field block.

V3—**Mandibular division** can be blocked as it exits **foramen ovale** in a similar fashion as the maxillary division at the foramen rotundum; after needle contacts lateral pterygoid plate, reorient needle more posteriorly to a depth of 6 cm where paresthesias are elicited and 5 cc of local anesthetic is injected. **Inferior alveolar nerve** block can be performed by passing 5 cm needle into retromolar tissue using **lingula** as landmark for injection and injecting 5 cc. **Mental nerve** (foramen usually below **apex of second pre-molar**: can lie 6–10 mm in either anterior or posterior direction) block is done by injecting 2 cc through mandibular gingivo-buccal sulcus between first and second pre-molars (in some cases, e.g., chin augmentation, additional sensation of chin below mentolabial sulcus may need to be anesthetized by injecting additional local to block contribution from nerve to mylohyoid sensory branch or in some cases the lower branch of the mental nerve). Mental nerve is 2.6–2.8 cm from midline and ~1 cm above inferior mandibular border.

Cervical plexus: Superficial branches (lesser occipital, great auricular, supraclavicular, and transverse cervical nerves) can be blocked at Erb's point 1/3 of the way between mastoid tip and sternoclavicular joint; ~10 cc injected 3 cm deep to skin at this point will suffice; great auricular nerve (C2/3) can be blocked 6.5 cm down from lower EAC on line drawn delineating mid-SCM.

Special Situations

Sedation/Analgesia in Pediatric Patients

- If IV sedation is used in pediatric procedures, the provider(s) must be prepared for poor patient tolerance as children often become restless and combative during standard IV sedation regimens.
- Similar to adult procedures, hypoxemia, presumably due to oversedation, was identified as the most common complication.
- Deeper levels of sedation are associated with a child that may be unconscious, is unable to cooperate, and has its protective reflexes blunted; increased risks of sedation and prolonged recovery room stays are significantly correlated with deeper levels of sedation; can be difficult to distinguish between under- and oversedation in an agitated child. Similar to adult ASA criteria, it is generally felt that ASA class 1 and 2 patients may safely undergo moderate sedation, those in class 3 need further evaluation, and those in class 4–5 may be better served by general anesthesia.
- Standard IV sedation with fentanyl and midazolam is safe and efficacious; however there is reported incidence of 20 % of patients with at least one side effect of sedation including bradycardia, agitation, skin rxn, vomiting, and hypoxemia.
- Diazepam metabolism is slower in infants compared to school-aged children; fentanyl

is variably metabolized in the liver and may not be an ideal sedative for infants as it has been associated with significant apneas in infants <3 months.
- Current standard of care for monitoring patients undergoing pediatric procedures is continuous pulse oximetry and visual assessment of the patient; additionally, ventilatory monitoring should be done continuously by a nurse dedicated to monitoring the child.

Malignant Hyperthermia (MH)

- **Autosomal dominant** transmission most commonly due to abnormality of **ryanodine receptor** (although other mutations exist) resulting in **abnormal calcium metabolism** triggered by a sensitivity to volatile anesthetics (halothane [most potent], enflurane, isoflurane, sevoflurane, desflurane) and depolarizing paralytic agent, succinylcholine. When used in combination, they can result in explosive onset of MH. These agents cause a perturbation of the skeletal muscle membrane resulting in skeletal muscle hypermetabolism, with prolonged contraction and state of O_2 consumption, and release of lactic acid, CO_2, phosphate, and heat. Anaerobic metabolism follows and the plasma membrane begins leaking intracellular ions and myoglobin with a subsequent rise in serum potassium and myoglobinuria.
- Signs of MH include tachycardia and tachypnea, increased $ETCO_2$, hyperkalemia, muscle rigidity and eventual rhabdomyolysis, acidosis, elevated body temperature (**may NOT be immediate**), and masseter rigidity/spasm (may be more pronounced in children) which may serve as an initial warning sign.
- MH-susceptible individuals (those with a first-degree relative with a h/o MH) can be tested via genetic testing or in vitro muscle biopsy (**caffeine halothane contracture test**).
- Local anesthetics, opiates, and non-depolarizing paralytic agents **do not trigger** MH.
- If MH-susceptible individual proceeds with procedure, preoperative precautions must be performed including washing out anesthetic machine (with high-flow oxygen and/or use of charcoal filters) as large amounts of volatile gases can remain in reservoirs of such machines.
- Tx: **Dantrolene should be given immediately** (acts to stabilize ryanodine receptor and reduce efflux of Ca^{2+} from sarcoplasmic reticulum, 2.5 mg/kg as rapid bolus through large-bore IV line repeating at 5-min intervals until signs of acute episode are reversed; once patient has been stabilized [usually following transfer] infusion at 10 mg/kg/day should be given for at least 24 h after initial successful tx). Transfer to an ICU (if procedure is being performed at an outpatient facility) should be instituted immediately. Additional measures that should be implemented immediately are **cooling of body temperature, correction of electrolyte imbalances, and immediate cessation of all volatile gases.**

Operating Room Fires

- Cutaneous facial surgery is second most commonly affected site (tonsil #1) for occurrence of OR fires. Additional high-risk procedures include airway procedures and laser procedures of the head and neck.
- Majority of cases occurred while supplemental oxygen was being used, and buildup of oxygen beneath a drape was cited as a reason for the fire in cases of cutaneous surgery.
- Requires vigilance to prevent the fire triad of ignition: **ignition source** (electrocautery, lasers, fiber-optic light cables), **fuel** (ETT, drapes, towels, sponges, pt's hair or skin, alcohol-based solutions), and **oxidizer** (oxygen or nitrous oxide).
- Provision of O_2 via nasopharyngeal tube may reduce the local concentration of O_2 in the environment, thereby reducing the risk of fire; cautery should be performed at least 5 cm away from supplemental oxygen source.

- Selective use of supplemental O_2 can limit the risk, as can the use of the lowest possible inspired O_2, waiting a 60-s period prior to use of ignition source if O_2 is given.
- Presence of fire may be heralded by abnormal sound (cracking) or burning odor, flash, or flame.
- **Management of or fire**
  - The presence of a fire should be noted and all team members should be made aware as to the presence of a surgical fire.
  - The flow of all gases should be stopped and the fire should be extinguished as rapidly as possible—initially using water and saline, and smothering the fire.
  - The fuel, such as drapes, tubes, and gauze, should be removed from the patient as rapidly as possible.

    If the fire occurs in the **Airway**, the ET tube should be removed as quickly as possible, the gas should be stopped, remove all flammable and burning materials from the airway, and pour saline into patient's airway to cool tissues and extinguish residual embers.

    Additionally, the tube should be evaluated for possibility of any residual pieces/remnants in the patient's airway and rigid bronchoscopy should be performed to assess tissue damage and remove any residual foreign materials.
  - Following control of the fire, airway support should be provided as quickly as possible with bag-mask ventilation without the use of supplemental oxygen and/or nitrous oxide, if possible.

Questions

1. **What is the first sign of local anesthetic toxicity? How does the toxicity of local anesthetics progress with further increases in plasma levels of lidocaine? What is the treatment of local anesthetic toxicity?**

   The first sign of local anesthetic toxicity is CNS excitation. This can be manifested by psychomotor agitation with muscle twitching that may progress to seizures. Additional early signs/symptoms are lightheadedness, euphoria, tingling of the lips, tinnitus, bitter/metallic taste, and hypertension. As the toxic plasma concentration of local anesthetic rises to higher levels, CNS depression can ensue followed by cardiorespiratory collapse. With the first signs of CNS excitation/toxicity, supplemental oxygen should be given and a benzodiazepine should be administered for its anticonvulsant effects to increase the seizure threshold. Additional measures include supportive care, such as ensuring that an IV is in place, and securing the airway in the case of progression of local anesthetic toxicity.

2. **What is the mechanism of action of cocaine? How is cocaine toxicity managed?**

   Cocaine is an ester local anesthetic that is broken down by plasma pseudocholinesterase. Vasoconstriction results from blockade of norepinephrine reuptake at the presynaptic membrane. Its local anesthetic effect is produced by blockage of sodium channels similar to other local anesthetics.

   Management of cocaine toxicity is primarily supportive. This includes provision of supplemental oxygen, anticonvulsants, antihypertensives, and support of the airway if progression of toxicity ensues.

3. **What causes malignant hyperthermia? What medication should be immediately available if an MH crisis occurs? What other measures should be taken if MH becomes evident?**

   Most commonly, malignant hyperthermia is caused by a ryanodine receptor abnormality that is transmitted in an autosomal dominant fashion. Following exposure to a trigger, usually a volatile anesthetic gas, or depolarizing paralytic, such as succinylcholine, membrane instability in skeletal muscle results. This leads to a rapid release of calcium with muscular hyper-contraction with rapid consumption of available oxygen

leading to a catabolic state with release of CO_2, and lactic acid. As this cascade progresses, muscle fibers are broken down leading to uncontrolled elevation of central body temperature and significant abnormalities of serum electrolytes, most prominently hyperkalemia. Early signs of MH include tachycardia and tachypnea with an increase of expired end-tidal CO_2. Muscular rigidity and elevated body temperatures are generally seen later on in the course of disease progression.

Dantrolene is the medication of choice if an MH crisis occurs. This medication acts at the ryanodine receptor and acts to stabilize the sarcoplasmic reticulum membrane, thereby limiting further calcium ion egress. Additional measures that should be undertaken if MH becomes evident are institution of cooling measures, correction of electrolyte abnormalities, immediate cessation of volatile gases, or other triggers. Transfer to a facility that has ICU capabilities for continued monitoring of the patient should also be considered.

4. **What are the four levels of sedation? What variables are used to identify the depth of sedation of a patient?**

Four levels of sedation: minimal sedation/anxiolysis, moderate/conscious, deep, and a state of general anesthesia. The four variables used to define sedation are (1) a patient's responsiveness, (2) airway function, (3) spontaneous ventilation, and (4) cardiovascular status. See also Fig. 1.1.

5. **How are valium and versed different? Which is more potent? Which has a more rapid onset of duration? What other patient factors should be considered when administering these agents?**

Versed is the more potent of the two benzodiazepines and has a more rapid onset of action. Additionally, valium is metabolized and some of its metabolites have a similar mechanistic effect, thereby prolonging the overall effect of valium as compared to versed, which does not have active metabolites. Age and hepatic status must also be considered when these medications are given as reduction in the dosage is usually advised in patients with advanced age and hepatic dysfunction.

6. **What is the primary cause of morbidity in procedures performed under sedation? How do you manage a patient who has entered a deeper state of sedation than anticipated, i.e., how do you treat a patient who has lost control of his/her airway or becomes cardiovascularly unstable?**

The primary cause of morbidity in procedures under sedation is drug-induced respiratory depression, often with loss of airway support, usually with loss of muscular tone and protective airway reflexes. If this is unrecognized, this can lead to significant complications including anoxic/hypoxic brain injury.

A patient who has lost control of his/her airway should receive immediate interventions that aim to reestablish airway support. This includes prompting the patient to breathe or stimulating the patient to do so, supplemental oxygen, chin thrust, bag-mask ventilation, and endotracheal intubation. Cardiovascular support may be administered via *intravenous administration of fluids and or pharmacologic means of blood pressure support*, i.e., *pressors, if necessary. Consideration of rescue medications in the form of pharmacologic antagonists should also be considered under these circumstances.*

7. **A patient presents for reconstruction of a 4 cm cheek defect and receives a combination of versed and fentanyl in addition to local anesthetic. She is no longer responding verbally and only reflexively withdraws from painful stimuli. What does this indicate with regard to the level of sedation and what should be done next for her?**

Reflexive withdrawal from noxious stimuli indicates at least a level of deep sedation, if not a state of general anesthesia, and requires that the patient be "rescued" from