

Anesthesia Complications: Management and Prevention

7

Elie M. Ferneini and Jeffrey Bennett

Abstract

The volume of cosmetic surgery continues to grow rapidly, so does the demand for skilled anesthesia care. Although anesthesia for facial cosmetic surgical procedures remains remarkably safe, no anesthesia should be considered minor. Complications can occur at any time. Proper preoperative evaluation, patient selection, incorporation of a safety checklist, and close collaboration with the surgeon will decrease adverse events leading to increased patient safety and improved outcomes.

7.1 Introduction

The volume of cosmetic surgery continues to grow rapidly. More than ten million cosmetic procedures (surgical and nonsurgical) were performed in 2014 alone, a tenfold increase from 1997 (one million procedures) [1]. In fact, Americans spent more than 12 billion dollars on cosmetic procedures during each calendar year in 2013 and 2014 [1]. More than 80% of these pro-

cedures were performed as outpatient and 60% in an office facility. These procedures have very low rates of perioperative mortality and complications, not exceeding 0.002% and 0.7%, respectively [2–7]. However, facial cosmetic surgery patients may be at a higher anesthesia risk [8].

7.2 Perioperative and Anesthetic Management

Anesthetic management of patients undergoing facial cosmetic surgery presents a unique challenge to the maxillofacial surgeon and anesthesiologist. The essential anesthesia requirements for facial cosmetic surgery include a quiet operating room, clear and clean surgical field, non-stimulating emergence from anesthesia, a rapid return of consciousness and protective airway reflexes, prevention of postoperative nausea and vomiting (PONV), as well as fast-tracking patients for discharge.

E.M. Ferneini, M.D., D.M.D., M.H.S., F.A.C.S. (✉)
Beau Visage Med Spa, Greater Waterbury OMS,
Cheshire, CT, USA

Division of Oral and Maxillofacial Surgery,
University of Connecticut, Farmington, CT, USA
e-mail: eferneini@yahoo.com

J. Bennett, D.M.D.
Indianapolis, IN, USA
e-mail: jb2omfs@gmail.com

Maintenance of a clear and clean surgical environment is a critical part of facial cosmetic surgical procedures. This is due to the high vascularity of the head and neck area where even a small amount of bleeding can have a significant impact on intra-operative exposure and visualization. Moderate hypotension, local anesthesia with epinephrine infiltration, adequate hemostasis, and meticulous surgical technique allow for optimal outcomes.

Smooth emergence from anesthesia and prevention of PONV are essential to prevent post-op complications such as hematoma formation, postoperative bleeding, and wound dehiscence [9, 10]. This is critical in rhytidectomy and neck procedures [9].

Like any surgery, preoperative risk assessment is essential. For facial cosmetic surgical procedures, patient's health status and type of procedure performed should be taken into consideration [11]. Patients presenting for maxillofacial cosmetic surgery are classified as ASA I (healthy patient), ASA II (patient with mild, controlled systemic disease), or ASA III (patient with severe systemic disease, definite functional impairment). Patients with ASA III physical status have not been shown to have an increased incidence of perioperative complications. Additionally, many patients presenting for esthetic surgery are older. While older age can increase the risks associated with surgery and anesthesia, older age is not a contraindication to ambulatory surgery. An appropriate and detailed preoperative evaluation is essential to decrease adverse events. Having a clear and detailed anesthetic plan is an important part of working up the cosmetic surgery patient. In fact, a wide variety of facial cosmetic surgical procedures can be safely and efficiently performed under monitored anesthesia care (MAC) sedation or general anesthesia [4, 12]. The choice is often dictated by the surgeon's comfort level and preference as well as the patient's desires.

According to the ASA Closed Claims Project database containing 8954 anesthesia malpractice claims, some of the most common major anesthesia-related complications included death (26%), nerve damage (22%), permanent brain damage (9%), airway injury (7%), and medication errors (7%) [13].

Adverse respiratory and cardiovascular incidents accounted for 17% and 13% of all claims associated with anesthesia-related sentinel events [13]. Among the respiratory incidents, difficult intubation, inadequate oxygenation or ventilation, pulmonary aspiration, and airway fires were the most common. In fact, inadequate oxygenation/ventilation has become a growing problem during MAC cases and administration of anesthesia in the non-OR environment [13]. Moreover, between 1997 and 2007, more than 40% of malpractice claims associated with MAC involved the patient's death or permanent brain damage; and as ambulatory esthetic surgery has increased in popularity, the incidence of airway fire claims has grown to be 17% of all anesthetic closed claims [14].

Cardiovascular incidents are less common. Most cardiovascular events leading to anesthesia claims between 1990 and 2007 were related to hemorrhage/blood replacement, fluid management/electrolyte abnormalities, and stroke [13]. Medication problems are relatively common and represented 7% of anesthesia claims between 1990 and 2007 [13]. These claims were equally distributed between adverse drug reactions and medication errors. Additionally, most medication errors were considered preventable [13].

Most anesthesia-related complications in the outpatient setting are minor. The most common include [15]:

- PONV 4.7%
- Shivering 2.2%
- Eye injury 0.056%
- Dental injury 0.02%
- Ulnar neuropathy 0.47%
- Sore throat 28%

7.2.1 MAC Sedation

Many facial cosmetic surgical procedures can be performed under MAC sedation. This eliminates the need for general anesthesia. MAC avoids invasive airway management, removes emergence phenomena, reduces the incidence of PONV, and allows quicker patient discharge [3].

However, oxygenation and ventilatory monitoring may be more challenging in the patient whose airway is open and unprotected. Additionally, high oxygen concentrations (>30% with or without nitrous oxide) and oxygen trapping must be avoided, especially when using electrocautery and lasers. Experience and comfort with MAC sedation is required to prevent adverse events.

MAC claims in facial cosmetic surgery are relatively common, accounting for more than 25% of all MAC claims between 1997 and 2007 [14]. Respiratory depression is the leading cause of inadequate oxygenation/ventilation and death or permanent brain damage in MAC malpractice claims [14]. This was the result of either absolute or relative overdose of sedative or opioid drugs. Nearly half of these claims were judged as preventable by [14]:

1. Better monitoring (including capnography)
2. Improved vigilance
3. Presence of audible monitoring alarms

Conducting safe and successful MAC requires the surgeon to appreciate pharmacokinetic and pharmacodynamic interactions of the chosen intravenous sedative agents, as well as the role of pharmacologic antagonists for opioids and benzodiazepines [16]. A synergistic effect of the sedating agents should be always kept in mind to avoid rapid onset of respiratory depression and upper airway obstruction. Additionally, the doses of the administered medications should be appropriately reduced [17].

Benzodiazepines and opioids are an appropriate combination for sedation and analgesia. Benzodiazepines provide anxiolysis, sedation, and amnesia. Opioids provide analgesia. However, opioids have a proportionately greater degree of respiratory depressant effects compared with benzodiazepines. Despite the respiratory depressant effects of opioids and benzodiazepines, both drugs have specific antagonists [18–20]. Appropriate and planned anesthetic management should avoid the need for reversal agents. Sedation and analgesia should be titrated slowly. The peak onset of midazolam and fentanyl is 8 and 6 min, respectively, and is

slower in the older and/or medically compromised patient. Maximal anesthetic effect may require several minutes.

7.2.2 General Anesthesia

Compared to MAC sedation, general anesthesia with an advanced airway protects the patient's airway, assures adequate ventilation and oxygenation, and removes patient movement. The deeper anesthetic depth more likely will provide more profound amnesia; however, this is dependent on the anesthetic technique and selection of anesthetic agents.

Detailed preoperative airway assessment should be performed on every cosmetic surgery patient. During this examination, risk factors for difficult mask ventilation, airway management, and tracheal intubation must be assessed [21, 22]. A detailed medical and surgical history must be obtained. For example, previous cosmetic procedures, such as chin implant, may mask preexisting retrognathia, thus leading to unanticipated difficult direct laryngoscopy and intubation [23]. Although tracheal intubation is usually performed for most cosmetic facial surgical procedures, the authors believe that a laryngeal mask airway (LMA) can be used for most of these procedures [12]. In fact, the use of a LMA for most cosmetic maxillofacial surgical procedures compared to an open airway provides airway protection and may provide an improved quality of the surgical field. The use of a LMA compared to endotracheal intubation can provide comparable airway protection, improved hemodynamic stability, reduced use of anesthetic medications, a shortened operative time, a decreased likelihood of airway reflex stimulation, and smoother emergence from anesthesia [24–26].

7.3 Complications

Anesthesia-related morbidity ranges from minor complications that can affect our patient's experience with minimal long-term effects to complications with long-term effects and dire adverse

events. The most common complications are cardiovascular and respiratory in nature. Thorough preoperative assessment can help identify risk factors and stratify patients so that we can optimize care.

7.3.1 Cardiovascular Complications

The most common perioperative cardiac complications include myocardial infarction (MI), thromboembolism, arrhythmias, and cardiac arrest.

7.3.2 Myocardial Infarction

The risk of a myocardial infarction (MI) is low: most recent studies suggest up to 5% of patients undergoing elective noncardiac surgery have MI [27]. Most perioperative ischemic events are usually silent without any clinical signs and symptoms. However, the incidence is reported at 4.4% in the presence of a cardiac risk factor [28].

Perioperative MI is usually hard to predict and prevent. It usually occurs in the first 48 h postoperatively [29]. Most of the cause of a perioperative MI is an oxygen-demand mismatch. Surgery in general confers a physiologic stress to the patient that leads to increased oxygen demand. Patient risk stratification is important in assessing the likelihood of perioperative cardiac complications. The most commonly used risk index is the Revised Cardiac Risk Index (RCRI). This index identifies six independent predictors of major cardiac complications. The risk increases with the presence of each additional risk factor. These include [30]:

- High-risk surgery, e.g., intraperitoneal, intrathoracic
- History of ischemic heart disease: history of MI, history of positive exercise test, current chest pain considered due to MI, use of nitrate therapy, ECG with pathological Q waves
- History of heart failure: pulmonary edema, paroxysmal nocturnal dyspnea
- History of cerebrovascular disease: prior TIA or CVA

- Diabetes requiring insulin
- Chronic renal impairment: preoperative creatinine >2 mg/dL

Depending on each patient's risk factors, measures should be taken to improve cardiac outcomes. β -Blockers have been shown to decrease the incidence of perioperative MI; however there is a potential increase in the incidence of stroke [28, 31]. In fact, perioperative β -blockade started within 1 day or less before noncardiac surgery prevents nonfatal MI but increases risks of stroke, death, hypotension, and bradycardia [32]. Perioperative statins can be beneficial: statin treatment in statin-naive patients reduces atrial fibrillation, myocardial infarction, and duration of hospital stay in patients undergoing cardiac or noncardiac surgery [33]. Clonidine and aspirin could be beneficial in reducing the risk of major cardiovascular events. However, in a recent study, the administration of aspirin before surgery and throughout the early postsurgical period had no significant effect on the rate of a composite of death or nonfatal MI but increased the risk of major bleeding [34].

7.3.3 Thromboembolism

Venous thromboembolism (VTE) is a significant cause of morbidity and mortality in the perioperative period. VTE includes both deep vein thrombosis (DVT) and pulmonary embolism (PE). In patients undergoing plastic surgery, the overall incidence of VTE is 1.69% [35]. The executive committee of the American Society of Plastic Surgeons (ASPS)-approved Venous Thromboembolism Task Force Report identified the best practices for DVT/PE prevention and treatment [36]. Additionally, the ASPS has published articles offering recommendation for DVT prophylaxis based on the risk levels [37, 38]. The highest risk is associated with liposuction and abdominoplasty procedures. For maxillofacial cosmetic surgical procedures, facelift procedures carry a higher risk of VTE. In fact, rhytidectomy and its association with DVT/PE have been documented by Rigg, Reinisch, and Abboushi [39–41].

Each Risk Factor = 1 point Age 41-60 years Minor surgery planned Prior major surgery (<1 month) Varicose veins History of inflammatory bowel disease Swollen legs Obesity (BMI>25) Acute myocardial infarction Congestive heart failure (<1 month) Sepsis (<1 month) Serious lung disease (<1 month) Abnormal pulmonary function (e.g. COPD) Medical patient currently at bed rest Other risk factors	Each Risk Factor = 2 points Age 60-74 Arthroscopic surgery Malignancy (present or previous) Major surgery (>45minutes) Laparoscopic surgery (>45minutes) Patient confined to bed (>72 hours) Immobilizing plaster cast (<1 month) Central venous access	Each Risk Factor = 5 points Elective major lower extremity arthroplasty Hip, pelvis or leg fracture (<1 month) Stroke (<1 month) Multiple trauma (<1 month) Acute spinal cord injury (<1 month)
	Each Risk Factor = 3 points Age > 75 years History of DVT/PE Family history of thrombosis Positive Factor V Leiden Positive Prothrombin 20210A Elevated serum homocysteine Positive lupus anticoagulant Elevated anticardiolipin antibodies Heparin-induced thrombocytopenia Other congenital/acquired thrombophilia	For Women Each Risk Factor = 1 point Oral contraceptives or hormone replacement therapy Pregnancy or postpartum (<1 month) History of unexplained stillborn infant, recurrent spontaneous abortion (>3), premature birth with toxemia or growth-restricted infant

Fig. 7.1 Thrombosis risk assessment (Data from Murphy RX, Schmitz D, Rosolowski K. Evidence based practices for thromboembolism prevention: a report from the ASPS

Venous Thromboembolism Task Force. Arlington Heights (IL): American Society of Plastic Surgeons; 2011)

The maxillofacial surgeon must assess and stratify the risk of VTE for each patient. The Caprini risk assessment model is a helpful tool to stratify your patients (Fig. 7.1) [42]. Current recommendations on thromboprophylaxis in surgery patients are based on the calculated risk of VTE and consideration of the risk of bleeding associated with any intervention. These recommendations are summarized in Table 7.1 [43].

7.3.4 Arrhythmias

Arrhythmias are rare in the perioperative period. In fact, less than 1% of all surgery patients experience a bradyarrhythmia or ventricular arrhythmia that is severe enough to require treatment [44]. In the perioperative period, atrial fibrillation is the most common arrhythmia with an incidence of 0.37–20% in noncardiac surgery patients [45]. Preoperative risk factors for the development of atrial fibrillation include increasing age, male gender, preexisting heart disease, ASA III and/or IV, and preoperative electrolyte disturbances [45].

For most new-onset arrhythmias in the perioperative period, no medical management is required. They are usually self-limiting with more than 80% reverting to sinus rhythm before

Table 7.1 Incidence of VTE and recommendations for thromboprophylaxis based on Caprini risk assessment model (RAM) score

Caprini RAM score	Risk of VTE (%)	Recommended thromboprophylaxis
0	<0.5	Nil/ambulate early
1–2	1–5	Mechanical prophylaxis (IPC)
3–4	3.0	If not high risk for major bleeding: LMWH or LDUH or mechanical prophylaxis (IPC) If high risk for major bleeding: mechanical prophylaxis (IPC)
>5	6.0	LMWH or LDUH and mechanical prophylaxis (IPC)

Abbreviations: IPC intermittent pneumatic compression, LDUH low-dose unfractionated heparin, LMWH low molecular weight heparin

Data from Gould MK. Prevention of VTE in nonorthopedic surgical patients: antithrombotic therapy and prevention of thrombosis. Chest 2012; 141:e2275–775

discharge [45]. Management includes recognition and initiating rate and rhythm control.

7.3.5 Cardiac Arrest

The risk of anesthesia-related cardiac arrest is 1.86:10,000 [46]. General anesthesia is a risk factor for cardiac arrest. In fact, more than

90% of anesthesia-related cardiac arrests are related to airway management or medication administration.

7.3.6 Respiratory Complications

Respiratory complications are major predictors of morbidity in the perioperative period. Its incidence is at 6.8% with serious complications occurring at 2.6% [47]. Adverse respiratory events were the primary contributing factors resulting in death in out-of-operating room locations, of which 50% were associated with MAC sedation [48]. The complications discussed in this section include hypoxia/hypopnea/apnea, aspiration, bronchospasm, atelectasis, and airway crisis.

7.3.7 Hypoxia/Hypopnea/Apnea

Anesthetic agents depress ventilatory drive and contribute to loss of upper airway patency. The latter results from the loss of pharyngeal dilator muscular support affecting the tensor palatini, genioglossus, and hyoid muscles, which dilate the nasopharynx, oropharynx, and laryngopharynx, respectively, and maintain airway patency. As patients age, these muscles become less efficacious and the patients are more susceptible to becoming obstructed. In order to detect impairment of airway exchange either from respiratory depression or obstruction, the patient must be monitored using both pulse oximetry (providing oxygen saturation) and capnography and auscultation (providing respiratory rate and end-tidal carbon dioxide). While oxygen saturation is obviously important, it provides limited information when registering 100% saturation in the oxygenated patient because it does not provide the actual arterial oxygen content. The limitation is illustrated by the following. The arterial oxygen content in a patient receiving 40% oxygen may approximate 180 mmHg with an oxygen saturation of 100%; the same patient whose airway

and/or ventilation is compromised may have an oxygen saturation of 100 mmHg with an oxygen saturation of 100%. The incorporation of ventilatory monitoring into routine anesthesia care can detect respiratory depression that may be the primary factor contributing to the lower arterial oxygen content. The first ASA closed claims report suggested the lack of ventilatory monitoring as the most significant component contributing to morbidity and mortality.

Capnography/capnometry is the standard of care for monitoring ventilation. It provides a waveform that shows the respiratory pattern, the respiratory rate, and a measurement of the end-tidal carbon dioxide. In the patient with an advanced airway (e.g., intubation), the practitioner can set ventilatory parameters based on end-tidal carbon dioxide. The absolute end-tidal carbon dioxide has limited value in the patient with an open airway. In this situation, the generated waveform and corresponding respiratory rate can monitor ventilation and demonstrate potentially ominous changes before they may be manifested by changes in oxygen saturation. There are several ways in which air sampling can be achieved. A nasal cannula in which oxygen is delivered via one naris and carbon dioxide is sampled via the other naris is noninterfering with most procedures. These authors also recommend the use of an electronic wireless stethoscope that magnifies sounds and is placed in the pre-tracheal region in the open-airway patient. Auscultation can identify respiratory changes not detected by capnography. Furthermore, the sampling line can become displaced or obstructed in the open-airway patient. The viewable capnographic screen and the stethoscope wirelessly connected to a speaker allow the entire team to be observant of real-time ventilatory changes.

7.3.8 Aspiration

Aspiration of gastric contents into the airway is the most common cause of airway-related death during anesthesia [49]. It occurs in 1:4000 patients undergoing general anesthesia. The

highest risk is at intubation as well as extubation [49, 50]. Prevention is an important step in decreasing the incidence of aspiration. It is aimed at identifying patients at risk. These factors are divided into four categories: patient factors,

surgical factors, anesthetic factors, and device factors. Box 7.1 summarizes these risk factors [51]. The surgeon must follow specific strategies to reduce the risk of aspiration. Box 7.2 discusses some of these strategies.

Box 7.1 Risk Factors for Aspiration (Adapted from Asai [6] with Permission from the British Journal of Anaesthesia)

Patient factors

- (a) Full stomach
 - Emergency surgery
 - Inadequate fasting time
 - Gastrointestinal obstruction
- (b) Delayed gastric emptying
 - Systemic diseases, including diabetes mellitus and chronic kidney disease
 - Recent trauma
 - Opioids
 - Raised intracranial pressure
 - Previous gastrointestinal surgery
 - Pregnancy (including active labor)
- (c) Incompetent lower esophageal sphincter
 - Hiatus hernia
 - Recurrent regurgitation

- Dyspepsia
- Previous upper gastrointestinal surgery
- Pregnancy
- (d) Esophageal diseases
 - Previous gastrointestinal surgery
 - Morbid obesity

Surgical factors

- Upper gastrointestinal surgery
- Lithotomy or head down position
- Laparoscopy
- Cholecystectomy

Anesthetic factors

- Light anesthesia
- Supraglottic airways
- Positive pressure ventilation
- Length of surgery >2 h
- Difficult airway

Device factors

- First-generation supraglottic airway devices

Box 7.2 A Summary of the Available Strategies for Reducing Aspiration Risk

Reducing gastric volume	Preoperative fasting
	Nasogastric aspiration
	Prokinetic premedication
Avoidance of general anesthetic	Regional anesthesia
Reducing pH of gastric contents	Antacids
	H ₂ histamine antagonists
	Proton pump inhibitors
Airway protection	Tracheal intubation
	Second-generation supraglottic airway devices
Prevent regurgitation	Cricoid pressure
	Rapid sequence induction
Extubation	Awake after return of airway reflexes
	Position (lateral, head down, or upright)

Guidelines to reduce the risk of aspiration include:

- Experienced anesthesia assistance and supervision available at all times.
- Applying appropriate cricoid pressure with all inductions using neuromuscular blocking agents.
- Consider intubation in patients with:
 - Delayed gastric emptying (opioids, diabetes mellitus, renal failure)
 - Increased intra-abdominal pressure (obesity)
- Extubating high-risk cases awake and on their side and extubating all others on their side.
- A LMA does not protect against aspiration and endotracheal intubation should be considered in at-risk patients.

7.3.9 Bronchospasm

Bronchospasm occurs in 0.2% of patients undergoing general anesthesia. It is a lower airway obstruction due to contraction or spasm of bronchial smooth muscle. It can lead to hypoxia, hypotension, or even death [52, 53]. Patients with preexisting airway disease, a recent or active upper respiratory tract infection, and smoking history are at increased risk of developing a bronchospasm. Manifestations of bronchospasm include rapidly increasing peak inspiratory pressure (with plateau pressure unchanged), wheezing, slowly increasing wave on the capnograph, and decreasing exhaled tidal volumes. The patient will actually exhibit dyspnea and wheezing attributed to chest obstruction. The key triggers include:

- Airway instrumentation
- Airway irritation
- Early surgical stimulation without adequate depth of anesthesia
- Some medications (e.g., β -blockers, neostigmine, morphine)
- Anaphylaxis

Prevention of bronchospasm relies on optimizing any underlying airway disease, encouraging smoking cessation, delaying elective cosmetic surgery if recent or acute upper respiratory infec-

Box 7.3 Bronchospasm: Acute Treatment Preoperative

- Supplemental oxygen
- Inhaled β_2 -agonists (e.g., albuterol)
- Intravenous steroids

Intraoperative

1. Deepen anesthetic—increase volatile anesthetic concentration
 - (a) All volatile anesthetic agents are bronchodilators
 - (b) Ketamine
 - (c) Propofol (protection against bronchoconstriction)
2. Consider alternative causes of high airway pressures, e.g., kinked tube, endobronchial intubation, etc.
3. Inhaled β_2 -agonists—delivered to the inspiratory limb of the circuit through a metered dose inhaler or nebulized
4. Epinephrine—subcutaneous (1:1000) versus intravenous (1:10,000). If the severity of bronchospasm prohibits delivery of inhaled beta-agonists, consider infusions of IV agonists such as terbutaline or epinephrine
5. Consider administering intravenous steroids

tion, and avoiding unnecessary airway manipulation. Box 7.3 discusses appropriate management of an acute bronchospasm.

7.3.10 Laryngospasm

Laryngospasm is a serious complication which can lead to airway obstruction and even death. It is a reflex spasm of the striated muscles of the larynx, resulting in partial or complete closure of the glottis and an inability to ventilate the lungs. Any patient under IV sedation or general anesthesia whose airway is not protected by an ET tube may experience laryngospasm. Laryngospasm can be also caused

by airway manipulation (extubation, insertion of a LMA, suctioning), secretions in the pharynx, surgical stimulus due to light sedation, vomiting, and patient movement. Signs include inspiratory stridor, complete airway obstruction, increased inspiratory efforts, paradoxical chest and abdominal movements, desaturation, and bradycardia.

Airway patency may be restored with jaw thrust and positive pressure mask ventilation with 100% oxygen. Deepening anesthesia with a rapidly acting intravenous agent, such as propofol, is frequently helpful. If these measures fail, a muscle relaxant, typically succinylcholine, is administered. Endotracheal intubation may not be necessary because succinylcholine is short acting. Mask ventilation until the return of spontaneous breathing is often sufficient. The patient may require intubation if he or she has difficult mask ventilation or if the laryngospasm doesn't resolve. The complications of laryngospasm include aspiration and negative inspiratory pressure pulmonary edema, and the patient may require ventilatory support in an intensive care setting [54].

7.3.11 Airway Crisis: "Cannot Ventilate, Cannot Intubate"

Patients with difficult airway usually require multiple attempts at intubation during airway crisis. These attempts have been associated with death or permanent brain damage [13]. The office should be capable of performing direct laryngoscopy with both a Macintosh and Miller blade and videolaryngoscopy. Videolaryngoscopy provides a more rapid view of the glottis but is an indirect technique that requires a different hand-eye coordination compared to traditional laryngoscopy. Videolaryngoscopy has become the standard of care in managing the difficult airway. Attempts at laryngoscopy and intubation should be limited. A good rule of thumb is to limit the intubation attempts to three before placement of a LMA. If ventilation with a LMA is not successful, a surgical airway should be secured.

The importance of preoperative airway assessment to identify patients with a difficult airway cannot be overemphasized. A patient with a suspected difficult airway should not

have a procedure in an outpatient facility, unless the cosmetic procedure is performed under minimal sedation.

7.3.12 Postoperative Nausea and Vomiting

The incidence of vomiting is about 30%, and the incidence of nausea is about 50%. In general, PONV is experienced by a high number of our cosmetic surgery patients: 20–30%. It can be as high as 70–80% in high-risk patients [55–57]. Unresolved PONV can result in prolonged postanesthesia care unit (PACU) stay and increased patient dissatisfaction. Additionally, it increases adverse events of maxillofacial cosmetic surgical procedures. The goal of PONV prophylaxis is to decrease the incidence of PONV leading to decreasing patient-related distress and healthcare costs [58–60].

Preoperative assessment of high-risk patients can lead to prophylactic management which leads to decreased incidence. Box 7.4 summarizes risk factors for PONV [55, 61]. Four factors (female sex, nonsmoking status, history of PONV or motion sickness, and opioid use) have been well documented. In fact, the incidence of PONV is estimated at 10%, 20%, 40%, 60%, and 80% depending on the presence of none, 1, 2, 3, or 4 risk factors, respectively [62]. Guidelines have been developed that identify patients at risk for PONV, approaches for

Box 7.4 PONV Risk Factors

- 1 Patient factors
 - Female gender postpuberty
 - Nonsmoking status
 - History of PONV or motion sickness
- 2 Anesthetic factors
 - Use of volatile anesthetics
 - Use of nitrous oxide
 - Use of intraoperative/postoperative opioids
 - Use of large-dose neostigmine
- 3 Surgical factors
 - Duration of surgery >30 min
 - Type of surgery (plastic and maxillofacial)

reducing baseline risks for PONV, effective antiemetic therapy for PONV prophylaxis, and strategies for treatment of PONV. Tables 7.2, 7.3, and 7.4 provide an algorithm for the

management of individuals at increased risk for PONV as well as steps to ensure PONV prevention and treatment are implemented [63, 64]. Some elements to consider in patient management are as follows: decadron is both an anti-inflammatory and antiemetic agent at 4 mg; midazolam 2 mg provides anxiolysis, amnesia, and sedation and a dose as little as 2 mg given a minimum of 30 min prior to the end of the case reduces PONV; several of the antiemetics act on the dopamine receptor and have the potential to produce extrapyramidal effects which if it occurs is treated with administration of diphenhydramine; several of the antiemetics (even the serotonin antagonists, more likely with higher

Table 7.2 Strategies to reduce risk of PONV

Table 7.3 Risk-adapted PONV prevention algorithm (with no prevention in low-risk patients) [63]

	Estimated risk for PONV, for example, as determined by a risk score		
	Low	Medium	High
Interventions for prophylaxis	No prevention (“wait and see”)	Drug A + Drug B or TIVA	Drug A + Drug B + TIVA On a case-by-case decision: further interventions
Interventions for treatment	1. Drug B 2. Drug C (in case of ineffectiveness of treatment in stage 1) (i.e., Drug B)	1. Drug C 2. Drug D (in case of ineffectiveness of treatment in stage 1) (i.e., Drug C)	1. Drug C 2. Drug D (in case of ineffectiveness of treatment in stage 1) (i.e., Drug C)

Example interventions: Drug A, dexamethasone 4 mg in adults/0.15 mg/kg of body weight in children; Drug B, ondansetron 4 mg in adults/0.1 mg/kg of body weight in children; Drug C, droperidol 1 mg in adults/10–15 µg/kg of body weight in children; Drug D, dimenhydrinate 1 mg/kg of body weight in adults/0.5–1.0 mg/kg of body weight in children. Given drug examples are used to illustrate how the algorithm may be actually implemented but may not represent the most favorable approach. The latter may be context sensitive (children, adults, or other issues). In the event of treatment failure, a timely assessment and alternative antiemetics should be used. A multimodal treatment approach may be appropriate to increase the likelihood of success

TIVA total intravenous anesthesia, that is, propofol induction and maintenance, no nitrous oxide

Table 7.4 PONV prevention algorithm in all patients including low-risk patients plus additional interventions for high-risk patients [63]

	Estimated risk for PONV, for example, as determined by a risk score		
	Low	Medium	High
Interventions for prophylaxis	Drug A + (Drug B or TIVA)	Drug A + (Drug B or TIVA)	Drug A + Drug B + TIVA On a case-by-case decision: further interventions
Interventions for treatment	1. Drug C 2. Drug D (in case of ineffectiveness of treatment in stage 1) (i.e., Drug C)	1. Drug C 2. Drug D (in case of ineffectiveness of treatment in stage 1) (i.e., Drug C)	1. Drug C 2. Drug D (in case of ineffectiveness of treatment in stage 1) (i.e., Drug C)

Example interventions: Drug A, dexamethasone 4 mg in adults/0.15 mg/kg of body weight in children; Drug B, ondansetron 4 mg in adults/0.1 mg/kg of body weight in children; Drug C, droperidol 1 mg in adults/10–15 µg/kg of body weight in children; Drug D, dimenhydrinate 1 mg/kg of body weight in adults/0.5–1.0 mg/kg of body weight in children. Given drug examples are used to illustrate how the algorithm may be actually implemented but may not represent the most favorable approach. The latter may be context sensitive (children, adults, or other issues). In the event of treatment failure, a timely assessment and alternative antiemetics should be used. A multimodal treatment approach may be appropriate to increase the likelihood of success

TIVA total intravenous anesthesia, that is, propofol induction and maintenance, no nitrous oxide

doses used with chemotherapy and/or in patients with cardiovascular disease) can cause QT prolongation [65, 66].

7.3.13 Malignant Hyperthermia

Malignant hyperthermia (MH) is a true anesthesia emergency and a potentially fatal condition which results in tachycardia, muscle rigidity, hypercapnia, and hyperthermia. It is an autosomal dominant, pharmacogenetic disorder of the skeletal muscle. When exposed to a trigger agent, patients susceptible to MH have disordered regulation of calcium within the skeletal muscle leading to a hypermetabolic state [67, 68]. Major triggers include all volatile anesthetic agents and depolarizing muscle relaxants (succinylcholine).

The incidence of MH is in the order of 1:10,000 to 1:50,000; however the prevalence of MH is up to 1:3000 patients [67]. The annual number of suspected MH cases per year in the United States is around 700. Larach et al. reported that in 291 MH episodes recorded in the North American Malignant Hyperthermia Registry database between 1987 and 2006, there were eight cardiac arrests and four deaths [69].

MH may occur either in the operating room (OR) or in the early postoperative period. The earliest sign is an increase in end-tidal carbon dioxide. A fulminant reaction then occurs with very high end-tidal carbon dioxide (>100 mmHg), a low pH with a metabolic component, tachycardia and dysrhythmias, rigidity, rapidly increasing temperature, hyperkalemia, myoglobinuria, muscle edema, increased cellular permeability, disseminated intravascular coagulopathy (DIC), and even cardiac and renal failure.

Treatment consists of early recognition, removal of the trigger agent (including the necessity for high flow oxygen from a machine not exposed to a potent inhalational agent), and administration of dantrolene. Dantrolene is available as Dantrium (Revonto) or Ryanodex. Dantrium comes as a vial containing 20 mg dantrolene powder that is reconstituted with 60 mL sterile water. The vial is shaken until the solution is clear. Ryanodex comes as a vial containing

250 mg dantrolene. The powder is reconstituted with 5 mL sterile water and is shaken creating an orange-colored opaque suspension. Preparation time comparing Dantrium to Ryanodex is 860 s compared to 51 s, respectively. The initial dose is 2.5 mg/kg for either agent. The Malignant Hyperthermia Association of the United States (MHAUS) recommends that any office in which an inhalational agent (excluding nitrous oxide) or succinylcholine may be administered must have dantrolene. A patient with a history of MH can be safely anesthetized if triggering agents are avoided. The caffeine halothane contracture test (CHCT) is the standard for establishing the diagnosis of MH. The test is performed on freshly biopsied muscle tissue [70].

The issue with managing a patient with MH pertains to the potential for emergent airway intubation when succinylcholine is absolutely contraindicated. Rocuronium 1.2 mg/kg administered intravenously has an onset of 60–90 s providing satisfactory intubating conditions. However, rocuronium provides a neuromuscular blockade of intermediate duration. The FDA approved use of sugammadex in December 2015 to reverse the neuromuscular blockade induced by rocuronium. Sugammadex 16 mg/kg can be administered 3 min after the administration of an intubating dose of rocuronium providing neuromuscular blockade reversal in approximately 4.4 min from the time of rocuronium administration.

7.3.14 Sore Throat

Sore throat is a common postoperative complaint and affects up to 12.1% of patients at 24 h after surgery [71, 72]. It is directly related to the degree of airway instrumentation. The use of a LMA reduces the incidence to between 17.5% and 34% compared to 50% with endotracheal tube [71, 72]. The incidence of sore throat increases with the diameter of the endotracheal tube, as a result of increased pressure at the tube-mucosal interface [73]. Other factors which increase the incidence of sore throat include high-volume pressure cuffs, uncuffed endotracheal tubes, and lidocaine gel use for lubrication. For most maxillofacial cosmetic surgical procedures, using a LMA

decreases the incidence of sore throat. Some factors that increase this incidence include:

- Larger size LMA
- Multiple attempts at insertion
- Longer surgery time

For most cosmetic surgery patients, a sore throat is a self-limiting complication that does not require specific treatment and responds well to simple analgesia.

7.3.15 Operating Room Fire

Fires in the operating room are unanticipated, devastating events that can lead to significant morbidity, even mortality, for the patient and medical personnel. As maxillofacial surgeons, the topic of fire safety demands attention and preparedness. Approximately 78% of all fires occur during facial, neck, and tonsil surgery [74]. In fact, operating room fires account for nearly a fifth of MAC-related closed claims [75]. The most common ignition source in operating room fires is the electrosurgical unit, which is used in most facial cosmetic surgical procedures. Additionally, supplemental oxygen was also present [76, 77]. Fires can occur in any setting, provided these three elements are in close proximity under the right conditions: (1) heat or an ignition source, (2) fuel, and (3) an oxidizer [78].

In maxillofacial cosmetic surgery, lasers and electrocautery are major causes of fires. Use of a bur on metal (e.g., sectioning a crown to facilitate extraction of a tooth) may also promote a spark which may cause a fire. Oxidizers are always present and can increase the chance of a fire. Oxygen is a primary oxidizer and is typically present in every MAC or general anesthetic. Additionally, nitrous oxide, when heated, decomposes into its constituent elements, thus increasing the ambient oxygen. In fact, in most MAC cases of a fire, supplemental oxygen was used. Ninety-five percent of cases involved the head or neck, while alcohol-containing prep solutions and/or drapes were the most common

fuel sources [75]. Special attention must be directed to patient draping to avoid oxygen from being trapped within the surgical field. The flow rate of supplemental oxygen, such as a nasal cannula, should be set to the lowest possible setting to reduce unnecessary excess oxygen while still maintaining safe blood oxygenation saturation levels [79, 80]. Ideally, if electrocautery or laser is being used, oxygen administration should be replaced with medical grade air. Consideration to using advanced airways (e.g., intubation) removes the potential for an oxygen-enriched surgical field. If an advanced airway, either a specific laser tube or protective measures must be employed to avoid igniting the airway.

The best method of fighting a fire is to avoid one in the first place. Great emphasis should be placed on prevention. Education and training in fire risk reduction strategies for nurses, surgical technicians, anesthetists, and surgeons is essential to promote and maintain a fire-safe environment (Table 7.5).

If a fire occurs, it is the responsibility of all team members to protect the patient. Each individual plays a vital role in fire control. Often, the RACE method is used to guide actions when a fire occurs.

Table 7.5 Training programs should include details on the role-specific actions that are to be taken by the operating room staff to prevent fires

Examples include
• Keep the electric cautery tip in a holster when it is not being used
• Turn off all high-intensity light sources when not in use
• Using cuffed endotracheal tubes and appropriate use of oxygen gas
• Beware of patient draping so as to not create occlusive “tent” to trap in oxidizing agents
• Attempt to use water-soluble preparation solutions and ointments
• Consider flame-resistant surgical drapes
• Know the location of the fire extinguishers, alarms, and gas valves
• Obtain proficiency with fire extinguishers and equipment
• Know the location of all fire alarm and fire exits [80]

The fire-protection mnemonic stands for [80]:

- **R**escue: In the event of a fire, rescue means moving patients out of harm's way. All airway gases should be stopped and the safety of the patient and team members should be ensured.
- **A**larm (or alert): The person who notices the fire should call out and alert others to the situation. The surgical and anesthesia teams should work together. The surgeon must determine the point at which it is safe to stop the cosmetic procedure, while the rest of the team will decide how and where to move the patient.
- **C**onfine (or contain): After moving the patient away from the immediate fire danger, team members should work on containing the fire in order to prevent smoke and fire from entering other surgical rooms.
- **E**xtinguish: Extinguishing the fire is an important step. Three types of portable extinguishers are available: ABC, carbon dioxide, and water mist [80].

7.3.16 Local Anesthetic Toxicity

Local anesthesia is a major adjunct to maxillofacial cosmetic surgery. Local anesthesia can be used by itself or in tumescent solution. In fact, tumescent solution is often used in many procedures such as facelift, neck lift, and brow lift.

Lidocaine with epinephrine is usually added to the tumescent solution. The addition of epinephrine is important for hemostasis, prolonged anesthesia, and lowering the peak lidocaine levels.

7.3.17 Lidocaine Safe Dosing

Potential lidocaine toxicity is an important consideration, especially when using tumescent anesthesia. Traditionally, 7 mg/kg of lidocaine (with epinephrine) is accepted as the maximum dose for dermal infusion [81]. However, when used for tumescence, the ceiling is dramatically increased to 35 mg/kg (others report as high as 55 mg/kg). With this dosage, plasma concentrations of lidocaine remain below toxic levels (<5 mg/L) [82, 83].

7.3.18 Lidocaine Toxicity

Potential lidocaine toxicity can occur with tumescent anesthesia. Maxillofacial surgeons must recognize the signs and symptoms. Some of these symptoms can occur at 6 µg/mL but usually manifest at concentrations exceeding 10 µg/mL. Some of the signs include drowsiness, disorientation, cardiovascular instability, and seizures. Table 7.6 reports the CNS and cardiovascular effects of lidocaine toxicity [84].

Table 7.6 Effects of lidocaine on the CNS and cardiovascular system (CVS) [84]

Concentration (mg/mL)	CNS effect ^a	CVS effect ^a
<5	Anticonvulsant activity Mild sedation Analgesia Circumoral paresthesia	Antiarrhythmic activity Mild increases in mean blood pressure with similar increases in cardiac output or peripheral vascular resistance
5–10	Light-headedness, slurred speech, drowsiness, restlessness, euphoria	Cardiovascular instability
10–15	Disorientation, uncontrollable tremors, respiratory depression, tonic-clonic seizures	
15–20	Coma Respiratory arrest	
>20		Profound myocardial depression, vasodilatation, cardiovascular collapse

^aCNS and CVS effects are listed in approximate order of occurrence with increasing blood concentration

Adapted from Yagiela JA. Local anesthetics. *Anesth Prog* 1991; 38:128–41; and Butterwick KJ. Goldman MP Sripachya-Anunt S. Lidocaine levels during the first 2 hours of Infiltration of dilute anesthetic solution for tumescent liposuction rapid versus slow delivery. *Dermatol Surg* 1999; 25(9):681–5

Table 7.7 Checklist for treatment of local anesthetic systemic toxicity [85]

1. Get help
2. Initial focus
(a) Airway management
(b) Seizure suppression: benzodiazepines are preferred
(c) Alert the nearest facility having cardiopulmonary bypass capability
3. Management of cardiac arrhythmias
(a) Basic and advanced cardiac life support
(b) Avoid vasopressin, calcium channel blockers, beta-blockers, or local anesthetic
(c) Reduce individual epinephrine dose to <1 µg/kg
4. Lipid emulsion (20%) therapy
(a) Bolus 1.5 mL/kg (lean body mass)
(b) Continuous infusion 0.25 mL/kg/min
(c) Repeat bolus once or twice for persistent cardiovascular collapse
(d) Double the infusion rate to 0.5 mL/kg/min if blood pressure remains low
(e) Continuous infusion for at least 10 min after attaining circulatory stability
(f) Recommended upper limit: approximately 10 mL/kg lipid emulsion over the first 30 min

7.3.19 Intravenous Lipid Emulsion Therapy for the Management of Local Anesthetic Toxicity

The administration of intravenous lipid emulsion for the management of local anesthetic systemic toxicity has evolved over the past several years. The American Society of Regional Anesthesia and Pain Management has published recommendations, although no prospective randomized trial has been performed (Table 7.7) [85].

7.4 Cosmetic Procedure-Related Anesthesia Safety Considerations

7.4.1 Rhytidectomy

Rhytidectomy can be performed using endotracheal intubation, LMA, or MAC sedation. If MAC sedation is performed, tumescent local anesthesia is often used. During a facelift procedure, monitoring the facial nerve twitches during local anesthetic

injection in the facial nerve planes is recommended. This reduces the possibility of direct trauma to and/or paralysis of the facial nerve.

If the surgeon prefers tracheal intubation, succinylcholine can be administered. However, in conjunction with the inhalational anesthetic agents, this may trigger MH in susceptible patients. A combination of intravenous remifentanyl 2 mg/kg, propofol 2 mg/kg, and lidocaine 1.5 mg/kg and only half of an intubating dose of rocuronium (0.3 mg/kg) will achieve intubating conditions similar to those from administration of intravenous succinylcholine 1.5 mg/kg [86]. Postoperative hematoma is an undesired adverse event. Aggressive control of postoperative hypertension and PONV is essential in preventing this complication. Additionally, limiting intravenous fluids and bladder distension will also help in reducing postoperative patient discomfort, agitation, and hypertensive responses.

As mentioned above, DVT and PE constitute the greatest cause of morbidity and mortality after ambulatory surgery and are the most feared complications of prolonged facial cosmetic surgery, such as a rhytidectomy [87]. However, their incidence is extremely low: 0.35% for a DVT and 0.14% after a PE [40]. Prophylaxis plays a major role in prevention. Additionally, early ambulation and adequate postoperative pain control are important in reducing the risk of a VTE [40, 88].

7.4.2 Blepharoplasty

Blepharoplasty was among the top 5 cosmetic surgical procedures performed in 2014 [1]. Most cases can be performed under local anesthesia with or without sedation [12]. Some surgeons prefer general anesthesia for this procedure. If performed under MAC sedation, deeper planes of sedation may be required while injecting local anesthesia into the lower eyelids to prevent patient discomfort [89]. In some cases, general anesthesia might be indicated in order to prevent patient's movement, thus decreasing the risk of complications such as an intravascular injection and globe damage [89, 90].

7.4.3 Rhinoplasty

Because of the increased risk of laryngospasm or intraoperative coughing because of increased aspiration of blood and/or secretions, general anesthesia is preferred over MAC sedation.

A rare postoperative complication is Tapia syndrome. It is characterized by concomitant paralysis of the hypoglossal and the recurrent laryngeal branch of the vagus nerve. It is usually associated with endotracheal anesthesia for rhinoplasty, possibly resulting from excessive pressure of the throat pack in the hypopharynx [91].

Other complications include postoperative skull base defects and tension pneumocephalus. Tension pneumocephalus, a medical emergency, is usually a result of a poor surgical technique [92].

Conclusion

As the number of cosmetic surgical procedures performed in the United States continues to grow, so does the demand for skilled anesthesia care. Although anesthesia for facial cosmetic surgical procedures remains remarkably safe, no anesthesia should be considered minor. Complications can occur at any time. Proper preparation, preoperative evaluation, patient selection, incorporation of a safety checklist, and close collaboration and communication with the surgeon will decrease adverse events leading to increased patient safety and patient satisfaction.

References

1. The American Society for Aesthetic Plastic Surgery's (ASAPS) statistics. 2014. <http://www.surgery.org/sites/default/files/2014-Stats.pdf>. Accessed 19 Jan 2016.
2. Keyes GR, Singer R, Iverson RE, et al. Mortality in outpatient surgery. *Plast Reconstr Surg*. 2008;12:245–50.
3. Blake DR. Office-based anesthesia: dispelling common myths. *Aesthet Surg J*. 2008;28:564–70.
4. Bitar G, Mullis W, Jacobs W, et al. Safety and efficacy of office-based surgery with monitored anesthesia care/sedation in 4778 consecutive plastic surgery procedures. *Plast Reconstr Surg*. 2003;111:150–6.
5. Melloni C. Anesthesia and sedation outside the operating room: how to prevent risk and maintain good quality. *Curr Opin Anaesthesiol*. 2007;20:513–9.
6. Byrd HS, Barton FE, Orenstein HH, et al. Safety and efficacy in an accredited outpatient plastic surgery facility: a review of 5316 consecutive cases. *Plast Reconstr Surg*. 2003;112:636–41.
7. Hancox JG, Venkat AP, Coldiron B, et al. The safety of office-based surgery: review of recent literature from several disciplines. *Arch Dermatol*. 2004;140:1379–82.
8. Coldiron B, Shreve E, Balkrishnan R. Patient injuries from surgical procedures performed in medical offices: three years of Florida data. *Dermatol Surg*. 2004;30:1435–43.
9. Caloss R, Lard MD. Anesthesia for office-based facial cosmetic surgery. *Atlas Oral Maxillofac Surg Clin North Am*. 2004;12:163–77.
10. Taub PJ, Bashey S, Hausman LM. Anesthesia for cosmetic surgery. *Plast Reconstr Surg*. 2010;125:1e–7e.
11. Prendiville S, Weiser S. Management of anesthesia and facility in facelift surgery. *Facial Plast Surg Clin North Am*. 2009;17:531–8.
12. Hoefflin SM, Bornstein JB, Gordon M. General anesthesia in an office-based plastic surgical facility: a report on more than 23,000 consecutive office-based procedures under general anesthesia with no significant anesthetic complications. *Plast Reconstr Surg*. 2001;107:243–51.
13. Metzner J, Posner KL, Lam MS, et al. Closed claims' analysis. *Best Pract Res Clin Anaesthesiol*. 2011;25:263–76.
14. Bhananker SM, Posner KL, Cheney FW, et al. Injury and liability associated with monitored anesthesia care: a closed claims analysis. *Anesthesiology*. 2006;104:228–34.
15. Waddle J, Coleman J. General anesthesia in an office-based plastic surgical facility: a report on more than 23,000 consecutive office-based procedures under general anesthesia with no significant anesthetic complications by Steven M. Hoefflin, M.D., John B. Bornstein, M.D., Martin Gordon, M.D [discussion]. *Plast Reconstr Surg*. 2001;107:256–7.
16. 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.
17. Bailey PL, Pace NL, Ashburn MA, et al. Frequent hypoxemia and apnea after sedation with midazolam and fentanyl. *Anesthesiology*. 1990;73:826–30.
18. Blouin RT, Conard PF, Perreault S, et al. The effect of flumazenil on midazolam-induced depression of the ventilator response to hypoxia during isohypercarbia. *Anesthesiology*. 1993;78:635.
19. Gross JB, Blouin RT, Zandsberg S, et al. Effect of flumazenil on ventilator drive during sedation with midazolam and alfentanil. *Anesthesiology*. 1996;85:713.
20. Gross JB, Weller RS, Conard P. Flumazenil antagonism of midazolam-induced ventilatory depression. *Anesthesiology*. 1991;75:179.
21. Kheterpal S, Han R, Tremper KK, et al. Incidence and predictors of difficult and impossible mask ventilation. *Anesthesiology*. 2006;105:885–91.

22. Kheterpal S, Martin L, Shanks AM, et al. Prediction and outcomes of impossible mask ventilation: a review of 50,000 anesthetics. *Anesthesiology*. 2009;110:891–7.
23. Haddow GR, Brodsky JB, Brock-Utne JG, et al. Difficult laryngoscopy masked by previous cosmetic surgery. *Plast Reconstr Surg*. 1991;87:1143–4.
24. Mandel JE. Laryngeal mask airways in ear, nose, and throat procedures. *Anesthesiol Clin*. 2010;28:469–83.
25. Atef A, Fawaz A. Comparison of laryngeal mask with endotracheal tube for anesthesia in endoscopic sinus surgery. *Am J Rhinol*. 2008;22:653–7.
26. Kaplan A, Crosby GJ, Bhattacharyya N. Airway protection and the laryngeal mask airway in sinus and nasal surgery. *Laryngoscope*. 2004;114:652–5.
27. Devereaux PJ, Xavier D, Pogue J, et al. Characteristics and short-term prognosis of perioperative myocardial infarction in patients undergoing noncardiac surgery: a cohort study. *Ann Intern Med*. 2011;154:523–8.
28. Devereaux PJ, Yang H, Yusuf S, et al. Effects of extended-release metoprolol succinate in patients undergoing non-cardiac surgery (POISE trial): a randomised controlled trial. *Lancet*. 2008;371:1839–47.
29. Landesberg G, Beattie WS, Mosseri M, et al. Perioperative myocardial infarction. *Circulation*. 2009;119:2936–44.
30. Lee TH, Marcantonio ER, Mangione CM, et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. *Circulation*. 1999;100:1043–9.
31. Fleisher LA, Beckman JA, Brown KA, et al. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: a report of the American College of Cardiology/American Heart Association task force on practice guidelines. *Circulation*. 2007;116:e418–500.
32. Wijeyesundera DN, et al. Perioperative beta blockade in noncardiac surgery: a systematic review for the 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;130:2246–64.
33. Chopra V, Wesorick DH, Sussman JB, et al. Effect of perioperative statins on death, myocardial infarction, atrial fibrillation, and length of stay: a systematic review and meta-analysis. *Arch Surg*. 2012 Feb;147(2):181–9.
34. Devereaux PJ, Mrkobrada M, et al. Aspirin in patients undergoing noncardiac surgery. *N Engl J Med*. 2014;370:1494–503.
35. Pannucci CJ, Bailey SH, Dreszer G, et al. Validation of the Caprini risk assessment model in plastic and reconstructive surgery patients. *J Am Coll Surg*. 2011;212:105–12.
36. Murphy RX Jr, Alderman A, Gutowski K, et al. Evidence-based practices for thromboembolism prevention: summary of the ASPS Venous Thromboembolism Task Force report. EBM special topic-online. *Plast Reconstr Surg*. 2012;130:259.
37. Iverson RE, ASPS Task Force. Patient safety in office-based surgery facilities: I. Procedures in the office-based surgery setting. *Plast Reconstr Surg*. 2002;110(5):1337–42.
38. Iverson RE, Lynch DJ, ASPS Task Force. Patient safety in office-based surgery facilities: II. Patient selection. *Plast Reconstr Surg*. 2002;110(7):1785–92.
39. Rigg BM. Deep vein thrombosis after face-lift surgery [letter]. *Plast Reconstr Surg*. 1997;100:1363.
40. Reinisch JF, Bresnick SD, Walker JWT, et al. Deep venous thrombosis and pulmonary embolus after facelift: a study of incidence and prophylaxis. *Plast Reconstr Surg*. 2001;107:1570–5.
41. Abboushi N, Yezhelyev M, Symbas J, et al. Facelift complications and the risk of venous thromboembolism: a single center's experience. *Aesthet Surg J*. 2012;32(4):413–20.
42. Murphy RX, Schmitz D, Rosolowski K. Evidence based practices for thromboembolism prevention: a report from the ASPS Venous Thromboembolism Task Force. American Society of Plastic Surgeons: Arlington Heights, IL; 2011.
43. Gould MK. Prevention of VTE in nonorthopedic surgical patients: antithrombotic therapy and prevention of thrombosis. *Chest*. 2012;141:e227S–77S.
44. Amar D. Strategies for perioperative arrhythmias. *Best Pract Res Clin Anaesthesiol*. 2004;18:565–77.
45. Walsh SR, Tang T, Wijewardena C, et al. Postoperative arrhythmias in general surgical patients. *Ann R Coll Surg Engl*. 2007;89:91–5.
46. Braz LG. Perioperative cardiac arrest: a study of 53718 anaesthetics over 9 yr from a Brazilian teaching hospital. *Br J Anaesth*. 2006;96:569–75.
47. Smetana GW, Lawrence VA, Cornell JE. Preoperative pulmonary risk stratification for noncardiothoracic surgery: systematic review for the American College of Physicians. *Ann Intern Med*. 2006;144:581.
48. Metzner J, Posner KL, Domino KB. The risk and safety of anesthesia at remote locations: the US closed claims analysis. *Cur Opin Anaesthesiol*. 2009;22:502–8.
49. Cook TM, Woodall N, Frerk C. Major complications of airway management in the UK: results of the Fourth National Audit Project of the Royal College of Anaesthetists and the Difficult Airway Society. Part 1: anaesthesia. *Br J Anaesth*. 2011;106:617–31.
50. Kluger MT, Short TG. Aspiration during anaesthesia: a review of 133 cases from the Australian Anaesthetic Incident Monitoring Study (AIMS). *Anaesthesia*. 1999;54:19–26.
51. Robinson M, Davidson A. Aspiration under anaesthesia: risk assessment and decision-making. *Contin Educ Anesth Crit Care Pain*. 2014;14:4171–5.

52. Westhorpe RN. Crisis management during anaesthesia: bronchospasm. *Qual Saf Health Care*. 2005;14:1–6.
53. Dewachter P, Mouton-Faivre C, Emala CW, et al. Case scenario: bronchospasm during anesthetic induction. *Anesthesiology*. 2011;114:1200–10.
54. Visvanathan T, Kluger MT, Webb RK, et al. Crisis management during anesthesia: laryngospasm. *Qual Saf Health Care*. 2005;14:e3. <http://www.qshc.com/cgi/content/full/14/3/e3>. Accessed 9 Feb 2016.
55. Gan TJ, Meyer TA, Apfel CC, et al. Society for Ambulatory Anesthesia guidelines for the management of postoperative nausea and vomiting. *Anesth Analg*. 2007;105:1615–28.
56. Koivuranta M, Läärä E, Snäre L, Alahuhta S. A survey of postoperative nausea and vomiting. *Anaesthesia*. 1997;52:443–9.
57. Sinclair DR, Chung F, Mezei G. Can postoperative nausea and vomiting be predicted? *Anesthesiology*. 1999;91:109–18.
58. Gold BS, Kitz DS, Lecky JH, Neuhaus JM. Unanticipated admission to the hospital following ambulatory surgery. *JAMA*. 1989;262:3008–10.
59. Hill RP, Lubarsky DA, Phillips-Bute B, Fortney JT, Creed MR, Glass PS, Gan TJ. Cost-effectiveness of prophylactic antiemetic therapy with ondansetron, droperidol, or placebo. *Anesthesiology*. 2000;92:958–67.
60. Tramèr MR. Strategies for postoperative nausea and vomiting. *Best Pract Res Clin Anaesthesiol*. 2004;18:693–701.
61. Apfel CC, Korttila K, Abdalla M, et al. A factorial trial of six interventions for the prevention of postoperative nausea and vomiting. *N Engl J Med*. 2004;350:2441–51.
62. Apfel CC, Laara E, Koivuranta M, et al. A simplified risk score for predicting postoperative nausea and vomiting: conclusions from cross validations between two centers. *Anesthesiology*. 1999;91:693–700.
63. Gan TJ, et al. Consensus guidelines for the management of postoperative nausea and vomiting. *Anesth Analg*. 2014;118(1):85–113.
64. Apfel CC, et al. Evidence-based analysis of risk factors for postoperative nausea and vomiting. *Br J Anaesth*. 2012;109(5):742–53.
65. Tricco AC, Soobiah AC, Blondal E, et al. Comparative safety of serotonin (5 HT₃) receptor antagonists in patients undergoing surgery: a systematic review and network meta-analysis. *BMC Med*. 2015;13:142–55.
66. Grant MC, Kim J, Page AJ, et al. The effect of intravenous midazolam on postoperative nausea and vomiting: a meta-analysis. *Anesth Analg*. 2015;122(3):656–63.
67. Halsall PJ, Hopkins PM. Malignant hyperthermia. *Contin Educ Anaesth Crit Care Pain*. 2003;3:5–9.
68. Wappler F. Anesthesia for patients with a history of malignant hyperthermia. *Curr Opin Anaesthesiol*. 2010;23:417–22.
69. Larach MG, Brandom BW, Allen GC, Gronert GA, Lehman EB. Cardiac arrests and deaths associated with malignant hyperthermia in North America from 1987 to 2006: a report from the North American malignant hyperthermia registry of the malignant hyperthermia association of the United States. *Anesthesiology*. 2008;108(4):603–11.
70. Metterlein T, Hartung E, Schuster F, Roewer N, Anetseder M. Sevoflurane as a potential replacement for halothane in diagnostic testing for malignant hyperthermia susceptibility: results of a preliminary study. *Minerva Anesthesiol*. 2011;77(8):768–73.
71. Higgins PP, Chung F, Mezei G. Postoperative sore throat after ambulatory surgery. *Br J Anaesth*. 2002;88:582–4.
72. McHardy FE, Chung F. Postoperative sore throat: cause, prevention and treatment. *Anaesthesia*. 1999;54:444–53.
73. Stout DM, Bishop MJ, Dwersteg JF, et al. Correlation of endotracheal tube size with sore throat and hoarseness following general anesthesia. *Anesthesiology*. 1987;67:419–21.
74. Daane SP, Toth BA. Fire in the operating room: principles and prevention. *Plast Reconstr Surg*. 2005;115(5):73e–5e.
75. Rinder CS. Fire safety in the operating room. *Curr Opin Anaesthesiol*. 2008;21(6):790–5. Web: 12 Jan 2016.
76. Preventing surgical fires. Sentinel Event Alert 29 (24 June 2003). http://www.jointcommission.org/sentinel_event_alert_issue_29_preventing_surgical_fires/.
77. Hart SR, Yajnik A, Ashford J, Springer R, Harvey S. Operating room fire safety. *Ochsner J*. 2011;11(1):37–42.
78. Kaye A, Kolinsky D, Urman R. Management of a fire in the operating room. *J Anesth*. 2014;28(2):279–87. Available from: Academic Search Premier, Ipswich, MA. Accessed 12 Jan 2016.
79. Seifert PC, Peterson E, Graham K. Crisis management of fire in the OR. *AORN J*. 2015;101(2):250–63. <https://doi.org/10.1016/j.aorn.2014.11.002>.
80. Norris J. Fire safety in the operating room. *AANA J*. 1994;62(4):342–5. Available from: CINAHL with Full Text, Ipswich, MA. Accessed 12 Jan 2016.
81. Kucera JJ, Lambert TJ, Klein JA, et al. Liposuction: contemporary issues for the anesthesiologist. *J Clin Anesth*. 2006;18(5):379–87.
82. Klein JA. Tumescence technique for regional anesthesia permits lidocaine doses of 35 mg/kg for liposuction: peak plasma lidocaine levels are diminished and delayed 12 hours. *J Dermatol Surg Oncol*. 1990;16:248–63.
83. Ostad A, Kageyama N, Moy RL. Tumescence anesthesia with a lidocaine dose of 55 mg/kg is safe for liposuction. *Dermatol Surg*. 1996;22:921–7.
84. Lozinski A, Huq N. Tumescence liposuction. *Clin Plastic Surg*. 2013;40:593–613.

85. Neal JM, Mulroy MF, Weinberg GL. American Society of Regional and Pain Medicine checklist for managing local anesthetic systemic toxicity: 2012 version. *Reg Anesth Pain Med.* 2012;37:16–9.
86. Siddik-Sayyid SM, Taha SK, Kanazi GE, et al. Excellent intubating conditions with remifentanyl propofol and either low-dose rocuronium or succinylcholine. *Can J Anaesth.* 2009;56:483–8.
87. Shapiro FE. Anesthesia for outpatient cosmetic surgery. *Curr Opin Anaesthesiol.* 2008;21:704–10.
88. Ferneini EM, Sohn A, Ferneini CM, Tobin H. Prevention of venous thromboembolism in abdominoplasty. *Am J Cosmet Surg.* 2013;30(3):175–80.
89. Whipple KM, Lim LH, Korn BS, et al. Blepharoplasty complications: prevention and management. *Clin Plast Surg.* 2013;40:213–24.
90. Oestreicher J, Mehta S. Complications of blepharoplasty: prevention and management. *Plast Surg Int.* 2012;2012:352–68.
91. Lykoudis EG, Seretis K. Tapia's syndrome: an unexpected but real complication of rhinoplasty: case report and literature review. *Aesthet Plast Surg.* 2012;36:557–9.
92. Naraghi M, Ghazizadeh M. Tension pneumocephalus: a life-threatening complication of septoplasty and septorhinoplasty. *B-ENT.* 2012;8:203–5.