Chapter 5
Anesthesia Complications

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General Perspective and Overview

The relative risks and complications increase proportionately according to preexisting comorbidities of the patient and the length and difficulty of anesthesia and surgery. The age of the patient is often more related to the previously mentioned issues, but in itself increased age (or very premature age) carries risk of mortality. Loose teeth, difficult airways, poor access, emergency procedures, and repeated intubation attempts all carry some degree of increased risk of airway trauma.

Anesthetists argue the benefits of one approach over the other, but data are unclear to demonstrate differences in terms of the observed or reported complications in many cases.

Possible reduction in the risk of misunderstandings over complications or consequences from anesthesia might be achieved by:

- Good explanation of the risks, aims, benefits, and limitations of the procedure(s)
- Careful planning considering the anatomy, approach, alternatives, and method
- Avoiding likely problems
- Adequate clinical follow-up (especially if problems arise)

With these factors and facts in mind, the information given in this chapter must be appropriately and discernibly interpreted and used.

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Important Note: It should be emphasized that the risks and frequencies that are given here represent derived figures. These are often representative of a number of studies, which include different patients with differing comorbidities. As such, the risks of complications in lower or higher risk patients may lie outside these estimated ranges, and individual clinical judgement is required as to the expected risks communicated to the patient and staff, or for other purposes. The range of risks is also derived from experience and the literature; while risks outside this range may exist, certain risks may be reduced or absent due to certain variations of procedures or approaches. It is recognized that different patients, practitioners, institutions, regions, and countries may vary in their requirements and recommendations.

For risks and complications associated with anesthesia assessment, monitoring, or safety, see Chapters 3 and 8, or the relevant volume or chapter.

Introduction

Historically, surgical procedures until the mid-nineteenth century were initially performed without anesthesia of any type. Inevitably, severe pain was the limiting factor, with the consequence of significant restriction in the type, timing, and extent of surgery possible. Pain caused significant movement and muscle spasm, which severely limited operative ease and duration. Early battlefield surgeons noted the relative ease of performing surgery on the unconscious patient and sought this "ideal" state for elective surgery in civilian life. Early anesthetics in Western cultures included the use of cold compresses applied to the region preoperatively, herbal preparations to "numb" nerves, induced concussion, alcohol, opium extracts, and other sedatives and in many Eastern cultures, hypnosis, acupuncture, opiates, deep meditation, and other herbal medicines. Even as recently as the 1800s, alcohol and a "good dose of courage" comprised the sole anesthetic used by naval and military surgeons before amputation of limbs. "Biting the bullet" offered diversionary advantage from pain for the patient while surgery was being performed. However, patient movement and maintenance of a satisfactory airway with sufficient oxygenation remained significant problems using any available method involving central nervous system depression.

During the twentieth century there were massive and rapid advances in general, regional, and local anesthetic methods, ventilation, drugs, and intensive care support, and this has continued into the twenty-first century. Surgeons initially performed the basic initial forms of "anesthesia," but this developed into an art and clinical science as the present day specialty of anesthesia or anesthesiology. Enormous improvements and refinements in anesthesia, patient safety, and comfort have permitted much more complex surgical procedures, many now undertaken as day-case surgery. In reality, these have provided some dramatic reductions in surgical complications, risks and consequences, and notable economic advantages for healthcare systems.

These advances will extend even further into this century as electronic, chemical, robotic, and nanotechnology developments become a reality.

The term "anaesthesia" (also anesthesia) means *without feeling,* derived from the Greek words *an*, without, and *aesthesia*, feeling (normal ability to experience

sensation, perception, or sensitivity). The precise definition of what "anesthesia" means continues to evolve as different agents and techniques are being developed.

Assessment of the complications and overall risk of general anesthesia, or anesthesia of any type, is not always straightforward. Effectively it is the compounded risk of:

1. The administration of general (or other) anesthetic and associated agents
2. The surgery being performed
3. The comorbidities of the individual patient

These may not be easily separable. For this reason, different definitions of *anesthetic morbidity and mortality* exist.

Whether the problem was classified as due to *anesthesia alone as the primary cause* or *anesthesia as either the primary or an associated cause* is an important consideration. The time frame is important also. For example, general anesthesia might be assumed to be from the time of "induction" to regaining "full awareness and airway control." It may include the period from entry to departure from theatre. However, it might also include preoperative sedation and postoperative recovery back to the ward. It might also include an intensive care or surgical high-dependency period, where anesthetic effects might still be operational. Coroners often use "death within 24 h of an anesthetic." All of these time periods have been used in calculations of "anesthetic" or "anesthesia-related" complications. The term *perioperative mortality* is also often used, but again this varies appreciably in reports. It can mean operative death, combined pre-/intra-/immediate postanesthetic mortality, 24 or 48 h mortality from induction, or even 30-day hospitalization mortality. The denominator and its accuracy are important also, as some studies use per 100,000 of *population* and others use per 100,000 of *anesthetics delivered* or *surgical procedures performed*. These and other factors can account for reported mortality (and morbidity) differences between studies.

There is an increasing awareness that pre- and postoperative factors are closely, if not inextricably, linked to the intraoperative factors (selection of mode(s) of anesthesia, duration, temperature control, surgical factors, events, etc.) and that these might more usefully be considered collectively in measuring and improving outcomes.

Current Anesthesia or Anesthesiology

Types of Anesthesia

The main types of anesthesia are as follows:

General Anesthesia

Full and complete depression of consciousness by gaseous or intravenous agents is usually termed *general anesthesia*, whereas "partial" or "light" depression of consciousness is often termed *sedation*. States of consciousness between these levels are obtainable, if required, and occur during induction of and recovery from anesthesia.

Regional Anesthesia

Epidural anesthesia is instillation of local anesthetic, narcotic, or other agents into the epidural space around the spinal cord in order to create anesthesia of the area below or around the level of injection.

Spinal anesthesia is instillation of local anesthetic, narcotic, or other agents into the subarachnoid (containing cerebrospinal fluid, CSF) space around the spinal cord in order to create anesthesia of the area below or around the level of injection.

Nerve blocks are instillation of local anesthetic with or without adrenaline (epinephrine) or close to peripheral nerves (e.g., sciatic, popliteal, anterior/posterior tibial, orbital, trigeminal, axillary, median, ulnar, intercostal, femoral, ankle, digital) in order to block conduction in those nerves.

Local Anesthesia

Local anesthesia is injection of local anesthetic agents intradermally or subcutaneously, intramuscularly, intratendinously, intra-articularly, or similarly into tissues or spaces to directly block nerves, causing local anesthesia of the area for surgery and/or pain relief.

Combinations of the types of anesthesia, it should be noted, are not infrequent, for example, general anesthesia followed by local anesthesia, to maximize surgical ease, pain relief, and comfort for the patient. Indeed, such combinations are increasingly used.

General Anesthesia

History

Historically, use of gaseous agents for anesthesia was attempted by a number of dentists in the UK, USA, and Europe in the early 1820s, with experimentation using carbon dioxide-induced coma and then the use of nitrous oxide. Diethyl ether, although discovered in 1540, was not used for inhalational induction of anesthesia until about 1842 when a dentist, William Clarke, used it for teeth extractions, and soon afterwards, Crawford Long used inhaled ether for excision of a neck cyst, although this went unreported. In October 1846, at the Massachusetts General Hospital in Boston, USA, William Morton gave the first public demonstration of the use of ether anesthesia to Gilbert Abbott for excision of a neck tumor, and Oliver Holmes proposed the name *anesthesia* for the ether induction procedure, which gained rapid popularity across Europe, as well as the USA. In Britain, chloroform was used by James Simpson in 1847 for anesthesia, and this was associated with less problematic vomiting and flammability than with ether. Chloroform gained Royal approval in 1853 when John Snow successfully administered anesthesia to Queen Victoria for the birth of Prince Leopold. For many years, both ether and

chloroform were used for induction of general anesthesia, with nitrous oxide gradually regaining popularity as an additive agent. The main problem was control of the amount of gas inhaled/delivered to maintain uniform and safe depth of anesthesia. Dr. H.E.G. Boyle developed a machine in 1917 to deliver mixed gases, including oxygen, and this was later modified to allow extraction of CO_2 using soda lime. This method remained the standard approach to induction of anesthesia for many years. Airway management then took another leap forward with the development of the cuffed endotracheal tube and a range of ventilation devices. The cuffed tube permitted positive-pressure ventilation and improved airway protection. Later refinements have included double-lumen endotracheal tubes, which permit selective deflation of one lung during thoracic surgical procedures, and more recently the inflatable laryngeal mask airway (LMA) by Dr. Archie Brain. Muscle relaxant agents were used to permit adequate muscle relaxation. Analgesics were utilized intraoperatively to provide better pain relief. The development of effective and safe anesthetic techniques and the training of specialist anesthetists have enabled a massive expansion in the range and complexity of surgical procedures.

General Anesthetic Procedures

The main elements of modern general anesthesia are adequate:

1. General anesthesia (general ± regional or local)
2. Analgesia
3. Muscle relaxation (this is less important for superficial surgery)
4. Anti-emesis
5. Monitoring of particularly BP, oxygen saturation, and end-tidal CO_2 concentration

General Anesthesia

Rapid sequence induction of general anesthesia is a technique where pressure is placed directly over the cricoid cartilage to push it backwards, blocking off the esophagus against the cervical vertebral bodies, thereby limiting the risk of regurgitation of gastric contents and aspiration into the respiratory system. It is also called *crash induction* and is used most frequently in the unprepared, non-fasted patient in the emergency setting for safer airway, intensive care, or surgical management. It is always used for obstetric patients, in whom the risk of reflux and aspiration is increased.

A period of *preoxygenation* prior to general anesthetic induction has become standard practice for most anesthetists, because it achieves a higher concentration of oxygen in the lungs and offers a greater reserve of oxygen in case of difficulty in achieving control of the airway. Maximal hemoglobin O_2 loading and higher dissolved O_2 levels allow a greater time before desaturation and oxygen depletion. This may afford a significant advantage during intubation by trainees, or if a difficult intubation is encountered unexpectedly.

Endotracheal Intubation

In 1543, the Italian anatomist and surgeon Andreas Vesalius reported endotracheal intubation of an animal with life-sustaining artificial respiration, but this went virtually unnoticed until the German surgeon Friedrich Trendelenburg reported the first tracheal intubation through a tracheostomy in 1869. However, the first human oral intubation appears to have been performed in 1878 by Macewen, a Glasgow surgeon. The finer historical details of tracheal intubation and tracheostomy appear unclear but were mentioned and illustrated as far back as ancient Greece, Rome, and Egypt. After the First World War, Magill and Macintosh (subsequently professors of anesthesia in Cardiff and (Nuffield) Oxford) refined endotracheal intubation, also devising the Macintosh spatula with a curved blade for direct laryngoscopy, Magill forceps, Magill introducer, and the curved form of the endotracheal tube.

Indications for *nasal intubation* include dental or oral surgery; oral injury; endolaryngeal, tracheal, or esophageal surgery; oral obstruction from jaw fractures; jaw wiring; and oral tumor obstruction. Nasal intubation may be used also in patients with reduced cervical spine mobility (e.g., fracture instability, arthritis, fusion), when intubation may be achieved by fiberoptic endoscopy.

Modern endotracheal (ET) tubes are streamlined devices with collapsible, slim cuffs that permit good visualization of the larynx during the procedure. A variety of introducing bougies are available for "railroading" the ET tube through the larynx into the trachea. In difficult cases, fiberoptic laryngoscopy, using a narrow flexible or rigid endoscope device, can be used to locate the larynx and direct the ET tube in the correct direction. Longer-term effects of the ET tube remaining in place in the intensive care setting include ulceration of the trachea, bleeding, stenosis, and even perforation. These complications are usually avoided by ET tube changes, good care, low seal pressures, and tracheostomy. *Laryngeal masks* were developed to avoid the need for endotracheal intubation, reduce trauma, yet protect the airway from aspiration. These devices are placed over the larynx, within the pharynx, and are, in effect, an oropharyngeal tube with an inflatable cuff to secure the airway in place, creating a seal. These have replaced the ET tube in many settings, especially for peripheral, short elective procedures as they provide a clear, protected airway, without passing through the vocal cords. Problems with laryngeal masks include failure to adequately seal, dislodgement, aspiration around the cuff, failure to adequately suck secreted material out before removal, and unexpected cuff deflation.

Some Adverse Effects Associated with Laryngoscopy and Endotracheal Intubation

- Injury to lips and teeth
- Injury to tongue, hard palate, gums, and pharynx
- Injury to larynx
- Hoarse voice

- Sore throat
- Tracheal injury
- Esophageal intubation
- Tube retraction
- Tube distal migration
- Air leakage
- Cervical spine injury
- Nasal injury (with nasal intubation)
- Base of skull, meningeal injury (nasal method especially with basal skull fracture)
- Inhalation of foreign material (teeth, plates, plastic, etc.)
- Vomiting and aspiration pneumonitis
- Airway bleeding

Some Adverse Effects Associated with Intubation and Ventilation

- Air leakage
- Pneumothorax
- Surgical emphysema
- Pulmonary edema
- Inhalation of foreign material (teeth, plates, plastic, etc.)
- Vomiting and aspiration pneumonitis
- CO_2 accumulation and CO_2 retention (hypercapnia)
- Circuit disconnection
- Overinflation
- Underinflation
- Basal atelectasis

Stages of Anesthesia

Historically, the four stages of general anesthesia relating to the depth of CNS depression are:

1. *Induction* where unconsciousness is "induced" in the patient
2. *Excitation phase* where the somatic and autonomic responses become erratic, with breath holding, gagging, respiratory irregularity, twitching, limb movements, spasticity, and pupillary dilation
3. *Surgical plane* where stability returns with regular breathing (unless muscle relaxant is used), and this plane can further be subdivided into four planes based on eye movements, eye reflexes, and pupillary size
4. *Recovery* where the anesthetic is gradually eliminated and the patient reenters the previous phases in reverse and then regains all reflexes and respiratory drive

Current Agents Used for General Anesthesia

Current inhalational general anesthetic agents include:

- Nitrous oxide
- Isoflurane
- Sevoflurane
- Desflurane
- Less commonly, xenon and halothane (enflurane[1])

Current IV general or sedative agents include:

- Thiopental
- Propofol
- Etomidate
- Ketamine
- Diazepam
- Midazolam

General Anesthesia: Some Associated Adverse Effects

The overall population-based risk of anesthesia is very low. This does not imply that there is negligible risk or no room for improvement(s). Complications and adverse consequences of general anesthesia (± regional or local) and intubation are well described. Some of these represent "iatrogenic" injury, while others are the consequences or side effects of the techniques or agents used or more related to the surgery being performed or to underlying patient comorbidities. It is estimated that some component of human error occurs in 70–80 % of anesthetic injury, but the contribution may be small; studies of complex systems have revealed that up to 85 % are primarily due to deficiencies in the layout and processes of the system (Runciman et al. 1993). Analysis of 13,389 anesthetics identified 116 errors: 9 (7.8 %) were human errors and 107 (92.2 %) were system errors (Lagasse et al. 1995). Although many complications can occur, some of these are shown in Table 5.1 with estimated frequency ranges.

Oral/Nasal Injury

Injuries to the lips, teeth, nose, gums, palate, tongue, pharynx, larynx, and esophagus have all been reported. Dislodgement of teeth and inhalation becomes a major complication, if recognized, often requiring immediate bronchoscopic retrieval.

[1] Essentially no longer used.

Table 5.1 General anesthesia complications

Complications, risks, and consequences	Estimated frequency
Most significant/serious complications	
Nausea and vomiting after surgery (hours to rarely several days)[a]	1–5 %
Dizziness, blurred vision[a]	1–5 %
Confusion or memory loss (increases with age or dementia)[a]	1–5 %
Rare significant/serious problems	
Awareness during anesthesia[a]	0.1–1 %
Esophageal intubation[a] (unrecognized)	0.1–1 %
Chest infection (higher in smokers)	[a]0.1–1 %
Bladder problems	[a]0.1–1 %
Damage to teeth, lips, or tongue[a]	0.1–1 %
Exacerbation of an existing medical condition[a]	0.1–1 %
Respiratory depression	0.1–1 %
Aspiration pneumonitis[a]	<0.1 %
Damage to the eyes	<0.1 %
Serious allergy to drugs[a]	<0.1 %
Nerve damage[a]	<0.1 %
Equipment failure[a]	<0.1 %
Death[a]	<0.1 %
Less serious complications	
Pain or discomfort during injection of drugs[a]	5–20 %
Hematoma and soreness	5–20 %
Sore throat[a]	5–20 %
Minor allergy to drugs[a]	0.1–1 %
Shivering[a]	1–5 %
Headache[a]	1–5 %
Itching[a]	1–5 %
Aches, pains, and backache[a]	1–5 %

Note: Death is related closely to inadequate monitoring (especially lack of pulse oximetry and CO_2) comorbidities, the extent of surgery, and intraoperative bleeding. The extent of intraoperative "awareness" is dependent on definition, detection, and methods of measurement. Muscle pains and aches are more common with use of succinylcholine; bladder problems are more common in older men with prostatism; chest infection is more common in those already with respiratory compromise; aspiration pneumonitis is more common in urgent non-fasted, GI-obstructed, and obstetric patients; IV discomfort/pain is more common with some agents; sore throat and lip damage are more common with difficult endotracheal intubation (Adapted from the Royal College of Anaesthetists, UK)

[a]Depending on agents and techniques used, individual patient characteristics, comorbidities, pathology, postanesthesia care, and surgery being performed

Ocular Injury

This includes corneal abrasions, lacerations, contamination, contusions, blunt and sharp eyeball, and eyelid trauma.

Positioning and Pressure Injuries

Positioning should be a priority before and after induction of anesthesia, and because the position may change during surgery, all staff should reappraise the

situation regularly to ensure that positioning is safe, not contacting metal or hard surfaces and not straining joints or other anatomy. Nerve compression and traction injuries are reported including the ulnar, common peroneal, brachial plexus, facial, trigeminal, median, radial, sciatic, and plantar nerves from atypical positioning or direct compression. Special attention is required for patients with casts or braces, or when raising or lowering the bed. The possibility exists that some of the later postoperative complications, such as sacral, heel, malleolar, and buttock decubitus ulcers, may have a component of their genesis during anesthesia/surgery from the pressure of the firm operating table and immobility. Backache and neck ache are common postoperative musculoskeletal sequelae.

Neck Injury

Soft Tissue

Neck stiffness can occur from lack of support and occasionally dry retching.

Cervical Spine Injury

This is very rare as the head is routinely carefully protected. However, it can occur after trauma or in association with rheumatoid or congenital instability.

Burns

Diathermy or static electricity in the presence of oxygen can ignite volatile gases, including vaporized alcohol from skin preparations and methane from the bowel, causing flash burns and even fire externally on the skin or internally within the airway or bowel. Direct burns can arise from contact with the metal of the operating table, from surgical instruments touching exposed wires, or wet drapes, to complete an electrical circuit.

Cardiac Complications

Most inhalational anesthetic agents depress myocardial function to some degree, with relatively greater effects from halothane and enflurane. This can reduce blood pressure, which may be countered partially by the effects of surgical stimulation. A wide range of drugs used by either the anesthetist or surgeon can influence cardiac rate, rhythm, or function. Simple examples are adrenaline (epinephrine), lignocaine, atropine, and antibiotics. Vasovagal episodes from mesenteric traction, or handling of the gut or heart, can occur, leading to bradycardia and hypotension or cardiac asystole, which may respond to atropine but may require cessation of surgery and cardiac

compression. Severe bradycardia can also occur from peritoneal distension during laparoscopy. Atrial fibrillation, supraventricular tachycardia, atrioventricular block, and ventricular arrhythmias can occur during surgery and require urgent attention to correct the problem to restore adequate cardiac output. Ketamine typically has a cardiovascular stimulatory effect and maintains blood pressure better than other agents.

Respiratory Complications

Inhalational anesthetic agents may irritate the airways during induction, producing coughing and laryngospasm if the reflexes are not sufficiently depressed prior to inhalation. Inhalational induction is used commonly in pediatric anesthesia, usually with sevoflurane, which is less irritating than other volatile anesthetic drugs. Inadvertent distal migration of the endotracheal tube may obstruct one main bronchus, usually the left, and lead to lung collapse. This can lead to hypoventilation, atelectasis, and pulmonary consolidation, in addition to intraoperative hypoxemia. Pulmonary barotrauma can arise from excessively high ventilation pressures and volumes leading to pulmonary contusion, edema, and even pneumothorax. Tension pneumothorax is a surgical emergency requiring immediate pleural catheterization to equalize the pressure, reduce cardiac compression, and restore venous return. Airway occlusion with marked negative pressures during inspiratory efforts may result in pulmonary edema. Dislodgement of the endotracheal tube is potentially catastrophic, requiring immediate restoration of the airway with urgent reintubation. Similarly, endotracheal tube obstruction requires immediate attention; the commonest cause is inspissated secretions, which require aspiration. Cuff deflation may occur due to a faulty cuff or inflation valve. Accidental disconnection of the anesthetic breathing system is another potential disaster, usually heralded by alarms on the anesthetic monitoring system. Similarly, a rising CO_2 level in the circuit is typically detected by the in-line CO_2 monitor (capnography), and a fall in the inspired oxygen tension is detected by the in-line O_2 monitor or pulse oximeter, both of which are programed to alarm when abnormal levels are reached. Narcotic analgesics can produce respiratory depression, which may be initially reversed in the recovery ward by naloxone, but which may recur when the shorter half-life of naloxone permits further respiratory depression from the residual narcotic. Labile asthma during surgery can be problematic and is often unpredictable. Prophylactic nebulized bronchodilator and steroids are sometime useful before surgery in averting an episode in susceptible individuals. Ventilation pressures may rise intraoperatively, and lung compliance may reduce, indicating airway obstruction.

Hepatotoxicity

In a small number of patients, halothane can produce an acute hepatic inflammation and even liver failure in some situations. Some antibiotics can also cause hepatitis, or cholestasis.

Renal Toxicity

Methoxyflurane (now unavailable in most Western countries) releases fluoride ions when metabolized and can induce nephrotoxicity, leading to renal impairment and renal failure, on occasions. Sevoflurane can react with alkali in the CO_2 absorber to produce a vinyl chloride compound, which can cause acute tubular necrosis, usually of an apparently clinically insignificant level unless renal function is already impaired.

Drug Complications

These are multitudinous and can be idiosyncratic. Fortunately, despite the large number of procedures and many drugs used, the incidence of complications is relatively small. However, a real risk of a minor or major drug reaction remains for any patient undergoing anesthesia.

Allergy and Anaphylaxis

Any drug can trigger allergy, but of the anesthetic drugs, muscle relaxants are the most common group. Atypical allergic (anaphylactic) reactions present most commonly with one or more of three signs: bronchospasm, hypotension, and edema. A wide range of drugs and blood products can cause allergic reactions during anesthesia. The *most effective treatment is IV adrenaline (epinephrine) 1–2 (micro) μg/kg body weight,* repeated with dose doubling every 3–5 min, as required. A severe allergy may require an overall total dose of 2 mg or more of adrenaline. Allergic responses vary from minor skin rashes and postoperative nausea to asthmatic episodes where ventilation is difficult, ranging to severe anaphylaxis where laryngeal edema, asthma, severe angioedema, and circulatory collapse may occur. Minor reactions may be treated with an antihistamine, like IM Phenergan, moderate allergy may respond well to IV hydrocortisone, whereas anaphylaxis requires IM or IV adrenaline (epinephrine). Patients with an established or strongly suspected latex rubber allergy should have a full history taken and blood testing for Hevea braziliensis (rubber) antibody may be useful. Ideally, such patients must be preplanned for the beginning of a morning operating list where a non-latex anesthetic circuit and equipment setup is used (including non-latex gloves) to avoid exposure.

Venous Stasis, Deep Venous Thrombosis (DVT), and Pulmonary Thromboembolism (PTE)

The precise effect of general anesthesia on the genesis of deep venous thrombosis is difficult to determine. The analysis is confounded by the effects of the surgical illness, other medical conditions, preoperative immobility and stasis, surgery,

genetics, dehydration and postoperative hypercoagulability, and possible further stasis. However, it is assumed that there is a contributing effect of stasis from the immobility induced by unconsciousness due to anesthesia. The effect may be increased by positive-pressure ventilation and head-up positioning and during laparoscopic procedures where insufflation pressure may impede venous return from the lower limbs.

The use of:

- Intraoperative full leg support stockings
- Intermittent pneumatic calf compression devices
- Preoperative fractionated or unfractionated subcutaneous heparin

is prophylactic measures that have been shown to reduce the incidence of DVT. Pulmonary embolism is reduced in situations where DVT can be reduced. However, a small, low residual incidence of PTE has remained despite diligent application of these prophylactic measures. Risk factors include prior history of DVT, cancer, prolonged surgery, hip and pelvic surgery, lower-limb joint replacement surgery, obesity, pregnancy, hormone therapy, prolonged immobility pre- or postoperatively, increasing age over 45, smoking, and trauma, especially multiple fractures.

Hypothermia

Low core temperature during anesthesia has become a significant risk factor for increased risk of surgical infection. Recent evidence shows that maintenance of a core temperature *above* 36 °C is associated with reduced wound infection and improved wound healing, especially in situations where anaerobic bacterial contamination has occurred intraoperatively. For this reason, patients should be considered for a hot-air blanket, covering as much of the body as is practicable, and the warming of all IV fluids during surgery.

Malignant Hyperthermia (Hyperpyrexia)

This relatively rare condition is also termed malignant hyperpyrexia (MH). It is an autosomal dominant genetic condition where the control of Ca^{++} release with skeletal muscle contraction is impaired in the presence of volatile anesthetics or after the administration of suxamethonium. The leaking of Ca^{++} triggers continual muscle contraction and generates heat, causing hyperthermia and lethal hyperkalemia. Any family history needs to be treated seriously and investigated further. Any possible drugs that can trigger MH should be avoided, which includes all of the volatile anesthetic agents, but not the gases nitrous oxide and xenon. Intravenous anesthetic agents are safe, as are all of the muscle relaxants except suxamethonium (succinylcholine). If a general anesthetic is required, then it can be achieved using nitrous oxide and intravenous agents such as midazolam and fentanyl or total intravenous anesthesia using propofol and an analgesic, such as remifentanil.

Hypoxia

Recent evidence demonstrates that avoidance of hypoxemia (a low oxygen tension or oxygen saturation in blood) and hypoxia (a low oxygen tension in the tissues) is associated with less wound infections postoperatively. When coupled with avoidance of hypothermia, these intraoperative anesthetic factors can potentially influence (later) surgical outcomes significantly. Because hypoxemia is difficult to detect clinically, all patients undergoing anesthesia should be monitored with pulse oximetry.

Central Nervous System Complications

Although it is usually thought that a death from anesthesia is the worst adverse event possible, it could be argued that brain injury (severe or even minor) arising from cerebral hypoxia might be equally, or possibly even more, catastrophic. The outcome of a patient who is unable to perform the tasks that they were able to undertake prior to anesthesia and surgery represents a devastatingly distressing situation for the patient, families, and community. Very small cerebral deficits may go unnoticed, but moderate or larger disturbances in cerebral function can cause a range of disabilities such as hemiparesis, dysarthria, cognitive loss, loss of sensory faculties, and even permanent coma. The risk of severe brain injury is difficult to ascertain precisely but has been estimated at 1:170,000 anesthetics (Aitkenhead 2005; Kawashima et al. 2003a, b). Economic costs from these injuries are often considerable personally in patient maintenance, rehabilitation, and negligence payouts.

Mortality

The overall risk of general anesthesia is effectively the compounded risk of (1) the administration of general anesthetic and associated agents, (2) the surgery being performed, and (3) the comorbidities of the individual patient. These may not be easily separable. For this reason, different definitions of *anesthetic mortality* exist. Whether deaths are classified as due to *anesthesia alone as the primary cause* or *anesthesia as either the primary or an associated cause* is influential. The term *perioperative mortality* is also often used, but again this varies appreciably, meaning operative death, combined pre-/intra-/immediate postanesthetic mortality, 48-h mortality, or even 30-day mortality. The denominator and its accuracy are important also, as some studies use per 100,000 of *population* and others use per 100,000 of *anesthetics delivered*. "Critical incidents" and "serious adverse events" are wider terms, sometimes used, that include mortality and morbidity. These and other factors can account for reported mortality differences between studies.

However, the risk of death from a *general anesthetic* in most Western countries is very low, with an annual mortality rate ranging from about 0.11 deaths per 100,000 *population* (Li et al. 2009) to around 1.8 per 100,000 *anesthetics given*,

Table 5.2 Rates of deaths totally or partially related to anesthesia according to age and ASA physical status

		Mortality rate per 100,000 anesthetic procedures
Age		
0–7 year		0.60
8–15 year		1.20
16–39 year		0.52
40–74 year		5.20
>75 year		21.00
ASA physical status		
I	Normal healthy individual	0.40
II	Mild systemic disease that does not limit activity	5.0
III	Severe systemic disease that limits activity but is not incapacitating	27.0
IV	Incapacitating systemic disease which is constantly life-threatening	55.0
V	Moribund, not expected to survive 24 h with or without surgery	Individual

Adapted from Lienhart et al. (2006)

depending on the patient mix (age, morbidity, urgency), definitions, location, data recording, and health system supports. Anesthetic mortality rates in Western Australia for the period 1980–2002 were <1:50,000 surgical procedures per annum, with confidentiality and legal protection having been ensured, with consistent definitions and classifications applied.

These overall mortality figures include a spectrum of risk of death ranging from extremely "low" up to "almost certain," depending on the individual risk profile of the patient and surgery. The relative risk ranges from about 0.6 per 100,000 anesthetics in those <15 years of age to over 21 per 100,000 in those over the age of 75 years. The American Society of Anesthesiologists (ASA) grading offers a refinement on this overall figure by including an evaluation of the comorbidities and relative risk of "anesthetic death" arising from these. In a French study by Lienhart et al. in 2006, death rates totally or partially related to anesthesia for 1999 were 0.69:100,000 and 4.7:100,000 *anesthetics given (estimated)*, respectively. The death rate increased from 0.4 to 55 per 100,000 *anesthetics given* for ASA physical status I and IV patients, respectively. Rates increased with increasing age. Aspiration of gastric contents, intraoperative hypotension, and anemia associated with postoperative ischemic complications were the associated factors most often encountered. Deviations from standard practice and organizational failure were often found to be associated with death. Comparing data from a 1978 to 1982 French nationwide study, the anesthesia-related mortality rate appeared to be reduced tenfold in 1999. It was noted that much remained to improve physician compliance to standard practice and improve the anesthetic system and institutional processes (Lienhart et al. 2006). Some 25 % of deaths occur in the hands of nonspecialist anesthetists, trainees, and other nonspecialists using ANZCA data (ANZCA 2002) (Table 5.2).

Table 5.3 Estimates of the incidence of mortality due to anesthesia between 1982 and 2006

Authors	Year of publication	Primary anesthetics	Primary cause	Associated cause
Lunn and Mushin	1982	1,147,362	1:10,000	1:700
Tiret et al.	1986	198,103	1:13,207	1:7,924
Buck et al.	1987	555,258	1:185,086	1:1,354
Holland	1987	–	–	1:26,000
Chopra et al.	1990	113,074	1:16,250	–
Pedersen	1994	200,000	1:2,500	–
Tikkanen and Hovi-Viander	1995	325,585	1:66,667	–
Warden and Horan	1996	–	–	1:20,000
Arbous et al.	2001	869,483	1:124,212	1:7,143
ANZCA	2002	2,586,000	1:168,000	1:56,000
Kawashima et al.	2003a, b	2,363,038	1:47,619	–
Lienhart et al.	2006	7,756,121	1:145,500	1:18,500

Adapted from Aitkenhead (2005), Lagasse (2002), and Lienhart et al. (2006)
Note: The denominator, the methodology, definitions, patient population, ASA grading, experience of anesthetists, data collection, and precision of calculations of these rates are unlikely to have been identical, so some caution in comparison and interpretation is advisable. These factors may account for the observed variability. The numerator of these studies is not always equivalent nor is the denominator – per head of population, surgical procedures performed, or anesthetics performed. Caution should be exercised on this aspect also

Overdose of anesthetics accounted for almost half (47 %) of anesthesia-related deaths and adverse effects of anesthetics in therapeutic use accounted for an additional 42 %. Men had about twice the anesthesia-related mortality rate of women and both increased markedly after the age of 65 years (Li et al. 2009).

Respiratory deaths (and coma) were found in 1:7,960 *anesthetics given* in a 1978–1982 French national mortality survey compared with 1:48,200 in 1999, based on death certificates. Deaths associated with intraoperative *failure of the breathing circuit and equipment* were no longer encountered, and no deaths due to recovery unit *undetected hypoxia* were found in the 1999 study. Deaths related to *difficult intubation* occurred in 1:13,000 in the 1978–1982 period compared with 1:46,000 in 1999, a fourfold reduction. In most cases, both *inadequate practice and systems failure* (inappropriate communication between staff, inadequate supervision, poor organization) were identified as contributory (Tables 5.3, 5.4, 5.5, and 5.6).

Sedation

Sedation is a state of reduced awareness, resulting from administration of a sedative agent, usually intravenously. Sedation is not usually as "deep" a state of unconsciousness as is desired in general anesthesia. Drugs also used for general anesthesia are often used in a lesser or titrated doses to produce sedative effects. The use of an intravenous sedative is frequently employed in combination with injected or topically applied local anesthesia, for example, for superficial surgery or endoscopic procedures. Sedation reduces awareness and discomfort associated

Table 5.4 Examples of the most commonly quoted critical incidents in the Australian Incident Monitoring Study (Holland et al. 1993a, b)

Problems with breathing system
Disconnections
Misconnections
Leaks
Problems in administration of drugs
Overdosage
Underdosage
Wrong drug
Problems with intubation and control of airway
Failed intubation
Esophageal intubation
Endobronchial intubation
Accidental or premature extubation
Aspiration
Failure of equipment
Laryngoscopes
Intravenous infusion devices
Breathing system valves
Monitoring devices

Table 5.5 Examples of the commonest underlying factors associated with critical incidents in the Australian Incident Monitoring Study (Holland et al. 1993a, b)

Inattention/carelessness
Inexperience
Haste
Drug effect, type, or dosage problem
Failure to check equipment
Failure of equipment
Unfamiliarity with equipment
Poor communication
Restricted visual field or access
Failure of planning
Lack of experience
Distraction
Lack of skilled assistance
Lack of supervision
Fatigue and decreased vigilance

with the procedure and is used for endoscopic (upper GI, urological, colonoscopic, laryngoscopic, bronchoscopic, and a range of ophthalmic and cutaneous surgical procedures), often abolishing memory of noxious stimuli while preserving respiratory function and most reflexes. Topical anesthetic agents may, however, abolish important reflexes, such as the gag reflex – which may be necessary for pharyngeal passage of instrumentation. If sedatives are given in high enough doses, they can abolish reflexes and effectively deliver intravenous anesthesia to the patient. In elderly patients, and those on sedative medications or with central nervous system depression, the therapeutic window may be very narrow; the effects of relatively small doses of sedative agents can be profound and cause respiratory depression. Pulse oximetry during sedation is usually considered mandatory, or at least prudent in most cases, with the sedation preferably delivered by an anesthetist.

Table 5.6 Most common complications found on analysis of US closed claims related to anesthetic care in the 1990s (Posner 2001)

Injury	Frequency
Death	23 %
Nerve injury	21 %
Brain damage	9 %
Burns/skin inflammation	6 %
Awareness	5 %
Eye injury	5 %
Backache	5 %
Headache	5 %
Pneumothorax	4 %
Aspiration pneumonitis	3 %
Injury to newborn	1.5 %

Current IV sedative agents include:

- Diazepam
- Midazolam
- Propofol

Some Adverse Effects Associated with Sedation

- Airway compromise
- Hypoxemia
- Reflux
- Aspiration pneumonitis

Analgesia

General anesthesia induces unconsciousness, but may not necessarily block the pain pathways, and therefore adequate intravenous analgesia is used to abolish or modulate pain sensation, transmission, and awareness. Pain awareness is different from awareness under inadequate general anesthesia, although both may occur together. Opioids and opioid derivatives and synthetic agents are typically used for analgesia, although intravenous nonsteroidal agents (e.g., paracetamol, ketoprofen), and more empirically steroidal and other agents, have been used to modulate operative and postoperative pain.

Analgesic Agents

Opioid agents include:

- Morphine
- Diamorphine (diacetyl morphine, also known as heroin)

- Codeine (methyl morphine)
- Fentanyl
- Alfentanil
- Sufentanil
- Remifentanil
- Meperidine, also called pethidine in the UK, New Zealand, Australia, and other countries
- Methadone
- Oxycodone
- Tramadol

Opiate antagonists include:

- Naloxone, similar chemically to some analgesics, reverses the effects of morphine-like agents, but is not a painkiller.
- Naltrexone.

Non-opiate analgesic agents include:

- Aspirin
- Ibuprofen
- Indomethacin
- Naproxen
- Piroxicam
- Paracetamol
- Ketorolac
- Ketoprofen
- Diclofenac
- Cox-II inhibitors (celecoxib, rofecoxib, valdecoxib)
- Ketamine

Analgesic modulating agents include:

- α2-adrenergic agonists
- Clonidine
- Dexmedetomidine
- Adenosine
- Magnesium
- Neostigmine
- Gabapentin
- Cannabinoids
- Inositol triphosphate
- Droperidol
- Amitriptyline
- Glucocorticoid steroids

Analgesic agents should be selected based on the desired actions and route of administration. Paracetamol, for example, can be given orally or intravenously.

Some Adverse Effects Associated with Analgesia

Narcotic overdosage can occur as a result of excess administration (e.g., dose or frequency too high) or in association with reduced clearance and accumulation of narcotic agent or its active metabolites (e.g., norpethidine as a metabolite of pethidine), especially in patients with liver disease. Relative overdosage can occur with concomitant administration of other central nervous system (respiratory) depressant agents, such as diazepam, barbiturates, other narcotics, antihistamines, or even alcohol. When the dose of narcotic-reversing agent (e.g., naloxone; shorter half-life) is inadequate for full reversal of the narcotic effect, the narcotic respiratory depression can return and require a further dose of naloxone.

- Intravenous/subcutaneous access site infections
- Respiratory depression, failure, and rarely mortality
- Chest infection
- Urinary retention
- Dry eyes and mouth
- Psychogenic effects, including psychoses
- Constipation or diarrhea

Constipation (which can be unpleasantly severe and require urgent intervention) is a main side effect of narcotic agents. Stool softeners (e.g., coloxyl) and good hydration are paramount in avoiding this unpleasant complication. Severe or increasing pain may, of course, signify a complication with the need for surgical intervention and/or return for acute surgical intervention. Acute pain service support may be useful for difficult situations where pain is uncontrolled and variable or requires special techniques such as epidural infusion or nerve blocks.

Paracetamol in high dose may be hepatotoxic, especially when used continuously or in susceptible individuals.

Aspirin (and other NSAIDs) may cause bleeding through reduction of platelet adhesion and may lead to peptic ulceration and gastrointestinal hemorrhage, which on occasions may be life-threatening.

Cannabinoids, droperidol, and amitriptyline can have additional psychogenic or hallucinogenic effects and may cause urinary retention and constipation.

Muscle Relaxation

In many procedures, especially superficial procedures, muscle relaxation is not required. However, in procedures that require entry through the abdominal wall or chest, or for manipulation of large joints, adequate muscle relaxation is usually required to permit the procedure to proceed optimally or with less difficulty. Muscle relaxants are also usually given if tracheal intubation is required.

Current muscle relaxants include:

Depolarizing agent:

- Succinylcholine (also known as suxamethonium in the UK, New Zealand, Australia, and some other countries)

Non-depolarizing agents:

- Vecuronium
- Rocuronium
- Pancuronium
- Atracurium
- Cisatracurium
- Mivacurium (plasma cholinesterase-dependent breakdown)
- Curare, the active ingredient of which is tubocurarine

Some Adverse Effects Associated with Muscle Relaxation

Succinylcholine (suxamethonium) is a "depolarizing" muscle relaxant, which is degraded by the enzyme plasma cholinesterase. Some people have a genetic autosomal recessive abnormality which makes their plasma cholinesterase less effective in metabolizing succinylcholine. This can be checked by a dibucaine number and measurement of plasma cholinesterase activity. In patients with slow cholinesterase activity, the depolarization is prolonged, leading to *excessively prolonged muscle relaxation*, which necessitates continued artificial ventilation. The effects gradually recover, sometimes over many hours. Although it is not a depolarizing agent, *mivacurium* is also deactivated by plasma cholinesterase, and prolonged paralysis can occur in patients with abnormal forms of the cholinesterase enzyme.

Succinylcholine may cause *hyperkalemia* in burn patients or paralyzed (quadriplegic, paraplegic) patients after several days as their muscle sensitivity increases. *Malignant hyperthermia* may be triggered by succinylcholine in susceptible patients.

Awareness during anesthesia is "unintended intraoperative awareness" which can occur during general anesthesia, when a patient has not had enough general anesthetic or analgesic to prevent consciousness and the recall of events. This experience may be extremely traumatic for the patient and occurs usually when patients have been paralyzed with a muscle relaxant but awaken from anesthesia and are then aware of their surroundings, but unable to move or indicate that they are awake. This is distinct and separate from the situation when sedation or regional anesthesia is used, and the patient is deliberately fully or partially aware of the procedure and may have some recall. Neurological (e.g., BIS, entropy, or EEG systems) and cardiac monitoring can help to minimize the incidence of awareness. Arguably the best method of avoiding awareness is end-tidal in-circuit monitoring of the administered volatile agent.

Some 0.15 % of patients undergoing general anesthesia are given insufficient anesthetic to maintain unconsciousness, either initially or during the operation. The incidence of anesthesia awareness in the USA is believed to be 20,000–40,000 cases per year, out of approximately 20 million general anesthetics administered, which represents between 0.1 and 0.2 % of all patients undergoing general anesthesia (JCAHO 2004; Sebel et al. 2004).

Antiemetics

Many anesthetic agents, both inhalational and intravenous, and analgesics may induce postoperative nausea and vomiting (PONV). Some patients and certain conditions are more often associated with higher risk of emesis. This is particularly so for patients who have a previous history of PONV or of motion sickness. Generally, the incidence is higher in younger women and in those who are nonsmokers. The range and efficacy of antiemetic agents have progressed rapidly in recent years, and classical agents such as metoclopramide, prochlorperazine, and droperidol are gradually being supplemented or replaced by serotonin ($5HT_3$) antagonists such as ondansetron, tropisetron, and dolasetron.

Antiemetics include:

- Metoclopramide
- Prochlorperazine
- Droperidol
- Ondansetron
- Tropisetron
- Dolasetron
- Cyclizine
- Glucocorticoid steroids

Some adverse effects of antiemetics include:

- Oculogyric crisis (especially in the young and adolescents)
- Dystonic reactions
- Failure to relieve vomiting
- Sedation

Regional Anesthesia

Regional anesthesia includes a range of selective regional nerve blocks and also epidural and spinal anesthetic procedures. Regional anesthesia has increased in popularity as better techniques and training have occurred, and electrophysiological and ultrasound methods of more accurate placement of local anesthesia instillation have developed. Better appreciation of the lower overall complication rates from regional anesthesia (e.g., lower incidence of chest infection, lower DVT risk, lower

cerebral effects) compared with general anesthesia and the increased understanding of concomitant use of regional and general anesthesia for better pain control have also increased usage.

Selective Regional Nerve Blocks

Anesthesia can be achieved in a specific body region using a range of techniques to block the nerve supply to the respective area. Digital ring blocks, ankle block, intercostal blocks, wrist block, periorbital block, arm (axillary) block, femoral block, caudal block, and sacral block are examples of regional anesthesia. Epidural and spinal blocks are special types of regional anesthesia and are often described separately.

All involve instillation of local anesthetic agents, sometimes in combination with an opioid analgesic, steroid, adrenaline (epinephrine), or other agents.

Types of selective regional nerve blocks include:

Peripheral blocks

- Arm (axillary)
- Wrist
- Ocular/ophthalmic
- Femoral
- Ankle
- Sciatic
- Digital

Caudal block
Paravertebral block

Regional Anesthesia Complications: Some Associated Adverse Effects

Pain/discomfort
Nerve injury

- Sensory (partial/complete; permanent/temporary)
- Motor (partial/complete; permanent/temporary)

Sympathetic dystrophy/dysesthesia
Drug reaction
Local anesthetic toxicity

- Seizures, cardiac arrhythmias, and cardiac arrest
- Perioral numbness and tingling

Adrenaline (epinephrine) toxicity

- Tachycardia
- Cardiac arrhythmias

Vasospasm of extremities

- Digital or flap ischemia and necrosis

Hematoma formation
Infection
Sterile abscess formation
Failure of anesthesia
Pneumothorax (intercostals or vertebral block)

Epidural and Spinal Anesthesia

Epidural anesthesia (block) is selective instillation of local anesthetic agent (with or without adrenaline (epinephrine)) via a cannula placed in the epidural (extradural) space, usually with the catheter remaining in place in the epidural space. A potential advantage of the epidural method is that a catheter can be placed in the space for later administration of local anesthetic or analgesic agents by continuous or intermittent dosing of the agent, postoperatively in the ward for several days.

Spinal anesthesia (block) is selective instillation of the local anesthetic agent (with or without adrenaline (epinephrine)) into the subarachnoid space, mixing directly with the cerebrospinal fluid (CSF). Over recent years, there has been a relative shift to the increased use of spinal anesthesia over epidural anesthesia in many centers, particularly for obstetric and lower abdominal, pelvic, and perineal surgery, as generally a much denser block is achieved than with epidural administration. Spinal anesthesia is sometimes preferred to general anesthesia for elderly patients and those with comorbidities, for suitable procedures below the umbilicus, because of the relatively lower risks from cardiorespiratory complications compared with general anesthesia. However, spinal anesthesia has the risk of postural headache. This risk is greater with larger bore needles for spinal injection – roughly double the risk when an 18G compared with a 25G needle is used.

Epidural and Spinal Anesthesia: Some Associated Adverse Effects

More Specific Effects

Headache (dural leak of CSF)
Spinal injury

- Minor – paresthesia, anesthesia or weakness, permanent or temporary
- Major – paraplegia or extremely rarely quadriplegia from injury

Epidural or intrathecal bleeding (especially in patients taking anticoagulants)
Inadvertently wider anesthetic field (e.g., respiratory or cardiac fiber paralysis from spinal anesthetic)
Permanent damage to nerve roots or spinal cord from injection of wrong drug
Paraspinal infection
Severe hypotension
Inadvertent retention of catheter part(s) (potential in epidural catheter insertion)
Dislodgement of epidural catheter
Failure of adequate anesthesia

More General Epidural and Spinal Anesthesia Effects

Pain/discomfort
Nerve injury

- Sensory (partial/complete; permanent/temporary)
- Motor (partial/complete; permanent/temporary)

Sympathetic dystrophy/dysesthesia
Drug reaction
Local anesthetic toxicity

- Seizures, cardiac arrhythmias, and cardiac arrest
- Perioral numbness and tingling

Adrenaline (epinephrine) toxicity

- Tachycardia
- Cardiac arrhythmias

Vasospasm of extremities

- Digital or flap ischemia and necrosis

Hematoma formation
Infection
Sterile abscess formation
Nonspecific memory effects

Local Anesthesia

The first recognized local anesthetic agent in Western medicine was cocaine, used in 1884 by Karl Koller for an ophthalmological procedure, possibly suggested by Sigmund Freud. Prior to that, ice, frozen saltwater slurry, cold alcohol, topical alcohol, topical ether, and ethyl chloride spray had been used. In Eastern cultures a variety of herbal preparations and other potions, and methods of acupuncture, had been used.

A variety of derivatives, synthetic analogues, and novel organic compounds have been developed since, including procaine (1905) and lidocaine (lignocaine; 1943). These agents alter the sodium permeability of the neuronal cell membrane and impede or eliminate conduction of the action potential, leading to loss of sensation in the region supplied by the injected and affected nerves.

The two basic classifications of local anesthetic agents are (1) the *esters* (procaine, amethocaine, and cocaine) and (2) the *amides* (lidocaine/lignocaine, prilocaine, bupivacaine, ropivacaine). The amides are usually slower acting, are more stable in solution, have a longer shelf life, and are associated with less allergic reactions than the esters.

Addition of adrenaline (epinephrine) to the local anesthetic (LA) agent (usually in 1:100,000 or 1:200,000 concentrations) can typically cause vasoconstriction, can reduce the loss of LA from the region, and may promote an increased duration of action. Prepackaged dental anesthetic agents may have much higher doses of adrenaline (e.g., 1:50,000), increasing the potential for side effects from these agents either from direct vessel puncture or absorption, especially in the very vascular regions such as the head and neck.

Toxic effects may arise from overdosage of the local anesthetic or vasoconstrictive agents.

The predicted upper safe doses:

Lignocaine is 3 mg/kg alone or 7 mg/kg with adrenaline added
Bupivacaine is 2 mg/kg either with or without adrenaline

Local Anesthesia Complications: Some Associated Adverse Effects

Pain and discomfort
Nerve injury

- Sensory
- Motor

Drug reaction
Local anesthetic toxicity

- Seizures, cardiac arrhythmias, and cardiac arrest
- Perioral numbness and tingling

Adrenaline (epinephrine) toxicity

- Tachycardia
- Cardiac arrhythmias

Vasospasm of extremities

- Digital or flap ischemia and necrosis

Hematoma formation
Infection
Sterile abscess formation
Failure of anesthesia

Summary

Anesthesia has been a major essential requirement for most aspects of surgical development over the years. Initially, early anesthesia was given by surgeons, but as anesthesiology has grow as a specialty, it has rapidly advanced technologically. The multifaceted elements of anesthesia include general anesthesia, airway protection, ventilation, sedation, muscle relaxation, analgesia, anti-emesis, regional nerve blocks, epidural anesthesia, spinal anesthesia and local anesthesia, and intensive care management, all of which have exerted profound effects on both the spectrum of surgery possible and patient comfort. Although anesthesia has a range of specific complications, good anesthesia has reduced the incidence of a range of surgical postoperative complications, for example, control of pain and emesis. However, appropriate selection and management of anesthesia is capable of reducing the risks of major adverse events such as DVT, pulmonary thromboembolism, pneumonia, cardiac events, and mortality in many situations. Close collaboration between anesthetic, medical, and surgical teams is required for optimization of the patient's condition prior to surgery, especially in complex situations and/or elderly patients (see Chap. 4). The main focus has developed on good supervision and training, good practices in patient monitoring for early detection of problems, detailed audit of perioperative adverse events, near misses and evaluation of deaths, and an ongoing commitment to improvement of patient safety including "designing out" potential system problems that lead to risk of human error (see Chaps. 3, 8, 9, and 10).

Despite our vast knowledge about anesthesia, we still do not know precisely how general anesthesia works! However, the successes do *not* mean there is no room for further progress, and rapid developments have occurred over the last decade and are set to continue into the future. These developments will undoubtedly change the possibilities and face of surgery and influence the range and incidence of complications that will be experienced in the future.

Consent and Risk Reduction

Main Points to Explain

- Oral/teeth/nasal injury
- Sore throat/pharyngeal injury
- Bleeding
- Nausea/vomiting

- IV access/infection
- Respiratory problems
- Muscle pain
- Risk of death
- Risks without anesthesia/surgery

Further Reading, References, and Resources

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