CHAPTER

54

Anesthetic Complications

KEY CONCEPTS

1. The rate of anesthetic complications will never be zero. All anesthesia practitioners, irrespective of their experience, abilities, diligence, and best intentions, will participate in anesthetics that are associated with patient injury.
2. Malpractice occurs when four requirements have been met: (1) the practitioner must have a duty to the patient; (2) there must have been a breach of duty (deviation from the standard of care); (3) the patient (plaintiff) must have suffered an injury; and (4) the proximate cause of the injury must have been the practitioner's deviation from the standard of care.
3. Anesthetic mishaps can be categorized as preventable or unpreventable. Of the preventable incidents, most involve human error, as opposed to equipment malfunctions.
4. The relative decrease in death attributed to respiratory rather than cardiovascular damaging events has been attributed to the increased use of pulse oximetry and capnometry.
5. Many anesthetic fatalities occur only after a series of coincidental circumstances, misjudgments, and technical errors coincide (mishap chain).
6. Despite differing mechanisms, anaphylactic and anaphylactoid reactions are typically clinically indistinguishable and equally life-threatening.
7. True anaphylaxis due to anesthetic agents is rare; anaphylactoid reactions are much more common. Muscle relaxants are the most common cause of anaphylaxis during anesthesia.
8. Patients with spina bifida, spinal cord injury, and congenital abnormalities of the genitourinary tract have a very increased incidence of latex allergy. The incidence of latex anaphylaxis in children is estimated to be 1 in 10,000.
9. Although there is no clear evidence that exposure to trace amounts of anesthetic agents presents a health hazard to operating room personnel, the United States Occupational Health and Safety Administration continues to set maximum acceptable trace concentrations of less than 25 ppm for nitrous oxide and 0.5 ppm for halogenated anesthetics (2 ppm if the halogenated agent is used alone).
10. Hollow (hypodermic) needles pose a greater risk than do solid (surgical) needles because of the potentially larger inoculum. The use of gloves, needleless systems, or protected needle devices may decrease the incidence of some (but not all) types of injury.

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11 Anesthesiology is a high-risk medical specialty for substance abuse.

12 The three most important methods of minimizing radiation doses are limiting total exposure time during procedures, using proper barriers, and maximizing one's distance from the source of radiation.

1 The rate of anesthetic complications will never be zero. All anesthesia practitioners, irrespective of their experience, abilities, diligence, and best intentions, will participate in anesthetics that are associated with patient injury. Moreover, unexpected adverse perioperative outcomes can lead to litigation, even if those outcomes did not directly arise from anesthetic mismanagement. This chapter reviews management approaches to complications secondary to anesthesia and discusses medical malpractice and legal issues from an American (USA) perspective. Readers based in other countries may not find this section to be as relevant to their practices.

LITIGATION AND ANESTHETIC COMPLICATIONS

All anesthesia practitioners will have patients with adverse outcomes, and in the USA most anesthesiologists will at some point in their career be involved to one degree or another in malpractice litigation. Consequently, all anesthesia staff should expect litigation to be a part of their professional lives and acquire suitably solvent medical malpractice insurance with coverage appropriate for the community in which they practice.

When unexpected events occur, anesthesia staff must generate an appropriate differential diagnosis, seek necessary consultation, and execute a treatment plan to mitigate (to the greatest degree possible) any patient injury. Appropriate documentation in the patient record is helpful, as many adverse outcomes will be reviewed by facility-based and practice-based quality assurance and performance improvement authorities. Deviations from acceptable practice will likely be noted in the practitioner's quality assurance file. Should an adverse outcome lead to litigation, the medical record documents the practitioner's actions at the time of the incident. Often years pass before litigation proceeds to the point where the anesthesia provider is asked about the case in question. Although memories fade, a clear and complete anesthesiology record can provide convincing evidence that a complication was recognized and appropriately treated.

A lawsuit may be filed, despite a physician's best efforts to communicate with the patient and family about the intraoperative events, management decisions, and the circumstances surrounding an adverse event. It is often not possible to predict which cases will be pursued by plaintiffs! Litigation may be pursued when it is clear (at least to the defense team) that the anesthesia care conformed to standards, and, conversely, that suits may not be filed when there is obvious anesthesia culpability. That said, anesthetics that are followed by unexpected death, paralysis, or brain injury of young, economically productive individuals are particularly attractive to plaintiff's lawyers. When a patient has an unexpectedly poor outcome, one should expect litigation irrespective of one's "positive" relationship with the patient or the injured patient's family or guardians.

2 Malpractice occurs when four requirements are met: (1) the practitioner must have a duty to the patient; (2) there must have been a breach of duty (deviation from the standard of care); (3) the patient (plaintiff) must have suffered an injury; and (4) the proximate cause of the injury must have been the practitioner's deviation from the standard of care. A duty is established when the practitioner has an obligation to provide care (doctor–patient relationship). The practitioner's failure to execute that duty

constitutes a breach of duty. Injuries can be physical, emotional, or financial. Causation is established; if but for the breach of duty, the patient would not have experienced the injury. When a claim is meritorious, the tort system attempts to compensate the injured patient and/or family members by awarding them monetary damages.

Being sued is stressful, regardless of the perceived "merits" of the claim. Preparation for defense begins before an injury has occurred. Anesthesiology staff should carefully explain the risks and benefits of the anesthesia options available to the patient. The patient grants informed consent following a discussion of the risks and benefits. Informed consent does not consist of handing the patient a form to sign. Informed consent requires that the patient understand the choices being presented. As previously noted, appropriate documentation of patient care activities, differential diagnoses, and therapeutic interventions helps to provide a defensible record of the care that was provided, resistant to the passage of time and the stress of the litigation experience.

When an adverse outcome occurs, the hospital and/or practice risk management group should be immediately notified. Likewise, one's liability insurance carrier should be notified of the possibility of a claim for damages. Some policies have a clause that disallows the practitioner from admitting errors to patients and families. Consequently, it is important to know and obey the institution's and insurer's approach to adverse outcomes. Nevertheless, most risk managers advocate a frank and honest disclosure of adverse events to patients or approved family members. It is possible to express sorrow about an adverse outcome without admitting "guilt." Ideally, such discussions should take place in the presence of risk management personnel and/or a departmental leader.

It must never be forgotten that the tort system is designed to be adversarial. Unfortunately, this makes every patient a potential courtroom adversary. Malpractice insurers will hire a defense firm to represent the anesthesia staff involved. Typically, multiple practitioners and the hospitals in which they work will be named to involve the maximal number of insurance policies that might pay in the event of a plaintiff's victory, and to ensure that the defendants cannot choose to attribute "blame" for the adverse event to whichever person or entity was not named in the suit. In some systems (usually when everyone in a health system is insured by the same carrier), all of the named entities are represented by one defense team. More commonly, various insurers and attorneys represent specific practitioners and institutional providers. In this instance, those involved may deflect and diffuse blame from themselves and focus blame on others also named in the action. One should not discuss elements of any case with anyone other than a risk manager, insurer, or attorney, as other conversations are not protected from discovery. Discovery is the process by which the plaintiff's attorneys access the medical records and depose witnesses under oath to establish the elements of the case: duty, breach, injury, and causation. False testimony can lead to criminal charges of perjury.

Oftentimes, expediency and financial risk exposure will argue for settlement of the case. The practitioner may or may not be able to participate in this decision depending upon the insurance policy. Settled cases are reported to the National Practitioner Data Bank and become a part of the physician's record. Moreover, malpractice suits, settlements, and judgments must be reported to hospital authorities as part of the credentialing process. When applying for licensure or hospital appointment, all such actions must be reported. Failure to do so can lead to adverse consequences.

The litigation process begins with the delivery of a summons indicating that an action is pending. Once delivered, the anesthesia defendant must contact his or her malpractice insurer/risk management department, who will appoint legal counsel. Counsel for both the plaintiff and defense will identify "independent experts" to review the cases. These "experts" are paid for their time and expenses and can arrive at dramatically different assessments of the case materials. Following review by expert consultants, the plaintiff's counsel may depose the principal actors involved in the case. Providing testimony can be stressful. Generally, one should follow the advice of one's defense attorney. Oftentimes, plaintiff's attorneys will attempt to anger or confuse the deponent,

hoping to provoke a response favorable to the claim. Most defense attorneys will advise their clients to answer questions as literally and simply as possible, without offering extraneous commentary. Should the plaintiff's attorney become abusive, the defense attorney will object for the record. However, depositions, also known as "examinations before trial," are not held in front of a judge (only the attorneys, the deponent, the court reporter[s], and [sometimes] the videographer are present). Obligatory small talk often occurs among the attorneys and the court reporters. This is natural and should not be a source of anxiety for the defendant, because in most localities, the same plaintiff's and defense attorneys see each other regularly.

Following discovery, the insurers, plaintiffs, and defense attorneys will "value" the case and attempt to monetize the damages. Items, such as pain and suffering, loss of consortium with spouses, lost wages, and many other factors, are included in determining what the injury is worth. Also during this period, the defense attorney may petition the court to grant defendants a "summary judgment," dismissing the defendant from the case if there is no evidence of malpractice elicited during the discovery process. At times, the plaintiff's attorneys will dismiss the suit against certain named individuals after they have testified, particularly when their testimony implicates other named defendants.

Settlement negotiations will occur in nearly every action. Juries are unpredictable, and both parties are often hesitant to take a case to trial. There are expenses associated with litigation, and, consequently, both plaintiff and defense attorneys will try to avoid uncertainties. Many anesthesia providers will not want to settle a case because the settlement must be reported. Nonetheless, an award in excess of the insurance policy maximum may (depending on the jurisdiction) place the personal assets of the defendant providers at risk. This underscores the importance of our advice to all practitioners (not only those involved in a lawsuit) to assemble their personal assets (house, retirement fund, etc.) in a fashion that makes personal asset confiscation difficult in the event of a negative judgment. One should remember that an adverse judgment may arise from a case in which most anesthesiologists would find the care to meet acceptable standards!

When a case proceeds to trial, the first step is jury selection in the process of *voir dire*—from the French—"to see, to say." In this process, attorneys for the plaintiff and defendant will use various profiling techniques to attempt to identify (and remove) jurors who are less likely to be sympathetic to their case, while keeping the jurors deemed most likely to favor their side. Each attorney is able to strike a certain number of jurors from the pool because they perceive an inherent bias. The jurors will be questioned about such matters as their educational level, history of litigation themselves, professions, and so forth.

Following empanelment, the case is presented to the jury. Each attorney attempts to educate the jurors—who usually have limited knowledge of healthcare (physicians and nurses will usually be struck from the jury)—as to the standard of care for this or that procedure and how the defendants did or did not breach their duty to the patient to uphold those standards. Expert witnesses will attempt to define what the standard of care is for the community, and the plaintiff and defendant will present experts with views that are favorable to their respective cause. The attorneys will attempt to discredit the opponent's experts and challenge their opinions. Exhibits are often used to explain to the jury what should or should not have happened and why the injuries for which damages are being sought were caused by the practitioner's negligence.

After the attorneys conclude their closing remarks, the judge will "charge" the jurors with their duty and will delineate what they can consider in making their judgment. Once a case is in the hands of a jury, anything can happen. Many cases will settle during the course of the trial, as neither party wishes to be subject to the arbitrary decisions of an unpredictable jury. Should the case not settle, the jurors will reach a verdict. When a jury determines that the defendants were negligent and negligence was the cause of the plaintiff's injuries, the jury will determine an appropriate award. If the award is so egregiously large that it is inconsistent with awards for similar injuries, the judge may reduce its amount. Of course, following any verdict, there are

numerous appeals that may be filed. It is important to note that appeals typically do not relate to the medical aspects of the case, but are filed because the trial process itself was somehow flawed.

Unfortunately, a malpractice action can take years to reach a conclusion. Consultation with a mental health professional may be appropriate for the defendant when the litigation process results in unmanageable stress, depression, increased alcohol consumption, or substance abuse.

Determining what constitutes the "standard of care" is increasingly complicated. In the United Sates, the definition of "standard of care" is made separately by each state. The standard of care is NOT necessarily "best practices" or even the care that another physician would prefer. Generally, the standard of care is met when a patient receives care that other reasonable physicians in similar circumstances would regard as adequate. The American Society of Anesthesiologists (ASA) has published standards, and these provide a basic framework for routine anesthetic practice (eg, monitoring). Increasingly, a number of "guidelines" have been developed by the multiple specialty societies to identify best practices in accordance with assessments of the evidence in the literature. The increasing number of guidelines proffered by the numerous anesthesia and other societies and their frequent updating can make it difficult for clinicians to stay abreast of the changing nature of practice. This is a particular problem when two societies produce conflicting guidelines on the same topic using the same data. Likewise, the information upon which guidelines are based can range from randomized clinical trials to the opinion of "experts" in the field. Consequently, guidelines do not hold the same weight as standards. Guidelines produced by reputable societies will generally include an appropriate disclaimer based on the level of evidence used to generate the guideline. Nonetheless, plaintiff's attorneys will attempt to use guidelines to establish a "standard of care," when, in fact, clinical guidelines are prepared to assist in guiding the delivery of therapy. However, if deviation from guidelines is required for good patient care, the rationale for such actions should be documented on the anesthesia record, as plaintiff's attorneys will attempt to use the guideline as a *de facto* standard of care.

ADVERSE ANESTHETIC OUTCOMES

Incidence

There are several reasons why it is difficult to accurately measure the incidence of adverse anesthesia-related outcomes. First, it is often difficult to determine whether the cause of a poor outcome is the patient's underlying disease, the surgical procedure, or the anesthetic management. In some cases, all three factors contribute to a poor outcome. Clinically important measurable outcomes are relatively rare after elective anesthetics. For example, death is a clear endpoint, and perioperative deaths do occur with some regularity. But, because deaths attributable to anesthesia are much rarer, a very large series of patients must be studied to assemble conclusions that have statistical significance. Nonetheless, many studies have attempted to determine the incidence of complications due to anesthesia. Unfortunately, studies vary in criteria for defining an anesthesia-related adverse outcome and are limited by retrospective analysis.

Perioperative mortality is usually defined as death within 48 hr of surgery. It is clear that most perioperative fatalities are due to the patient's preoperative disease or the surgical procedure. In a study conducted between 1948 and 1952, anesthesia mortality in the United States was approximately 5100 deaths per year or 3.3 deaths per 100,000 population. A review of cause of death files in the United States showed that the rate of anesthesia-related deaths was 1.1/1,000,000 population or 1 anesthetic death per 100,000 procedures between 1999 and 2005 (**Figure 54–1**). These results suggest a 97% decrease in anesthesia mortality since the 1940s. However, a 2002 study reported an estimated rate of 1 death per 13,000 anesthetics. Due to differences in methodology, there are discrepancies in the literature as to how well anesthesiology is doing in achieving safe practice. In a 2008 study of 815,077 patients (ASA class 1, 2, or 3) who underwent elective surgery at US Department of Veterans Affairs hospitals, the mortality rate was 0.08% on the day of surgery. The strongest association with perioperative death was the type of surgery (**Figure 54–2**). Other factors associated with increased risk of death

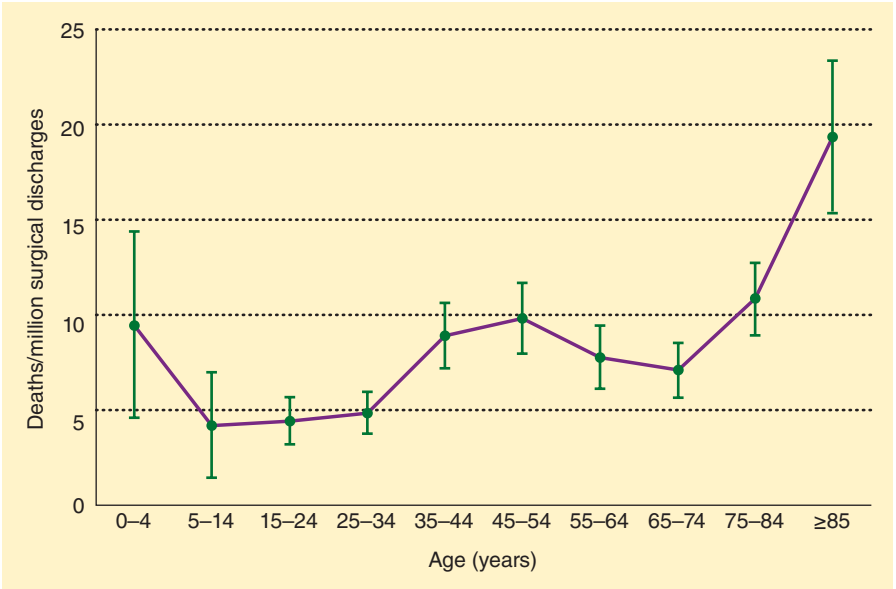

FIGURE 54–1 Annual in-hospital anesthesia-related deaths rates per million hospital surgical discharges and 95% confidence intervals by age, United States, 1999-2005. (Reproduced, with permission, from Li G, Warner M, Lang B, et al: Epidemiology of anesthesia-related mortality in the United States 1999-2005. Anesthesiology 2009;110:759.)

included dyspnea, reduced albumin concentrations, increased bilirubin, and increased creatinine concentrations. A subsequent review of the 88 deaths that occurred on the surgical day noted that 13 of the patients might have benefitted from better anesthesia care, and estimates suggest that death might have been prevented by better anesthesia practice in 1 of 13,900 cases. Additionally, this study reported

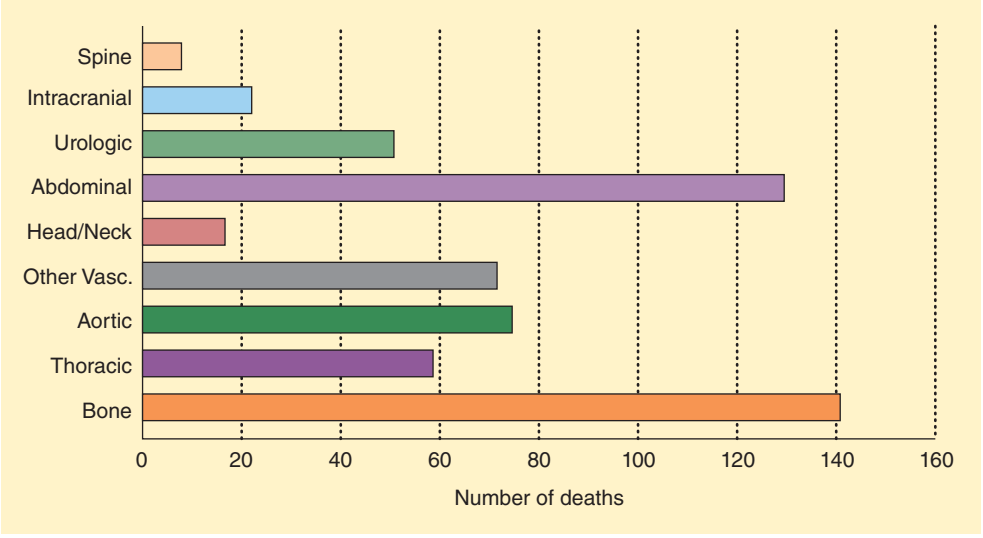

FIGURE 54–2 Total number of deaths by type of surgery in Veterans Affairs hospitals. (Reproduced, with permission, from Bishop M, Souders J, Peterson C, et al: Factors associated with unanticipated day of surgery deaths in Department of Veterans Affairs hospitals. Anesth Analg 2008;107:1924.)

that the immediate postsurgical period tended to be the time of unexpected mortality. Indeed, often missed opportunities for improved anesthetic care occur following complications when "failure to rescue" contributes to patient demise.

American Society of Anesthesiologists Closed Claims Project

The goal of the ASA Closed Claims Project is to identify common events leading to claims in anesthesia, patterns of injury, and strategies for injury prevention. It is a collection of closed malpractice claims that provides a "snapshot" of anesthesia liability rather than a study of the incidence of anesthetic complications, as only events that lead to the filing of a malpractice claim are considered. The Closed Claims Project consists of trained physicians who review claims against anesthesiologists represented by some US malpractice insurers. The number of claims in the database continues to rise each year as new claims are closed and reported. The claims are grouped according to specific damaging events and complication type. Closed Claims Project analyses have been reported for airway injury, nerve injury, awareness, and so forth. These analyses provide insights into the circumstances that result in claims; however, the incidence of a complication cannot be determined from closed claim data, because we know neither the actual incidence of the complication (some with the complication may not file suit), nor how many anesthetics were performed for which the particular complication might possibly develop. Other similar analyses have been performed in the United Kingdom, where National Health Service (NHS) Litigation Authority claims are reviewed.

Causes

3 Anesthetic mishaps can be categorized as preventable or unpreventable. Examples of the latter include sudden death syndrome, fatal idiosyncratic drug reactions, or any poor outcome that occurs despite proper management. However, studies of anesthetic-related deaths or near misses suggest that many accidents are preventable. Of these preventable incidents, most involve human error (Table 54–1), as opposed to equipment malfunctions (Table 54–2). Unfortunately, some rate of human error is inevitable, and a preventable accident is not necessarily evidence of incompetence. During the 1990s, the top three causes for claims in the ASA Closed Claims Project were death (22%), nerve injury (18%), and brain damage (9%). In a 2009 report based on an analysis of NHS litigation records, anesthesia-related claims accounted for 2.5% of total claims filed and 2.4% of the value of all NHS claims. Moreover, regional and obstetrical anesthesia were responsible for 44% and 29%, respectively, of anesthesia-related claims filed. The authors of the latter study noted that there are two ways to examine data related to patient harm: critical incident and closed claim analyses. Clinical (or critical) incident data consider events that either cause harm or result in a "near-miss." Comparison between clinical incident datasets and closed claims analyses demonstrates that not all critical events generate claims and that claims may be filed in the absence of negligent care. Consequently, closed claims reports must always be considered in this context.

TABLE 54–1 Human errors that may lead to preventable anesthetic accidents.

Unrecognized breathing circuit disconnection
Mistaken drug administration
Airway mismanagement
Anesthesia machine misuse
Fluid mismanagement
Intravenous line disconnection

TABLE 54–2 Equipment malfunctions that may lead to preventable anesthetic accidents.

Breathing circuit
Monitoring device
Ventilator
Anesthesia machine
Laryngoscope

MORTALITY AND BRAIN INJURY

Trends in anesthesia-related death and brain damage have been tracked for many years. In a Closed Claims Project report examining claims in the

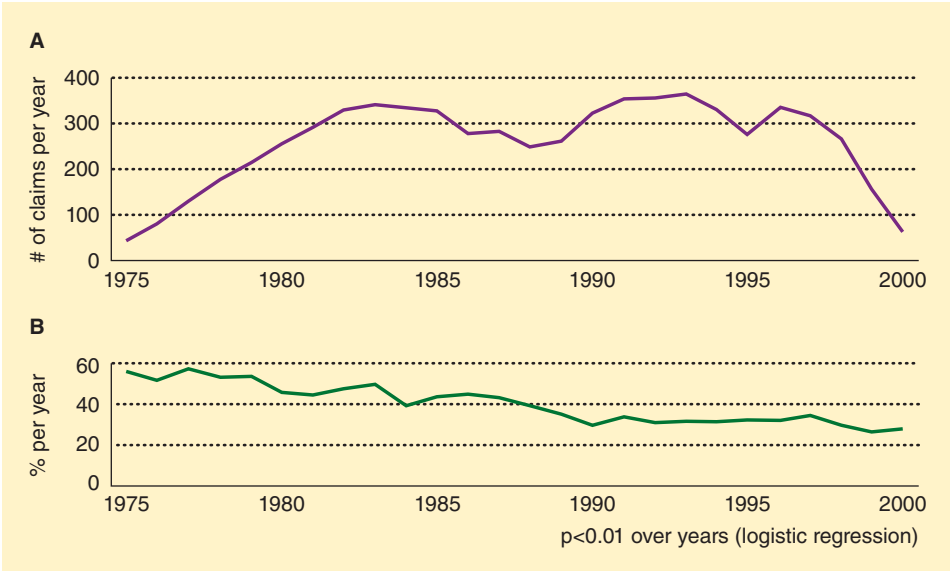

FIGURE 54–3 **A:** The total number of claims by the year of injury. Retrospective data collection began in 1985. Data in this analysis includes data collected through December 2003. **B:** Claims for death or permanent brain damage as percentage of total claims per year by year of injury. (Reproduced, with permission, from Cheney FW, Domino KB, Caplan RA, Posner KL: Nerve injury associated with anesthesia: a closed claims analysis. Anesthesiology 1999;90:1062.)

period between 1975 and 2000, there were 6750 claims (Figure 54–3A and B), 2613 of which were for brain injury or death. The proportion of claims for brain injury or death was 56% in 1975, but had decreased to 27% by 2000. The primary pathological mechanisms by which these outcomes occurred were related to cardiovascular or respiratory problems. Early in the study period, respiratory-related damaging events were responsible for more than 50% of brain injury/death claims, whereas cardiovascular-related damaging events were responsible for 27% of such claims; however, by the late 1980s, the percentage of damaging events related to respiratory issues had decreased, with both respiratory and cardiovascular events being equally likely to contribute to severe brain injury or death. Respiratory damaging events included difficult airway, esophageal intubation, and unexpected extubation. Cardiovascular damaging events were usually multifactorial. Closed claims reviewers found that anesthesia care was substandard in 64% of claims in which respiratory complications contributed to brain injury or death, but in only 28% of cases in which the primary mechanism of patient injury was cardiovascular in nature. Esophageal intubation, premature extubation, and inadequate ventilation were the primary mechanisms by which less than optimal anesthetic care was thought to have contributed to patient injury related to respiratory events. The relative decrease in causes of death being attributed to respiratory rather cardiovascular damaging events during the review period was attributed to the increased use of pulse oximetry and capnometry. Consequently, if expired gas analysis was judged to be adequate, and a patient suffered brain injury or death, a cardiovascular event was more likely to be considered causative.

A 2010 study examining the NHS Litigation Authority dataset noted that airway-related claims led to higher awards and poorer outcomes than did nonairway-related claims. Indeed, airway manipulation and central venous catheterization claims in this database were most associated with patient death. Trauma to the airway also generates significant claims if esophageal or tracheal rupture occur. Postintubation mediastinitis should always

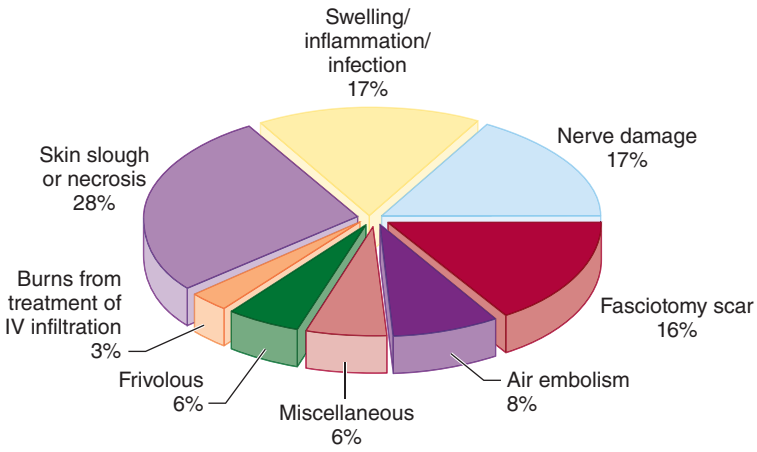

FIGURE 54–4 Injuries related to IV catheters (n = 127). (Reproduced, with permission, from Bhananker S, Liau D, Kooner P, et al: Liability related to peripheral venous and arterial catheterization: a closed claims analysis. Anesth Analg 2009;109:124.)

be considered whenever there are repeated unsuccessful airway manipulations, as early intervention presents the best opportunity to mitigate any injuries incurred.

VASCULAR CANNULATION

Claims related to central venous access in the ASA database were associated with patient death 47% of the time and represented 1.7% of the 6449 claims reviewed. Complications secondary to guidewire or catheter embolism, tamponade, bloodstream infections, carotid artery puncture, hemothorax, and pneumothorax all contributed to patient injury. Although guidewire and catheter embolisms were associated with generally lower level patient injuries, these complications were generally attributed to substandard care. Tamponade claims following line placement were often for patient death. The authors of a 2004 closed claims analysis recommended reviewing the chest radiograph following line placement and repositioning lines found in the heart or at an acute angle to reduce the likelihood of tamponade. Brain damage and stroke are associated with claims secondary to carotid cannulation. Multiple confirmatory methods should be used to ensure that the internal jugular and not the carotid artery is cannulated.

Claims related to peripheral vascular cannulation in the ASA database accounted for 2% of 6849 claims, 91% of which were for complications secondary to the extravasation of fluids or drugs from peripheral intravenous catheters that resulted in extremity injury (Figure 54–4). Air embolisms, infections, and vascular insufficiency secondary to arterial spasm or thrombosis also resulted in claims. Of interest, intravenous catheter claims in patients who had undergone cardiac surgery formed the largest cohort of claims related to peripheral intravenous catheters, most likely due to the usual practice of tucking the arms alongside the patient during the procedure, placing them out of view of the anesthesia providers. Radial artery catheters seem to generate few closed claims; however, femoral artery catheters can lead to greater complications and potentially increased liability exposure.

OBSTETRIC ANESTHESIA

Both critical incident and closed claims analyses have been reported regarding complications and mortality related to obstetrical anesthesia.

In a study reviewing anesthesia-related maternal mortality in the United States using the Pregnancy Mortality Surveillance System, which collects data on all reported deaths causally related to pregnancy, 86 of the 5946 pregnancy-related deaths reported to the Centers for Disease Control were thought to be anesthesia related or approximately 1.6% of total pregnancy related-deaths in the period 1991–2002. The anesthesia mortality rate in this period was 1.2 per million live births, compared with 2.9 per million live births in the

period 1979–1990. The decline in anesthesia-related maternal mortality may be secondary to the decreased use of general anesthesia in parturients, reduced concentrations of bupivacaine in epidurals, improved airway management protocols and devices, and greater use of incremental (rather than bolus) dosing of epidural catheters.

In a 2009 study examining the epidemiology of anesthesia-related complications in labor and delivery in New York state in the period 2002–2005, an anesthesia-related complication was reported in 4438 of 957,471 deliveries (0.46%). The incidence of complications was increased in patients undergoing cesarean section, those living in rural areas, and those with other medical conditions. Complications of neuraxial anesthesia (eg, postdural puncture headache) were most common, followed by systemic complications, including aspiration or cardiac events. Other reported problems related to anesthetic dose administration and unintended overdosages. African American women and those aged 40–55 years were more likely to experience systemic complications, whereas Caucasian women and those aged 30–39 were more likely to experience complications related to neuraxial anesthesia.

ASA Closed Claims Project analyses were reported in 2009 for the period 1990–2003. Four hundred twenty-six claims from this period were compared with 190 claims in the database prior to 1990. After 1990, the proportion of claims for maternal or fetal demise was lower than that recorded prior to 1990. After 1990, the number of claims for maternal nerve injury increased. In the review of claims in which anesthesia was thought to have contributed to the adverse outcome, anesthesia delay, poor communication, and substandard care were thought to have resulted in poor newborn outcomes. Prolonged attempts to secure neuraxial blockade in the setting of emergent cesarean section can contribute to adverse fetal outcome. Additionally, the closed claims review indicated that poor communication between the obstetrician and the anesthesiologist regarding the urgency of newborn delivery was likewise thought to have contributed to newborn demise and neonatal brain injury.

Maternal death claims were secondary to airway difficulty, maternal hemorrhage, and high neuraxial blockade. The most common claim associated with obstetrical anesthesia was related to nerve injury following regional anesthesia. Nerve injury can be secondary to neuraxial anesthesia and analgesia, but also due to obstetrical causes. Early neurological consultation to identify the source of nerve injury is suggested to discern if injury could be secondary to obstetrical rather than anesthesia interventions.

REGIONAL ANESTHESIA

In a closed claims analysis, peripheral nerve blocks were involved in 159 of the 6894 claims analyzed. Peripheral nerve block claims were for death (8%), permanent injuries (36%), and temporary injuries (56%). The brachial plexus was the most common location for nerve injury. In addition to ocular injury, cardiac arrest following retrobulbar block contributed to anesthesiology claims. Cardiac arrest and epidural hematomas are two of the more common damaging events leading to severe injuries related to regional anesthesia. Neuraxial hematomas in both obstetrical and nonobstetrical patients were associated with coagulopathy (either intrinsic to the patient or secondary to medical interventions). In one study, cardiac arrest related to neuraxial anesthesia contributed to roughly one-third of the death or brain damage claims in both obstetrical and nonobstetrical patients. Accidental intravenous injection and local anesthesia toxicity also contributed to claims for brain injury or death.

Nerve injuries constitute the third most common source of anesthesia litigation. A retrospective review of patient records and a claims database showed that 112 of 380,680 patients (0.03%) experienced perioperative nerve injury. Patients with hypertension and diabetes and those who were smokers were at increased risk of developing perioperative nerve injury. Perioperative nerve injuries may result from compression, stretch, ischemia, other traumatic events, and unknown causes. Improper positioning can lead to nerve compression, ischemia, and injury, however not every nerve injury is the result of improper positioning. The care received by patients with ulnar nerve injury was rarely judged to be inadequate in the ASA Closed Claims database. Even awake patients undergoing

spinal anesthesia have been reported to experience upper extremity injury. Moreover, many peripheral nerve injuries do not become manifest until more than 48 hr after anesthesia and surgery, suggesting that some nerve damage that occurs in surgical patients may arise from events taking place after the patient leaves the operating room setting.

PEDIATRIC ANESTHESIA

In a 2007 study reviewing 532 claims in pediatric patients aged <16 years in the ASA Closed Claims database from 1973–2000 (**Figure 54–5**), a decrease in the proportion of claims for death and brain damage was noted over the three decades. Likewise, the percentage of claims related to respiratory events also was reduced. Compared with before 1990, the percentage of claims secondary to respiratory events decreased during the years 1990–2000, accounting for only 23 % of claims in the latter study years compared with 51% of claims in the 1970s. Moreover, the percentage of claims that could be avoided by better monitoring decreased from 63% in the 1970s to 16% in the 1990s. Death and brain damage constitute the major complications for which claims are filed. In the 1990s, cardiovascular events joined respiratory complications in sharing the primary causes of pediatric anesthesia litigation. In the study mentioned above, better monitoring and newer airway management techniques may have reduced the incidence of respiratory events leading to litigation-generating complications in the latter years of the review period. Additionally, the possibility of a claim being filed secondary to death or brain injury is greater in children who are in ASA classes 3, 4, or 5.

In a review of the Pediatric Perioperative Cardiac Arrest Registry, which collects information from about 80 North American institutions that provide pediatric anesthesia, 193 arrests were reported in children between 1998 and 2004. During the study period, 18% of the arrests were "drug related," compared with 37% of all arrests during the years 1994–1997. Cardiovascular arrests occurred most often (41%), with hypovolemia and hyperkalemia being the most common causes. Respiratory arrests (27%) were most commonly associated with laryngospasm. Central venous catheter placement with resultant vascular injury also contributed to some perioperative arrests. Arrests from cardiovascular causes occurred most frequently during surgery, whereas arrests from respiratory causes tended to occur after surgery. The reduced use of halothane seems to have decreased the incidence of arrests secondary to medication administration. However, hyperkalemia and electrolyte disturbances

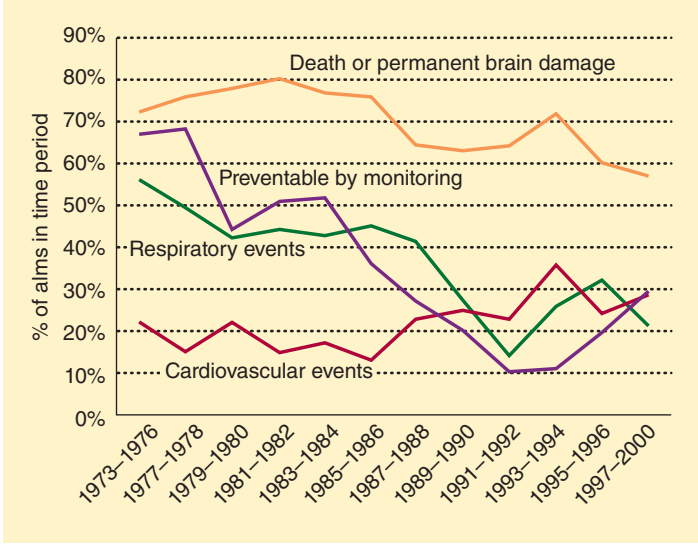

FIGURE 54–5 Trends over time. Outcome, type of event, and prevention by better monitoring. Years are grouped for illustration. (Reproduced, with permission, from Jimenez N, Posner K, Cheney F, et al: An update on pediatric anesthesia liability: a closed claims analysis. Anesth Analg 2007;104:147.)

associated with transfusion and hypovolemia also contribute to sources of cardiovascular arrest in children perioperatively.

A review of data from the Pediatric Perioperative Cardiac Arrest Registry with a focus on children with congenital heart disease found that such children were more likely to arrest perioperatively secondary to a cardiovascular cause. In particular, children with a single ventricle were at increased risk of perioperative arrest. Children with aortic stenosis and cardiomyopathy were similarly found to be at increased risk of cardiac arrest perioperatively.

OUT OF THE OPERATING ROOM ANESTHESIA AND MONITORED ANESTHESIA CARE

Review of the ASA Closed Claims Project database indicates that anesthesia at remote (out of the operating room) locations poses a risk to patients secondary to hypoventilation and excessive sedation. Remote location anesthesia care was more likely than operating room anesthesia care to involve a claim for death (54% vs 29%, respectively). The endoscopy suite and cardiac catheterization laboratory were the most frequent locations from which claims were generated. Monitored Anesthesia Care (MAC) was the most common technique employed in these claims. Overwhelmingly, adverse respiratory events were most frequently responsible for the injury.

An analysis of the ASA Closed Claims Project database focusing on MAC likewise revealed that oversedation and respiratory collapse most frequently lead to claims. Claims for burn injuries suffered in operating room fires were also found in the database. Supplemental oxygen, draping, pooling of flammable antiseptic preparatory solutions, and surgical cautery combine to produce the potential for operating room fires.

EQUIPMENT PROBLEMS

"Equipment problems" is probably a misnomer; the ASA Closed Claims Project review of 72 claims involving gas delivery systems found that equipment *misuse* was three times more common than equipment *malfunction*. The majority (76%) of adverse outcomes associated with gas delivery problems were either death or permanent neurological damage.

TABLE 54–3 Factors associated with human errors and equipment misuse.

Factor	Example
Inadequate preparation	No machine checkout or preoperative evaluation; haste and carelessness; production pressure
Inadequate experience and training	Unfamilarity with anesthetic technique or equipment
Environmental limitations	Inability to visualize surgical field: poor communication with surgeons
Physical and emotional factors	Fatigue; distraction caused by personal problems

Errors in drug administration also typically involve human error. It has been suggested that as many as 20% of the drug doses given to hospitalized patients are incorrect. Errors in drug administration account for 4% of cases in the ASA Closed Claims Project, which found that errors resulting in claims were most frequently due to either incorrect dosage or unintentional administration of the wrong drug (syringe swap). In the latter category, accidental administration of epinephrine proved particularly dangerous.

Another type of human error occurs when the most critical problem is ignored because attention is inappropriately focused on a less important problem or an incorrect solution (fixation error). Serious anesthetic mishaps are often associated with distractions and other factors (Table 54–3). The impact of most equipment failures is decreased or avoided when the problem is identified during a routine preoperative checkout performed by adequately trained personnel. Many anesthetic fatalities occur only after a series of coincidental circumstances, misjudgments, and technical errors coincide (mishap chain).

Prevention

Strategies to reduce the incidence of serious anesthetic complications include better monitoring and anesthetic techniques, improved education, more

comprehensive protocols and standards of practice, and active risk management programs. Better monitoring and anesthetic techniques imply more comprehensive monitoring and ongoing patient assessments and better designed anesthesia equipment and workspaces. The fact that most accidents occur during the maintenance phase of anesthesia—rather than during induction or emergence—implies a failure of vigilance.

Inspection, palpation, percussion, and auscultation of the patient provide important information. Instruments should supplement (but never replace) the anesthesiologist's own senses. To minimize errors in drug administration, drug syringes and ampoules in the workspace should be restricted to those needed for the current specific case. Drugs should be consistently diluted to the same concentration in the same way for each use, and they should be clearly labeled. Computer systems for scanning bar-coded drug labels are available that may help to reduce medication errors. The conduct of all anesthetics should follow a predictable pattern by which the anesthetist actively surveys the monitors, the surgical field, and the patient on a recurrent basis. In particular, patient positioning should be frequently reassessed to avoid the possibility of compression or stretch injuries. When surgical necessity requires patients to be placed in positions where harm may occur or when hemodynamic manipulations (eg, deliberate hypotension) are requested or required, the anesthesiologist should note on the record the surgical request and remind the surgeon of any potential risks to the patient.

QUALITY MANAGEMENT

Risk management and continuous quality improvement programs at the departmental level may reduce anesthetic morbidity and mortality rates by addressing monitoring standards, equipment, practice guidelines, continuing education, quality of care, and staffing issues. Specific responsibilities of peer review committees include identifying (and, ideally, preventing) potential problems, formulating and periodically revising departmental policies, ensuring the availability of properly functioning anesthetic equipment, enforcing standards required for clinical privileges, and evaluating the appropriateness and quality of patient care. A quality improvement system impartially and continuously reviews complications, compliance with standards, and quality indicators.

AIRWAY INJURY

The daily insertion of endotracheal tubes, laryngeal mask airways, oral/nasal airways, gastric tubes, transesophageal echocardiogram (TEE) probes, esophageal (bougie) dilators, and emergency airways all involve the risk of airway structure damage. Common morbid complaints, such as sore throat and dysphagia, are usually self-limiting, but may also be nonspecific symptoms of more ominous complications.

The most common persisting airway injury is dental trauma. In a retrospective study of 600,000 surgical cases, the incidence of injury requiring dental intervention and repair was approximately 1 in 4500. In most cases, laryngoscopy and endotracheal intubation were involved, and the upper incisors were the most frequently injured. Major risk factors for dental trauma included tracheal intubation, preexisting poor dentition, and patient characteristics associated with difficult airway management (including limited neck motion, previous head and neck surgery, craniofacial abnormalities, and a history of difficult intubation).

Other types of airway trauma are rare. Although there are scattered case reports in the literature, the most comprehensive analysis was performed by the ASA Closed Claims Project. This report describes 266 claims, of which the least serious were temporomandibular joint (TMJ) injuries that were all associated with otherwise uncomplicated intubations and occurred mostly in females younger than age 60 years. Approximately 25% of these patients had previous TMJ disease. Laryngeal injuries included vocal cord paralysis, granuloma, and arytenoid dislocation. Most tracheal injuries were associated with emergency surgical tracheotomy, but a few were related to endotracheal intubation. Some injuries occurred during seemingly easy, routine intubations. Proposed mechanisms include excessive tube movement in the trachea, excessive cuff inflation

leading to pressure necrosis, and inadequate relaxation. Esophageal perforations contributed to death in 5 of 13 patients. Esophageal perforation often presents with delayed-onset subcutaneous emphysema or pneumothorax, unexpected febrile state, and sepsis. Pharyngoesophageal perforation is associated with difficult intubation, age over 60 years, and female gender. As in tracheal perforation, signs and symptoms are often delayed in onset. Initial sore throat, cervical pain, and cough often progressed to fever, dysphagia, and dyspnea, as mediastinitis, abscess, or pneumonia develop. Mortality rates of up to 50% have been reported after esophageal perforation, with better outcomes attributable to rapid detection and treatment.

Minimizing the risk of airway injury begins with the preoperative assessment. A thorough airway examination will help to determine the risk for difficulty Documentation of current dentition (including dental work) should be included. Many practitioners believe preoperative consent should include a discussion of the risk of dental, oral, vocal cord, and esophageal trauma in every patient who could potentially need any airway manipulation. If a difficult airway is suspected, a more detailed discussion of risks (eg, emergency tracheotomy) is appropriate. In such cases, emergency airway supplies and experienced help should be available. The ASA algorithm for difficult airway management is a useful guide. After a difficult intubation, one should seek latent signs of esophageal perforation and have an increased level of suspicion for airway trauma. When intubation cannot be accomplished by routine means, the patient or guardian should be informed to alert future anesthesia providers of potential airway difficulty.

Emergent nonoperating room intubations present unique challenges. In a review of 3423 out of the operating room intubations, 10% were considered to be "difficult," and 4% of these intubations were associated with some form of complication, including aspiration, esophageal intubation, or dental injury. In this report, intubation bougies were employed in 56% of difficult intubations. The increased availability of video laryngoscopes and bougies have made emergent intubations less stressful and less likely to be unsuccessful.

PERIPHERAL NERVE INJURY

Nerve injury is a complication of being hospitalized, with or without surgery, regional, or general anesthesia. Peripheral nerve injury is a frequent and vexing problem. In most cases, these injuries resolve within 6–12 weeks, but some are permanent. Because peripheral neuropathies are commonly associated (often incorrectly!) with failures of patient positioning, a review of mechanisms and prevention is necessary.

The most commonly injured peripheral nerve is the ulnar nerve (**Figure 54–6**). In a retrospective study of over 1 million patients, ulnar neuropathy (persisting for more than 3 months) occurred in approximately 1 in 2700 patients. Of interest, initial symptoms were most frequently noted more than 24 hr after a surgical procedure. Risk factors included male gender, hospital stay greater than 14 days, and very thin or obese body habitus. More than 50% of these patients regained full sensory and motor function within 1 yr. Anesthetic technique was not implicated as a risk factor; 25% of patients with ulnar neuropathy underwent monitored care or lower extremity regional technique. The ASA Closed Claims Project findings support most of these results, including the delayed onset of symptoms and the lack of relationship between anesthesia technique and injury. This study also noted that many neuropathies occurred despite notation of extra padding over the elbow area, further negating compression as a possible mechanism of injury. Finally, the ASA Closed Claims Project investigators found no deviation from the standard of care in the majority of patients who manifested nerve damage perioperatively.

The Role of Positioning

Other peripheral nerve injuries seem to be more closely related to positioning or surgical procedure. They may involve the peroneal nerve, the brachial plexus, or the femoral and sciatic nerves. External pressure on a nerve could compromise its perfusion, disrupt its cellular integrity, and eventually result in edema, ischemia, and necrosis. Pressure injuries are particularly likely when nerves pass through closed compartments or take a superficial course (eg, the

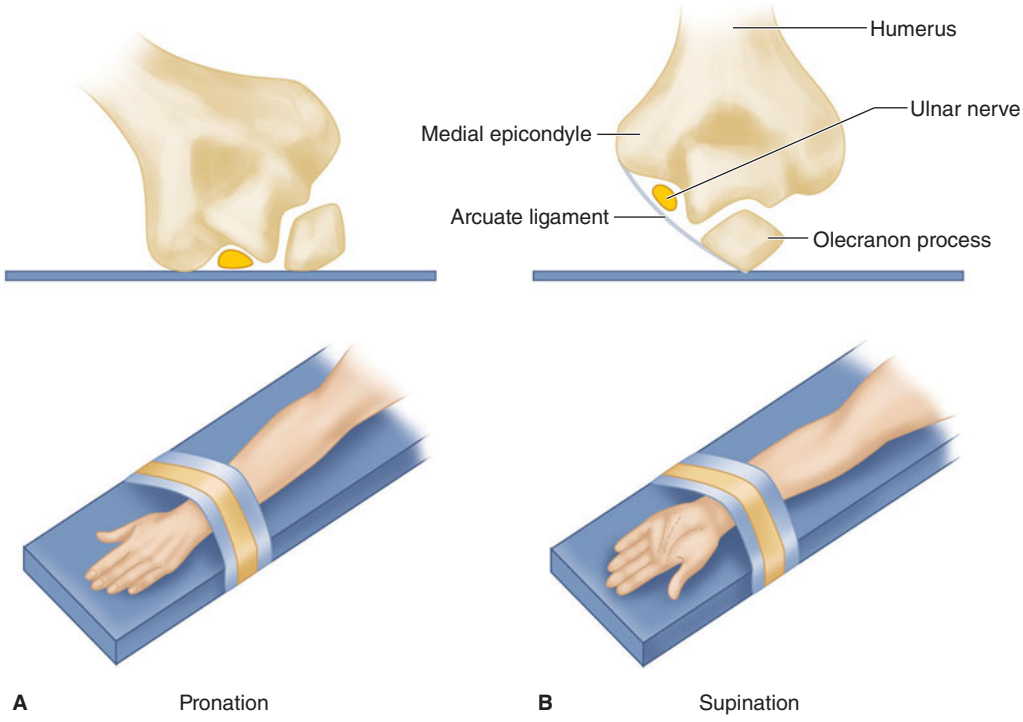

FIGURE 54–6 **A:** Pronation of the forearm can cause external compression of the ulnar nerve in the cubital tunnel. **B:** Forearm supination avoids this problem.

(Modified and reproduced, with permission, from Wadsworth TG: The cubital tunnel and the external compression syndrome. Anesth Analg 1974;53:303.)

peroneal nerve around the fibula). Lower extremity neuropathies, particularly those involving the peroneal nerve, have been associated with such factors as extreme degrees (high) and prolonged (greater than 2 h) durations of the lithotomy position. But, these nerve injuries also sometimes occur when such conditions are not present. Other risk factors for lower extremity neuropathy include hypotension, thin body habitus, older age, vascular disease, diabetes, and cigarette smoking. An axillary (chest) "roll" is commonly used to reduce pressure on the inferior shoulder of patients in the lateral decubitus position. This roll should be located caudad to the axilla to prevent direct pressure on the brachial plexus and large enough to relieve any pressure from the mattress on the lower shoulder.

The data are convincing that some peripheral nerve injuries are not preventable. The risk of peripheral neuropathy should be included in discussions leading to informed consent. When reasonable, patients with contractures (or other causes of limited flexibility) can be positioned before induction of anesthesia to check for feasibility and discomfort. Final positioning should be evaluated prior to draping. In most circumstances, the head and neck should be kept in a neutral position. Shoulder braces to support patients maintained in a Trendelenberg position should be avoided if possible, and shoulder abduction and lateral rotation should be minimized. The upper extremities should not be extended greater than 90° at any joint. (There should be no continuous external compression on the knee, ankle, or heel.) Although injuries may still occur, additional padding may be helpful in vulnerable areas. Documentation should include information on positioning, including the presence of padding. Finally, patients who complain of sensory or motor dysfunction in the postoperative period should be

reassured that this is usually a temporary condition. Motor and sensory function should be documented. When symptoms persist for more than 24 hr, the patient should be referred to a neurologist (or a physiatrist or hand surgeon) who is knowledgeable about perioperative nerve damage for evaluation. Physiological testing, such as nerve conduction and electromyographic studies, can be useful to document whether nerve damage is a new or chronic condition. In the latter case, fibrillations will be observed in chronically denervated muscles.

Complications Related to Positioning

Changes of body position have physiological consequences that can be exaggerated in disease states. General and regional anesthesia may limit the cardiovascular response to such a change. Even positions that are safe for short periods may eventually lead to complications in persons who are not able to move in response to pain. For example, the alcoholic patient who passes out on a hard floor or a park bench may awaken with a brachial plexus injury. Similarly, regional and general anesthesia abolish protective reflexes and predispose patients to injury.

Complications of postural hypotension, the most common physiological consequence of positioning, can be minimized by avoiding abrupt or extreme position changes (eg, sitting up quickly), reversing the position if vital signs deteriorate, keeping the patient well hydrated, and having vasoactive drugs available to treat hypotension. Whereas maintaining a reduced level of general anesthesia will decrease the likelihood of hypotension, light general anesthesia will increase the likelihood that movement of the endotracheal tube during positioning will cause the patient to cough and become hypertensive.

Many complications, including air embolism, blindness from sustained pressure on the globe, and finger amputation following a crush injury, can be caused by improper patient positioning (Table 54–4). These complications are best prevented by evaluating the patient's postural limitations during the preanesthetic visit; padding pressure points, susceptible nerves, and any area of the body that will *possibly* be in contact with the operating table or its attachments; avoiding flexion or extension of a joint to its limit; having an awake patient assume the position

TABLE 54–4 Complications associated with patient positioning.

Complication	Position	Prevention
Venous air embolism	Sitting, prone, reverse Trendelenburg	Maintain adequate venous pressure; ligate "open" veins
Alopecia	Supine, lithotomy, Trendelenburg	Avoid prolonged hypotension, padding, and occasional head turning.
Backache	Any	Lumbar support, padding, and slight hip flexion.
Extremity compartment syndromes	Especially lithotomy	Maintain perfusion pressure and avoid external compression.
Corneal abrasion	Any, but especially prone	Taping and/or lubricating eye.
Digit amputation	Any	Check for protruding digits before changing table configuration.
Nerve palsies		
Brachial plexus	Any	Avoid stretching or direct compression at neck, shoulder, or axilla.
Common peroneal	Lithotomy, lateral decubitus	Avoid sustained pressure on lateral aspect of upper fibula.
Radial	Any	Avoid compression of lateral humerus.
Ulnar	Any	Avoid sustained pressure on ulnar groove.
Retinal ischemia	Prone, sitting	Avoid pressure on globe.
Skin necrosis	Any	Avoid sustained pressure over bony prominences.

to ensure comfort; and understanding the potential complications of each position. Monitors must often be disconnected during patient repositioning, making this a time of greater risk for unrecognized hemodynamic derangement.

Compartment syndromes can result from hemorrhage into a closed space following a vascular puncture or prolonged venous outflow obstruction, particularly when associated with hypotension. In severe cases, this may lead to muscle necrosis, myoglobinuria, and renal damage, unless the pressure within the extremity compartment is relieved by surgical decompression (fasciotomy) or in the abdominal compartment by laparotomy.

AWARENESS

A continuing series of media reports have imprinted the fear of awareness under general anesthesia into the psyche of the general population. Accounts of recall and helplessness while paralyzed have made unconsciousness a primary concern of patients undergoing general anesthesia. When unintended intraoperative awareness does occur, patients may exhibit symptoms ranging from mild anxiety to posttraumatic stress disorder (eg, sleep disturbances, nightmares, and social difficulties).

Although the incidence is difficult to measure, approximately 2% of the closed claims in the ASA Closed Claims Project database relate to awareness under anesthesia. Analysis of the NHS Litigation Authority database from 1995–2007 revealed that 19 of 93 relevant claims were for "awake paralysis." Clearly, awareness is of great concern to patients and may lead to litigation. Certain types of surgeries are most frequently associated with awareness, including those for major trauma, obstetrics, and major cardiac procedures. In some instances, awareness may result from the reduced depth of anesthesia that can be tolerated by the patient. In early studies, recall rates for intraoperative events during major trauma surgery have been reported to be as frequent as 43%; the incidence of awareness during cardiac surgery and cesarean sections is 1.5% and 0.4%, respectively. As of 1999, the ASA Closed Claims Project reported 79 awareness claims; approximately 20% of the claims were for awake paralysis, and the remainder of the claims were for recall under general anesthesia. Most claims for awake paralysis were thought to be due to errors in drug labeling and administration, such as administering paralytics before inducing narcosis. Since the 1999 review, another 71 cases have appeared in the database. Claims for recall were more likely in women undergoing general anesthesia without a volatile agent. Patients with long term substance abuse may have increased anesthesia requirements which if not met can lead to awareness.

Other specific causes of awareness include inadequate inhalational anesthetic delivery (eg, from vaporizer malfunction) and medication errors. Some patients may complain of awareness, when, in fact, they received regional anesthesia or monitored anesthesia care; thus, anesthetists should make sure that patients have reasonable expectations when regional or local techniques are employed. Likewise, patients requesting regional or local anesthesia because they want to "see it all" and/ or "stay in control" often can become irate when sedation dulls their memory of the perioperative experience. In all cases, frank discussion between anesthesia staff and the patient is necessary to avoid unrealistic expectations.

Some clinicians routinely discuss the possibility of intraoperative recall and the steps that will be taken to minimize it as part of the informed consent for general anesthesia. This makes particular sense for those procedures in which recall is more likely. It is advisable to also remind patients who are undergoing monitored anesthesia care with sedation that awareness is expected. Volatile anesthetics should be administered at a level consistent with amnesia. If this is not possible, benzodiazepines (and/or scopolamine) can be used. Movement of a patient may indicate inadequate anesthetic depth. Documentation should include end-tidal concentrations of anesthetic gases (when available) and dosages of amnesic drugs. Use of a bispectral index scale (BIS) monitor or similar monitors may be helpful although randomized clinical trials have failed to demonstrate a reduced incidence of awareness with use of BIS when compared with a group receiving appropriate concentrations of volatile agents. Finally, if there is evidence of intraoperative awareness during postoperative rounds, the practitioner should obtain a detailed account of the experience, answer patient

questions, be very empathetic, and refer the patient for psychological counseling if appropriate.

EYE INJURY

A wide range of conditions from simple corneal abrasion to blindness have been reported. Corneal abrasion is by far the most common and transient eye injury. The ASA Closed Claims Project identified a small number of claims for abrasion, in which the cause was rarely identified (20%) and the incidence of permanent injury was low (16%). It also identified a subset of claims for blindness that resulted from patient movement during ophthalmological surgery. These cases occurred in patients receiving either general anesthesia or monitored anesthesia care.

Although the cause of corneal abrasion may not be obvious, securely closing the eye lids with tape after loss of consciousness (but prior to intubation) and avoiding direct contact between eyes and oxygen masks, drapes, lines, and pillows (particularly during monitored anesthesia care, in transport, and in nonsupine positions) can help to minimize the possibility of injury. Adequate anesthetic depth (and, in most cases, paralysis) should be maintained to prevent movement during ophthalmological surgery under general anesthesia. In patients scheduled for MAC, the patient must understand that movement under monitored care is hazardous and, thus, that only minimal sedation may be administered to ensure that he or she can cooperate.

Ischemic optic neuropathy (ION) is a devastating perioperative complication. ION is now the most common cause of postoperative vision loss. Postoperative vision loss is most commonly reported after cardiopulmonary bypass, radical neck dissection, and spinal surgeries in the prone position. Both preoperative and intraoperative factors may be contributory. Many of the case reports implicate preexisting hypertension, diabetes, coronary artery disease, and smoking, suggesting that preoperative vascular abnormalities may play a role. Intraoperative deliberate hypotension and anemia have also been implicated (in spine surgery), perhaps because of their potential to reduce oxygen delivery. Finally, prolonged surgical time in positions that compromise venous outflow (prone, head down, compressed abdomen) have also been found to be factors in spine surgery. Symptoms are usually present immediately upon awakening from anesthesia, but have been reported up to 12 days postoperatively. Such symptoms range from decreased visual acuity to complete blindness. Analysis of case records submitted to the ASA Postoperative Vision Loss Registry revealed that vision loss was secondary to ION in 83 of 93 cases. Instrumentation of the spine was associated with ION when surgery lasted more than 6 hr and blood loss was more than 1 L. ION can occur in patients whose eyes are free of pressure secondary to the use of pin fixation, indicating that direct pressure on the eye is not required to produce ION.

Increased venous pressure in patients in the Trendelenberg position may reduce blood flow to the optic nerve.

It is difficult to formulate recommendations to prevent this complication because risk factors for ION are often unavoidable. Steps that might be taken include: (1) limiting the degree and duration of hypotension during controlled (deliberate) hypotension, (2) administering a transfusion to severely anemic patients who seem to be at risk of ION, and (3) discussing with the surgeon the possibility of staged operations in high-risk patients to limit prolonged procedures.

Of note, postoperative vision loss can be caused by other mechanisms as well, including angle closure glaucoma or embolic phenomenon to the cortex or retina. Immediate evaluation is advised.

CARDIOPULMONARY ARREST DURING SPINAL ANESTHESIA

Sudden cardiac arrest during an otherwise routine administration of spinal anesthetics is an uncommon complication. The initial published report was a closed claims analysis of 14 patients who experienced cardiac arrest during spinal anesthesia. The cases primarily involved young (average age 36 years), relatively healthy (ASA physical status I–II) patients who were given appropriate doses of local anesthetic that produced a high dermatomal level of block prior to arrest (T4 level). Respiratory insufficiency with hypercarbia due to sedatives was thought to be a potential contributing factor. The

average time from induction of spinal anesthesia to arrest was 36 min, and, in all cases, arrest was preceded by a gradual decline in heart rate and blood pressure. Just prior to arrest, the most common signs were bradycardia, hypotension, and cyanosis. Treatment consisted of ventilatory support, ephedrine, atropine, cardiopulmonary resuscitation (average duration 10.9 min), and epinephrine. Despite these interventions, 10 patients remained comatose and 4 patients regained consciousness with significant neurological deficits. A subsequent study concluded that such arrests had little relationship to sedation, but were related more to extensive degrees of sympathetic blockade, leading to unopposed vagal tone and profound bradycardia. Rapid appropriate treatment of bradycardia and hypotension is essential to minimize the risk of arrest. Early treatment of bradycardia with atropine may prevent a downward spiral. Stepwise doses of ephedrine, epinephrine, and other vasoactive drugs should be given to treat hypotension. If cardiopulmonary arrest occurs, ventilatory support, cardiopulmonary resuscitation, and full resuscitation doses of atropine and epinephrine should be administered without delay.

HEARING LOSS

Perioperative hearing loss is usually transient and often goes unrecognized. The incidence of low-frequency hearing loss following dural puncture may be as high as 50%. It seems to be due to cerebrospinal fluid leak, and, if persistent, can be relieved with an epidural blood patch. Hearing loss following general anesthesia can be due to a variety of causes and is much less predictable. Mechanisms include middle ear barotrauma, vascular injury, and ototoxicity of drugs (aminoglycosides, loop diuretics, nonsteroidal antiinflammatory drugs, and antineoplastic agents). Hearing loss following cardiopulmonary bypass is usually unilateral and is thought to be due to embolism and ischemic injury to the organ of Corti.

ALLERGIC REACTIONS

Hypersensitivity (or allergic) reactions are exaggerated immunological responses to antigenic stimulation in previously sensitized persons. The antigen, or allergen, may be a protein, polypeptide, or smaller molecule. Moreover, the allergen may be the substance itself, a metabolite, or a breakdown product. Patients may be exposed to antigens through the respiratory tract, gastrointestinal tract, eyes, skin and from previous intravenous, intramuscular, or peritoneal exposure.

TABLE 54–5 Hypersensitivity reactions.

Type I (immediate)
Atopy
Urticaria—angioedema
Anaphylaxis
Type II (cytotoxic)
Hemolytic transfusion reactions
Autoimmune hemolytic anemia
Heparin-induced thrombocytopenia
Type III (immune complex)
Arthus reaction
Serum sickness
Acute hypersensitivity pneumonitis
Type IV (delayed, cell-mediated)
Contact dermatitis
Tuberculin-type hypersensitivity
Chronic hypersensitivity pneumonitis

Anaphylaxis occurs when inflammatory agents are released from basophils and mast cells as a result of an antigen interacting with the immunoglobulin (Ig) E. Anaphylactoid reactions manifest themselves in the same manner as anaphylactic reactions, but are not the result of an interaction with IgE. Direct activation of complement and IgG-mediated complement activation can result in similar inflammatory mediator release and activity.

Depending on the antigen and the immune system components involved, hypersensitivity reactions are classically divided into four types (Table 54–5). In many cases, an allergen (eg, latex) may cause more than one type of hypersensitivity reaction. Type I reactions involve antigens that cross-link IgE antibodies, triggering the release of inflammatory mediators from mast cells. In type II reactions, complement-fixing (C1-binding) IgG antibodies bind to antigens on cell surfaces, activating the classic complement pathway and lysing the cells. Examples of type II reactions include hemolytic transfusion reactions and heparin-induced thrombocytopenia. Type III reactions occur when antigen–antibody

(IgG or IgM) immune complexes are deposited in tissues, activating complement and generating chemotactic factors that attract neutrophils to the area. The activated neutrophils cause tissue injury by releasing lysosomal enzymes and toxic products. Type III reactions include serum sickness reactions and acute hypersensitivity pneumonitis. Type IV reactions, often referred to as delayed hypersensitivity reactions, are mediated by $CD4^+$ T lymphocytes that have been sensitized to a specific antigen by prior exposure. Prior TH1 response causes expression of a T-cell receptor protein that is specific for the antigen. Reexposure to the antigen causes these lymphocytes to produce lymphokines—interleukins (IL), interferon (IFN), and tumor necrosis factor-γ (TNF-γ)—that attract and activate inflammatory mononuclear cells over 48–72 hr. Production of IL-1 and IL-6 by antigen-processing cells amplifies clonal expression of the specific sensitized T cells and attracts other types of T cells. IL-2 secretion transforms $CD8^+$ cytotoxic T cells into killer cells; IL-4 and IFN-γ cause macrophages to undergo epithelioid transformation, often producing granuloma. Examples of type IV reactions are those associated with tuberculosis, histoplasmosis, schistosomiasis, and hypersensitivity pneumonitis and some autoimmune disorders, such as rheumatoid arthritis and Wegener's granulomatosis.

1. Immediate Hypersensitivity Reactions

Initial exposure of a susceptible person to an antigen induces $CD4^+$ T cells to lymphokines that activate and transform specific B lymphocytes into plasma cells, producing allergen-specific IgE antibodies (**Figure 54–7**). The Fc portion of these antibodies then associates with high affinity receptors on the cell surface of tissue mast cells and circulating basophils. During subsequent reexposure to the antigen, it binds the Fab portion of adjacent IgE antibodies on the mast cell surface, inducing degranulation and release of inflammatory lipid mediators and additional cytokines from the mast cell. The end result is the release of histamine, tryptase, proteoglycans (heparin and chondroitin sulfate), and carboxypeptidases. Prostaglandin (mainly prostaglandin D_2) and leukotriene (B_4, C_4, D_4, E_4, and platelet-activating factor) synthesis is also increased. The combined effects of these mediators can produce arteriolar vasodilatation, increased vascular permeability, increased mucus secretion, smooth muscle contraction, and other clinical manifestations of type I reactions.

Type I hypersensitivity reactions are classified as atopic or nonatopic. Atopic disorders typically affect the skin or respiratory tract and include allergic rhinitis, atopic dermatitis, and allergic asthma. Nonatopic hypersensitivity disorders include urticaria, angioedema, and anaphylaxis; when these reactions are mild, they are confined to the skin (urticaria) or subcutaneous tissue (angioedema), but when they are severe, they become generalized and a life-threatening medical emergency (anaphylaxis). Urticarial lesions are characteristically well-circumscribed skin wheals with raised erythematous borders and blanched centers; they are intensely pruritic. Angioedema presents as deep, nonpitting cutaneous edema from marked vasodilatation and increased permeability of subcutaneous blood vessels. When angioedema is extensive, it can be associated with large fluid shifts; when it involves the pharyngeal or laryngeal mucosa, it can rapidly compromise the airway.

2. Anaphylactic Reactions

Anaphylaxis is an exaggerated response to an allergen (eg, antibiotic) that is mediated by a type I hypersensitivity reaction. The syndrome appears within minutes of exposure to a specific antigen in a sensitized person and characteristically presents as acute respiratory distress, circulatory shock, or both. Death may occur from asphyxiation or irreversible circulatory shock. The incidence of anaphylactic reactions during anesthesia has been estimated at a rate of 1:3500 to 1:20000 anesthetics. Mortality from anaphylaxis can be as frequent as 4% of cases with brain injury, occurring in another 2% of surviving patients. A French study evaluating 789 anaphylactic and anaphylactoid reactions reported that the most common sources of perioperative anaphylaxis were neuromuscular blockers (58%), latex (17%), and antibiotics (15%).

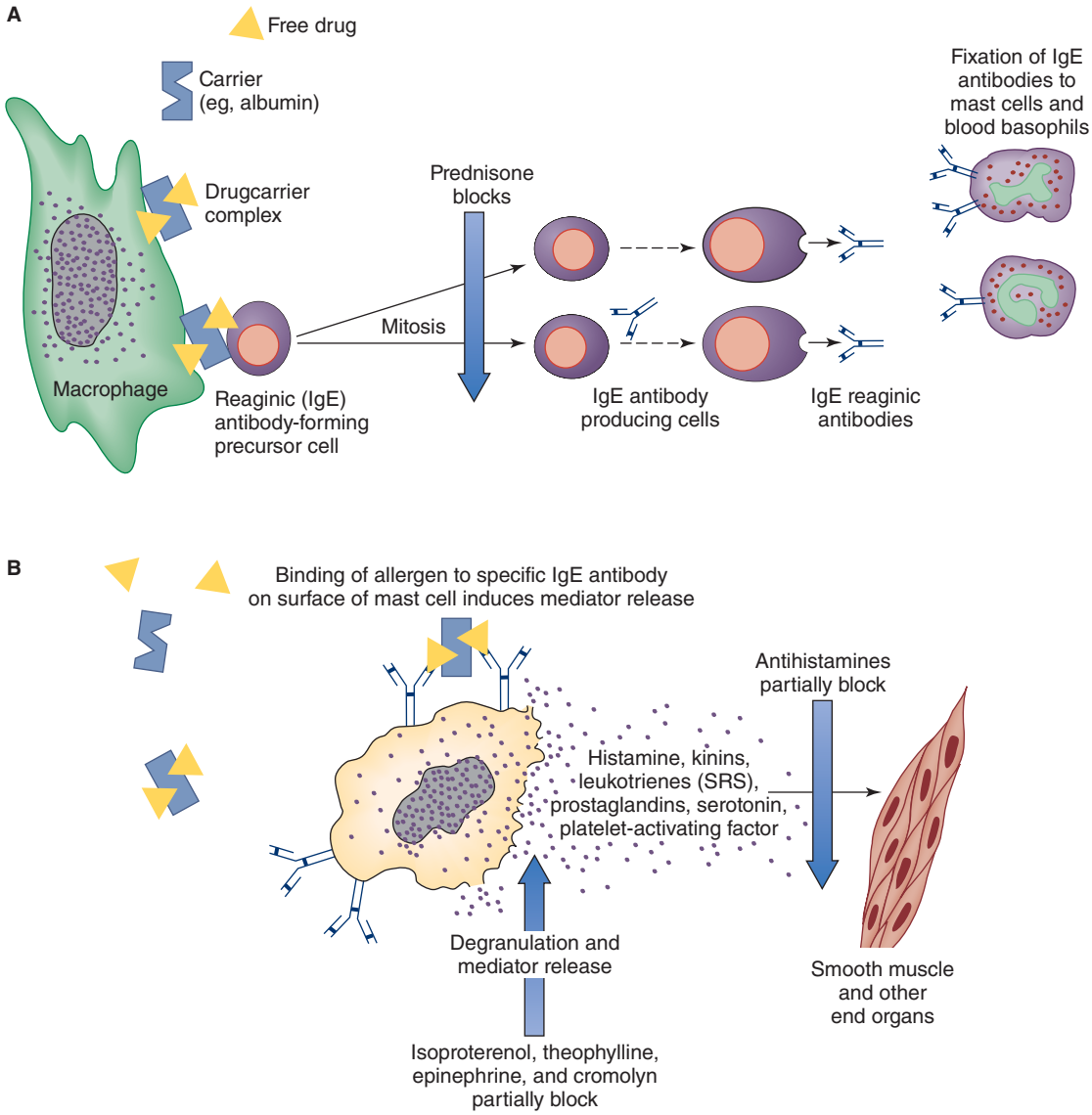

FIGURE 54–7 **A:** Induction of IgE-mediated allergic sensitivity to drugs and other allergens. **B:** Response of IgE-sensitized cells to subsequent exposure to allergens. Ig, immunoglobulin. (Reproduced, with permission, from Katzung BG [editor]: *Basic & Clinical Pharmacology,* 8th ed. McGraw-Hill, 2001.)

The most important mediators of anaphylaxis are histamine, leukotrienes, basophil kallikrein (BK-A,) and platelet-activating factor. They increase vascular permeability and contract smooth muscle. H_1-receptor activation contracts bronchial smooth muscle, whereas H_2-receptor activation causes vasodilatation, enhanced mucus secretion, tachycardia, and increased myocardial contractility. BK-A cleaves bradykinin from kininogen; bradykinin increases vascular permeability and vasodilatation and contracts smooth muscle. Activation of Hageman factor can initiate intravascular coagulation.

TABLE 54–6 Clinical manifestations of anaphylaxis.

Organ System	Signs and Symptoms
Cardiovascular	Hypotension,[1] tachycardia, arrhythmias
Pulmonary	Bronchospasm,[1] cough, dyspnea, pulmonary edema, laryngeal edema, hypoxia
Dermatological	Urticaria,[1] facial edema, pruritus

[1]Key signs during general anesthesia.

Eosinophil chemotactic factor of anaphylaxis, neutrophil chemotactic factor, and leukotriene B_4 attract inflammatory cells that mediate additional tissue injury. Angioedema of the pharynx, larynx, and trachea produce upper airway obstruction, whereas bronchospasm and mucosal edema result in lower airway obstruction. Histamine may preferentially constrict large airways, whereas leukotrienes primarily affect smaller peripheral airways. Transudation of fluid into the skin (angioedema) and viscera produces hypovolemia and shock, whereas arteriolar vasodilatation decreases systemic vascular resistance. Coronary hypoperfusion and arterial hypoxemia promote arrhythmias and myocardial ischemia. Leukotriene and prostaglandin mediators may also cause coronary vasospasm. Prolonged circulatory shock leads to progressive lactic acidosis and ischemic damage to vital organs. Table 54–6 summarizes important manifestations of anaphylactic reactions.

Anaphylactoid reactions resemble anaphylaxis but do not depend on IgE antibody interaction with antigen. A drug can directly release histamine from mast cells (eg, urticaria following high-dose morphine sulfate) or activate complement. Despite differing mechanisms, anaphylactic and anaphylactoid reactions typically are clinically indistinguishable and equally life-threatening. Table 54–7 lists common causes of anaphylactic and anaphylactoid reactions.

Factors that may predispose patients to these reactions include pregnancy, known atopy, and previous drug exposure. Such reactions are more common in younger than older patients. Laboratory identification of patients who have experienced an adverse allergic reaction or who may be particularly

TABLE 54–7 Causes of anaphylactic and anaphylactoid reactions.

Anaphylactic reactions against polypeptides	Venoms (Hymenoptera, fire ant, snake, jellyfish) Airborne allergens (pollen, molds, danders) Foods (peanuts, milk, egg, seafood, grain) Enzymes (trypsin, streptokinase, chymopapain, asparaginase) Heterologous serum (tetanus antitoxin, antilymphocyte globulin, antivenin) Human proteins (insulin, corticotropin, vasopressin, serum and seminal proteins) Latex
Anaphylactic reactions against hapten carrier	Antibiotics (penicillin, cephalosporins, sulfonamides) Disinfectants (ethylene oxide, chlorhexidine) Local anesthetics (procaine)
Anaphylactoid reactions	Polyionic solutions (radiocontrast medium, polymyxin B) Opioids (morphine, meperidine) Hypnotics (propofol, thiopental) Muscle relaxants (rocuronium, succinylcholine, cisatracurium) Synthetic membranes (dialysis) Nonsteroidal antiinflammatory drugs Preservatives (sulfites, benzoates) Protamine Dextran Steroids Exercise Idiopathic

Adapted and reproduced, with permission, from Bochner BS, Lichtenstein LM: N Engl J Med 1991;324:1786.

TABLE 54–8 Treatment of anaphylactic and anaphylactoid reactions.

Discontinue drug administration
Administer 100% oxygen
Epinephrine (0.01–0.5 mg IV or IM)[1]
Consider intubation
Intravenous fluid bolus
Diphenhydramine (50–75 mg IV)
Ranitidine (150 mg IV)
Hydrocortisone (up to 200 mg IV) or methylprednisolone (1–2 mg/kg)

[1]The dose and route of epinephrine depend on the severity of the reaction. An infusion of 1–5 mcg/min may be necessary in adults.

susceptible is often aided by intradermal skin testing, leukocyte or basophil degranulation testing (histamine release test), or radio-allergosorbent testing (RAST). The latter is capable of measuring the level of drug-specific IgE antibody in the serum. Serum tryptase measurement is helpful in confirming the diagnosis of an anaphylactic reaction. Prophylactic pretreatment with histamine receptor antagonists and corticosteroids decreases the severity of the reaction. Treatment must be immediate and tailored to the severity of the reaction (Table 54–8).

3. Allergic Reactions to Anesthetic Agents

7 True anaphylaxis due to anesthetic agents is rare; anaphylactoid reactions are much more common. Risk factors associated with hypersensitivity to anesthetics include female gender, atopic history, preexisting allergies, and previous anesthetic exposures. Muscle relaxants are the most common cause of anaphylaxis during anesthesia, with an estimated incidence of 1 in 6500 patients. They account for almost 60% of perioperative anaphylactic reactions. In many instances, there was no previous exposure to muscle relaxants. Investigators suggest that over-the-counter drugs, cosmetics, and food products, many of which contain tertiary or quaternary ammonium ions, can sensitize susceptible individuals. A French study found that, in decreasing order of frequency, rocuronium, succinylcholine, and atracurium were most often responsible; this likely reflects the propensity to cause anaphylaxis, together with frequency of use.

Although rarer, hypnotic agents can also be responsible for some allergic reactions. The incidence of anaphylaxis for thiopental and propofol is 1 in 30,000 and 1 in 60,000, respectively. Allergic reactions to etomidate, ketamine, and benzodiazepines are exceedingly rare. True anaphylactic reactions due to opioids are far less common than nonimmune histamine release. Similarly, anaphylactic reactions to local anesthetics are much less common than vasovagal reactions, toxic reactions to accidental intravenous injections, and side effects from absorbed or intravenously injected epinephrine. IgE-mediated reactions to ester-type local anesthetics, however, are well described secondary to reaction to the metabolite, para-aminobenzoic acid, In contrast, true anaphylaxis due to amide-type local anesthetics is very rare; in some instances, the preservative (paraben or methylparaben) was believed to be responsible for an apparent anaphylactoid reaction to a local anesthetic. Moreover, the cross-reactivity between amide-type local anesthetics seems to be low. There are no reports of anaphylaxis to volatile anesthetics.

4. Latex Allergy

The severity of allergic reactions to latex-containing products ranges from mild contact dermatitis to life-threatening anaphylaxis. Latex allergy is the second most common cause of anaphylaxis during anesthesia. Most serious reactions seem to involve a direct IgE-mediated immune response to polypeptides in natural latex, although some cases of contact dermatitis may be due to a type IV sensitivity reaction to chemicals introduced in the manufacturing process. Nonetheless, a relationship between the occurrence of contact dermatitis and the probability of future anaphylaxis has been suggested. Chronic exposure to latex and a history of atopy increases the risk of sensitization. Healthcare workers and patients undergoing frequent procedures with latex items (eg, repeated urinary bladder catheterization, barium enema examinations) should therefore be consid-

8 ered at increased risk. Patients with spina bifida, spinal cord injury, and congenital abnormalities of the genitourinary tract have an increased incidence of latex allergy. The incidence of

latex anaphylaxis in children is estimated to be 1 in 10,000. A history of allergic symptoms to latex should be sought in all patients during the preanesthetic interview. Foods that cross-react with latex include mango, kiwi, chestnut, avacado, passion fruit, and banana.

IL-18 and IL-13 single nucleotide polymorphisms may affect the sensitivity of individuals to latex and promote allergic responses.

Anaphylactic reactions to latex may be confused with reactions to other substances (eg, drugs, blood products) because the onset of symptoms can be delayed for more than 1 hr after initial exposure. Treatment is the same as for other forms of anaphylactic reactions. Skin-prick tests, intradermal tests, basophil histamine-release tests, and RAST have been used to evaluate high-risk patients. Preventing a reaction in sensitized patients includes pharmacological prophylaxis and absolute avoidance of latex. Preoperative administration of H_1 and H_2 histamine antagonists and steroids may provide some protection, although their use is controversial. Although most pieces of anesthetic equipment are now latex-free, some may still contain latex (eg, gloves, tourniquets, some ventilator bellows, intravenous injection ports, and older reusable face masks). An allergic reaction has even been documented from inhalation of latex antigen contained within aerosolized glove powder. Manufacturers of latex-containing medical products must label their products accordingly. **Only devices specifically known not to contain latex (eg, polyvinyl or neoprene gloves, silicone endotracheal tubes or laryngeal masks, plastic face masks) can be used in latex-allergic patients.** Rubber stoppers should be removed from drug vials prior to use, and injections should be made through plastic stopcocks, if latex has not been eliminated from containers and injection ports.

5. Allergies to Antibiotics

Many true drug allergies in surgical patients are due to antibiotics, mainly β-lactam antibiotics, such as penicillins and cephalosporins. Although 1% to 4% of β-lactam administrations result in allergic reactions, only 0.004% to 0.015% of these reactions result in anaphylaxis. Up to 2% of the general population is allergic to penicillin, but only 0.01% of penicillin administrations result in anaphylaxis. Cephalosporin cross-sensitivity in patients with penicillin allergy is estimated to be 2% to 7%, but a history of an anaphylactic reaction to penicillin increases the cross-reactivity rate up to 50%. Patients with a prior history of an anaphylactic reaction to penicillin should therefore not receive a cephalosporin. Although imipenem exhibits similar cross-sensitivity, aztreonam seems to be antigenically distinct and reportedly does not cross-react with other β-lactams. Sulfonamide allergy is also relatively common in surgical patients. Sulfa drugs include sulfonamide antibiotics, furosemide, hydrochlorothiazide, and captopril. Fortunately, the frequency of cross-reactivity among these agents is low.

Like cephalosporins, vancomycin is commonly used for antibiotic prophylaxis in surgical patients. Unfortunately, it is associated with adverse reactions. An anaphylactoid-type reaction, "red man syndrome," consists of intense pruritus, flushing, and erythema of the head and upper torso, often with arterial hypotension; this syndrome seems to be related to a rapid rate of administration more than to dose or allergy. Isolated systemic hypotension is a much more frequent side effect and seems to be primarily mediated by histamine release, because pretreatment with H_1 and H_2 antihistamines can prevent hypotension, even with rapid rates of administration.

Immunologic mechanisms are associated with other perioperative pathologies. Transfusion-related lung injury may be secondary to the activity of antibodies in the donor plasma, producing a hypersensitivity reaction that results in lung infiltrates and respiratory failure. IgG antibody formation directed at heparin–PF4 complexes results in platelet activation, thrombosis, and heparin-induced thrombocytopenia.

OCCUPATIONAL HAZARDS IN ANESTHESIOLOGY

Anesthesiologists spend much of their workday exposed to anesthetic gases, low-dose ionizing radiation, electromagnetic fields, blood products,

TABLE 54–9 Relative rate ratios for drug and suicide deaths comparing anesthesiologists with internists before and after January 1, 1987.

		Anesthesiologists (*N*)	Internists (*N*)	RR[1]	95% CI
All drug-related deaths	<1987	36	14	2.65	1.42–4.91
	≥1987	55	19	2.87	1.71–4.84
Drug-related suicides	<1987	16	11	1.48	0.69–3.20
	≥1987	32	11	2.88	1.45–5.71
Suicides	<1987	41	33	1.25	0.79–1.97
	≥1987	62	38	1.60	1.07–2.39

CI, confidence interval.

[1]Ratio (RR) of anesthesiologists compared with internists for that time period, RR is adjusted for age, gender, and race.

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and workplace stress. Each of these can contribute to negative health effects in anesthesia practitioners. A 2000 paper compared the mortality risks of anesthesiologists and internists. Death from heart disease or cancer did not differ between the groups; however, anesthesiologists had an increased rate of suicides and drug-related deaths (Table 54–9). Anesthesiologists also had a greater chance of death from external causes, such as boating, bicycling, and aeronautical accidents compared with internists. Nevertheless, both anesthesiologists and internists had lower mortality than the general population, likely due to their higher socioeconomic status. Anesthesiologists' access to intravenous opioids likely contributes to a 2.21 relative risk for drug-related deaths compared with that of internists.

1. Chronic Exposure to Anesthetic Gases

9 There is no clear evidence that exposure to trace amounts of anesthetic agents presents a health hazard to operating room personnel. However, because previous studies examining this issue have yielded flawed but conflicting results, the US Occupational Health and Safety Administration continues to set maximum acceptable trace concentrations of less than 25 ppm for nitrous oxide and 0.5 ppm for halogenated anesthetics (2 ppm if the halogenated agent is used alone). Achieving these low levels depends on efficient scavenging equipment, adequate operating room ventilation, and conscientious anesthetic technique. Most people cannot detect the odor of volatile agents at a concentration of less than 30 ppm. If there is no functioning scavenging system, anesthetic gas concentrations reach 3000 ppm for nitrous oxide and 50 ppm for volatile agents.

Early studies indicated that female operating room staff might be at increased risk of spontaneous abortion compared with other women. It is unclear if other factors related to operating room activity could also contribute to the possibly increased potential for pregnancy loss.

2. Infectious Diseases

Hospital workers are exposed to many infectious diseases prevalent in the community (eg, respiratory viral infections, rubella, and tuberculosis).

Herpetic whitlow is an infection of the finger with herpes simplex virus type 1 or 2 and usually involves direct contact of previously traumatized skin with contaminated oral secretions. Painful vesicles appear at the site of infection. The diagnosis is confirmed by the appearance of giant epithelial cells or nuclear inclusion bodies in a smear taken from the base of a vesicle, the presence of a rise in herpes simplex virus titer, or identification of the virus with antiserum. Treatment is conservative and includes

topical application of 5% acyclovir ointment. Prevention involves wearing gloves when contacting oral secretions. Patients at risk of harboring the virus include those suffering from other infections, immunosuppression, cancer, and malnutrition. The risk of this condition has virtually disappeared now that anesthesia personnel routinely wear gloves during manipulation of the airway, which was not the case in the 1980s and earlier.

Viral DNA has been identified in the smoke plume generated during laser treatment of condylomata. The theoretical possibility of viral transmission from this source can be minimized by using smoke evacuators, gloves, and appropriate OSHA approved masks.

More disturbing is the potential of acquiring serious blood-borne infections, such as hepatitis B, hepatitis C, or human immunodeficiency virus (HIV). Although parenteral transmission of these diseases can occur following mucous membrane, cutaneous, or percutaneous exposure to infected body fluids, accidental injury with a needle contaminated with infected blood represents the most common occupational mechanism. The risk of transmission can be estimated if three factors are known: the prevalence of the infection within the patient population, the incidence of exposure (eg, frequency of needlestick), and the rate of seroconversion after a single exposure. The seroconversion rate after a specific exposure depends on several factors, including the infectivity of the organism, the stage of the patient's disease (extent of viremia), the size of the inoculum, and the immune status of the healthcare provider. Rates of seroconversion following a single needlestick are estimated to range between 0.3% and 30%. Hollow (hypodermic) needles pose a greater risk than do solid (surgical) needles because of the potentially larger inoculum. The use of gloves, needleless systems, or protected needle devices may decrease the incidence of some (but not all) types of injury.

The initial management of needlesticks involves cleaning the wound and notifying the appropriate authority within the health care facility. After an exposure, anesthesia workers should report to their institution's emergency or employee health department for appropriate counseling on postexposure prophylaxis options. All OR staff should be made aware of the institution's employee health notification pathway for needle stick and other injuries

Fulminant hepatitis B (1% of acute infections) carries a 60% mortality rate. Chronic active hepatitis (<5% of all cases) is associated with an increased incidence of cirrhosis of the liver and hepatocellular carcinoma. Transmission of the virus is primarily through contact with blood products or body fluids. The diagnosis is confirmed by detection of hepatitis B surface antigen (HBsAg). Uncomplicated recovery is signaled by the disappearance of HBsAg and the appearance of antibody to the surface antigen (anti-HBs). A hepatitis B vaccine is available and is strongly recommended prophylactically for anesthesia personnel. The appearance of anti-HBs after a three-dose regimen indicates successful immunization.

Hepatitis C is another important occupational hazard in anesthesiology; 4% to 8% of hepatitis C infections occur in healthcare workers. Most (50% to 90%) of these infections lead to chronic hepatitis, which, although often asymptomatic, can progress to liver failure and death. In fact, hepatitis C is the most common cause of nonalcoholic cirrhosis in the United States. There is currently no vaccine to protect against hepatitis C infection.

Anesthesia personnel seem to be at a low, but measureable, risk for the occupational contraction of HIV. The risk of acquiring HIV infection following a single needlestick contaminated with blood from an HIV-infected patient has been estimated at 0.4% to 0.5%. Because there are documented reports of transmission of HIV from infected patients to healthcare workers (including anesthesiologists), the Centers for Disease Control and Prevention proposed guidelines that apply to all categories of patient contact. These universal precautions, which are equally valid for protection against hepatitis B or C infection, are as follows:

- No recapping and the immediate disposal of contaminated needles
- Use of gloves and other barriers during contact with open wounds and body fluids
- Frequent hand washing

- Use of proper techniques for disinfection or the disposal of contaminated materials
- Particular caution by pregnant healthcare workers, and no contact with patients by workers who have exudative or weeping skin lesions

3. Substance Abuse

11 Anesthesiology is a high-risk medical specialty for substance abuse. Probable reasons for this include the stress of anesthetic practice and the easy availability of drugs with addiction potential (potentially attracting people at risk of addiction to the field). The likelihood of developing substance abuse is increased by coexisting personal problems (eg, marital or financial difficulties) or a family history of alcoholism or drug addiction.

The voluntary use of nonprescribed mood-altering pharmaceuticals is a disease. If left untreated, substance abuse often leads to death from drug overdose—intentional or unintentional. One of the greatest challenges in treating drug abuse is identifying the afflicted individual, as denial is a consistent feature. Unfortunately, changes evident to an outside observer are often both vague and late: reduced involvement in social activities, subtle changes in appearance, extreme mood swings, and altered work habits. Treatment begins with a careful, well-planned intervention. Those inexperienced in this area would be well advised to consult with their local medical society or licensing authority about how to proceed. The goal is to enroll the individual in a formal rehabilitation program. The possibility that one may lose one's medical license and be unable to return to practice provides powerful motivation. Some diversion programs report a success rate of approximately 70%; however, most rehabilitation programs report a recurrence rate of at least 25%. Long-term compliance often involves continued participation in support groups (eg, Narcotics Anonymous), random urine testing, and oral naltrexone therapy (a long-acting opioid antagonist). Effective prevention strategies are difficult to formulate; "better" control of drug availability is unlikely to deter a determined individual. It is unlikely that education about the severe consequences of substance abuse will bring new information to the potential drug-abusing physician. There remains controversy regarding the rate at which anesthesia staff will experience recidivism. Many experts argue for a "one strike and you're out" policy for anesthesiology residents who abuse injectable drugs. The decision as to whether a fully trained and certified physician who has been discovered to abuse injectable drugs should return to anesthetic practice after completing a rehabilitation program varies and depends on the rules and traditions of the practice group, the medical center, the relevant medical licensing board, and the perceived likelihood of recidivism. Physicians returning to practice following successful completion of a program must be carefully monitored over the long term, as relapses can occur years after apparent successful rehabilitation. Alcohol abuse is a common problem among physicians and nurses, and anesthesia personnel are no exception. Interventions for alcohol abuse, as is true for injectable drug abuse, must be carefully orchestrated. Guidance from the local medical society or licensing authority is highly recommended.

4. Ionizing Radiation Exposure

The use of imaging equipment (eg, fluoroscopy) during surgery and interventional radiologic procedures exposes the anesthesiologist to the potential 12 risks of ionizing radiation. The three most important methods of minimizing radiation doses are limiting total exposure time during procedures, using proper barriers, and maximizing the distance from the source of radiation. Anesthesiologists who routinely perform fluoroscopic image guided invasive procedures should consider wearing protective eyeware incorporating radiation shielding. Lead glass partitions or lead aprons with thyroid shields are mandatory protection for all personnel who are exposed to ionizing radiation. The inverse square law states that the dosage of radiation varies inversely with the square of the distance. Thus, the exposure at 4 m will be one-sixteenth that at 1 m. The maximum recommended occupational whole-body exposure to radiation is 5 rem/yr. This can be monitored with an exposure badge. The health impact on operating room personnel of exposure to electromagnetic radiation remains unclear.

CASE DISCUSSION

Unexplained Intraoperative Tachycardia & Hypertension

A 73-year-old man is scheduled for emergency relief of an intestinal obstruction from a sigmoid volvulus. The patient had a myocardial infarction 1 month earlier that was complicated by congestive heart failure. His blood pressure is 160/90 mm Hg, pulse 110 beats/min, respiratory rate 22 breaths/min, and temperature 38.8°C.

Why is this case an emergency?

Strangulation of the bowel begins with venous obstruction, but can quickly progress to arterial occlusion, ischemia, infarction, and perforation. Acute peritonitis could lead to severe dehydration, sepsis, shock, and multiorgan failure.

What special monitoring is appropriate for this patient?

Because of the history of recent myocardial infarction and congestive heart failure, an arterial line would be useful. Transesophageal echocardiography and pulse contour analysis monitors of cardiac output could be used. Pulmonary arterial flotation catheters have often been used in the past, but they are associated with significant complications and current evidence does not indicate that their use improves patient outcomes. Large fluid shifts should be anticipated. Furthermore, information regarding myocardial supply (diastolic blood pressure) and demand (systolic blood pressure, left ventricular wall stress, and heart rate) should be continuously available. Central venous pressure may not track left atrial pressure in a patient with significant left ventricular dysfunction.

What cardiovascular medications might be useful during induction and maintenance of general anesthesia?

Drugs causing severe tachycardia or extremes in arterial blood pressure should be avoided.

During the laparotomy, gradual increases in heart rate and blood pressure are noted. ST-segment elevations appear on the electrocardiogram. A nitroglycerin infusion is started. The heart rate is now 130 beats/min, and the blood pressure is 220/140 mm Hg. The concentration of volatile anesthetic is increased, and metoprolol is administered intravenously in 1-mg increments. This results in a decline in heart rate to 115 beats/min, with no change in blood pressure. Suddenly, the rhythm converts to ventricular tachycardia, with a profound drop in blood pressure. As amiodarone is being administered and the defibrillation unit prepared, the rhythm degenerates into ventricular fibrillation.

What can explain this series of events?

A differential diagnosis of pronounced tachycardia and hypertension might include pheochromocytoma, malignant hyperthermia, or thyroid storm. In this case, further inspection of the nitroglycerin infusion reveals a labeling error: although the tubing was labeled "nitroglycerin," the infusion bag was labeled "epinephrine."

How does this explain the paradoxic response to metoprolol?

Metoprolol is a β_1-adrenergic antagonist. It inhibits epinephrine's β_1-stimulation of heart rate, but does not antagonize α-induced vasoconstriction. The net result is a decrease in heart rate, but a sustained increase in blood pressure.

What is the cause of the ventricular tachycardia?

An overdose of epinephrine can cause life-threatening ventricular arrhythmias. In addition, if the central venous catheter was malpositioned, with its tip in the right ventricle, the catheter tip could have stimulated ventricular arrhythmias.

What other factors may have contributed to this anesthetic mishap?

Multiple factors will often combine to create an anesthetic misadventure. Incorrect drug labels are but one example of errors that can result in patient injury. Inadequate preparation, technical failures, knowledge deficits, and practitioner fatigue or distraction can all contribute to adverse outcomes. Careful adherence to hospital policies, checklists, patient identification procedures, and surgical and regional block timeouts can all help to prevent iatrogenic complications.

GUIDELINES

Practice advisory for the prevention of perioperative peripheral neuropathies: a report by the American Society of Anesthesiologists Task Force on prevention of peripheral neuropathies. Anesthesiology 2000;92:1168.

SUGGESTED READING

Alexander B, Checkoway H, Nagahama S, Domino K: Cause-specific mortality risks of anesthesiologists. Anesthesiology 2000;93:922.

Berge E, Seppala M, Lanier W: The anesthesiology community's approach to opioid and anesthetic abusing personnel: time to change course. Anesthesiology 2008;109:762.

Bhananker S, Posner K, Cheney F, et al: Injury and liability associated with monitored anesthesia care. Anesthesiology 2006;104:228.

Bhananker S, Liau D, Kooner P, et al: Liability related to peripheral venous and arterial catheterization; a closed claims analysis. Anesth Analg 2009;109:124.

Bishop M, Souders J, Peterson C, et al: Factors associated with unanticipated day of surgery deaths in Department of Veterans Affairs hospitals. Anesth Analg 2008;107:1924.

Bowdle TA: Drug administration errors from the ASA closed claims project. ASA Newslett 2003;67:11.

Brown RH, Schauble JF, Miller NR: Anemia and hypotension as contributors to perioperative loss of vision. Anesthesiology 1994;80:222.

Bryson E, Silverstein J: Addiction and substance abuse in anesthesiology. Anesthesiology 2008;109:905.

Caplan RA, Ward RJ, Posner K, Cheney FW: Unexpected cardiac arrest during spinal anesthesia: a closed claims analysis of predisposing factors. Anesthesiology 1988;68:5.

Caplan RA, Vistica MF, Posner KL, Cheney FW: Adverse anesthetic outcomes arising from gas delivery equipment: a closed claims analysis. Anesthesiology 1997;87:741.

Chadwick HS: An analysis of obstetric anesthesia cases from the American Society of Anesthesiologists closed claims project database. Int J Obstet Anesth 1996;5:258.

Cheesman K, Brady J, Flood P, Li G: Epidemiology of anesthesia-related complications in labor and delivery, New York state, 2002-2005. Anesth Analg 2009;109:1174.

Cheney FW: The American Society of Anesthesiologists closed claims project: what have we learned, how has it affected practice, and how will it affect practice in the future? Anesthesiology 1999;91:552.

Cheney FW, Posner KL, Caplan RA, Gild WM: Burns from warming devices in anesthesia. Anesthesiology 1994;80:806.

Cheney FW, Domino KB, Caplan RA, Posner KL: Nerve injury associated with anesthesia: a closed claims analysis. Anesthesiology 1999;90:1062.

Cheney F, Posner K, Lee L, et al: Trends in anesthesia-related death and brain damage. Anesthesiology 2006;105:1071.

Cook T, Bland L, Mihai R, Scott S: Litigation related to anaesthesia: an analysis of claims against the NHS in England 1995-2007. Anaesthesia 2009;64:706.

Cook T, Scott S, Mihai R: Litigation related to airway and respiratory complications of anaesthesia: an analysis of claims against the NHS in England 1995-2007. Anaesthesia 2010;65:556.

Coppieters MW, Van De Velde M, Stappaerts KH: Positioning in anesthesiology. Toward a better understanding of stretch-induced perioperative neuropathies. Anesthesiology 2002;97:75.

Cranshaw J, Gupta K, Cook T: Litigation related to drug errors in anaesthesia: an analysis of claims against the NHS in England 1995-2009. Anaesthesia 2009;64:1317.

Crosby E: Medical malpractice and anesthesiology: literature review and role of the expert witness. Can J Anesth 2007;54:227.

Davies J, Posner K, Lee L, et al: Liability associated with obstetric anesthesia. Anesthesiology 2009;110:131.

Domino KB, Posner Kl, Caplan RA, Cheney FW: Awareness during anesthesia: a closed claims analysis. Anesthesiology 1999;90:1053.

Domino KB, Posner KL, Caplan RA, Cheney FW: Airway injury during anesthesia: a closed claims analysis. Anesthesiology 1999;91:1703.

Domino K, Bowdle T, Posner K, et al: Injuries and liability related to central vascular catheters. Anesthesiology 2004;100:1411.

Edbril SD, Lagasse RS: Relationship between malpractice litigation and human errors. Anesthesiology 1999;91:848.

Fisher DM: New York State guidelines on the topical use of phenylephrine in operating rooms. Anesthesiology 2000;92:858.

Fitzgibbon DR, Posner KL, Domino KB, et al: Chronic pain management: American Society of Anesthesiologists Closed Claims Project. Anesthesiology 2004;100:98.

Gayer S: Prone to blindness: answers to postoperative visual loss. Anesth Analg 2011;112:11.
Ghoneim MM: Awareness during anesthesia. Anesthesiology 2000;92:597.
Gild WM, Posner KL, Caplan RA, Cheney FW: Eye injuries associated with anesthesia. Anesthesiology 1992;76:204.
Hawkins J, Chang J, Palmer S, et al: Anesthesia-related maternal mortality in the United States: 1979-2002. Obstet Gynecol 2011;117:69.
Hepner DL, Castells MC: Anaphylaxis during the perioperative period. Anesth Analg 2003;97:1381.
Jimenez N, Posner K, Cheney F, et al: An update on pediatric anesthesia liability: a closed claims analysis. Anesth Analg 2007;104:147.
Lagasse R: Anesthesia safety: model or myth? Anesthesiology 2002;97:1336.
Lagasse R: To see or not to see. Anesthesiology 2006; 105:1971.
Lagasse R: Innocent prattle. Anesthesiology 2009;110:698.
Lee LA: Postoperative visual loss data gathered and analyzed. ASA Newslett 2000;64:25.
Lee L, Posner K, DominoK, et al: Injuries associated with regional anesthesia in the 1980s and 1990s. Anesthesiology 2004;101:143.
Lee L, Posner K, Cheney F, et al: Complications associated with eye blocks and peripheral nerve blocks: an American Society of Anesthesiologists Closed Claims analysis. Reg Anesth Pain Med 2008;33:416.
Lee LA, Rothe S, Posner KL, et al: The American Society of Anesthesiologists Postoperative Visual Loss Registry: analysis of 93 spine surgery cases with postoperative visual loss. Anesthesiology 2006;105:652.
Lesser JB, Sanborn KV, Valskys R, Kuroda M: Severe bradycardia during spinal and epidural anesthesia recorded by an anesthesia information management system. Anesthesiology 2003;99:859.
Levy J, Adkinson N: Anaphylaxis during cardiac surgery: implications for clinicians. Anesth Analg 2008;106:392.
Li G, Warner M, Lang B, et al: Epidemiology of anesthesia related mortality in the United States 1999-2005. Anesthesiology 2009;110:759.
Liang B: "Standards" of anesthesia: law and ASA guidelines. J Clin Anesth 2008;20:393.
Lineberger C: Impairment in anesthesiology: awareness and education. Int Anesth Clin 2008;46:151.
Marco A: Informed consent for surgical anesthesia care: has the time come for separate consent? Anesth Analg 2009;110:280.
Martin L, Mhyre J, Shanks A, et al: 3,423 emergency tracheal intubations at a university hospital: airway outcomes and complications. Anesthesiology 2011;114:42.
Martin JT: Compartment syndromes: concepts and perspectives for the anesthesiologist. Anesth Analg 1992;75:275.
Metzner J, Posner K, Domino K: The risk and safety of anesthesia at remote locations: the US closed claims analysis. Curr Opin Anaesthesiol 2009;22:502.
Monitto C, Hamilton R, Levey E, et al: Genetic predisposition to natural rubber latex allergy differs between health care workers and high risk patients. Anesth Analg 2010;110:1310.
Morray JP, Geiduschek JM, Caplan RA: A comparison of pediatric and adult anesthesia closed malpractice claims. Anesthesiology 1993;78:461.
Newland MC, Ellis SJ, Lydiatt CA, et al: Anesthetic-related cardiac arrest and its mortality. Anesthesiology 2002;97:108.
Pollard JB: Cardiac arrest during spinal anesthesia: common mechanisms and strategies for prevention. Anesth Analg 2001;92:252.
Ramamoorthy C, Haberkern C, Bhananker S, et al: Anesthesia-related cardiac arrest in children with heart disease: data from the pediatric perioperative cardiac arrest (POCA) registry. Anesth Analg 2010;110:1376.
Ranta SOV, Lauila R, Saario J, et al: Awareness with recall during general anesthesia: incidence and risk factors. Anesth Analg 1998;86:1084.
Roh J, Kim D, Lee S, et al: Intensity of extremely low frequency electromagnetic fields produced in operating rooms during surgery at the standing position of anesthesiologists. Anesthesiology 2009;111:275.
Rose G, Brown R: The impaired anesthesiologist: not just about drugs and alcohol anymore. J Clin Anesthesiol 2010;22:379.
Sharma AD, Parmley CL, Sreeram G, Grocott HP: Peripheral nerve injuries during cardiac surgery: risk factors, diagnosis, prognosis, and prevention. Anesth Analg 2000;91:1358.
Silverstein JH, Silva DA, Iberti TJ: Opioid addiction in anesthesiology. Anesthesiology 1993;79:354.
Sprung J, Bourke DL, Contreras MG, et al: Perioperative hearing impairment. Anesthesiology 2003;98:241.
Tait AR: Occupational transmission of tuberculosis: implications for anesthesiologists. Anesth Analg 1997;85:444.

Warner MA, Warner ME, Martin JT: Ulnar neuropathy. Incidence, outcome, and risk factors in sedated or anesthetized patients. Anesthesiology 1994;81:1332.

Warner MA, Warner DO, Harper CM: Lower extremity neuropathies associated with lithotomy positions. Anesthesiology 2000;93:938.

Warner ME, Benenfeld SM, Warner MA, et al: Perianesthetic dental injuries. Anesthesiology 1999;90:1302.

Weinger MB, Englund CE: Ergonomic and human factors affecting anesthetic vigilance and monitoring performance in the operating room environment. Anesthesiology 1990;73:995.

Welch M, Brummett C, Welch T, et al: Perioperative nerve injuries: a retrospective study of 380,680 cases during a 10-year period at a single institution. Anesthesiology 2009;111:490.

Williams EL, Hart WM Jr, Tempelhoff R: Postoperative ischemic optic neuropathy. Anesth Analg 1995;80:1018.

Yuill G, Saroya D, Yuill S: A national survey of the provision for patients with latex allergy. Anaesthesiology 2003;58:775.