

**Chapter 1 : Anesthesia Complications - Before and After Surgery - calendrierdelascience.com**

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Open in a separate window Table adapted from reference 8. The effect of advanced age becomes particularly notable at approximately 60 years of age and worsens from there. The most important mechanisms will be discussed at length. Atelectasis causes pulmonary shunt and, therefore, undoubtedly contributes to the impairment of gas exchange during general anesthesia. Although shunt does not increase with age, regions with poor ventilation in relation to their perfusion show a dependency on age. Furthermore, larger atelectatic areas are present in obese patients whereas patients with COPD may show less or even no atelectasis. Atelectasis appears immediately with induction of anesthesia and is present after muscle paralysis and during spontaneous breathing regardless of whether inhalational or intravenous anesthetics are used<sup>11</sup>. The use of ketamine is the only exception to this rule. Three possible mechanisms may cause atelectasis including gas resorption, impaired function of surfactant and compression atelectasis<sup>17</sup>. It has been shown that a lung unit will ultimately collapse if it is not ventilated. Hence, lung collapse may occur if induction of anesthesia results in an increased number of lung units with poor or no ventilation, and if such lung units are filled with an easily resorbed gas. It is known, however, that there may be a decreased content of active forms of alveolar surfactant due to a lack of intermittent deep breaths, as is usual during mechanical ventilation. Furthermore, anesthesia and atelectasis per se may impede the function of surfactant. Such decreased function results in reduced alveolar stability and causes alveolar collapse. It may also contribute to liquid bridging in the airway lumen and cause airway closure. The end-expiratory intrathoracic pressure is normally lower than the abdominal pressure because the diaphragm separates two spaces with different pressures as well as vertical pressure gradients. In an awake patient, the vertical pressure gradient in the pleural space is 0. This abdominal pressure will be transferred into the thoracic cavity if the diaphragm no longer acts as a rigid wall between these two spaces, thus increasing the pleural pressure in dependent lung regions. This process could result in compression atelectasis. This was indirectly reflected in a study by Tokics et al<sup>16</sup>, who showed that no atelectasis developed during anesthesia with ketamine, a drug known to maintain respiratory muscle tone and rib cage function. Furthermore, several studies<sup>29, 30</sup> showed a cephalad shift of the diaphragm with anesthesia and muscle paralysis. The attenuation of hypoxic pulmonary vasoconstriction<sup>31, 32</sup> may further contribute to impairment in gaseous exchange by increasing pulmonary shunt because atelectasis is present throughout anesthesia. This effect may be apparent with the inhalational anesthetics<sup>33</sup> but is less prominent or even absent with intravenous anesthetics. Two mechanisms most commonly involved in the perioperative formation of atelectasis are compression and resorption. The early formation of atelectasis and the increase in pulmonary shunt are unavoidable adverse effects of anesthesia leading to respiratory complications in the postanesthesia period. Alveolar hypoventilation CO<sub>2</sub> retention is the hallmark of hypoventilation and is always present. Review of the alveolar gas equation according to West<sup>34</sup> indicates that partial pressure of oxygen PaO<sub>2</sub> is both directly and inversely proportional to alveolar ventilation; therefore, when patients breathe room air while hypoventilating, hypoxia results secondary to an increase in alveolar CO<sub>2</sub>. Furthermore, hypoxemia of pure hypoventilation can be readily improved by increasing the FiO<sub>2</sub>. This is especially important when monitoring patients for airway or ventilation adequacy in the PACU. According to Stemp and Ramsay<sup>35</sup>, a fall in arterial oxygen saturation on pulse oximetry in patients breathing room air is indicative of alveolar hypoventilation or possible airway obstruction. Rapid detection of this phenomenon in the PACU thus enables early intervention. In the immediate postoperative period, causes of hypoventilation are myriad and include the following<sup>34</sup>. Morbid obesity is another example of increasing inspiratory load on breathing leading to hypoventilation. Severe cases of pulmonary edema or obstructive airways disease can also cause respiratory muscle fatigue and hypercapnia. The associated hypoxemia is more severe in relation to hypercapnia than in other forms of hypoventilation. Furthermore, injudicious use of oxygen therapy in such patients can worsen hypercarbia and ventilatory failure. This effect is often attributed to a decrease in hypoxic

ventilatory drive associated with this group of patients. In most patients with hypoventilation, hypoxemia is reversed with supplemental oxygen therapy and the main focus of management is treating the cause of hypoventilation. It is important to note, however, that the use of supplemental oxygen in these settings can mask the progression of bradypnea to apnea, preventing the onset of hypoxemia as evidenced by pulse oximetry and, thus, can lead to unrecognized severe hypoventilation with potentially catastrophic consequences. In this situation, the pulse oximeter becomes an important tool for monitoring not only oxygenation but also the adequacy of ventilation when supplemental oxygen is not used. Such a reduction is caused by a change in rib cage configuration<sup>42</sup> and cranial shift of the diaphragm mostly of its dependent parts<sup>43</sup>. The magnitude of such changes depends on several factors including the type of anesthetic used and whether muscle relaxation is added. Rib cage contribution to normal tidal breathing is reduced<sup>45</sup> or unchanged with inhalation anesthetics<sup>46</sup>, whereas ketamine, on the other hand, increases rib cage contribution<sup>47</sup> or may at least keep the contribution unchanged. A change in intrathoracic blood volume is an additional factor relevant to the decrease in FRC. These changes may also result in a change in regional ventilation. During spontaneous breathing, there is some gravity-dependent distribution of ventilation, with an increase in regional ventilation from nondependent to dependent lung regions<sup>50</sup>, as well as marked gravity-independent inhomogeneity of regional ventilation<sup>51</sup>. There is a marked change in ventilation distribution with induction of anesthesia and muscle paralysis caused, at least, in part, by a change in the position and movement of the diaphragm. Inspiratory gas predominantly shifts to nondependent lung regions during normal tidal breathing, whereas dependent regions are less ventilated. There is also a change in the distribution of pulmonary blood flow in addition to the change in ventilation distribution. Increased intrathoracic pressure, present during positive pressure ventilation, may reduce cardiac output and affect pulmonary vascular resistance. These changes contribute significantly to respiratory complications in the postanesthesia period.

**Pulmonary shunt** Hypoxemia attributable to shunt is characterized by a decrease in arterial oxygen content in the setting of constant alveolar ventilation. Consequently, venous blood passes to the arterial system through areas of unventilated lung. An important diagnostic element of shunt is apparent in the shape of the oxygen-hemoglobin dissociation curve<sup>36</sup>; the addition of supplemental oxygen cannot ameliorate the hypoxemia. This is because the saturation of nonshunted blood is on the flat portion of the oxygen-hemoglobin dissociation curve and, thus, additional oxygen has little impact in raising the PaO<sub>2</sub>. The PaO<sub>2</sub> drops precipitously when this blood mixes with poorly oxygenated shunted blood. Pulmonary disorders, such as pulmonary edema both cardiogenic and negative pressure, transfusion-related lung injury and pneumonia, represent potential causes of shunt commonly encountered in the PACU. The less common extrapulmonary causes of shunt may include congenital cardiac defects such as atrial septal defect, patent ductus arteriosus and ventricular septal defect, which are associated with increased right heart pressures.

**Diffusion impairment** Diffusion impairment reflects the lack of equilibrium between PaO<sub>2</sub> in the alveolar gas and pulmonary capillary blood. Such a situation can occur when the alveolar capillary membrane is thickened, thus limiting the rate of diffusion of oxygen between the capillary and alveoli. Underlying lung disease, such as emphysema, interstitial lung disease, primary pulmonary hypertension or pulmonary fibrosis, are classic examples. Damage or loss of whole lung units reduces alveolar capillary surface area and capillary volumes, resulting in a lack of equilibrium and decreased red cell transit time through alveolar capillaries. Such a decreased transit time through alveolar capillaries may be due to increases in cardiac output, as observed in sepsis for example, thus worsening arterial hypoxemia in the context of an already injured lung. Increased oxygen extraction Increased oxygen extraction typically refers to low cardiac output states and is due to the mixing of desaturated venous blood with oxygenated arterial blood. However, blood returns to the heart severely desaturated in low cardiac output states. At the conclusion of surgery, it is therefore especially important that residual effects of anesthetic agents be adequately reversed or dissipated. As long as the airway is maintained, adequate spontaneous ventilation is readily possible during surgical levels of anesthesia, but a picture resembling sleep apnea may be induced at lighter planes of anesthesia that requires control of the airway. Thus, many of the problems noted in the PACU are related to the loss of mechanical support of the upper airway. Pharmacological effects are of little importance in the control of breathing throughout general

anesthesia and mechanical ventilation; however, during and after the weaning process, any depression of ventilator control may require the reinstatement of mechanical ventilation. Furthermore, the effects of these drugs on the control of breathing are complex and can affect breathing by alterations in: Thus, during periods of patient sedation in the PACU, adequate ventilation may depend solely on the chemoreflexes as opposed to when the patient is fully awake, when feedforward influences from the higher centres typically predominate 59 , Ventilation may, therefore, be adequate when the patient is awake and aroused but may become inadequate during oversedation Opioids and sedatives Opioids are commonly used for analgesia in the PACU and are mainstays in the treatment of acute severe pain. It is well known, however, that they are the classic respiratory depressants and produce a dose-related depression of total ventilation through a decrease in both respiratory frequency and tidal volume. The pharmacokinetic characteristics of the different opioids may, however, have a profound impact and those that are short acting after a single dose may produce extended respiratory depression manifesting in the PACU after prolonged infusion in the operating room Thus, the increase in CO<sub>2</sub> secondary to the reduction in spontaneous minute ventilation when an opioid is given will tend to counter the depression of ventilation if the onset of the opioid is slow. On the other hand, a bolus of a rapid-onset opioid may induce apnea before the CO<sub>2</sub> can increase sufficiently to stimulate ventilation Neuraxially administered opioids, either epidurally or intrathecally, depress both the hypoxic and hypercapnic responses even though the plasma levels are not significant and, thus, the route by which the opioid reaches the brainstem does not appear to be as important Delayed respiratory depression must, however, be considered with neuraxial administration, particularly if lipophilic opioids are used. Thus, postoperatively, such opioid administration is potentially associated with upper airway obstruction and desaturation The effects of midazolam on the upper airway may be more important than its effects on decreasing respiratory drive It reduces both the hypoxic and hypercapnic chemoreflexes, and it is the loss of the wakefulness drive that may account for the mild reduction in hypercapnic sensitivity Both the sedative and respiratory depressant effects of midazolam can be reversed by flumazenil, although the sedation reversal may outlast reversal of the respiratory depression The combination of opioids and sedatives can be synergistic in the extent of respiratory depression produced This synergism may result from the effect of the sedative on the wakefulness drive a hypothesis originally proposed by Fink [ 69 ], that cerebral activity associated with wakefulness is a component of the normal respiratory drive and the effect of the opioid on the chemoreflex and, thus, should be closely monitored in the PACU. Neuromuscular blocking drugs Neuromuscular blocking drugs are frequently used intraoperatively and residual neuromuscular blockade is commonly observed in the PACU. Residual neuromuscular blockade may produce postoperative hypoxemia by several mechanisms. These include the deleterious effects on both chemoreception and upper airway patency in addition to their predictable effects on the phrenic nerve-diaphragm neuromuscular junction In an study by Eikermann et al 73 , significant upper airway obstruction was detected in eight of 12 volunteers at a train-of-four TOF ratio of 0. At the receptor level, it is generally accepted that the neuromuscular blocking drugs act to block the neuromuscular junction nicotinic receptors, with little effect on neuronal nicotinic receptors. As a result, low doses of vecuronium appear to depress hypoxic ventilatory drive through depression of carotid body chemosensitivity in both rats 75 and humans Cardiovascular medications Medications used to support the cardiovascular system can also have significant respiratory effects, but auspiciously, most of them do not cause major clinical problems in the PACU. Of these agents, dopamine has the most pronounced ventilator effects because even low doses significantly blunt the hypoxic ventilatory response Several studies have demonstrated that a low-dose dopamine infusion has depressant effects on minute ventilation when given during hypoxia or during states with compromised oxygen delivery to tissues such as during exacerbation of congestive heart failure Other cardiovascular drugs, such as digoxin and adenosine, have ventilatory effects that can be readily measured; however, there is no evidence that any of these effects are clinically significant Identifying various procedures in the operating room that can prevent atelectasis or reopen collapsed alveoli are, therefore, worth mentioning. At the outset, using an anesthetic, such as ketamine, that enables maintenance of respiratory muscle tone will likely preclude the formation of atelectasis 17 , Nonetheless, if combined with muscle paralysis, atelectasis will most likely develop In anesthetized adults with healthy

lungs, alveolar recruitment reduces the amount of atelectasis and pulmonary shunt and, despite a concomitant increase in perfusion to poorly ventilated lung units, it also improves ventilatory efficiency as measured by CO<sub>2</sub> elimination

**Chapter 2 : Important Complications of Anaesthesia. Information | Patient**

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Complications in future pregnancies Some cesarean delivery complications “ like a hysterectomy ” make it impossible for a woman to have another baby. However, even if the surgery goes well and the mother heals, she may have difficulties with pregnancy in the future. This can happen because of scar tissue at the site of the cesarean delivery. In some cases, cesarean delivery scarring can connect the uterus to the bladder. When they are connected, future cesarean deliveries are more likely to damage the bladder. Future pregnancies may also implant in dangerous areas, like the cesarean delivery scar. The surgery may also leave the wall of the uterus weak, making a future vaginal birth difficult or even dangerous. Though many women can have a successful vaginal birth after a prior cesarean delivery, in some cases the uterus will tear open at the site of the old cut. If this happens, another cesarean delivery is needed to protect the mother and the baby.

**Infection after cesarean delivery** After the membranes have ruptured, the uterus is especially susceptible to infection “ the bacteria that normally inhabit the vagina which are generally harmless can easily spread to the uterus. If bacteria are in the uterus, a cesarean delivery incision may result in endometritis infection of the uterus. In very rare cases, the infection may be serious and require a hysterectomy. In extremely rare cases, the infection may result in death. Serious infections are rare in women who have planned cesarean deliveries before labor and before the membranes are ruptured. Problems like this are more common after long labors, when the membranes have been ruptured for a long time before the surgery begins.

**Post-cesarean wound infection** Some women develop an infection at the site of the incision on the outer skin layers, instead of in the uterus. This is often called post-cesarean wound infection. Infections of the wound are often associated with fever and abdominal pain. Infection of the skin or any layer of tissue that was cut can normally be treated with antibiotics. These infections can also cause abscesses that fill with pus. If an abscess exists, a doctor may have to re-open the wound to drain and clean the infected area. Sometimes, the infection can spread to other organs or the type of bacteria that infects the wound can be very aggressive. These infections are rare but can be dangerous. With proper treatment, such as antibiotics and hospitalization, even the most serious infections can be cured.

**Puerperal or postpartum fever and sepsis** Cesarean delivery is the single most important risk factor for postpartum infection. This infection often starts in the uterus or vagina. If it spreads throughout the body, it is called sepsis. Most of the time, the infection is caught early. It can usually be cured with antibiotics. If the infection is untreated and sepsis occurs, it is harder to treat. In rare cases, sepsis can be deadly. A fever in the first 10 days after the cesarean delivery is a warning sign for puerperal fever. Infections like urinary tract infections or mastitis infections in the breasts can be a sign of this complication. They should be treated quickly to avoid the spread of the infection.

**Bleeding** While the average blood loss for a vaginal birth is about cc about two cups , the average blood loss with a cesarean delivery is twice that much: This is because the pregnant uterus has one of the greatest blood supplies of any organ in the body. In every cesarean delivery, large blood vessels are cut as the surgeon opens the wall of the uterus to gain access to the baby. Most healthy pregnant women can tolerate this much blood loss without any difficulty. Occasionally, however, blood loss can be greater than this and cause or arise from complications. The following forms of dangerous blood loss can happen during or after a cesarean delivery:

**Postpartum hemorrhage** It is normal to lose a lot of blood during a cesarean delivery. When you bleed too much, this may be called a postpartum hemorrhage. They can also be caused by a tear in the vagina or nearby tissue, a large episiotomy, or a ruptured uterus. Some women have problems clotting blood, which makes it hard to stop bleeding after any type of cut, tear, or bruise. About 6 percent of deliveries result in postpartum hemorrhaging. Hemorrhages are emergencies, though, and should be treated immediately by a doctor. If you continue bleeding heavily after you are sent home from the hospital, call a health professional immediately for advice. After receiving treatment, most women make a full recovery in a few weeks. In some cases, women are given a blood transfusion during or after the cesarean delivery to replace lost blood. Medicine, IV fluids, iron supplements, and nutritious foods or vitamins are recommended

to help you regain your strength and blood supply after hemorrhaging. **Atony** After the baby and the placenta are delivered, the uterus must contract to close the blood vessels that supplied the placenta during pregnancy. Uterine atony is when the uterus remains relaxed, without tone or tension. This can happen after a long labor or the birth of a big baby or twins. When the uterus has atony, bleeding may be very fast. Fortunately, a number of very effective medications have been developed to treat uterine atony. Most of these drugs are variations of natural substances in the body called prostaglandins. With the use of prostaglandins, long-term complications from uterine atony are extremely rare. As the baby is delivered through the incision, the incision may tear into areas not intended by the surgeon. The areas to the right and left of the uterus have big arteries and veins that can be torn accidentally. Often, there is nothing the surgeon can do to avoid such tears; every obstetrician will see this problem many times. If the doctor notices a tear quickly, it can be safely repaired before too much blood loss occurs. Sometimes, these tears affect blood vessels near the uterus. Other times, the surgeon may accidentally cut into arteries or nearby organs during the operation. For instance, the knife sometimes hits the bladder during a cesarean delivery because it is so close to the uterus. These lacerations can cause heavy bleeding. They also might require extra stitches and repairs. In rare cases, damage to other organs requires a second surgery to fix.

**Placenta accreta** When the tiny embryo travels into the uterus, the cells that will form the placenta begin to collect on the walls of the uterus. These cells are called trophoblasts. Trophoblasts generally grow through the walls of the uterus and into the blood vessels of the mother. These cells play an important role in moving oxygen and nutrients from mother to fetus. They also move waste products from fetus to mother. As the fetus and placenta grow, the trophoblasts keep seeking blood vessels to support the growing fetus. They may even spread into other organs, such as the bladder. This condition is called placenta accreta. Placenta accreta is especially common in women who have had a cesarean delivery in the past and whose embryo, during a later pregnancy, implants in the area of the cesarean delivery scar. Although this complication is rare, doctors are now seeing it more often because of the large number of cesarean deliveries that have been performed in the last 10 years. The good news is that doctors are now able to recognize when women are at risk for this condition and are usually ready to deal with it. The bad news is that almost all cases require a hysterectomy to save the life of the mother. Since the chances of this happening tend to increase with each cesarean delivery a woman has, some women try vaginal birth after a previous cesarean delivery to reduce their risk of placenta accreta or a hysterectomy.

**Hysterectomy** Cesarean hysterectomy is the removal of the uterus right after a cesarean delivery. Even though the risk of a hysterectomy is higher after a cesarean delivery, bleeding requiring a hysterectomy may happen even after a seemingly normal vaginal birth. As with all of the complications listed above, cesarean hysterectomy is very rare. Most obstetricians will probably need to do an emergency hysterectomy only a few times in their careers. Women who have had a hysterectomy cannot have more children, but there are usually no extra side effects from this operation. Obviously, this is a terrible situation, and physicians try their best to avoid it. There is no question that cesarean hysterectomies save lives, though, especially when bleeding cannot be controlled by simpler measures.

**Planned cesarean hysterectomy** Cesarean hysterectomy Although a hysterectomy immediately following cesarean delivery is probably easier than performing one later, blood loss is greater. Under certain circumstances, however, a cesarean hysterectomy may be planned. This is only done when there is a serious need to do the hysterectomy for reasons unrelated to pregnancy. Otherwise, cesarean hysterectomies are done only in the case of emergency, as in the cases above. These blood clots can break off and travel to the lungs. If this happens, it is called a pulmonary embolism. This complication is the leading cause of death among pregnant women in most developed countries. If a blood clot is found early, it can be treated with use of a blood thinner such as Coumadin or Warfarin. Occasionally, there are no warning signs until after the clots have broken off and reached the lungs. Most women recover with treatment, but sometimes the clot can be so large that the mother dies. Blood clots are more common in the following situations: The mother is overweight.

## Chapter 3 : Complications of regional and general anaesthesia in obstetric practice

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Fasting is usually necessary starting about six hours before your surgery. You may be able to drink clear fluids until a few hours prior. Your doctor may tell you to take some of your regular medications with a small sip of water during your fasting time. Discuss your medications with your doctor. You may need to avoid some medications, such as aspirin and some other over-the-counter blood thinners, for at least a week before your procedure. These medications may cause complications during surgery. Some vitamins and herbal remedies, such as ginseng, garlic, Ginkgo biloba, St. Discuss the types of dietary supplements you take with your doctor before your surgery. If you have diabetes, talk with your doctor about any changes to your medications during the fasting period. If you take insulin, your doctor may recommend a reduced dose. If you have sleep apnea, discuss your condition with your doctor. The anesthesiologist or anesthetist will need to carefully monitor your breathing during and after your surgery. What you can expect Before the procedure Before you undergo general anesthesia, your anesthesiologist will talk with you and may ask questions about: Your health history Your prescription medications, over-the-counter medications and herbal supplements Allergies Your past experiences with anesthesia This will help your anesthesiologist choose the medications that will be the safest for you. During the procedure Your anesthesiologist usually delivers the anesthesia medications through an intravenous line in your arm. Sometimes you may be given a gas that you breathe from a mask. Children may prefer to go to sleep with a mask. The tube ensures that you get enough oxygen and protects your lungs from blood or other fluids, such as stomach fluids. Your doctor may use other options, such as a laryngeal airway mask, to help manage your breathing during surgery. Someone from the anesthesia care team monitors you continuously while you sleep. He or she will adjust your medications, breathing, temperature, fluids and blood pressure as needed. Any issues that occur during the surgery are corrected with additional medications, fluids and, sometimes, blood transfusions. Blood transfusions may sometimes be necessary, such as during complex surgeries. The anesthesia care team monitors your condition and delivers blood transfusions when needed. Blood transfusions may involve risks. These risks are greater in people who are older, have low red blood cell volume or are undergoing complex heart surgeries. After the procedure When the surgery is complete, the anesthesia medications are stopped, and you slowly wake either in the operating room or the recovery room. You may experience common side effects such as:

**Chapter 4 : Effects of Anesthesia on Brain & Body - When Seconds Count**

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Print Possible Complications of Anesthesia Anesthesia is generally safe, but complications can occur. Local anesthesia carries the lowest risk, and general anesthesia the highest. An allergic reaction to an anesthetic agent can be life threatening and can occur with any type of anesthesia. Drug allergies remain unknown until the substance is ingested, so many people are unaware of them. There are generally few adverse reactions to local anesthesia. Some patients experience nausea and vomiting, but that is usually caused by the sedative. There may also be soreness at the injection site. Regional anesthesia has a higher risk of side effects and complications, including the following: Temporary weakness or paralysis of the affected area Headache following spinal and epidural anesthesia. This usually begins within 12 to 24 hours after surgery and can last a week or longer. It may be caused by a loss of spinal fluid that occurs when the anesthetic is injected. Headache is often accompanied by nausea, vomiting, dizziness, light sensitivity, and a stiff neck. Hypotension low blood pressure Inability to urinate usually temporary and relieved by catheterization Backache Much less frequently, infection, nerve damage, or permanent paralysis can occur. Because it affects the entire body, general anesthesia has the potential to cause the greatest number of side effects and complications. Most side effects clear up within 24 hours or so. The most common ones are: Sore throat caused by the devices used to keep the airway open Drowsiness or feeling tired hours after surgery Nausea and vomiting Headache, dizziness, and vision problems Damage to teeth caused by airway devices General anesthesia also carries the risk for serious complications. Serious complications include the following: Stroke Brain damage Death In August , results of a study published in Pediatrics indicated that anesthesia before the age of 3 may increase the risk for deficits in language and abstract reasoning. Certain inhaled anesthetics can trigger a disorder called malignant hyperthermia in people who carry the gene for it. If this disorder is known to run in the family, the patient must inform the anesthesiologist before surgery.

*Some specific complications of general anaesthesia Anaphylaxis. Anaphylaxis can occur to any anaesthetic agent and in all types of anaesthesia. [] The severity of the reaction may vary but features may include rash, urticaria, bronchospasm, hypotension, angio-oedema, and vomiting.*

To identify common human errors; how to avoid and identify them early enough that the complication does not turn into a disaster. To identify how to safely navigate your patient through a crisis to a successful recovery. Preparation including a thorough patient history, knowledge of anesthetic drugs and equipment, treating each patient as an individual, and careful monitoring enables the anesthetist to identify and take the appropriate actions in a timely manner before a complication turns into a disaster. Precautions such as an individual emergency drug sheet and knowing the resuscitation code for each animal will save time and in turn lives. All surgery rooms should minimally contain a crash cart or box with emergency drugs and syringes available at all times. The goal is not only to know the appropriate action to prevent complications but also be prepared to handle common complications when they arise. Previous planning prevents poor performance. Aspiration should be identified, noted in the record, and treated. If aspiration is treated correctly, you will rarely have complications. Depressed respiratory drive is a common side effect of many drugs we use in anesthesia and this can produce hypoventilation. Administering intermittent positive pressure ventilation IPPV one to two times a minute is an easy way to help the patient ventilate adequately. A capnogram or capnograph is an excellent monitor to assist in recognizing hypoventilation. These devices monitor end-tidal carbon dioxide ETCO<sub>2</sub>. If you are certain that you are providing adequate minute volume, some other causes of increased ETCO<sub>2</sub> are faulty oneway valves, expired absorbent granules, a faulty scavenging system, or an inadequate oxygen flow rate. Respiratory obstruction and laryngospasms typically go hand in hand. Prevention starts with correct intubation. In felines, avoid anticholinergics, unless warranted. Another reason for obstruction is a kinked ET tube. Two common causes of kinked ET tubes are flexion of the neck and an ET tube that extends past the incisors which becomes bent outside the oral cavity. Feline intubation can be a challenge if done without all the proper tools. To avoid laryngospasms and trauma, use lidocaine dripped onto the laryngeal folds and ensure your patient is in a proper plane of anesthesia prior to intubating. When administering lidocaine, wait 30 to 60 seconds for it to take effect! In felines, extubate earlier than in canines, leaving the ET tube in too long may cause laryngospasm. If you are in doubt as to how a patient may recover, be prepared, with all the necessary equipment, including an oxygen source and an induction agent, to reintubate at any time in the anesthesia procedure, including during recovery. Airway trauma may occur from multiple causes. One cause is forceful intubation, which may irritate or even damage the laryngeal folds. Another cause of airway trauma is when the stylet extends past the endotracheal ET tube causing a tear in the trachea. Always have safety practices in place to prevent a metal stylet from coming into contact with the trachea. ALWAYS disconnect your patient from the anesthetic circuit when repositioning the patient on the table. The last cause of tracheal damage is pressure necrosis of the trachea due to over inflation of the ET tube cuff. The goal is to use an ET tube sized so that there is little need for air in the cuff. Complications from airway trauma can range from minor coughing for a few days after anesthesia to subcutaneous emphysema. This is the minimum pressure to ensure adequate perfusion and oxygen delivery to major organ systems. Hypotension can lead to shock, ischemia of vital organs resulting in organ dysfunction or even organ failure. Causes of hypotension in anesthetized patients include drug overdose, hypovolemia, hypoxia, hypercarbia and the normal effects of some anesthetic drugs. Treating the underlying cause is the first step. When blood pressure is trending downward, a simple action, such as reducing your inhalant by a fraction can have positive results. Often a fluid bolus and decrease in anesthetic will return the patient to a normotensive state. If your patient is on an anesthetic CRI, this may also need to be reduced. Known hypovolemia should always be corrected prior to surgery. If hemorrhage occurs during surgery, crystalloid fluids should be administered to treat hypovolemia at 2. If fluid boluses and a drop in anesthetic does not correct the hypotension the cause may be due to reduced myocardial contraction. If this is the case, an inotropic drug such as dopamine or

dobutamine can be administered as a constant rate infusion CRI to improve the contractility of the heart. To be effective, the patient must be normovolemic prior to administration of inotropic drugs. Inotropic drugs increase the contractility of the heart muscle. Decreased venous return is another cause for hypotension due to the compression of the vena cava. Pressure on the vena cava will restrict blood return to the heart. This may be seen anytime there is a large mass, fluid or pressure in the abdominal area taking up space. Examples include pregnant bitches, large abdominal masses, gastric dilatation-volvulus GDV, hemoabdomen, etc. These patients benefit from being prepped in lateral recumbency and placed in dorsal recumbency at the last minute possible prior to surgery. Hypothermia will decrease anesthetic requirements and may produce hypotension if anesthesia is not decreased. Warming the patient and decreasing the inhalant might increase blood pressure. Cardiac arrhythmias are another anesthetic complication seen in the anesthetized patient. Bradycardia, atrioventricular AV blocks, sinus arrest and atrial standstill may all be seen in anesthetized patients. Some arrhythmias are a direct result of drugs used in anesthesia but they can also be due to increased vagal tone, surgical stimulation or hyperkalemia. Treatment will depend on the cause. Hyperkalemia should always be treated prior to induction of anesthesia. Often when a bradyarrhythmia is due to a drug or increased vagal tone it can be successfully treated with an anticholinergic. The exception is when an alpha-2 agonist has been used. Another common arrhythmia is ventricular premature complexes VPCs. These are often seen in patients with gastric dilatation volvulus, trauma hit by car, hypercarbia, electrolyte imbalances, hypoxia, myocardial ischemia, splenic masses, and pancreatitis. VPCs may not need to be treated if cardiac output is adequate. Monitoring mucous membrane color, capillary refill time and blood pressure will help you assess cardiac output. Runs of two to three VPCs per electrocardiogram ECG screen are typically not treated if they are transient and cardiac output is deemed adequate. If the underlying cause of the VPC is pain, administering an analgesic easily treats the cause. Doing a check of your anesthetic machine first thing each and every morning can eliminate most of these errors. This should include pressure checking the machine as well as visual inspection of all the machine parts. By following this guideline, you have ample time to ensure the anesthetic machine is set up correctly, there is an adequate supply of oxygen, the ventilator is working properly, the vaporizer is filled, absorbent granules are viable, and all parts of the anesthetic machine are working correctly. Previous planning prevents poor performance Absorbent granules should be changed after 6 to 10 hours of use. If your granules are still viable, they will produce heat when a patient is connected to the breathing circuit and, if using an ETCO<sub>2</sub> monitor, the reading will be 0 mmHg on inspiration. Do not rely on color if a machine is not being used, some granules will revert back to white when the patient is disconnected. After applying gloves, touch the granules; if they are viable they will be easy to crumble in your hands. Expired granules will not filter out carbon dioxide and will cause the patient to breath carbon dioxide. Overfilled vaporizers and those that are tipped beyond 45 degrees result in anesthetic leaking into the oxygen bypass that results in uncontrolled amounts of inhalant being delivered to the patient. To be safe, vaporizers should be filled so that ANY part of the fluid line should reach a level even with top etched line in the sight glass. Individual vaporizer instructions may be different, please refer to the owner manual or call tech services. If the vaporizer is overfilled or tipped beyond degree actions must be taken prior to the vaporizer being used. Please refer to your individual vaporizer instructions or call tech services. The most common causes for the pop off valve to be in the closed position is when assisted breathing is provided or during the pressure check of the machine and the valve was not re-opened. You may purchase inexpensive valves that can eliminate this problem during intermittent positive pressure ventilation IPPV but you must manually close the pop off valve to pressure check a machine so squeezing the air out of the reservoir bag at the end of the pressure check is a necessity. Another option when providing IPPV is to never take your hand off the pop off valve while administering a breath, and when done, give the bag one last squeeze to ensure the pop off valve is open. A closed pop off valve can cause death due to barotrauma! One of the first signs of the increased pressure in the thoracic cavity will be bradycardia. Administering the wrong drug or the wrong dosage is a preventable human error. When this occurs, inform the veterinarian immediately! Drug mistakes can be avoided by having checks and balances in place. All drugs and fluid rates should be checked by at least two highly skilled employees. All syringes should be clearly marked with the name of the drug they contain. When in doubt, throw it out! Two

common complications that are easily avoided are thermal burns and corneal abrasions. Corneal abrasions are easily avoided by applying sterile ophthalmic lubricant to the eyes of all patients who are undergoing an anesthetic protocol. Thermal burns happen when inappropriate warming methods are used. Thermal burns show up days after the procedure and can be catastrophic, leading to skin necrosis and loss of skin. Do not use unapproved heating methods. Signs of impending cardiac arrest include any of the following: Respiratory arrest, if not treated, may lead to cardiac arrest. There are many emergencies that occur during recovery. Turning a patient at the end of surgery often results in hypotension. Regurgitation can be a problem if the airway is not protected.

**Chapter 6 : General anesthesia - Mayo Clinic**

*Anesthesia is generally safe, but complications can occur. Local anesthesia carries the lowest risk, and general anesthesia the highest. An allergic reaction to an anesthetic agent can be life threatening and can occur with any type of anesthesia.*

This article has been cited by other articles in PMC. Abstract Any anaesthetic technique, either regional or general, has potential for complications. Moreover, it has been seen that in obstetric patients, the complications are potentiated due to pregnancy-related changes in physiology and due to various other factors. This review has highlighted the possible complications of regional and general anaesthesia encountered during the obstetric anaesthesia practice. Changes in maternal physiology during pregnancy and the care of both mother and foetus present unique challenges to the obstetric anaesthetists. Although new systems and technologies are developing to provide consistent and safe anaesthetic care to pregnant mothers, the modern-day obstetric anaesthetist has to also grapple with issues related to changing population characteristics, including maternal obesity, advanced maternal age and an increased complexity of medical diseases including cardiac diseases, which may affect women with a reproductive potential. Complications of regional anaesthesia and general anaesthesia that are commonly encountered during obstetric anaesthesia are discussed in this review. One study from the UK has shown that the rate of regional anaesthesia for elective caesarean section CS rose from The following complications can occur with central neuraxial blockades CNB. Post-dural puncture headache PDPH: PDPH is a common complication of neuraxial blockade. There is the potential for considerable morbidity due to PDPH. Therefore, the authors recommend approximately 24 h of conservative therapy. Serious neurological complications related to regional anaesthesia are, fortunately, very rare. Direct trauma to the nervous tissue may occur at the level of the spinal cord, nerve root or peripheral nerve. The epidural needle or spinal needles may touch the nerve roots or may directly injure the spinal cord. Scott and others, monitored, epidural blocks in parturients, finding only 38 single-root neuropathies 0. Cauda equina syndrome is another annoying complication of CNB. Rigler and others, postulated that the combination of trauma, maldistribution and a relatively high dose of local anaesthetic resulted in this neurotoxic injury. Epidural abscess is usually due to infection in the body seeding the epidural space. In one review, epidural anaesthesia was associated with only one in 39 epidural abscesses. Other symptoms include lower extremity pain, weakness, bowel and bladder dysfunction and paraplegia. Urgent surgical treatment is necessary. Low-molecular weight heparins have been responsible for over 35 epidural haematomas following regional anaesthesia, and should be considered a strong relative contraindication. Hypotension following neuraxial blockade is due to sympathetic inhibition, which causes a significant decrease in the venous return due to dilatation of the resistance and capacitance vessels. Decreased pre-load after spinal anaesthesia initiates reflexes that cause severe bradycardia. Atropine is typically used as the first line of therapy and also for prophylaxis. Sometimes, severe syncope may occur along with hypotension and bradycardia due to reflex cardiovascular depression. The cause was identified as compression of the inferior vena cava by the gravid uterus, reducing the venous return and right atrial pressure. An overall incidence of seven cases of cardiac arrest for every 10, spinal anaesthetics versus one case for every 10, epidural anaesthetics has been reported. Three possible mechanisms, e. Greater sedation has been observed with high spinal blocks. The possible mechanisms are the rostral spread of local anaesthetic agents or a reduction in the function of the reticular activating system caused by an interruption of the afferent inputs. There is some evidence in the early literature that cerebral hypoxia might occur during spinal anaesthesia in some patients. A circulatory etiology for cardiac arrest during spinal anaesthesia is directly or indirectly related to the blockade of sympathetic afferents and decrease of catecholamine release by the adrenal medulla. However, it may occur with normal dosage also due to rostral spread of anaesthetic drug. Initially, it was thought that increased pressure in the epidural space can compress the subarachnoid space, thereby disseminating the local anaesthetic. Recent research has shown that it is due to a hormonal progesterone effect. Subdural or subarachnoid blocks can happen unintentionally during epidural placement, causing an accidentally high block. This incidence is more

in spinal anaesthesia than in epidural anaesthesia. Previous studies reported that epidural anaesthesia for labour and delivery was associated with long-term backache. However, randomized controlled trials and prospective cohort studies have convincingly proved that new, long-term, post-partum back pain is not caused by intrapartum epidural analgesia. Epidural catheters may rarely break or shear. If part of a catheter is left in a patient, the patient should be informed. However, no surgery or attempts to retrieve the catheter are warranted unless there are persistent neurologic symptoms. Invariably, this happens with accidental intravascular injection. The previously reported incidence was 0.0%. Prompt recognition and management is essential for better prognosis. The incidence of inadequate analgesia in uniport catheters ranges from 31 to 38%.

**Complications with non-central blockade regional anaesthetic techniques** These techniques are to be employed when the facilities for central blockade are not available or CNB is contraindicated. General anaesthesia may lead to loss of airway control, with anoxia and aspiration of gastric contents. This risk associated with obstetric general anaesthesia has led to regional techniques being used wherever possible. General anaesthesia is now used mainly for true emergency cases where there is insufficient time for a regional technique. The major concerns regarding the use of general anaesthesia for the obstetric population are difficulty in airway management, failed intubation and acid aspiration. Awareness and drug toxicity are few other complications associated with general anaesthesia. It is well known that pregnant women and women giving birth offer special anaesthetic challenges, where a four to five-times higher frequency of intubation problem and faster development of hypoxia are the main cause of mortality. However, the characteristics of pregnant women have changed, whereby average maternal age, body mass index and number of comorbidities have all risen. When general anaesthesia is to be used in obstetrics, the method of airway management will depend on the urgency of the procedure and the anticipated ease or difficulty of intubation and ventilation. All equipment for routine and emergency airway management should be immediately available [ Table 1 ]. If tracheal intubation is unsuccessful in the first attempt, steps outlined in the difficult airway algorithm should be initiated [ Figure 1 ].

**Table 1 Equipment for airway management in obstetrics**

Routine	Laryngoscope, multiple blades Mac 3,4, Miller 2,3
Endotracheal tube	5.