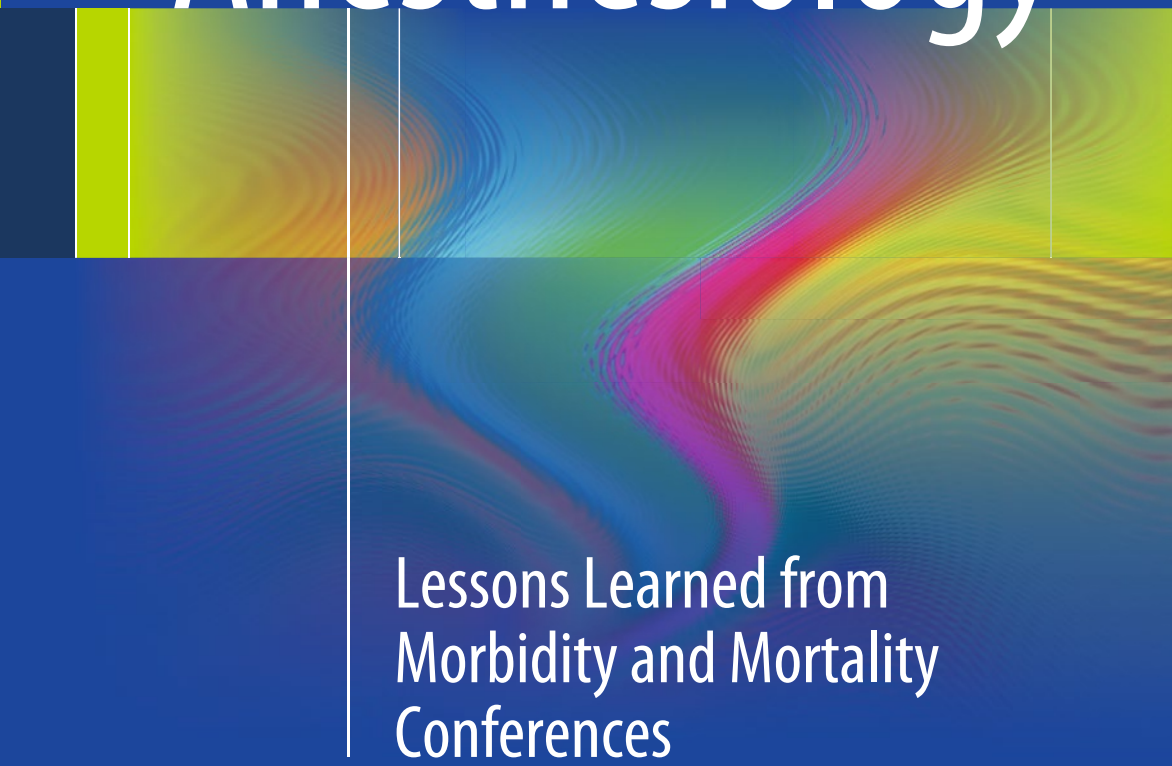
Jonathan L. Benumof *Editor*

Clinical Anesthesiology



Lessons Learned from Morbidity and Mortality Conferences

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Foreword

One of the first things I did when I joined the UCSD faculty in January of 2000 was attend the Wednesday morning Anesthesiology Morbidity and Mortality Conference (we now call it the Departmental "Continuous Quality Improvement Conference" for the sake of "Quality Improvement Correctness"). I was joining as general faculty, having served as Clinical Director for a number of years at my previous institution. I considered myself a "go to" person for complex clinical issues, and was accustomed to being the "big kid on the block."

The Departmental Morbidity and Mortality Conference was amazing. First, I noticed that the conference was moderated by a senior faculty with superb teaching skills (Dr. Benumof). The resident was expected to present the case, but the learning points, and, yes, arguments about the medical and anesthetic care were provided by the faculty. The presenting resident was virtually immune from harm! This was a learning environment far superior to the classic conference in which the presenting resident would simply stand alone at the front of the room, receiving criticism (daggers?) from all angles by the faculty. I later found that it was not uncommon for experts from other services to be invited, and that this was the flagship teaching exercise for the department. To this day we have all resident applicants attend this conference, so they experience, first hand, this learning experience our residents and faculty enjoy weekly.

The second thing I noticed was that the academic quality and information provided at the conference were outstanding. I may have previously considered myself a clinical expert, but these UCSD folks were in a completely different league. When the conference was over, I proceeded directly to the campus bookstore, recognizing that my level of knowledge was not going to suffice in this place, with the likes of Dr. Jon Benumof moderating the case conferences. The moderators here have inspired me to "raise my game" many notches, and I will forever be grateful to them for this.

When I was a resident, I was told by my Chair, Dr. Paul Poppers, that one should take copious notes at the Morbidity and Mortality conferences and save the write-ups in a binder with the notes for later review. By doing this I would be prepared for very complex cases, and I'd also be prepared for the board examination. I found his

advice to be sage, and I advise our residents similarly. The residents, however, almost never follow this advice, occupying the conference time with listening, answering the occasional "pimping" question, and eating muffins. The residents just don't seem to want to take notes. My fear about their not taking notes is that they will forget all the great teaching points presented at the conferences.

By writing this book, Dr. Benumof and the authors have fully addressed this issue for all of us! Herein is a wonderful compilation of UCSD case discussions, with chapters that are far superior to any notes one could take. The chapters have very readable prose, tables, figures, and other information that will be invaluable to any practitioner or student of anesthesiology. I still think the attendees of our conferences should take notes, and I still do. This relevant, well-written collection, however, allows us the luxury of simply listening (or reading), and eating muffins if we want to, while we learn the critical points of caring for some of the most complex patients in some of the most challenging circumstances.

San Diego, CA, USA Gerard R. Manecke Jr., MD

Preface

This book is a compilation of selected cases presented at the weekly University of California San Diego Medical Center Department of Anesthesiology Morbidity and Mortality (M&M)/Quality Improvement (QIC) conference that I moderated over the last 4–5 years. For all cases presented, a narrative of the case was prepared and published to the department by the person presenting the case (usually an anesthesia resident) prior to the actual case presentation. Whenever I moderated a case, I made notes as to what were the questions and the lessons going to be during the actual presentation of the case at the conference; these questions and lessons were those that I thought arose naturally from the narrative of the case.

During the actual presentation of the case, the answers to questions and the teaching of lessons was provided by the audience (usually the learned faculty) and by me by the communication vehicles of speech, using an eraser board, and by projection of prepared slides. Immediately after the case conference I made additional notes of what actually happened and the educational dialogue that had occurred at the conference. It is the preconference and immediate post-conference notes on the M&Ms/QICs that form the basis of the book, i.e., what were the questions answered and lessons learned.

For each of the previously presented cases that I selected for this book, I selected a senior resident (majority of the case chapters) or a faculty person to write the case chapter. The senior residents who wrote the case chapters took a department-approved writing-of-the-book 1 month elective in their third year with me. With both the senior residents and faculty, I discussed in detail the narrative of the case and my notes on questions answered and lessons learned that had naturally popped out of the narrative of the case and actually happened at the conference. Each author wrote multiple drafts of each chapter in close consultation with me until a high-quality final teaching product was arrived at. Each chapter consists of the narrative of the case followed by the lessons learned/questions answered.

The manner of preparation of each case chapter, as described above, has one limitation, and that is occasionally the data in the narrative is incomplete. I cannot remember why a piece of data, that may seem logical and desirable to have been included in the narrative in the first place, was not in the narrative or in my notes:

unfortunately these occasional little informational pieces are lost. Usually the answers to questions and the lessons learned will make a small comment on this minor problem.

Why produce this book and what is unique about this book? This book is a unique valuable teaching-of-anesthesia contribution for several reasons. First, it is unique in that the questions answered and lessons learned flow naturally out of real cases that actually happened. As a consequence of this format, many of the questions raised and lessons offered are unique because real cases generate issues in a way that traditional textbooks cannot do and do not cover. Just a few examples are: Why does a right main-stem bronchial intubation sometimes cause a hyperexpanded right lung? How to make anesthesia for office and same day surgery safe and efficient; practical aspects of doing many procedures and managing situations and complications that are simply not covered anywhere else. Simply browsing through the titles of the lessons learned for each case in this book will prove this point. Second, if the facts of these real cases happened once, then they are going to happen again; so it is no leap in logic that this book will be instructive, and preventive of, the same problems that will come up in the future. Third, since the answers to questions and lessons learned flow naturally from real cases, it is simply more fun and much easier to understand, absorb, learn, and remember the educational material from this book than from a traditional book. Indeed, it is very possible that the way readers remember all the teaching material in all the cases is that they remember the real cases.

Finally, for all the reasons above, the reader gets to learn anesthesia in a way that has never been done before; try it, you will like it.

San Diego, CA, USA Jonathan L. Benumof, MD

Contents

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Part I
Respiration-Related Cases

Chapter 1
Cannot Ventilate, Cannot Intubate Due to Airway Hemorrhage

Ankur P. Patel

A code blue was called at approximately 2100. Upon arrival to the patient's room, cardiopulmonary resuscitation with chest compressions and ventilation via mask was in progress for a pulseless electrical activity (PEA) arrest. A respiratory therapist was at the head of the bed attempting to ventilate via mask using a self-inflating Ambu bag (see **Lesson 1** [**L-1**]). Oxygen saturation of the patient was noted to be 60 % and downtrending. While the responding anesthesiologist was preparing to secure the airway, pertinent medical history and preceding events were sought from the primary team. This was a 42-year-old Hispanic male transferred from an outside hospital for further treatment of neck abscesses who became unstable after a witnessed episode of coughing resulting in copious bleeding from his mouth and left neck, leading to PEA arrest. He was given 1 mg of epinephrine and 40 units of vasopressin with continued cardiopulmonary resuscitation (CPR) as described above.

Once at the head of the bed, frank blood was coming out of the patient's mouth, running over his chin and down his neck. Two large external neck wounds were noted with concern of communication into the oropharynx. An attempt at laryngoscopy was made with a Macintosh 4 blade and a grade IV view was obtained. The patient's oropharynx was full of abundant clotted blood with active bleeding pooling in the posterior oropharynx. Tissue anatomy was grossly deformed from blood clots and edematous tissue, and the epiglottis was unable to be identified.

Oropharyngeal suctioning was attempted, but blood continued to accumulate, as the suction was unable to keep up with the profuse bleeding. After attempting to optimize the patient's sniffing position by placing several rolled towels under the patient's shoulders, another direct laryngoscopy attempt with a Macintosh 4 blade was undertaken. A grade III view was transiently visualized but was quickly lost by the pooling of blood. With oxygen saturation continuing to decrease into the low

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50 % level, blind intubation with a silver-coated 7.0 mm internal diameter ETT was attempted but was unsuccessful as determined by negative end-tidal CO_2 on the Easy Cap detector (**L-2**). The endotracheal tube was quickly removed. Another anesthesiologist attempted direct laryngoscopy with a Macintosh 4 blade and another grade IV view was obtained secondary to the continued oropharyngeal bleeding and grossly distorted anatomy. No attempt was made to intubate in this instance (**L-3**, **L-4**). A size 4.5 Cookgas laryngeal mask airway (LMA) was immediately placed and inflated. End-tidal CO_2 was detected and the patient could now be ventilated and oxygenated with oxygen saturations increasing to mid-1970s, and no blood was noted to be accumulating in the LMA. Patient eventually converted to sinus tachycardia after the third milligram of epinephrine; total time of PEA was approximately 10 min. The on-call anesthesiology attending made the decision to perform emergent cricothyroidotomy since the patient was only maintaining oxygen saturation in low 80 % level (**L-5**). The trauma surgery service was called for placement of an emergency surgical airway. While awaiting the arrival of the trauma surgery team, the patient's neck was sterilely prepped, the patient was paralyzed with 100 mg of rocuronium, and the instruments were prepared for surgical airway. Trauma surgery arrived and an emergent cricothyroidotomy was performed, and a 6.0 mm endotracheal tube was placed through the surgical airway. Oxygen saturation eventually increased to 100 % and patient was transferred to the coronary care unit (CCU) where he was eventually taken to interventional radiology (IR) for emergent embolization of a left lingual artery pseudoaneurysm secondary to abscess invasion.

Lessons Learned

L-1: How do you know when you are effectively positive pressure ventilating a patient? What is the utility of the self-inflating bag?

Effective ventilation via mask can be confirmed when end-tidal CO_2 is detected (sine qua non), there is visual observation of adequate chest rise, and there are bilateral breath sounds and by the tactile feel of the breathing bag during positive pressure (PP) inhalation and exhalation. Portable end-tidal CO_2 monitors are sometimes unavailable in code situations. Often in a CPR situation with active chest compressions, it is impossible to tell if there is bilateral chest movement during ventilation via mask. Auscultating breath sounds while ventilating via mask during a code with external compressions is virtually useless. The sensitivity and accuracy of the tactile feel of the breathing bag during PP inhalation and exhalation is far superior with a Mapleson circuit (*see* Fig. 1.1) than a self-inflating (Ambu) bag.

A Mapleson circuit allows the user to immediately know the following:

1. If the seal is adequate

   If so, the bag will inflate on expiration and the pop-off valve can be adjusted to optimize the amount of positive pressure needed. In educated hands, this tactile feedback is extremely important and simply not obtainable with a self-inflating (Ambu) bag.

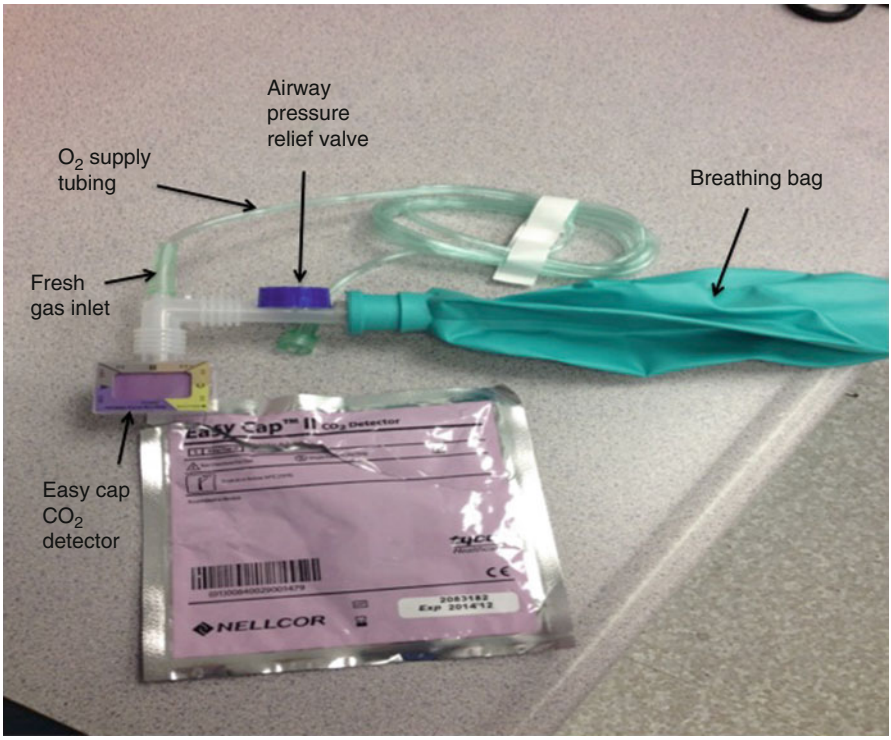

Fig. 1.1 This is a photograph of a Mapleson circuit attached to an Easy Cap II CO_2 detector. The Mapleson circuit consists of a dark-blue airway-pressure relief valve and fresh O_2 gas inlet connected to oxygen supply tubing at the universal elbow connector

2. If fresh gas is flowing

   Again, with the pop-off valve closed and a good seal, the practitioner will know if there is sufficient fresh gas flows to deliver to the patient instantly.

3. The compliance of the patient's airways

   If the bag is tight, with a good seal, the anesthesiologist should begin to form a differential for increased airway resistance.

4. If there is color change on the Easy Cap CO_2

   The anesthesiologist knows that there is gas exchange taking place and that there is enough cardiac output to generate blood flow to the lungs where gas exchange can take place.

The only real utility of the self-inflating Ambu bag is when there is no external source for supplemental oxygen. In this situation, one cannot rely on fresh gas flows to inflate the bag for positive pressure ventilation. A self-inflating bag therefore can deliver up to 18 % FiO_2 when not connected to an oxygen source. The self-inflating bag can be connected to an external oxygen source and provide an FiO_2 near 100 %,

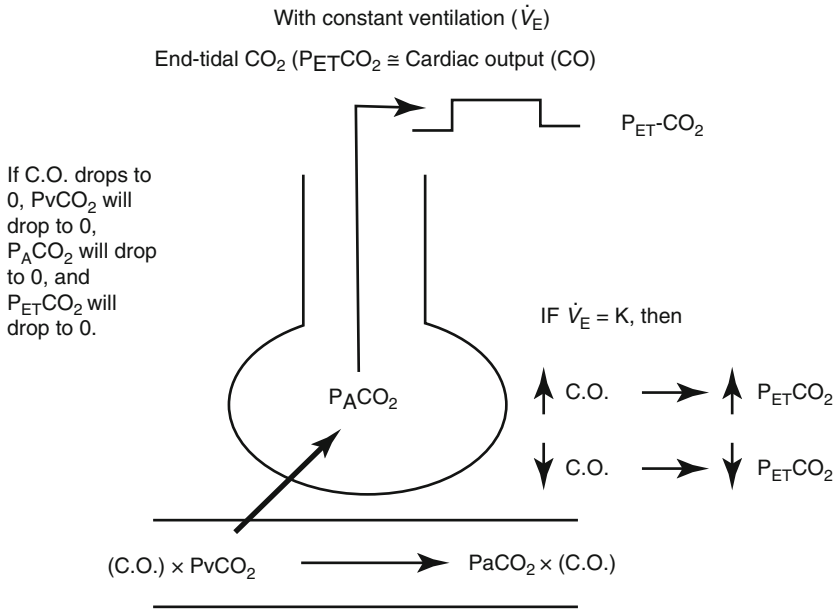

Fig. 1.2 When there is no CO_2 reaching the lungs (P_vCO_2), there will be no alveolar CO_2 (P_ACO_2) and no end-tidal CO_2 ($P_{ET}CO_2$)

but the tactile ability to detect the seal and the compliance of the patient's respiratory mechanics are lost.

L-2: What are the causes for false-negative end-tidal CO_2 readings?
There are a number of situations where an endotracheal tube is in the trachea, but there is no detected expired end-tidal carbon dioxide. It's important for the anesthesiologist to have a high index of suspicion for these events, especially when the endotracheal tube was visualized to go through the vocal cords with conventional laryngoscopy.

1. There may not be enough cardiac output [Q_t] for pulmonary gas exchange to take place. In this instance, there will be no end-tidal carbon dioxide and the P_aCO_2 on a blood gas reading will be elevated and the P_aO_2 will be low (*see* Fig. 1.2).
2. There may be kinks in the system leading exhaled gas to the monitor; these kinks can occur in the sampling line, in the endotracheal tube (ETT), and biting on the tube by the patient. Alternatively and oppositely, there may be a disconnect.
3. There may be obstruction in the ETT from four types of fluids:

   - Blood
   - Pulmonary secretions (edema, mucus)
   - Pus
   - Gastric contents

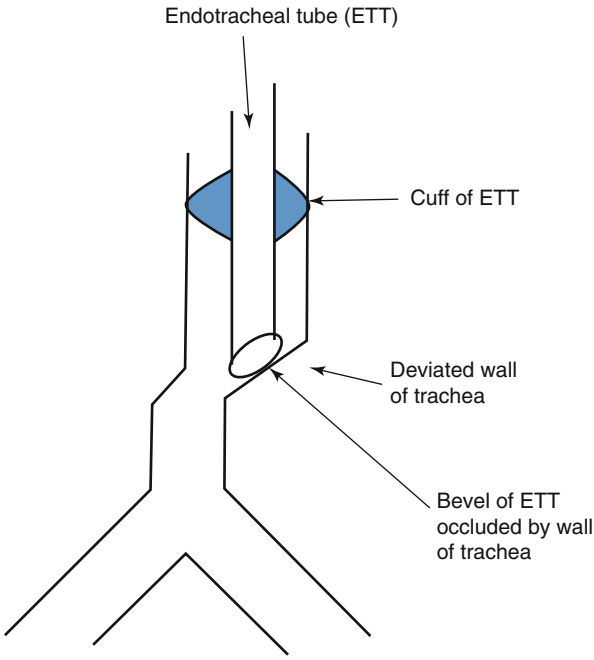

Fig. 1.3 The tip of the ETT may be occluded by the wall of a deviated trachea, thereby resulting in no $P_{ET}CO_2$ detection

4. Obstruction of the tracheobronchial tree and/or the ETT by masses in the airway. The masses may be intraluminal or extraluminal.
   (a) Intraluminal – If these masses were located in the trachea distal to the tube, CO_2 detection would be negative with an ETT in situ in the trachea. However, if the object was in the mainstem bronchi and the contralateral airway was patent, a normal CO_2 value may be detected.
   (b) Extaluminal – If a mass compresses the airway from outside the lumen of the airway, distal to the ETT, then the end-tidal CO_2 would be negative despite having an endotracheal tube in the trachea. This is an all-too-common story in patients with mediastinal masses that compress the carinal area on loss of spontaneous ventilation.
5. Events in a hemithorax may cause the tip of the ETT to become obstructed by the airway itself. For example, contralateral pneumothorax, hemothorax, chylothorax, or bulging thoracic aortic aneurysms may cause tracheal deviation so that the wall of the deviated trachea occludes the distal lumen of an ETT (*see* Fig. 1.3).

A swift assessment of the monitors and the breathing circuit for disconnects and passing a suction catheter down the endotracheal tube will begin a differential diagnosis work-up of these problems. A fiberoptic bronchoscope through the ETT will allow the anesthesiologist to confirm tracheal intubation as well as diagnose any foreign material or obstruction in the airway from the tube to the distal airways.

L-3: How does an anesthesiologist know when there is a cannot ventilate, cannot intubate situation?

As a prerequisite to knowing when further attempts at ventilation via mask and intubation by conventional laryngoscopy are likely to be futile, it is important to understand that:

1. Laryngeal edema and bleeding due to laryngoscopy are directly proportional to the number of attempts at laryngoscopy.
2. It is natural for an anesthesiologist to use an increasing amount of force with each subsequent laryngoscopy, potentially causing harm to teeth, lips, eyes, gums, and laryngeal structures with the laryngoscope and/or endotracheal tube.
3. By definition, if you have given laryngoscopy your optimal effort, then all further attempts accrue only risk and no further benefit.

L-4: What is the best/optimal attempt at (A) laryngoscopy and (B) ventilation by mask?

A. Laryngoscopy
   See Fig. 1.4.
B. Ventilation by mask
   See Figs. 1.5, 1.6, and 1.7.

L-5: How does an anesthesiologist decide whether to use a supraglottic or subglottic rescue approach in a cannot ventilate, cannot intubate situation?

A cannot ventilate, cannot intubate (CVCI) situation can be due to the given natural anatomy of the patient or to the presence of periglottic pathology.

Prototypical examples of given natural anatomy that can cause a CVCI situation are very large tonsils; a very large tongue, severe retrognathia; severe micrognathia; very prominent incisors; relatively anterior larynx; very narrow, high-arched palate; and a very small mouth opening.

Prototypical examples of periglottic pathology that can cause a CVCI situation are large periglottic masses, large abscesses (retropharyngeal, tonsillar), large hematomas, hemorrhage in the airway, radiation-related changes, severe epiglottitis, and laryngeal stenosis.

If given anatomy without significant pathology is the cause of the CVCI situation, then supraglottic airways are good choices because they separate the tongue from the posterior pharynx very well. If periglottic pathology is the cause of the CVCI situation, then subglottic airways are good choices because they are below the obstruction (*see* Table 1.1).

If it is thought that periglottic pathology is causing a CVCI situation and the alternative to performing a surgical airway is the death of the patient, then it is logical that the resuscitating physician be committed to performing the surgical airway and do so immediately. To perform this maneuver, the anesthesiologist should position the patient on a roll placed between the patient's shoulder blades so as to extend the neck and chest. Then, quickly sterilize the neck, and make a vertical incision in the cricothyroid membrane (*see* Fig. 1.8) and place an ETT or tracheostomy for emergent airway.

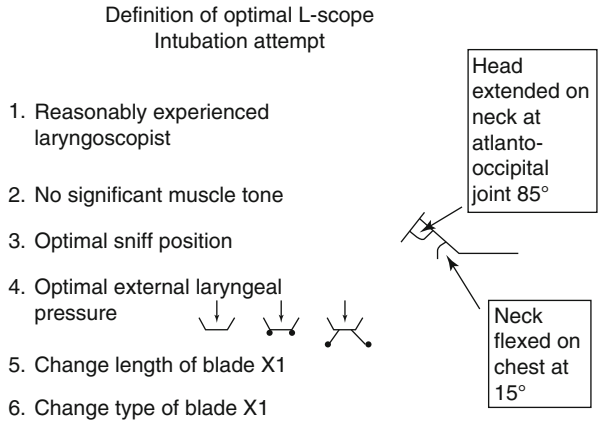

Fig. 1.4 Definition of optimal laryngoscopic intubation attempt. *1* A reasonably experienced laryngoscopist means that the laryngoscopist is a late second-year resident or higher in training. An early first-year resident or CRNA in training would not qualify as an experienced laryngoscopist. *2*. No significant muscle tone means that the mouth can be opened reasonably widely to permit laryngoscopy. *3* The optimal sniffing position is shown in the figure with the neck flexed on the chest at 15° and the head extended at the atlanto-occipital joint so that the angle between the occiput and the neck is 85°. *4* Optimal external laryngeal manipulation means that the right hand should manipulate the larynx by pressing on the midpoint of the thyroid cartilage externally during the first laryngoscopy so that the laryngeal structures and vocal cords can be better visualized (see Fig. 1.5). The above four criteria should be met on the first attempt at laryngoscopy. It is reasonable to proceed to step *5* and change the length of the blade either when the tip of a Macintosh blade is too short for the tip to be advanced into the vallecula or when a Miller blade cannot reach and trap the epiglottis. *6* Finally, changing the type of blade may be appropriate. For example, if the epiglottis is obscuring the view (long floppy epiglottis) and the tip of the blade is in the vallecula (if using a Macintosh) or if there is a decreased thyromental distance, then a Miller blade may be more appropriate. If a narrow palate or small inter-incisor distance is encountered, a Macintosh blade may create more space for the intubating practitioner. Points *5* and *6* will need to be done at the discretion of the experienced laryngoscopist, knowing that additional attempts may lead to harm

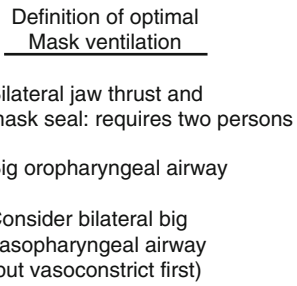

Fig. 1.5 The definition of optimal ventilation by mask is that there are *1* bilateral jaw thrust and mask seal. This requires two persons; Figs. 1.5 and 1.6 show the two possible configurations of these two people and what they do with respect to holding the mask, performing jaw thrust, and squeezing the bag. *2* An oropharyngeal airway needs to be in place to ensure that the tongue and posterior pharynx are separated. *3* A nasopharyngeal airway may be used to keep the nasopharynx patent and to provide a conduit from the nasopharynx to the laryngopharynx for gas exchange; however, serious consideration should be given to vasoconstricting the nasal passages with Afrin before instrumenting the nasal passage in order to decrease nasal mucosal bleeding

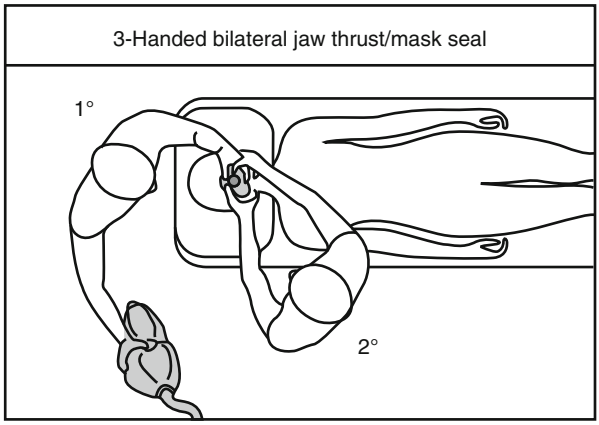

Fig. 1.6 Ventilation by mask with bilateral jaw thrust and mask seal takes a two-person effort. This illustrates a two-person approach to ventilation by mask whereby the second person (*2°*) assists providing bilateral jaw thrust and mask seal. The second person should be trained in airway management

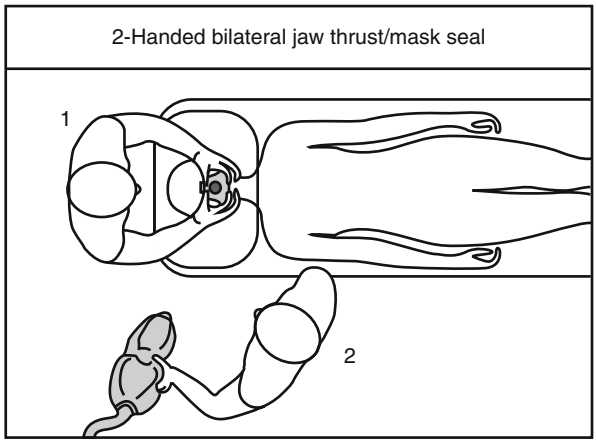

Fig. 1.7 Ventilation by mask with bilateral jaw thrust and mask seal takes two-person effort. In this figure, the second person (*2*) assists by squeezing the bag as the first person (*1*) provides bilateral jaw thrust and seals the mask. If a second person is not available, an alternative is to turn on the ventilator and optimize the airway with bilateral jaw thrust, making use of an oral and possible nasal airway as described above

Table 1.1 CVCI options: choosing between the supraglottic mechanisms (laryngeal mask airway cricothyroidotomy) and subglottic mechanisms (transtracheal jet ventilation, surgical airway)

Cause of CVCI	Ventilatory mechanism	Specific choice
Given anatomy, no pathology	Supraglottic	LMA, combitube
Periglottic pathology	Subglottic	TTJV, surgical airway

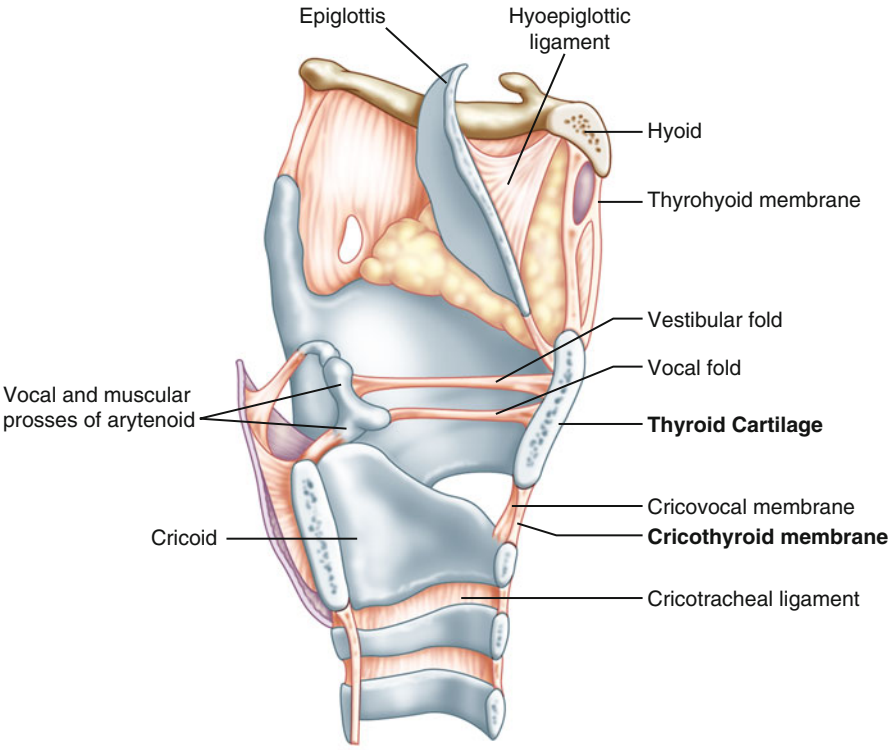

Fig. 1.8 This is a sagittal view of the larynx. Note the cricothyroid membrane is the largest membrane connecting the various cartilages of the airway (note large letters). Also note that the vocal cords are under the midpoint of the thyroid cartilage, and that is why pressing on the thyroid cartilage moves the larynx posteriorly and improves the laryngoscopic grade of view

Chapter 2
Pulmonary Edema Following Attempted Nasal Intubation for Mandibular Fracture Repair

Zakir Rangwala

The patient is a 28-year-old-male with an angle fracture of the right mandible (**L-1**, **L-2**) from assault trauma from the previous night. CT of the jaw is consistent with right-sided mandibular angle fracture, as well as soft tissue swelling of the right cheek and hematoma. The patient was scheduled for an ORIF of the mandible. The patient had nothing pertinent on past medical history. Past surgical history was significant for chest tube placement and a possible splenectomy following trauma, during which he received general anesthesia without complication. He had no known drug allergies and was not taking any medications daily. He stated his last meal was at 2100 the night before.

Physical exam showed a young healthy-appearing male, 102 kg, with significant swelling of his right jaw and face. He had a Mallampati class IV airway secondary to pain causing him inability to open his mouth. He had approximately 2.5 cm mouth opening, with thyromental distance >3 fingerbreadths. Dentition was intact, there was fairly good range of motion in the neck, and he was unable to prognath. Vital signs and preliminary labs were unremarkable.

The patient was taken back to the OR at 1720 and standard ASA monitors applied. A nasotracheal intubation was planned with a nasal RAE tube (**L-3**, **L-4**). The patient was preoxygenated for >5 min, twitch monitor applied. Intravenous induction was done with fentanyl 250 mcg, lidocaine 100 mg, propofol 200 mg, and rocuronium 80 mg. Patient was easily ventilated via mask until he lost twitches. Phenylephrine was sprayed into his nares, and the right nare was serially dilated with lubricated nasal trumpets size 26 through 32 (**L-3**, **L-4**). A nasal RAE size 7.0 cuffed tube was introduced and direct laryngoscopy with MAC #3 blade was performed (**L-3**). The tip of the ETT was visualized and McGill forceps introduced to grasp the ETT proximal to the cuff. However, the posterior pharynx was notable for significant soft tissue swelling and some blood. The ETT could not be passed

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through the vocal cords and a second DL was attempted, again without success. O2 saturation was in the 80s at this point and the ETT was removed, and patient was ventilated via mask with O2 saturation coming up into the high 90s. FOB was brought in and nasal intubation over the FOB was attempted; however, significant swelling and bleeding made this unsuccessful (**L-3**, **L-4**). Again, the nasal RAE tube was removed and patient was ventilated via mask to improve oxygenation. At this point, a disposable LMA, size 3.5, was placed and the patient could be successfully ventilated. The anesthesia team decided to postpone surgery until airway edema was relieved and the patient was better optimized. After reversing the paralytic and weaning off the anesthesia, the patient was spontaneously ventilating with an LMA in place. The patient was allowed to wake up from anesthesia, and as he did, medium-pitched, noisy breath sounds emanated from the LMA, and his saturations remained in the high 90s and he was taking approximately 500 cc tidal volumes. However, as the patient fully woke up, he began coughing up frothy pink sputum in the LMA and the LMA was removed. He was sat up and continued to have respiratory distress with bloody sputum coming from his mouth (**L-5**, **L-6**). The decision to reintubate him was made and anesthesia was quickly re-induced. A second LMA was placed and an FOB was placed through the LMA and a grade 1 view of the cords was seen. There was no edema of the cords. An ETT sized 7.0 mm ID was placed over the FOB orally and secured at 22 cm at the teeth. The FOB was advanced into the trachea and no pathology was visualized. The ETT tip was confirmed to be 5 cm above the carina. Patient was maintained on the ventilator and admitted to the SICU. His ventilation continued to improve over the next 24 h later and, on day 2, was extubated successfully. He was taken back to the OR 4 days later and had a successful ORIF of his right jaw after an awake FOB nasal intubation.

Lessons Learned

L-1: **What is the anatomy of the mandible**? **Where can fractures occur**? (See Fig. 2.1.)

L-2: **What fractures are of particular concern**? **Airway concerns**?
Condylar Fracture
The condyle is a bony protuberance that sits in the temporal bone fossa. During mouth opening, the condyle first rotates within the fossa and then translates out of the fossa across the anterior tubercle. Condylar fractures will limit mouth opening via two ways:

1. Failure of rotation of the condyle within the temporal bone fossa
2. Failure of t ranslation of the mandibular condyle from the temporal bone fossa across the articular tubercle of the temporal bone

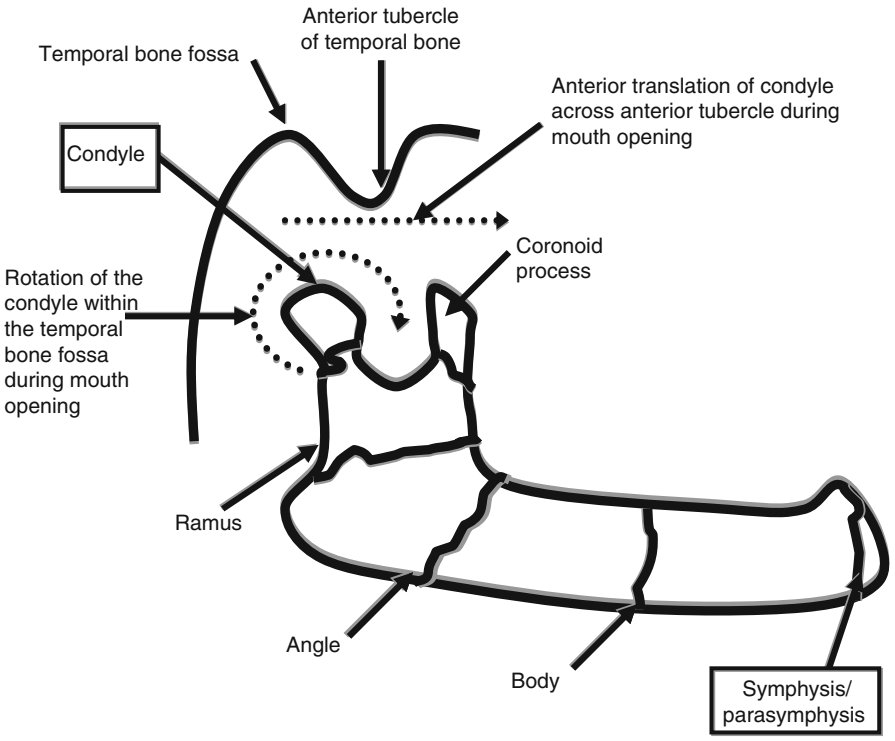

Fig. 2.1 Anatomy of the mandible with the most common fracture sites. During the first part of mouth opening, the condyle rotates counterclockwise within the temporal bone fossa. In the latter half of mouth opening, the condyle translates anteriorly across the anterior tubercle of the temporal bone. Fractures of the condyle and the parasymphysis are of particular concern to the anesthesiologist because of limited mouth opening and posterior movement of the mandible, respectively. Note: Figure not drawn to scale

Bilateral Parasymphysis Fracture

A bilateral fracture at the parasymphysis will allow the suprahyoid muscles to contract unopposed and pull the free-floating posterior segment of the mandible posteriorly, causing the tongue to obstruct against the posterior pharynx.

L-3: **What is optimal preparation for nasal intubation**?

Avoidance of bleeding and trauma to the nasal passage is key to a successful nasal intubation.

Regarding vasoconstriction of the nasal blood vessels, there is differing opinion on whether or not application of a vasoconstrictor before or after anesthetic induction is superior. It is not known whether nasal mucosal vessels are better constricted by applying topical vasoconstriction prior to inducing general anesthesia or vice versa, but we believe the former is more effective than the latter. Topical alpha$_1$

agonists like oxymetazoline or phenylephrine are ideal for this purpose and should be sprayed in both nares for backup.

Serial dilation with lubricated nasal trumpets of increasing diameter creates an increasingly open space in the nare and reduces the amount of trauma and bleeding with passage of the endotracheal tube.

Regarding which nare to use when performing a nasal intubation, it is important to know the anatomy of the nasal cavity so as to avoid any unnecessary trauma and bleeding. The nasal turbinates reside on the lateral aspect of each nare, and trauma to the turbinates can cause significant bleeding, as this tissue is very friable. It is important to orient the endotracheal tube such that the very tip of the tube cannot impact against the turbinates. When intubating through the left nare, no rotation of the tube is needed as the tip of the tube passes along the septum and the bevel is facing the lateral wall. When intubating through the right nare, the anesthesiologist should rotate the tube 180^0 so that the bevel will face the right lateral wall and the tip of the tube is against the medial septum. Once the tip of the tube is past the turbinates (approximately 2–3 cm), the nasal tube can be rotated back to its original orientation and be advanced further down the airway (Fig. 2.2).

When using the McGill forceps to advance the endotracheal tube, it is important to grasp the tube proximal to the inflatable cuff so as to not potentially rupture the cuff and for ease of guidance into the trachea.

L-4: **What are the specific preparations for fiberoptic nasal intubation**?
The aforementioned steps of nasal dilation and vasoconstriction should be carried out to minimize trauma and bleeding.

Suction tubing should be available and attached to the fiberoptic scope in order to optimize viewing.

The patient should be placed in the sniff position, and the tongue should be displaced anteriorly, either by manual traction on the tongue or by elevated jaw lift, by an assistant.

The anesthesiologist must not underestimate the amount of hands necessary to accomplish the set of tasks in a fiberoptic nasal intubation. The anesthesiologist should have an appropriate number of assistants to aid in securing the airway. One individual should be responsible for displacing the tongue anteriorly, one should be responsible for advancing the ETT over the fiberoptic scope, and one should be using two hands to manipulate the fiberoptic scope itself for guidance.

L-5: **What is the mechanism of pulmonary edema**?
Pulmonary edema is defined as an abnormal accumulation of interstitial fluid in the lung after lymphatic flow is exhausted and can no longer clear the fluid.

The forces that govern transcapillary/interstitial space fluid movement are as follows: The net flow of fluid (F) out of the pulmonary capillaries is equal to the difference between pulmonary capillary hydrostatic pressure (P_i) and the interstitial capillary hydrostatic pressure (P_o) and to the difference between the capillary oncotic pressure (π_i) and the interstitial oncotic pressure (π_o). These four forces

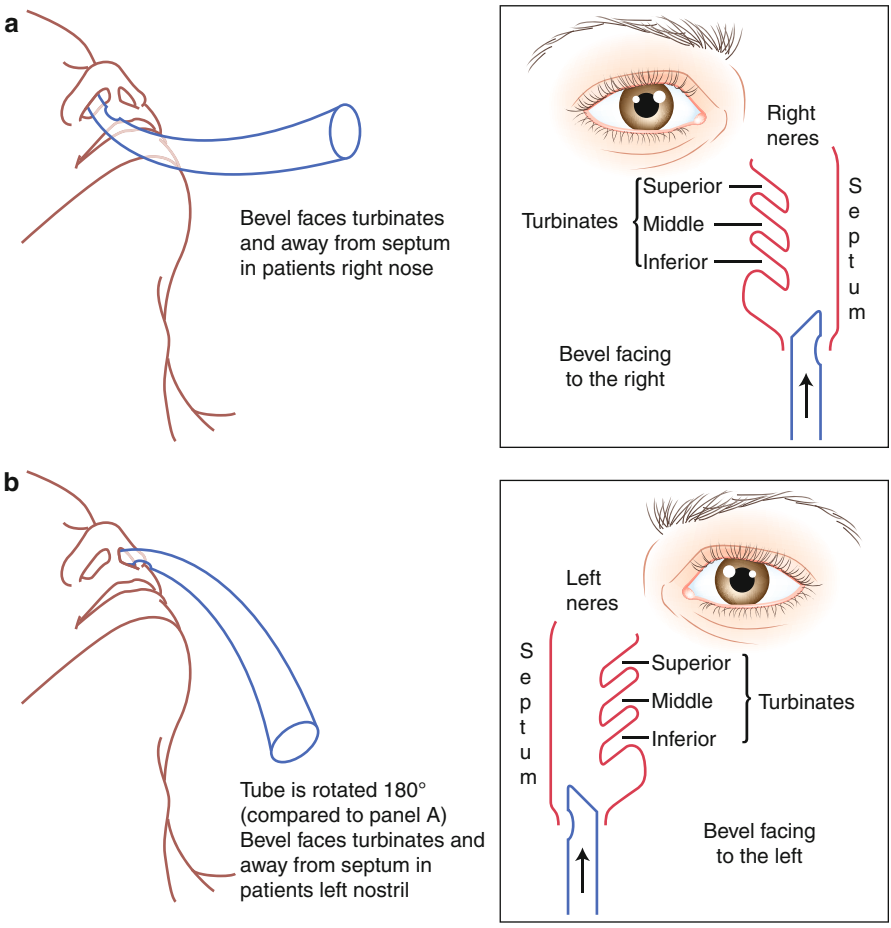

Fig. 2.2 Insertion of a nasotracheal tube into the nares. (**a**) When the nasotracheal tube is passed into the right nares, the bevel should be facing to the right toward the turbinates (see *inset*). In this way the tip of the tube is against the septum and the risks of catching the tip of the tube on a turbinate and tearing or dislocating it are minimized. In this orientation, the concavity of the tube is pointing anteriorly. (**b**) When the nasotracheal tube is passed into the left nares, the bevel should be facing to the left toward the turbinates (see *inset*). In this way the tip of the tube is against the septum and the risks of catching the tip of the tube on a turbinate and tearing or dislocating it are minimized. In this orientation, the concavity of the tube is pointing posteriorly

will produce a steady-state fluid flow (F) during a constant capillary permeability (K) (Fig. 2.3).

$$P = K\left[\left(P_{\mathrm{i}} - P_{\mathrm{o}}\right) - \left(\pi_{\mathrm{i}} - \pi_{\mathrm{o}}\right)\right]$$

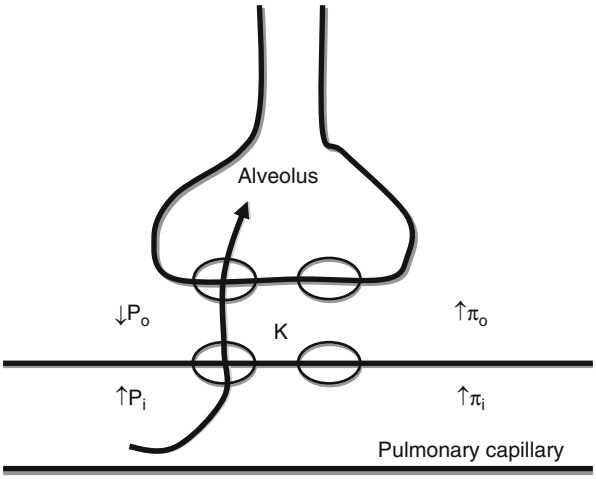

Fig. 2.3 Transcapillary migration of fluid from the pulmonary capillaries into the alveolus in the setting of pulmonary edema is dependent on five factors: hydrostatic pressure inside the vessel (P_i), hydrostatic pressure in the interstitial space (P_o), oncotic pressure inside the vessel (π_i), oncotic pressure in the interstitial space (π_o), and the capillary permeability constant (K), determined by the size of the holes between the endothelial and epithelial junctions in the vasculature and alveolus, respectively

L-6: **What is negative pressure pulmonary edema**? **How should it be treated**?

A. Negative pressure pulmonary edema (NPPE) can occur in one of three manners:

1. The generation of negative intrapleural pressures during spontaneous ventilation against an obstructed airway
   - Upper airway mass/swelling
   - Laryngospasm (most common cause)
   - Infection (croup, epiglottitis)
   - Vocal cord paralysis
   - Strangulation
   - Terminal obstruction in a patient with severe OSA
2. Rapid re-expansion of a collapsed lung
3. Vigorous pleural suction

B. In the setting of NPPE, the hydrostatic pressure outside of the pulmonary capillaries (P_o) becomes very negative as a result of the generated negative pulmonary pressure either from spontaneous ventilation (descent of the diaphragm while the airway is obstructed) or artificially through re-expansion of a collapsed lung or vigorous suctioning. This creates a net movement of fluid (F) across the capillary membrane into the alveolus.

C. Treatment of NPPE is to secure the airway, maintain oxygenation, mechanical ventilation, and the institution of PEEP to increase Po.

Chapter 3
Loss of Critical Airway

Christopher Edwards

The patient is a 40-year-old male for which the anesthesiology service was paged for a reintubation after loss of a "critical airway" while being turned electively in the MICU. The patient was 168 cm and 304 kg; body mass index (BMI) was 108 kg/m^3. Recent airway history included an intubation by anesthesia in the operating room 7 days prior with an awake fiberoptic technique (**L-1**, **L-2**, **L-3**). A percutaneous tracheostomy was then electively performed by the pulmonary critical care team 3 days prior to the loss of the critical airway. Upon arrival to the bedside, the patient was hypertensive and tachycardic with oxygen saturations in the low 50s. By report from the nursing staff, after positioning the patient, they were unable to ventilate him via the tracheostomy (**L-5**). The anesthesia team was able to ventilate him via a facemask Mapleson D circuit and large oral airway. There was no evidence of subcutaneous emphysema. Ventilation was synchronized with the patient's spontaneous respiratory effort via the facemask Mapleson D circuit and a green facemask over the tracheostomy site with improvement in the oxygen saturation to the low 90s. At that time the pulmonary critical care attending attempted to pass a fiberoptic bronchoscopy (FOB) scope through the tracheostomy site but was unable to identify the tracheal lumen (V). Each time FOB was attempted, the saturations fell rapidly requiring ventilatory support. Next, a disposable Cookgas 4.5 LMA was placed and ventilation was successful (**L-6**). A FOB was introduced through the LMA and the vocal cords were easily visualized. At that time, intravenous (**L-4**) etomidate and succinylcholine were used and the bronchoscope was advanced into the trachea without incident. A 7.0 ETT was then passed over the bronchoscope and visualized to be in the trachea (**L-6**). The ETT was positioned distal to the tracheostomy site and exhaled carbon dioxide was detected. The patient was then hand ventilated with the Mapleson D circuit, paralyzed with rocuronium, and care was transferred to the pulmonary critical care team.

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Table 3.1 Advantages of intubation in the operating room

Well-lit room
Adjustable bed
Anesthesia machine
Anesthesia cart
Extra help
Extra space
Access to the head and neck
Familiar monitors

Lessons Learned

L-1: **Why is it a good idea to perform a semi-elective intubation in a morbidly obese patient in the OR**?
Recent data from the anesthesia closed claims database shows that morbid obesity was a factor in adverse outcomes in 38 % of anesthetic inductions and 58 % of tracheal extubations. This data shows that intubating and extubating the morbidly obese population can be difficult and unpredictable. They are often difficult to ventilate via mask and may require multiple trained anesthesiologists to help secure the airway. Given these patients and the difficulties that arise secondary to their anatomy, it may be prudent to perform an elective intubation in the operating room. The operating room provides many tools that the anesthesiologist may take for granted. Common aids such as a well-lit room, adjustable bed, anesthesia machine, anesthesia cart, extra help, extra space, excellent access to the head and neck, and familiar monitors are just a few of advantages that are often overlooked. Having these tools close by and in a familiar location will save precious time if a difficult airway is encountered (Table 3.1).

L-2: **Why should the superobese be intubated awake**?
There are a series of questions an anesthesia provider should ask before manipulating the airway of a morbidly obese patient (Table 3.2). Given many of the answers to the questions posed in this table, performing an awake intubation will safely bypass most of these issues.

L-3: **How to perform an awake intubation**
There are many technical aspects of performing an awake fiberoptic intubation that are required to successfully intubate the trachea. While these skills need to be developed, there is one aspect of the awake intubation that needs to be dealt with first. *Psychological buy-in is the first and foremost task of performing an awake intubation*. Without a fully cooperative patient, an awake intubation is difficult. The entire process needs to be discussed with the patient and family members if necessary. If there is any hesitation from the patient, they need to understand that this is a life and death matter. Once you have the patient's full cooperation, performing the intubation requires following a specific set of detailed steps.

Table 3.2 General airway considerations for the morbidly obese patient

Questions	Answers
1. Must the airway be managed?	Depends on many factors: cardiopulmonary condition of the patient (e.g., patient with a BMI of 108 kg/m^2), type of surgery, location of surgery, local/regional vs. general anesthesia
2. Is there potential for difficult laryngoscopy?	In the superobese population, this answer is likely yes. Indicators are *MP classification*, *neck circumference*, *pretracheal adipose tissue*, *and OSA*. *Inability to place patient in the "sniffing" position*
3. Can supralaryngeal ventilation be used? Mask ventilation vs. LMA	Mask ventilation can be difficult secondary to adipose tissue in face and cheeks, facial hair, and large tongue LMA can be a rescue device to help with oxygenation and ventilation. LMA setbacks are unsecured airway (aspiration risk), development of leak, and high ventilatory pressures, leading to gastric insufflation
4. Is the stomach empty?	Morbid obesity is not a risk factor for high gastric residuals. However, many of these patients have gastroesophageal reflux or diabetes, which increases gastric contents
5. Will the patient tolerate an apneic period?	Obesity is associated with a marked reduction in lung volumes and FRC as well as increased oxygen consumption. Desaturation after preoxygenation *WILL* occur before return of spontaneous ventilation after induction. These patients *will not* tolerate even short apneic periods

1. *Anti-sialogue*: Glycopyrrolate 0.3 mg IV should be given to help dry the airway as airway manipulation stimulates secretions.
2. *Sedation*: Any sedation should be used with caution as even small amounts may lead to airway compromise. Commonly used sedatives include dexmedetomidine, midazolam, fentanyl, or remifentanil. All sedatives should be used conservatively and titrated slowly to effect. The desired effect is a sedated patient who is rational, oriented, and following commands.
3. *Topicalization:* There are many ways to anesthetize the airway; lidocaine is the most commonly used local anesthetic. Various methods to anesthetize the airway include 5 % lidocaine ointment applied with a tongue depressor, 4 % atomized lidocaine sprayed liberally throughout the oropharynx and larynx, and/or performing superior laryngeal nerve, recurrent laryngeal nerve, and transtracheal blocks. Each of these methods has advantages and disadvantages; despite what method is chosen, the goal is to have a well-anesthetized airway in a cooperative patient.
4. *Flexible fiberoptic bronchoscope*: A flexible fiberoptic bronchoscope is the most commonly used device to perform the awake intubation. This technique allows for direct visualization of the entire path from the teeth to the trachea. The fiberoptic scope is directed into the trachea and an endotracheal tube is passed over the fiberoptic scope with visual conformation of tracheal rings before the fiberoptic bronchoscope is withdrawn. Once the endotracheal tube is confirmed with direct visualization and end-tidal carbon dioxide, the patient can safely be fully anesthetized.

Table 3.3 Types of tracheostomy tubes

Type of tracheal tube	Advantages	Disadvantages
Single cannula	Large internal diameter, short-term use	Easily blocked with secretions, needs changing every 5–7 days
Double cannula	Can remove the inner cannula to clean, can be left in for 1 month	Decreases internal diameter by 1–1.5 mm which may increase work of breathing
Uncuffed tube	Long-term, easy to replace, can still breath around plugged tube, decreased incidence of tracheal mucosal injury	Cannot provide positive pressure ventilation, not suitable for providing FiO_2 >40 %, does not protect against aspiration
Cuffed tube	Can provide positive pressure ventilation, protection against aspiration, FiO_2 = 1.0	If tube is dislodged or plugged, a complete airway obstruction can develop, tracheal mucosal damage secondary to cuff pressure
Fenestrated tube (has small hole or holes in the tracheostomy tube)	Used to wean patients from a temporary tracheostomy tube, allows vocalization	Aspiration risk, cannot positive pressure ventilate
Adjustable flange	Are used in patients that have a deep-set trachea such as obese patients, tumor, or edema in the neck	Not recommended to move the flange because of high risk of decannulation

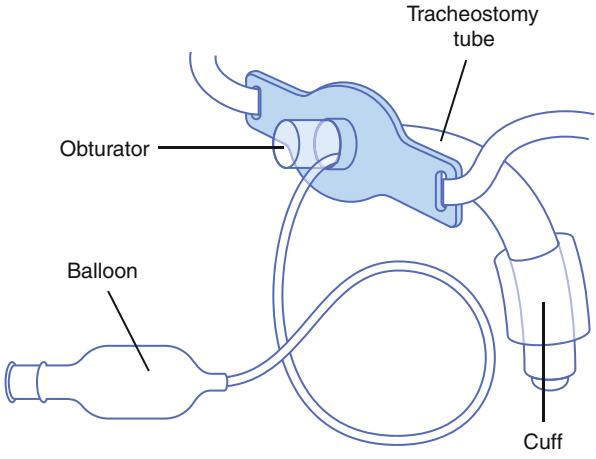

Fig. 3.1 Cuffed tracheostomy tube

L-4: **General overview of tracheostomy tubes and considerations for tracheostomy tube selection for obese patients**

A tracheostomy is a tube inserted into the trachea through a surgical incision in the anterior portion of the neck (*see* Table 3.3 and Fig. 3.1). A typical tracheostomy tube has three main portions: outer cannula with a flange, an inner removable cannula, and an obturator (Fig. 3.2). The outer cannula holds the tracheostomy site open and has a flange, which is used to secure the tube to the skin with twill ties or

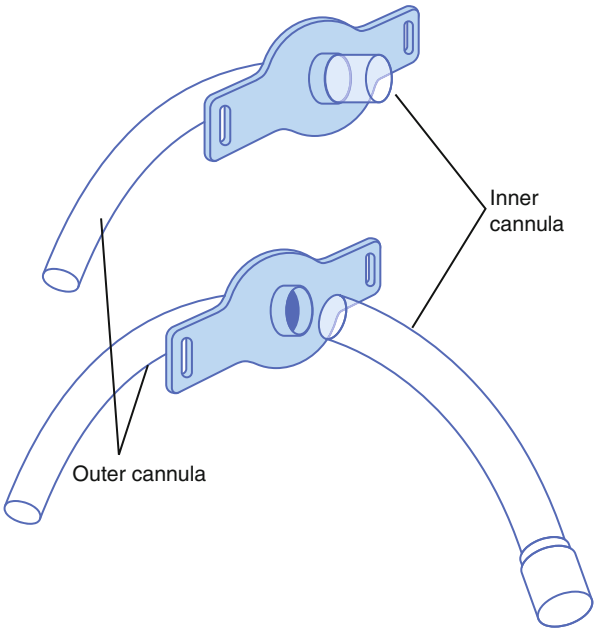

Fig. 3.2 Tracheostomy tube showing inner and outer cannula

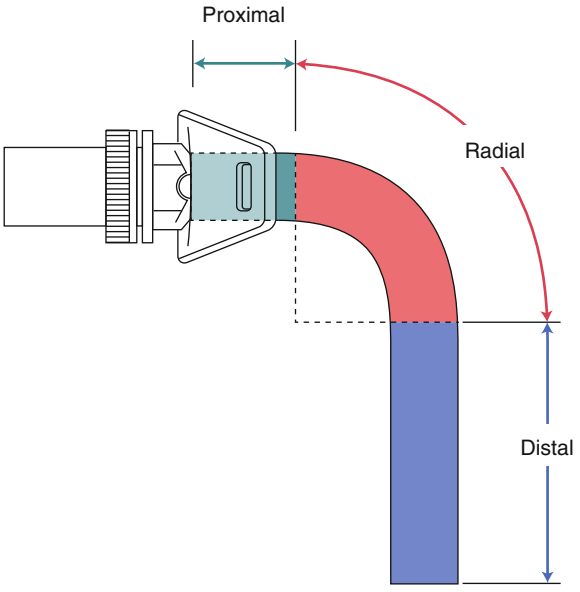

Fig. 3.3 Shiley XLT. This figure shows the various proximal, radial, and distal segments of a tracheostomy tube. Each segment may be individually selected to best fit a patient's anatomy

sutures. The inner cannula is inserted and locked in place inside the outer cannula. The proximal end of the inner cannula is the connection to all breathing circuits. The inner cannula can be removed and cleaned or are disposable. The obturator is a stiff device with a rounded leading tip used as an aid to insert the tube. The tube itself is broken down into three distinct zones: proximal, radial, and distal (Fig. 3.3).

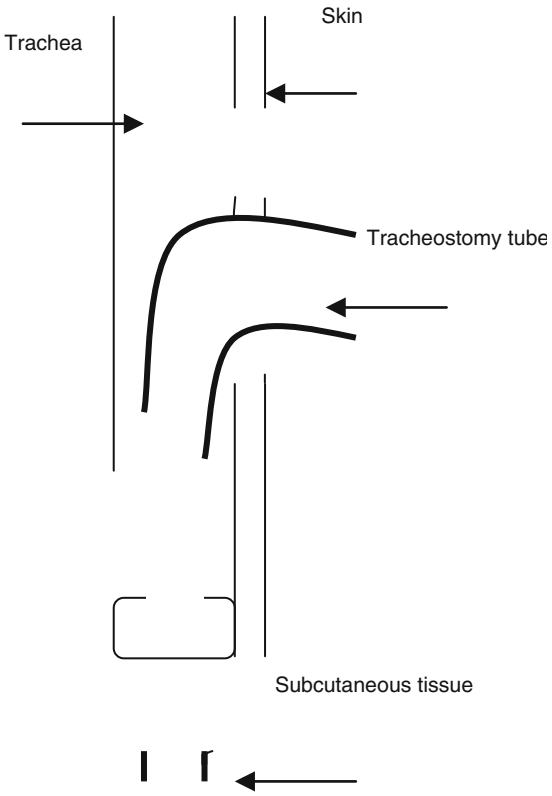

Fig. 3.4 Tracheostomy tube in adequate position

These various zones can independently have different lengths, internal diameter, and external diameter. These features allow the physician placing the tube to have flexibility in selecting a tube which best conforms to a patient's anatomy.

The tracheostomy tube in a standard percutaneous tracheostomy set (the tracheostomy tube used in this case) is a standard tracheostomy tube and does not accommodate an increased skin to tracheal distance in a morbidly obese patient; therefore, there will be a smaller portion of the tube in the trachea increasing the risk of decannulation (*see* Figs. 3.3 and 3.4). If decannulation occurs in these patients, it can be difficult to replace the tracheostomy tube through the tracheostomy site secondary to adipose tissue. In morbidly obese patients, there are options to help to limit this from occurring. Using a tracheostomy tube with an adjustable flange or a tracheostomy tube designed for increased skin to tracheal distance will allow for proper positioning of the tracheostomy tube (Fig. 3.5). The other option is to perform a "cervical lipectomy" procedure during surgical placement of the tracheostomy. Cervical lipectomy is removal of pretracheal and anterior cervical fat, which allows for the standard tracheostomy tube to fit better. By having an understanding of tube types and sizes as well as the procedures to place them, an anesthesia provider can be better informed when providing anesthesia for morbidly patients requiring a tracheostomy.

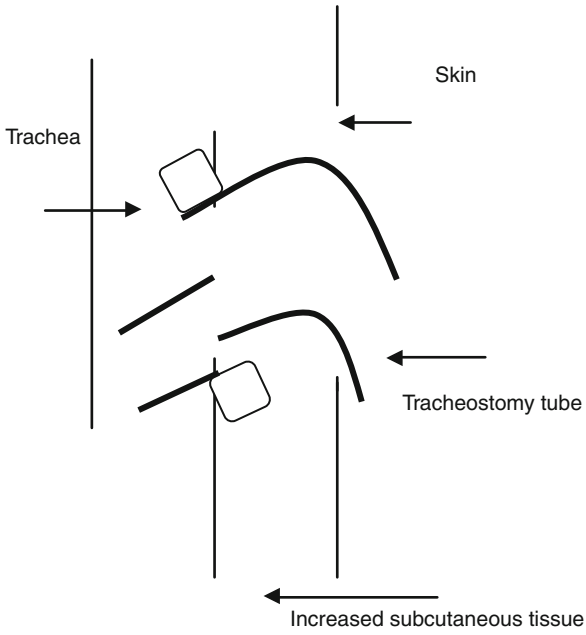

Fig. 3.5 Tracheostomy tube in a partially backed out position with a partially herniated cuff

L-5: **What happened in this case**? **Two critical errors occurred in series leading to dislodgment of the tracheostomy tube**

There were multiple critical errors which occurred during this hospitalization leading to loss of this patient's critical airway. After the elective intubation the patient was brought to the operating room and a percutaneous tracheostomy tube was chosen as opposed to placing a surgical tracheostomy. Choosing to do the percutaneous tracheostomy limited the type of tracheostomy tube to a standard size and length which in a patient with a BMI of 108 kg/m^2 was an error in judgment. There was no ability to choose either an adjustable flange tube or Shiley XLT (this is an extended length tracheostomy tube. Both Bivona and Rusch have adjustable flange tracheostomy tubes). The Shiley XLT was designed for patients with a thick or full neck or long tracheal anatomy, which allows the tube to properly fit in obese patients. Additionally the Shiley XLT allows the practitioner to choose tubes with varying proximal, radial, and distal portions to best fit a particular patient's needs (Fig. 3.6). Because of the increased skin to tracheal distance in this patient, the tracheostomy tube likely was in a partially backed out position (*see* Fig. 3.4). The second error occurred in the MICU when the nursing staff was turning the patient during routine care. While turning the patient there was no human hand holding onto the tracheostomy tube and it was subsequently dislodged. This case represents a flagrant example of not taking care of an absolutely critical airway. An anesthesiologist would never think of moving a patient without dedicating a hand to holding the endotracheal tube. Simply having a nurse dedicated to holding the tracheostomy tube anytime a patient is moved will limit potentially fatal errors similar to this one.

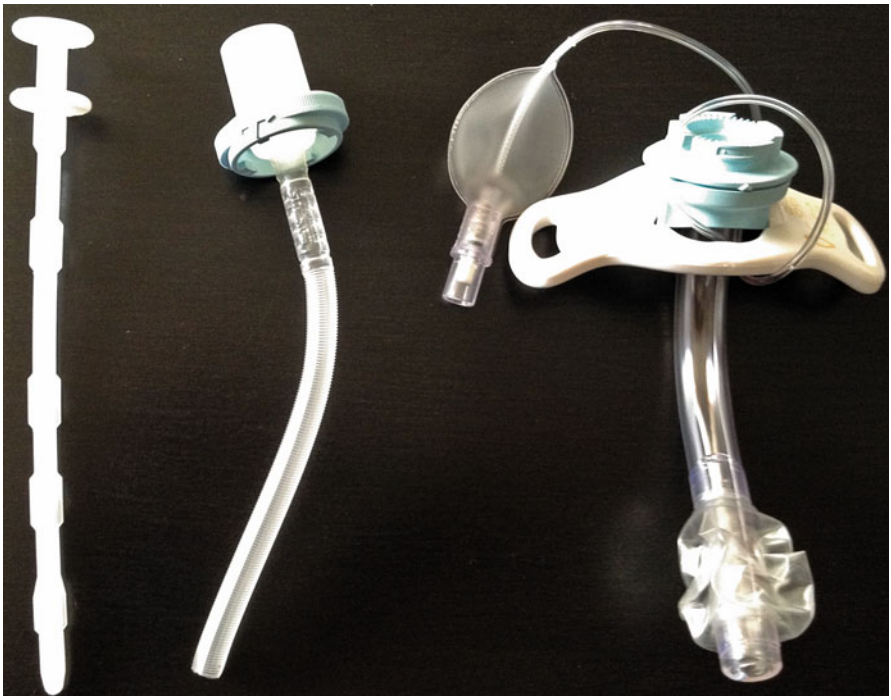

Fig. 3.6 Shiley Tracheosoft XLT tracheostomy tube. *From left to right*: obturator, inner cannula, and outer cannula

L-6: **Continuous ventilation with the Cookgas LMA intubating technique**
Morbidly obese patients are often difficult to intubate for reasons discussed earlier. One of the biggest challenges is that with a decreased functional residual capacity

(FRC) and increased oxygen consumption, they will desaturate much more quickly than a nonobese patient. Using the Cookgas LMA with a continuous ventilation technique can circumvent this challenge. This provides the practitioner with more time to secure the airway in a safe and controlled fashion. The following guidelines outline the steps needed to effectively intubate using the Cookgas LMA. It should be remembered that this technique can only safely be used in a patient that is able to be ventilated with a supraglottic device and has no risk of aspiration. In addition a Cookgas 3.5 LMA should be used in females and a Cookgas 4.5 should be used in males.

1. The first step is to assemble and set up all the required instruments. A fiberoptic bronchoscope with an outer diameter (OD) of 5.0 mm should be used. This will easily fit inside a 6.0 mm (without adapter), 7.0 mm (with adapter), and 8.0 mm (with adapter) endotracheal tube. A lubricating solution should be liberally applied to the fiberoptic bronchoscope, inside the Cookgas LMA, and inside and outside the endotracheal tube so there is minimal friction. Other required tools are a bronchoscopic elbow adapter and an endotracheal tube pusher.

2. Once the patient is anesthetized and paralyzed, the Cookgas LMA should be inserted. The LMA should be checked to make sure ventilation is adequate. Once LMA placement is confirmed with pressure seal to roughly 20 mmHg and end-tidal carbon dioxide is detected, the bronchoscopic elbow can be attached and the ventilator turned on.
3. The lubricated fiberoptic bronchoscope should be inserted through the bronchoscopic adapter attached to the LMA. All movements should be slow and deliberate and the area of interest should be kept in the center of the screen. The bronchoscope is advanced until the bowl of the LMA is encountered and vocal cords are visualized. If vocal cords are off-center or not visualized, the LMA position may need to be adjusted.
4. The fiberoptic bronchoscope is withdrawn and the bronchoscopic elbow is removed. The endotracheal tube is then inserted to a depth of 16 cm. The bronchoscopic elbow is then attached to the endotracheal tube and ventilation is once again confirmed. The bronchoscope is then advanced through the bronchoscopic elbow through the endotracheal tube. Once the tip of the bronchoscope passes the end of the endotracheal tube, it is guided through the vocal cords, making sure to identify carina and tracheal rings.
5. Next the previously lubricated endotracheal tube is advanced over the bronchoscope until the endotracheal tube is confirmed to be in the trachea. Resistance is occasionally encountered while passing the endotracheal tube through the glottic opening. A simple $90–180^0$ counterclockwise rotation of the endotracheal tube often relieves the resistance and allows the tube to pass into the trachea. The bronchoscope can be safely withdrawn once both end-tidal carbon dioxide and visual confirmation have been established.
6. The final step is removing the LMA. The bronchoscopic elbow is removed and a small “tube pusher” is placed in the adapterless proximal end of the endotracheal tube. A constant inward pressure applied to the endotracheal tube as the LMA is slowly pulled back. Once the LMA has been partially withdrawn, the index finger can be used to secure the tube in the mouth pinching the tube between the index finger and hard palate. The LMA is completely removed and the endotracheal circuit adapter is attached. The endotracheal is then secured in standard fashion.

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Chapter 4
Anterior Mediastinal Mass

Christopher Edwards

The patient is a 42-year-old female with an anterior mediastinal mass (**L-1**). She presented 1 month earlier with progressive shortness of breath, dry cough, weight loss, and chest pressure. The shortness of breath was worse while lying supine and better in the lateral position. During the preoperative workup, a CT scan had shown a large anterior mediastinal mass and a large pericardial effusion. A subsequent biopsy of the mass determined it to be a neuroendocrine tumor. The patient had denied symptoms of flushing, diarrhea, or abdominal cramps. A pericardiocentesis was performed draining approximately 800 mL of fluid with some resolution of symptoms. Chemotherapy failed to decrease the tumor burden. She was scheduled for a left thoracotomy for tumor debulking and possible pneumonectomy. Transthoracic echocardiogram and CT scan of the chest showed no further increase in the pericardial effusion, mild compression of the main pulmonary artery, and left main bronchus compression with diffuse left lung atelectasis.

Once in the operating room an awake thoracic epidural and arterial line was placed. General anesthesia was induced without incident and a 7.0 single-lumen endotracheal tube was placed. With the fiberoptic bronchoscope, the endotracheal tube was advanced into the right mainstem bronchus. A bronchial blocker was placed in the left main bronchus, which functioned well throughout the operation. The surgery proceeded uneventfully. The mass was removed without the need for pneumonectomy. The remainder of the operation was uneventful and the patient was extubated and transferred to the ICU without incident.

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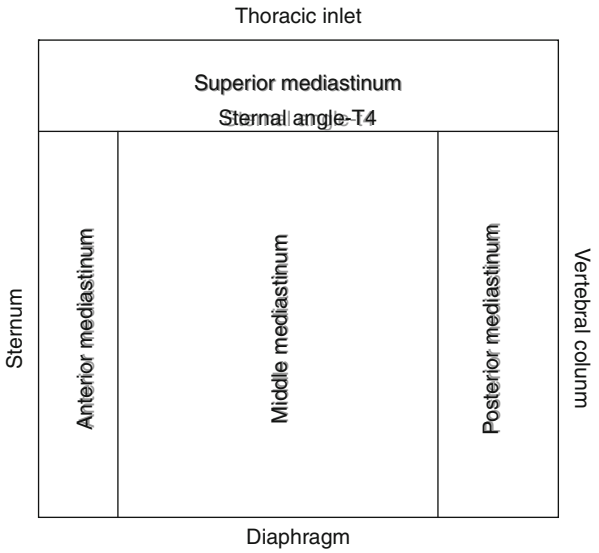

Fig. 4.1 Schematic of the boundaries and divisions of the mediastinum

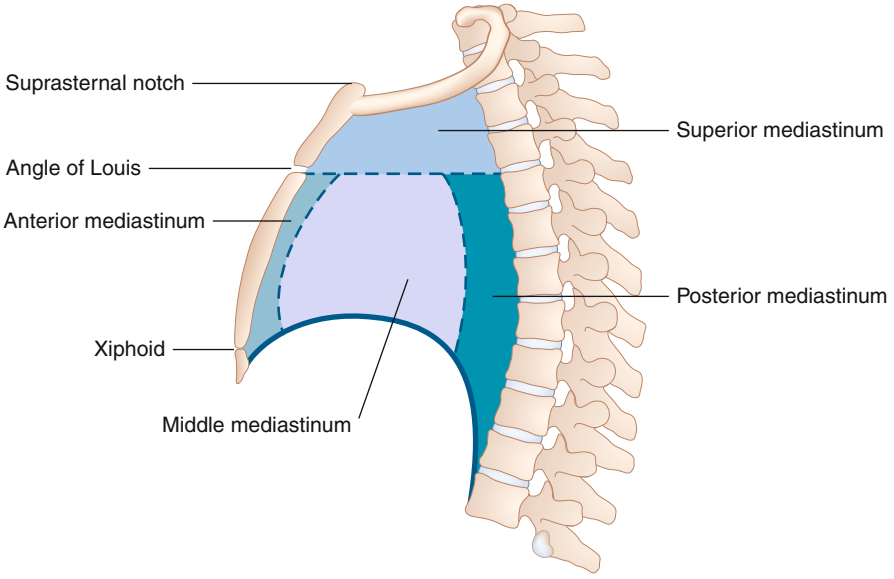

Fig. 4.2 Schematic and anatomic diagram of the boundaries and divisions of the mediastinum

Lessons Learned

L-1: **What are the divisions of the mediastinum**?
The divisions of the mediastinum are shown in Figs. 4.1 and 4.2, and Table 4.1.

Table 4.1 Divisions of the mediastinum

Name	Structures
Superior	Thymus, trachea, esophagus, thoracic duct
Lower-anterior	Thymus (in children)
Lower-middle	Heart, pericardium, great vessels, mainstem bronchi
Lower-posterior	Descending aorta, esophagus

L-2: **What are the cardiorespiratory problems caused by mediastinal masses**?

Tracheobronchial Tree Obstruction

There are two main concerns when anesthetizing a patient with a tracheobronchial tree (TBT) obstruction. The first is that the obstruction is often distal to the tracheal bifurcation. In such a case, intubation would not relieve the obstruction and a rigid conduit may be needed to provide ventilation. Another possible cause is loss of chest wall and airway tone. During normal spontaneous respiration, negative intrathoracic pressure helps to keep the airway open (Fig. 4.3a). After intubation and positive pressure ventilation, the intrathoracic pressure is only slightly less positive relative to airway pressure and with the weight of the mass pressing in on the airway, collapsing forces predominate (Fig. 4.3b). These various forces may help to explain signs and symptoms seen with TBT obstruction. Masses affecting the TBT do not always have associated respiratory symptoms. If the mass had an insidious course, symptoms may not have developed or developed slowly. While respiratory symptoms may be a good indicator that the airway may be compromised, one still needs to be careful in a patient without such symptoms.

Compression of Pulmonary Artery and Heart

There are only a few case reports of isolated pulmonary artery and heart compression by an anterior mediastinal mass. The pulmonary artery is protected by the aortic arch and TBT, thus making isolated compression of the pulmonary artery relatively rare. Hemodynamic effects of pulmonary artery compression include right ventricular failure, decreased left ventricular preload, decreased cardiac output, and eventual systemic hypoxia. If the mass involves the myocardium or pericardium, arrhythmias and pericardial effusions are not uncommon.

Superior Vena Cava Syndrome

Superior vena cava (SVC) syndrome is mechanical obstruction of the SVC by external compression, thrombosis, or invasion by tumor. The signs include distended veins in the upper extremities and edema of the face and neck. Symptoms are related to the time course and the ability of the body to shunt blood through collateral circulation. As blood backs up in the various organ systems, symptoms start to develop; headaches, shortness of breath, nasal congestion, dizziness, and lightheadedness are most common. Therapies for SVC syndrome include chemotherapy, radiation, and surgical resection. Surgical resection is often difficult and incomplete secondary to distortion of tissue planes surrounding to the SVC and tumor.

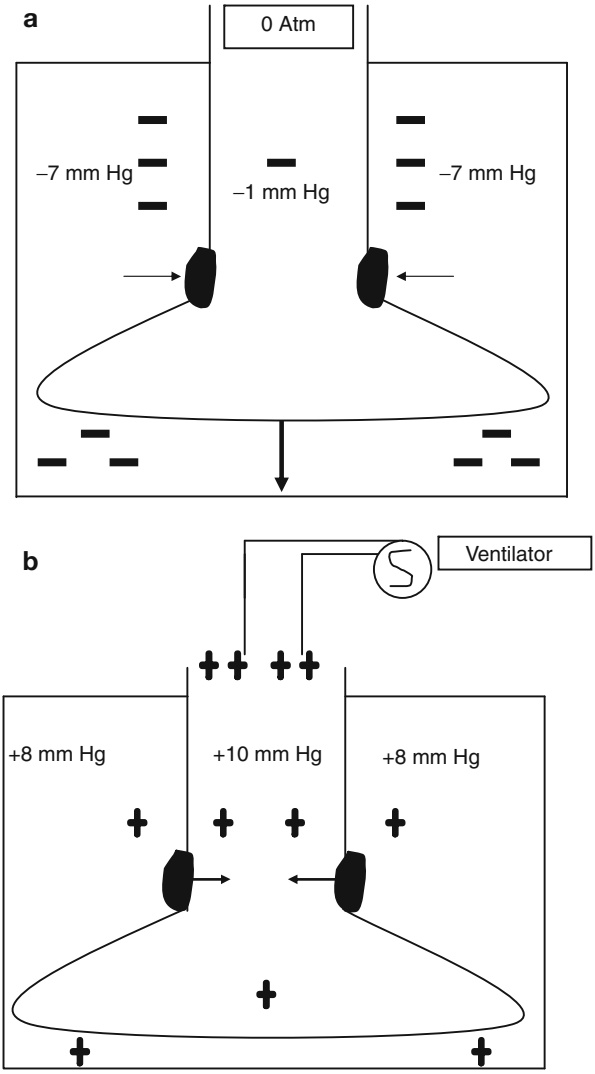

Fig. 4.3 (**a**) Relatively favorable transmural intrathoracic distending airway pressures during spontaneous inspiration (*black circle* indicates tumor compressing the airway). The numbers are intended to show relative airway pressures. (**b**) Relatively unfavorable transmural intrathoracic distending airway pressures during positive pressure ventilation (*black circle* indicates tumor compressing the airway). The numbers are intended to show relative airway pressures

L-3: **What are the anesthetic concerns for a patient with an anterior mediastinal mass**?

The main anatomic structures at risk during general anesthesia are the aforementioned tracheobronchial tree, SVC, and pulmonary artery and heart (Fig. 4.4). A patient can have one or all of these vital structures involved, and a thorough preoperative evaluation with detailed imaging of the mass is necessary. Imaging should include a CT scan and a sitting and prone echocardiogram as this will help determine the severity of position-related hemodynamic changes. Particular attention should be given to positional respiratory symptoms, especially in the supine position. Compromise during induction of anesthesia of any of these structures can lead to quick cardiovascular collapse. *Local anesthesia for all diagnostic procedures should be used unless there is a strong indication otherwise* (*see* Fig. 4.4).

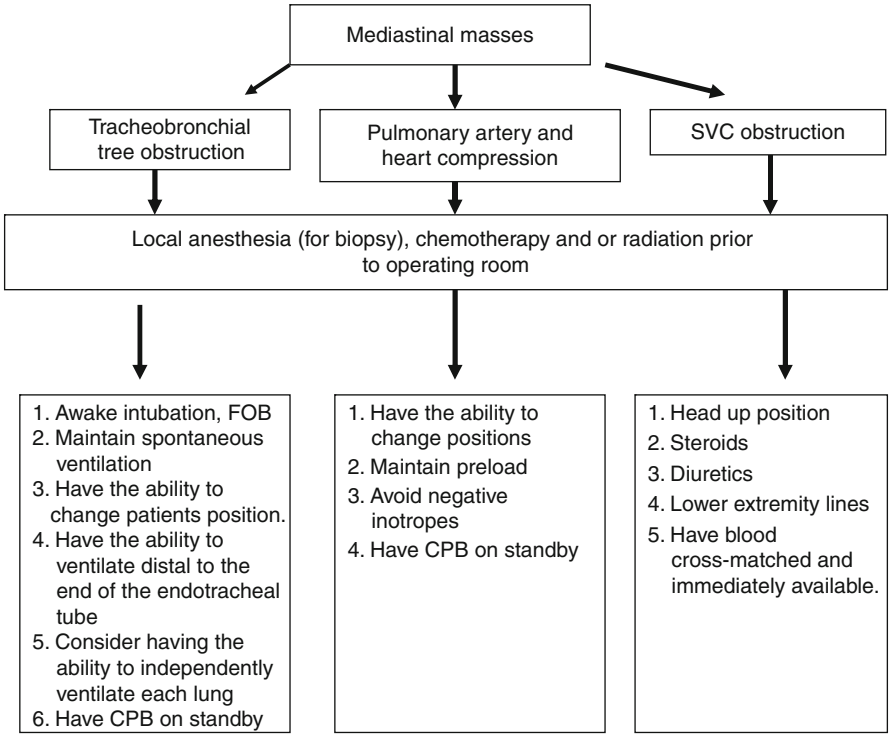

Fig. 4.4 Flow diagram of the anesthetic plan for a mediastinal mass

Between the various types of anterior mediastinal masses, there are common themes in delivering a safe anesthetic. Always have a firm understanding of the anatomy of the mass and what structures are affected. Take a detailed history and physical exam prior to inducing anesthesia as this may indicate what position is most suitable to maintain the blood pressure and airway in the event of collapse. Finally, have a detailed plan from intubation technique to IV access. The surgeons and operating room staff should be briefed on how to aid in quick positional changes. These patients are often on the edge of cardiopulmonary collapse, but with a predetermined plan, an anesthetic can be delivered in a safe and effective manner.

L-4: **Commentary on anesthetic management of this case**
This particular case deviates in some regards as how to best manage a patient with a mediastinal mass. Based on symptoms and imaging this patient likely had tracheobronchial tree compression (as shown in Fig. 4.4). Following the flow diagram (left side of Fig. 4.4, bullets 1, 2, and 5), there are a few points that demand further discussion. Given the fragile nature of this airway, an awake intubation would have been the safest and most controlled way to secure the airway. Performing an awake intubation allows you to maintain control of the airway (point 1) at all times while maintaining spontaneous ventilation (point 2). Given the specific location of this tumor and compression of the left mainstem bronchus, intubating the right mainstem bronchus under direct fiberoptic visualization guaranteed the ability to

ventilate the right lung independently from the left (point 5). In this case a single-lumen endotracheal tube was likely used because of the high likelihood of performing a total left pneumonectomy. The rest of the therapeutic logic in Fig. 4.4 is self-explanatory.

Recommended Reading

Benumof J. Anesthesia for special elective therapeutic procedures. In: Benumof J, editor. Anesthesia for thorcacic surgery. 2nd ed. Philadelphia: W.B. Saunders Company; 1995. p. 567–75.

Chapter 5
Awake Intubation with a NIM Tube: How Is It Done?

Bahareh Khatibi

A 24-year-old male with a large right-sided neck mass, likely a vagal schwannoma, was scheduled for a transcervical excision of skull base tumor with cranial nerve dissection. The patient reported right-sided neck fullness for over a year but denied associated symptoms including dysphagia, hoarseness, or difficulty breathing. Magnetic resonance imaging (MRI) 2 days prior to surgery showed a 7.4 × 5.3 × 3.9 cm heterogeneous mass in the right carotid space with splaying of the internal and external carotid arteries, internal jugular vein compression, and a significant mass effect on the pharynx and hypopharynx. Mass was unchanged in size compared to an MRI 6 months previously.

The patient was otherwise healthy with normal vital signs and 97 % O_2 saturation on room air. He was 5′8″ and 79 kg. His surgical history consisted of tonsillectomy at a young age, knee surgery, and a left-hand incision and drainage in 2009. Airway exam was significant for class I Mallampati with the right-sided mass reaching the midline with the uvula deviated to the left, right-sided neck fullness and firmness, full range of motion of the neck and 4 cm thyromental distance.

After discussion with the ear, nose, and throat (ENT) surgeon regarding special considerations for the case, it was determined that an orotracheal tube would be acceptable. Our plan was to perform an awake fiberoptic intubation (**L-1**) through the Williams pink airway after the oral mucosa and upper airway were topicalized with 4 % aerosolized lidocaine. The importance of an awake intubation was discussed with the patient. The patient verbalized understanding and agreed with the plan. The patient received glycopyrrolate 0.3 mg IV in the preoperative area and was taken to the OR. During the brief, the attending surgeon notified us that a *NIM tube* (**L-2**, **L-3**, **L-4**) was required. We proceeded with a new plan for securing the airway with a NIM tube (**L-5**).

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Lessons Learned

L-1: **In this case, difficulties with securing the airway were anticipated. Based on the clinical pathology of the lesion, preparations for awake intubation were made. What is the proper preparation for an awake intubation?**

1. The most important step is the *psychological preparation* of the patient during the preoperative visit. The goal is that the patient truly understands why an awake intubation is necessary and the safety it provides. Inform the patient that securing the airway awake is the safest way to secure the airway and minimizes the risk of complications including hypoxic brain injury and death; the patient must understand that it is his/her life that is at stake. Involve family members if appropriate (or anyone that means anything to the patient). Provide a detailed explanation of the procedure so the patient knows what to expect.
2. Second, intravenous *glycopyrrolate* is necessary to decrease secretions and allows for visualization and facilitates topical anesthesia of the airway. There are very few contraindications to glycopyrrolate in the context of securing the airway; severe coronary artery disease could constitute a relative contraindication.
3. Third, take the patient to the *OR* as this is the most secure place for airway management. Place the patient in the *semi-upright position* since this is better for breathing than the supine position. Apply *full standard monitors*. Provide for maximal *oxygenation* throughout the procedure by high-flow oxygen via nasal prongs. Remember, nasal prongs do not get in the way of any method of intubation.
4. Fourth, provide *conscious sedation* as allowed, given specific patient characteristics. The goal of sedation during an awake intubation is to have an awake, calm patient who can follow commands and maintain adequate oxygenation and ventilation. A small dose of midazolam significantly increases the seizure threshold.
5. Fifth, *topical local anesthetic* application to the oropharynx (or nasopharynx if nasal intubation is to be performed) with 4 % aerosolized lidocaine. Application of the topical local anesthetic should be repeated over and over to all areas of the upper airway until the patient can tolerate whatever intubation conduit is going to be used (Williams pink airway, LMA, laryngoscope, etc.).
6. Sixth, consider *nerve blocks* of the airway as appropriate. Avoid if there is altered anatomy or if there is malignancy in close proximity to the airway. Nerve blocks were not appropriate in this case given the proximity of the schwannoma and that recurrent laryngeal nerve function needed to be intact for monitoring.

L-2: **What is a NIM tube**? **What does It do**?
NIM stands for "Nerve Integrity Monitor."

The NIM tube is an endotracheal tube that provides for intraoperative monitoring of electromyographic (EMG) activity of the intrinsic laryngeal musculature. It contains two pairs of electrodes: one pair at the 2 o'clock and 4 o'clock positions and

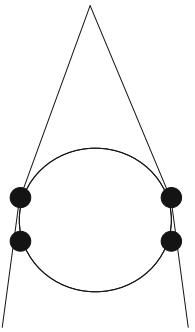
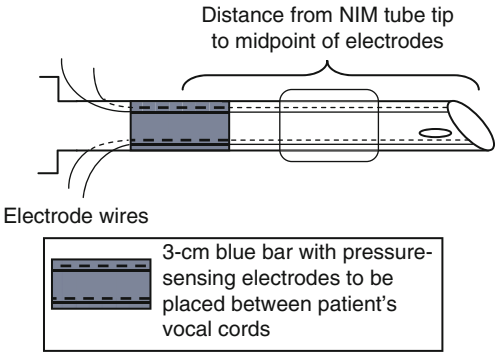


Fig. 5.1 Schematic of NIM tube in situ between vocal cords. ● Point of contact of vocal cords with NIM tube electrodes, ⋀ vocal cords, ○ NIM tube



Internal Diameter (mm)	Distance from NIM tube tip to midpoint of electrodes (cm)
6.0	9.0
7.0	10.5
8.0	11.5

Fig. 5.2 Schematic of the anatomy of NIM tube and relationship between internal diameter of NIM tube and distance of NIM tube tip to midpoint of blue electrode bar

one pair at the 8 o'clock and 10 o'clock positions along the tube (Fig. 5.1). The electrodes are exposed on the 3-cm-long blue bar that is to be positioned between the patient's vocal cords, so that the electrodes can make contact with the vocal cords. When the recurrent laryngeal nerve (RLN) is stimulated electrically in the surgical field by the surgeon, the vocal cords will adduct (if RLN function is intact), creating a pressure change sensed by the electrodes at the 2, 4, 8, and 10 o'clock positions on the NIM tube; the pressure changes are then converted to graphical changes on the NIM monitor screen or audible sounds.

L-3: **Dimensions of a NIM tube**
See Fig. 5.2 NIM tube dimensions.

L-4: **Key points for use of a NIM tube**
Medtronic recommends that a tube that is one size larger than standard selection is used whenever possible to improve electrode contact with vocal cords.
Lubricate the tube and cuff with an aqueous lubricant, avoiding local anesthetic gels which may impair monitoring vocal cord function.
Use a lubricated stylet for intubation.

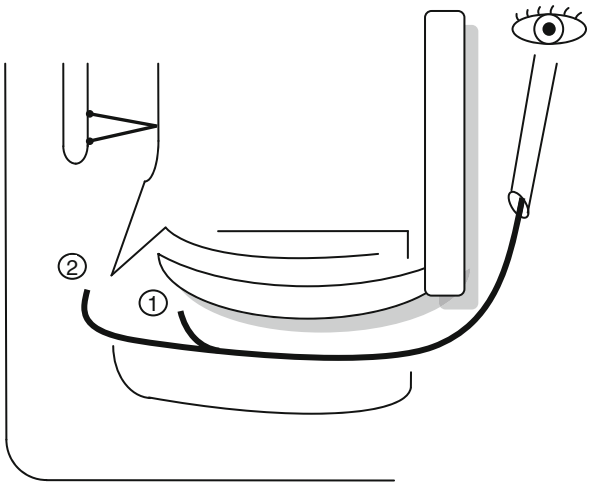


Fig. 5.3 Trajectory of the fiberoptic bronchoscope within the airway. The bronchoscope is first passed under the laryngoscope and identifies tip of the epiglottis (*1*). Then the bronchoscope is directed posteriorly around the epiglottis and then toward and through the vocal cords (*2*)

L-5: **How must a NIM tube be placed in a patient who needs an awake intubation**?

Remember, the NIM tube adapter and electrode wires preclude, passing the NIM tube through any 360° conduit.

In our patient, after proper preparation for an awake intubation, a gentle direct laryngoscopy was performed with Mac 3 laryngoscope which revealed that the right pharyngeal wall was deviated past midline to the left. The fiberoptic bronchoscope was passed under the laryngoscope and through the vocal cords (*see* Fig. 5.3). The NIM tube was advanced into the trachea without difficulty. After confirmation of tracheal intubation by FOB and end-tidal CO_2 with the patient's spontaneous ventilation, general anesthesia was induced with propofol. The patient tolerated the procedure well.

Recommended Reading

NIM™ EMG endotracheal tube, product information and instructions. Medtronic. 2006.

Chapter 6
Hemodynamic Collapse Following a Mainstem Intubation

Sameer J. Shah and Preetham Suresh

The patient was a 32-year-old female scheduled for a right needle-localized excisional breast biopsy. Aside from an abnormal mammogram and breast biopsy, the patient's history was significant for febrile seizures as a child, seasonal allergies, and marijuana use approximately once per week. She also stated that she had had an upper respiratory infection (URI) associated with sinus congestion about 1 week ago, and now had an ongoing dry cough, but denied fever or wheezing. Past surgical history was significant for a cesarean section for breech presentation, for which she underwent epidural anesthesia, and oral surgery performed under monitored anesthesia care but converted to general anesthesia because of patient discomfort. The patient's home meds included cetirizine, an oral contraceptive, and a multivitamin. She denied drug allergies. Physical exam revealed a 56 kg, 59″ (BMI = 27 kg/m^2) female (**L-1**). All vitals were within normal limits; of note her room air saturation (SpO_2) was 98 % and she was afebrile. Her throat was clear, she had no lymphadenopathy, and her lungs were clear to auscultation. Her airway exam revealed a Mallampati class II airway, with a thyromental distance of 5 cm, intact dentition, full range of motion of her neck, and the ability to prognath her jaw. Preoperative labs, EKG, and chest x-ray were deferred.

In the preoperative holding area, a 20-gauge peripheral IV was placed. When asked about her URI, she stated that she was back to her baseline and that she had run three miles the day prior to surgery. Patient's initial preference was to have the procedure under paravertebral block. But after a discussion about risks and benefits with the patient and the surgeon, the patient agreed to a general anesthetic. The patient was administered midazolam and taken to the operating room. Routine monitors were placed and she was fully preoxygenated. Preinduction vitals were as follows: BP of 125/70 mmHg, HR of 86 bpm, respiratory rate of 18 breaths per minute, and SpO_2 of 100 %. An IV induction was performed with lidocaine 60 mg

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and propofol 140 mg. A Cookgas size 3.5 laryngeal mask airway (LMA) was easily placed on the first attempt. Although there was no audible leak, high resistance was encountered with ventilation (**L-2**). There was no end-tidal CO_2 ($etCO_2$), no chest movement, or exhaled tidal volume. Additional propofol was given and an attempt to adjust the LMA was performed, but there was no improvement in the ability to ventilate (**L-3**). Bronchospasm was suspected, succinylcholine was given, and the LMA removed (**L-4**). Although initially there was no $etCO_2$ with mask ventilation, we slowly began to get the return of $etCO_2$ and adequate chest rise with positive pressure ventilation (**L-5**). Throughout this period the saturation was 100 % and the BP was 110/60 mmHg. Direct laryngoscopy was performed, revealing a grade I view of the vocal cords. A size 7-mm ETT was placed atraumatically under direct visualization past the vocal cords. Hand ventilation revealed $etCO_2$ in the mid-30s mm Hg, PIPs around 25 cm of water (cmH_20) with exhaled tidal volumes of 450–500 cc, and auscultation revealed bilateral lung sounds. The ETT was taped at 20 cm at the teeth and the patient was placed on the ventilator (**L-6**). PIPs remained at about 25 cmH_20, and hemodynamics and saturation were within normal limits for the next 5 min. One dose of dexamethasone 4 mg was administered for postoperative nausea prophylaxis. No major changes were made to the patient's position, but the circulating nurse and surgical resident did make minor adjustments to head and neck positioning while prepping and draping. Cefazolin 1,000 mg was then given as an IV bolus. Within the 30 s of administration, peak pressures were noted to increase to 28–30 cmH_20, and SpO_2 began to drop rapidly. Approximately 7 min had passed since succinylcholine was administered (**L-7**). The ventilator was switched to manual and the FiO_2 was turned to 100 %. Hand ventilation revealed increased resistance to air movement. At this time, $etCO_2$ was noted to sharply decline, going from the low 30s to the mid-20s mmHg. SpO_2 slowly increased with hand ventilation from 94 % to 95 %. The lungs were auscultated, revealing positive breath sounds on the right and questionable wheezes on left, but a paucity of air movement bilaterally. Given the temporal relation to cefazolin administration and dropping BP (90s/40s and then 70s/30s mmHg), the diagnosis of anaphylaxis was entertained (**L-8**). Epinephrine 5 μg was given, and additional anesthesia staff was called to the room. Diphenhydramine 50 mg and famotidine 20 mg were administered. Epinephrine boluses (a total of three were given) were associated with a definite easing of hand ventilation and increase in BP, though these effects were transient. An arterial line and second IV were placed during this time, and an epinephrine infusion was started at 0.05 μg/kg/min. After improvement in hemodynamics and lung mechanics, the patient was transitioned to the ventilator on previous settings. An ABG showed a pH of 7.4, $PACO_2$ of 36 mmHg, PaO_2 of 78 mmHg, base deficit of −2.2 mEq/L, and SaO_2 of 96 % on an FiO_2 of 100 %. At this point, the gown was removed to look for a rash and none was found. The trachea was examined and found to be midline. The surgeon was asked regarding the possibility of pneumothorax but she was able to palpate the end of the needle localizer which was superficially placed in the right breast. The chest was auscultated again and breath sounds were heard greater on the right than left. Inspection of the ETT showed that it was situated now at 22 cm at the teeth. FOB was performed, and

what was believed to have been the true carina was identified. The ETT was pulled back to 20 cm.

Auscultation still revealed diminished breath sounds on the left side, and a stat intraoperative chest x-ray (CXR) was performed. PIPs were down to approximately 24 cm H_2O at this time, and the patient was saturating still around 94–95 % with a BP of 80–90s/50s mmHg. The CXR revealed the ETT terminating in the right mainstem bronchus with significant volume loss of the left lung and shift of the mediastinum to the left (**L-9**).

FOB was again performed, and as the ETT was withdrawn over the scope, it became clear that the "carina" visualized earlier has been a division of the right mainstem bronchus. The ETT was secured at 17 cm, and auscultation revealed clear breath sounds bilaterally. SpO_2 increased rapidly to 100 % and PIP decreased to 18 cm H_2O with tidal volumes of 500 cc. The epinephrine infusion was discontinued and BP remained in the 90–100s/60–70s mmHg range. Recruitment breaths were given. Repeat CXR confirmed tracheal intubation and reinflation of the left lung. ABG showed a pH of 7.36, $PaCO_2$ of 40, PaO_2 of 462, base deficit of −2.8 mEq/L, and SaO_2 of 100 % on an FiO_2 of 100 %. After discussion with the ICU attending and the surgical team, it was decided that the period of right lung hyperinflation was unlikely to have persistent untoward sequelae and the decision was made to proceed with the case. The case proceeded uneventfully, and prior to extubation, the patient's lung fields were clear to auscultation and she had a normal SpO_2 and a normal ABG on an FiO_2 of 30 %. Extubation and postoperative course were uneventful, and the patient was discharged the following day.

Lessons Learned

L-1: **Variability in tracheal length and distance to vocal cords**
A study done by Drs. Lozano, Lee, and Benumof at the University of California, San Diego (UCSD) aimed to measure the upper incisor to vocal cord length and tracheal length in male and female participants and to determine if placement of an ETT at a "standard" 23 cm for males and 21 cm for females is a safe practice. Using fiberoptic bronchoscopy to gather data from 30 participants, the group found the measurements shown in Table 6.1.

The findings show that while there is a linear correlation between height and airway length, there exists considerable variability. The group also found that the

Table 6.1 Tracheal length, upper incisor to vocal cord length, and upper incisor to carina length in males and females

Gender (*n*)	Tracheal length (cm)	Upper incisor to vocal cord length (cm)	Upper incisor to carina length (cm)
Males (16)	11.0 ± 0.7 (95 % CI)	16.3 ± 0.7 (95 % CI)	27.4 ± 0.8 (95 % CI)
Females (14)	10.6 ± 0.4 (95 % CI)	14.3 ± 0.5 (95 % CI)	24.9 ± 0.4 (95 % CI)

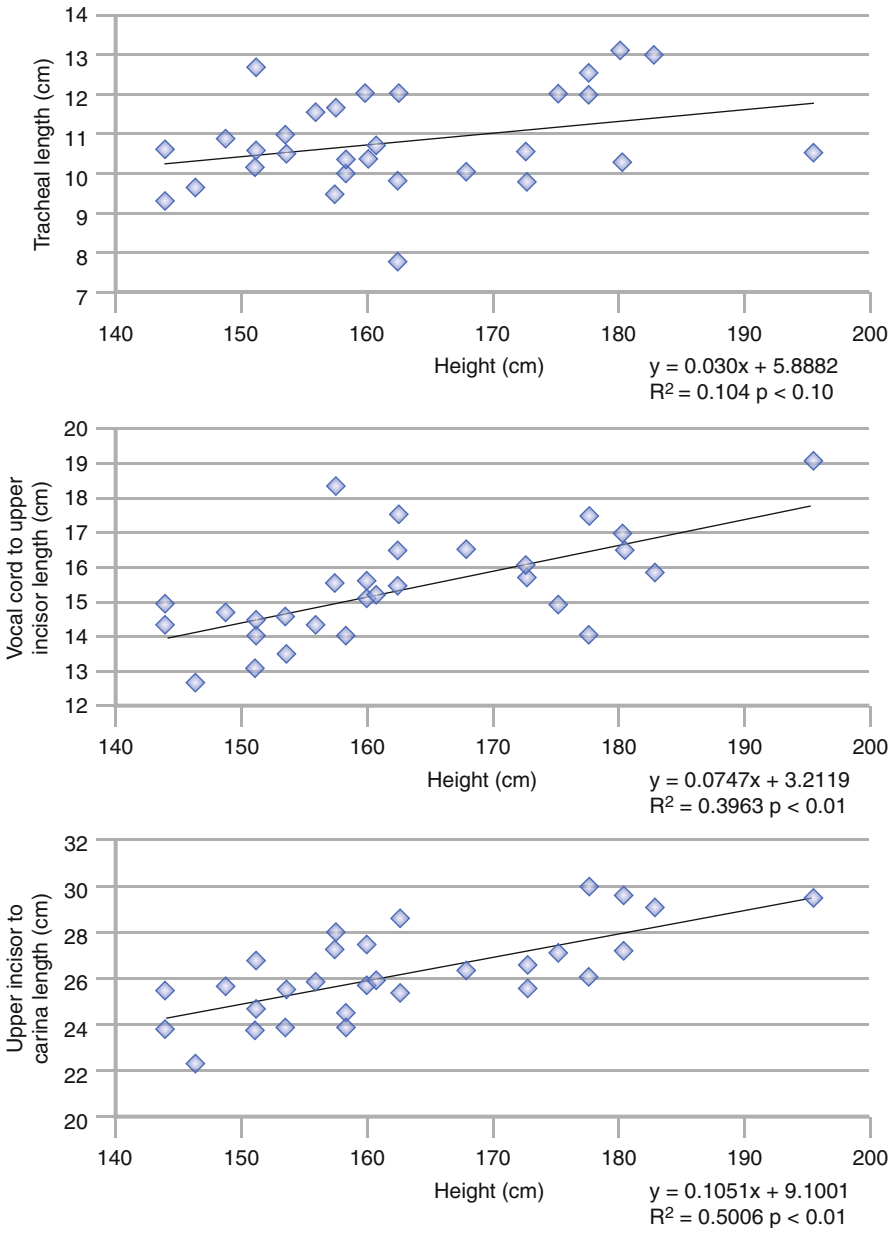


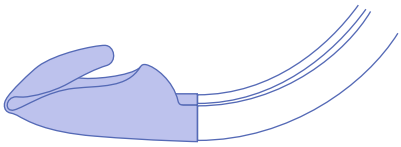
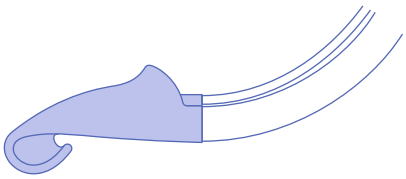
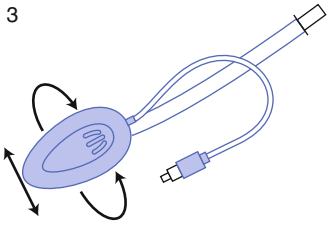
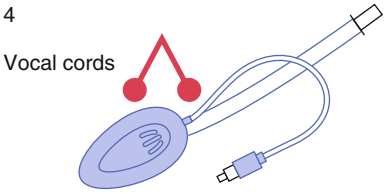
Fig. 6.1 The *top panel* shows the relationship between height and tracheal length, the *middle panel* shows the relationship between height and upper incisor to vocal cord length, and the *bottom panel* shows the relationship between height and upper incisor to carina length. Note that while each demonstrates a correlation, there exists considerable variability between individuals of a given height

routine taping of the ETT at 23 cm in males and 21 cm in females would result in an unacceptable distal position of the tip of the ETT (≤3 cm from carina) in 31 and 36 % of participants, respectively. Looking at this data, we would have expected our patient, whose height was 4′11″ or 150 cm, to have a tracheal length of around 10 cm and an upper incisor to vocal cord length of around 14 cm. Figure 6.1 shows the correlation of the three lengths with height in the combined male and female groups; p values were $p < 0.10$ for tracheal length, $p < 0.01$ for upper incisor to vocal cord length, and $p < 0.01$ for upper incisor to carina length.

L-2: **Possible malpositions of an LMA**
There are several possible malpositions of an LMA, as seen in Table 6.2.

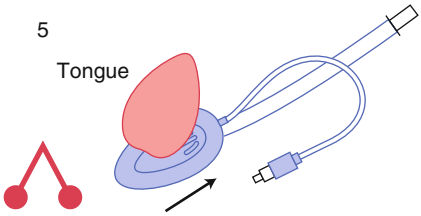
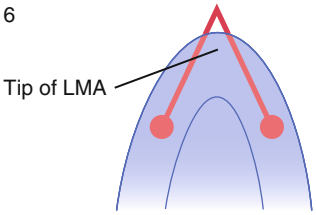
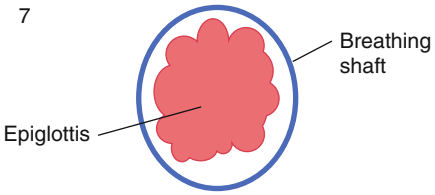
L-3: **Adjustments to a malpositioned LMA**
A malpositioned LMA may present with difficulty ventilating (no air movement or low tidal volumes with high positive pressure) or large air leaks at low inspiratory pressures or volumes. As a general rule, blindly (i.e., without an FOB), moving an LMA in or out or rotating it is unlikely to correct a malposition (see Table 6.2).

Table 6.2 Malpositions of an LMA, as observed by FOB

Illustration of malposition	Description of malposition	Findings with malposition
1 	Tip folded forward	Very large leak
2 	Tip folded backward	Very large leak
3 	Laterally displaced or rotated	Very large leak
4 	In too deep, bowl of LMA distal to vocal cords	High resistance

(continued)

Table 6.2 (continued)

Illustration of malposition	Description of malposition	Findings with malposition
	In too shallow, tongue is occluding bowl of LMA	High resistance or low resistance (with a large leak) depending on degree of occlusion of the bowl of the LMA by the tongue
	Tip of LMA abutting against vocal cords	Ventilation with normal limits, minimal leak, increased risk for laryngospasm
	Epiglottis occluding distal end of breathing shaft	Normal to high resistance with minimal leak depending on degree of occlusion

A corollary to this is that accurate diagnosis of a malposition can only be done with the aid of an FOB. Correction of a malpositioned LMA should start with complete removal of the device and then starting the process of inserting it over again:

1. Ensure you have the correct size LMA for the patient, using the weight-based guide on the LMA and your own preoperative airway exam and clinical judgment.
2. Inspect the LMA for tears and verify that the cuff does not leak when inflated. Depending on the type of LMA and the practitioner, you may want to deflate the cuff to form a smooth flat wedge shape.
3. Lubricate the posterior surface of the mask.
4. Optimally position the patient in "sniff" position – flex the neck and extend the head (see Fig. 6.2). This position best aligns the oropharyngeal, laryngeal, and tracheal axes in a straight line. In larger patients a ramp is critical in bringing the ear canal higher than the level of the sternum.
5. Adequately anesthetize the patient – a lightly anesthetized, tight-jawed patient will make placement of the LMA difficult or impossible.
6. Hold the LMA like a pen, with the index finger of the dominant hand at the junction of the mask and breathing shaft (see Fig. 6.3).
7. Slide the LMA along the hard palate, pushing it back against the palate as it is advanced towards the hypopharynx. Using your index finger as a guide for the tip of the LMA can help prevent the tip from folding over and helps get the tongue out of the way (see Fig. 6.4).

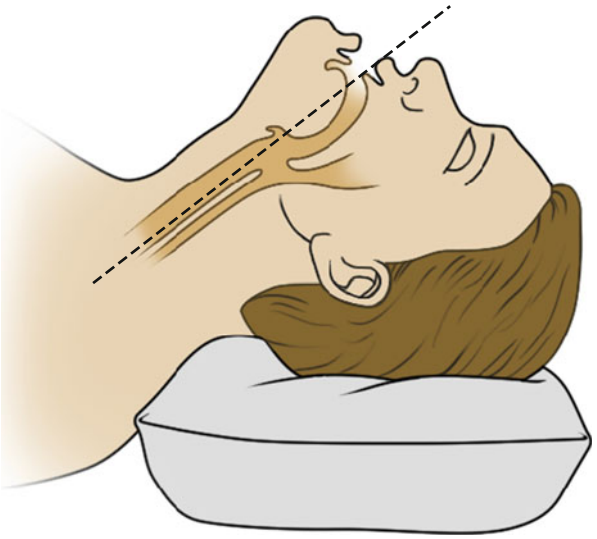


Fig. 6.2 "Sniff" position, optimally aligning oropharyngeal, laryngeal, and tracheal axes by flexing the neck on the chest and extending the head on the neck at the atlantooccipital joint

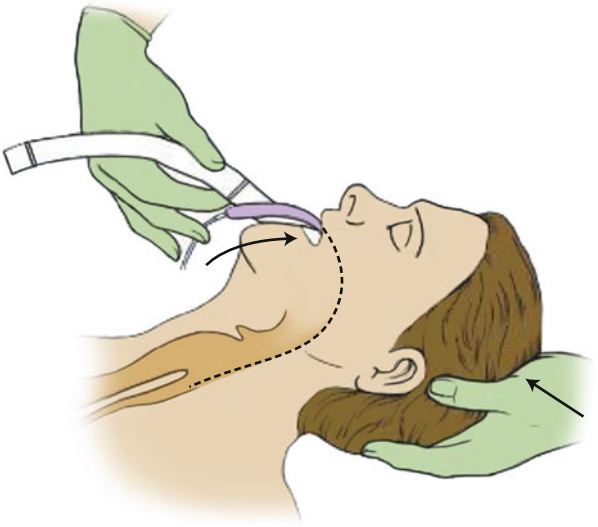


Fig. 6.3 Hold the LMA at the junction of the mask and breathing shaft

8. Alternatively, if the tip of the LMA impacts against the posterior pharynx and will not turn towards the vocal cords, the index finger of the nondominant hand can be positioned posterior to the tip of the LMA in the hypopharynx and can flex the tip anteriorly towards the vocal cords (a "kicking a field goal" motion; see Fig. 6.5).
9. Advance gently until resistance is met.
10. Inflate the cuff with an appropriate volume of air.
11. Verify placement with etCO_2 and auscultation.

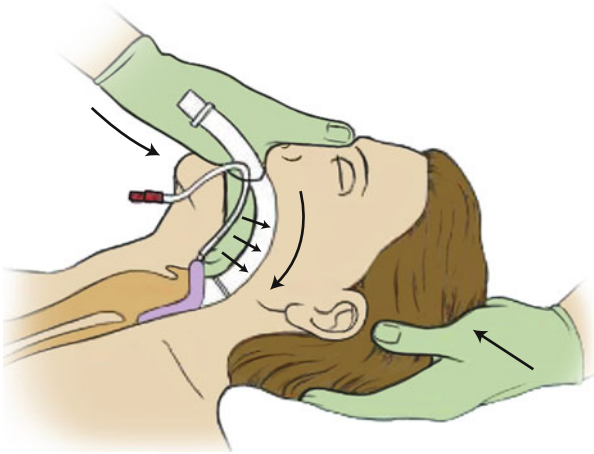


Fig. 6.4 Slide the LMA along the hard palate using the index finger of your dominant hand

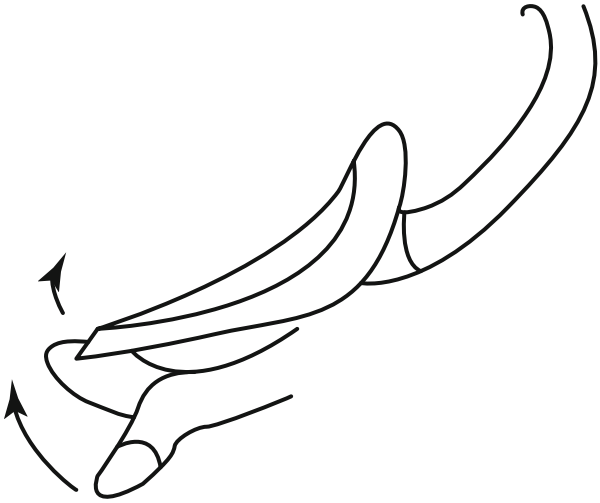


Fig. 6.5 A "kicking a field goal" motion can direct the LMA towards the vocal cords

L4: **Differential diagnosis of the inability to ventilate/no $etCO_2$ scenario**
Every practicing anesthesiologist should have a basic approach to the inability to ventilate/no $etCO_2$ scenario. One approach has a practitioner start at the gas analyzer and work her way down to the patient, from the oropharynx down to the lung parenchyma and pulmonary circulation, as detailed below in Table 6.3.

L5: **How to optimally ventilate via mask**
In our case, we attempted to mask ventilate after administration of succinylcholine and removal of the LMA. At first, we were unable to ventilate with positive pressure, but within about thirty seconds, the paralytic began taking effect and we were able to move air. Figure 6.6 shows how to perform optimal ventilation via mask.

Figure 6.7 illustrates the increasing difficulty of mask ventilation and endotracheal intubation, with the extremes being relatively easy (0) and impossible (infinity). Review the steps one must take to ventilate the progressively more and more difficult patient.

L6: **How to properly tape an ETT**

Proper fixation of the ETT is a simple but crucial step in maintaining its position throughout an operation, as seen in our case. For most patients, securing the ETT with adhesive tape is standard.

1. Note the cm mark at the teeth.
2. Adhesive tape should be applied to the skin of the maxilla, preferably getting purchase on as much skin as possible bilaterally. Caution should be exercised when wrapping anything completely around the neck however as this may impair venous drainage. Lay the tape down smoothly, avoiding pinches and creases in the skin (see Fig. 6.8).
3. The tape should be wrapped around the ETT as close as possible to the teeth: it is our observation that the majority of the time, the tape is placed above the teeth, applying inward traction when the tape is once again brought into contact with the skin. The inward traction can result in subsequent inward migration of the ETT 1–2 cm, easily the distance necessary for a mainstem intubation (see Figs. 6.9 and 6.10).
4. A second piece of tape should be applied horizontally over the first (see Fig. 6.10).
5. If the lateral anchors are not sticking to the patient, consider benzoin or Tegaderm. If using twill ties, make sure to again choke up on the ETT as close to the teeth as possible with your knot.

L7: **Succinylcholine**: **a brief review**

Succinylcholine, or more accurately succinyldicholine (SDC), is a depolarizing neuromuscular blocker composed of two linked molecules of acetylcholine. Like acetylcholine (Ach), SDC stimulates cholinergic receptors at the neuromuscular junction (NMJ) and at nicotinic (ganglionic) and muscarinic autonomic sites, thereby opening the ionic channel in the acetylcholine receptor. Table 6.4 covers the basic pharmacokinetics and pharmacodynamics of SDC.

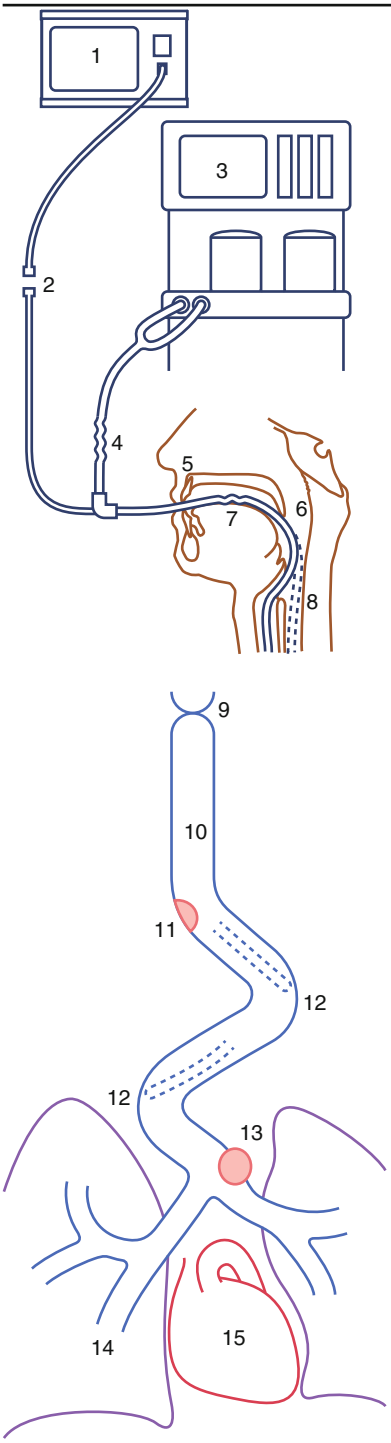
People with atypical butyrylcholinesterase can exhibit prolonged durations of action (see Table 6.5). Return of twitches should be checked with a twitch monitor before administration of a longer-acting paralytic after SDC has been given, such as for an intubation (Table 6.5).

While the combination of rapid onset and ultrashort duration makes SDC a necessary drug in the anesthesiologist's armamentarium, the adverse effect profile is long and significant. See Table 6.6 to review important adverse effects.

L8: **Causes of decreasing $etCO_2$**

It is important to understand $etCO_2$ in terms of the ventilation/perfusion ratio ($\dot{V}/\dot{Q}$), namely, that with increased $\dot{V}/\dot{Q}$, $etCO_2$ will decrease. In other words, an increase in ventilation (relative to perfusion) or a decrease in perfusion (relative to ventilation) will cause a decrease in $etCO_2$. The latter is what likely occurred in the case above, as the $etCO_2$ fell as blood pressure (and cardiac output) fell. There are myriad causes

Table 6.3 Basic approach to inability to ventilate/no $etCO_2$ scenario

Schematic	Description
	1. Gas analyzer malfunction 2. Sampling line disconnect 3. Anesthesia machine malfunction 4. Circuit leak or kink 5. Teeth clamped down on airway 6. Oropharyngeal masses, enlarged lingual tonsils 7. ETT kinked 8. ETT in esophagus or oropharynx 9. Laryngospasm 10. ETT occluded with mucous plug, blood, edema, pus, vomit 11. Intraluminal mass occluding the bevel of the ETT 12. Pneumothorax, hemothorax, chylothorax, or aortic aneurysm (left of trachea) causing "S"-shaped deviation of trachea, bringing the bevel of the ETT up against wall of the airway 13. Extraluminal (mediastinal) mass compressing the airway (usually at carinal area) 14. Very intense bronchospasm 15. No pulmonary circulation/cardiac arrest

Airway pressures	Diagnostic maneuvers, signs and symptoms
Normal	Disconnect sampling line and exhale into it
Normal	
Variable	Switch to hand ventilation and progressively isolate various parts of machine (valve incompetence, CO_2 canister misconnect, bag leaks, etc.). Switch to O_2 tank and Mapleson circuit if necessary
Low (leak) or high (kink)	
High	
High	
High	
Low	Auscultate, repeat laryngoscopy, and consider use of esophageal detector bulb or fiberoptic bronchoscopy in low cardiac output situation
High	Auscultate for stridor and restricted air movement. Assess the patient's depth of anesthesia. Watch for negative pressure pulmonary edema
High	Pass a suction catheter through ETT
High	
High	Auscultate, look for asymmetric chest rise, check for tracheal deviation
High	
High	Auscultate, can give a trial of bronchodilators. Look for signs of anaphylaxis (rash, urticaria, hemodynamic collapse)
Normal	Check other vital signs

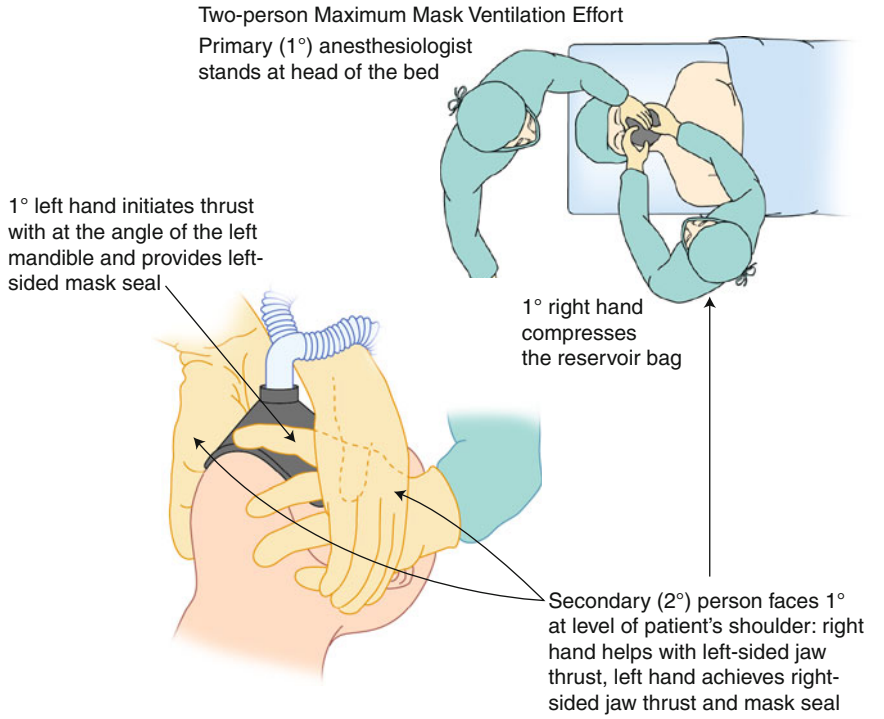


Fig. 6.6 Schematic detailing the correct position and technique for two-person mask ventilation

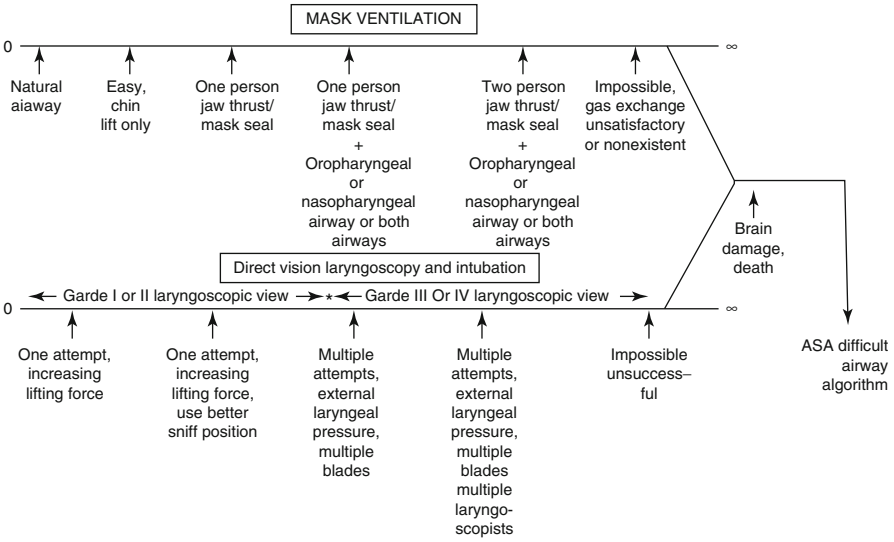


Fig. 6.7 Traveling down each line towards infinity, one can review the progressive steps taken when a difficult mask ventilation or intubation situation is encountered. At infinity one reaches a "cannot ventilate/cannot intubate" scenario that is incorporated into the ASA difficult airway algorithm

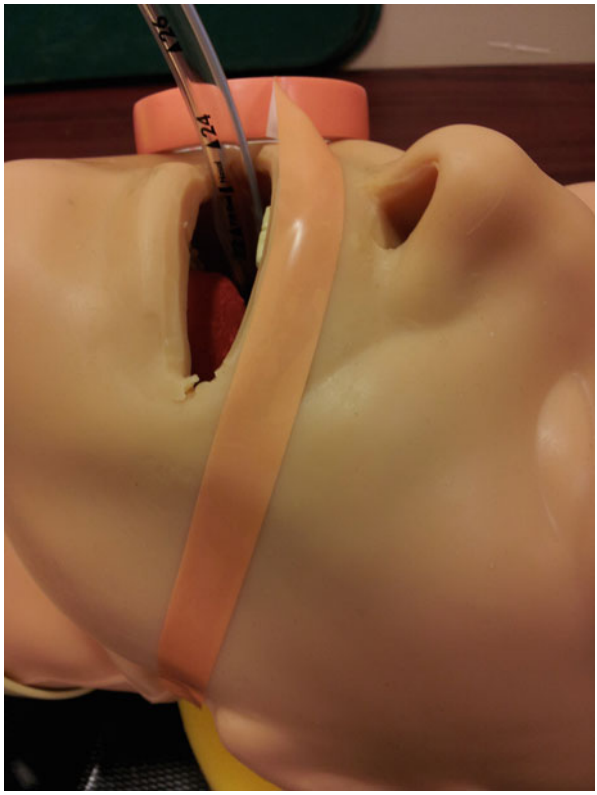


Fig. 6.8 Tape is applied smoothly, with purchase on as much skin as possible

of decreased $\dot{Q}$. Hyperventilation, either iatrogenic or spontaneous, will increase $\dot{V}$ relative to $\dot{Q}$ and result in a gradual decline in $etCO_2$. It should be stated that if production of CO_2 decreases with constant minute ventilation (such as in hypothermia or hypothyroid states), this will also result in an increase in $\dot{V}$ relative to $\dot{Q}$ and a gradual decline in $etCO_2$.

L9: **So what actually happened**?

This case emphasizes a simple lesson that cannot be overstated: do a thorough preoperative evaluation, paying close attention to the patient's height and weight! We also see from the data presented in **L-1** that significant variability in airway lengths exists even for patients of equal height. At a height of 150 cm, we would have expected our patient's trachea to be around 14 cm long and her incisor-to-carina distance to be around 24 cm. As we learned through the events of the case, her airway anatomy fell well on the short side of the normal bell curve. The high resistance we encountered after placing the LMA was likely due to a type 4 malposition, with the tip of the LMA in too deep, distal to the vocal cords and in the esophagus (see Table 6.2). Though we correctly removed the LMA and reinserted it, we again likely placed it too deep. The initial difficulty in ventilating via mask may have been due to some degree of bronchospasm due to repeated instrumentation of the patient's airway and the patient's history of a recent upper respiratory infection, ongoing cough,

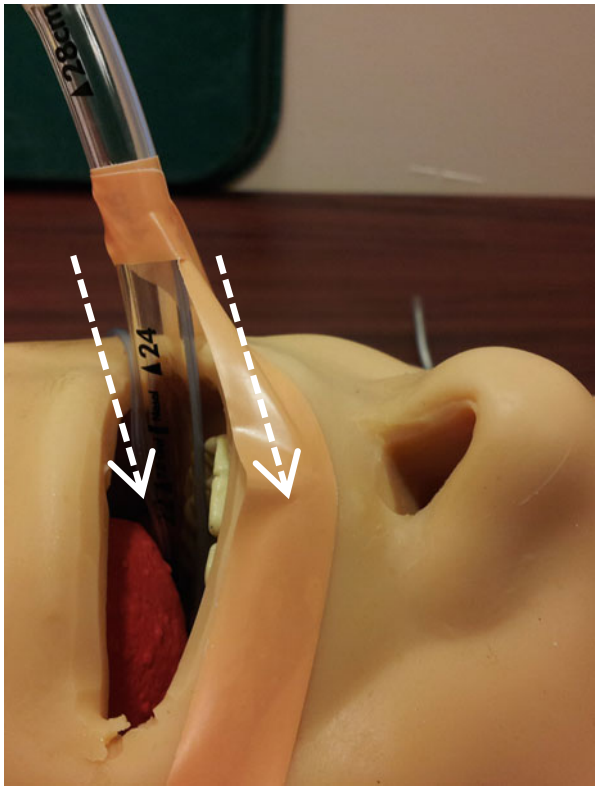


Fig. 6.9 Taping too high above the teeth can cause inward traction

and marijuana use. Another possibility, perhaps more likely, was suboptimal ventilation via mask (not making use of an oropharyngeal airway) and then relaxation of the oropharyngeal and airway muscles once the succinylcholine took effect (see Fig. 6.7).

Once the ETT was in, we likely made the mistake of taping the tube too high above the teeth (see Fig. 6.9); the inward traction is what likely brought the ETT down to 22 cm at the teeth, as was subsequently discovered. The rapid drop in $etCO_2$, concomitant drop in BP, and recent administration of cefazolin and succinylcholine (both potential inciting agents) made the presumptive diagnosis of anaphylaxis a reasonable one. The hemodynamic collapse associated with anaphylaxis could lead to a hypoperfusion state that would decrease perfusion even more than it decreased ventilation (through widespread bronchospasm), resulting in an overall increased $\dot{V}/Q$ and a drop in $etCO_2$. Bronchospasm would have also accounted for the increase in peak inspiratory pressures. However, upon examining the intraoperative radiographs and analyzing what we saw with fiberoptic bronchoscopy, it became evident that what we were primarily dealing with was actually a right mainstem intubation that developed "tension" physiology. Figure 6.11 is the first radiograph taken. The ETT is clearly seen deviating to the right. The nonventilated left lung has almost totally collapsed and the overexpanded right lung has shifted mediastinal structures dramatically to the left, likely severely limiting venous return.

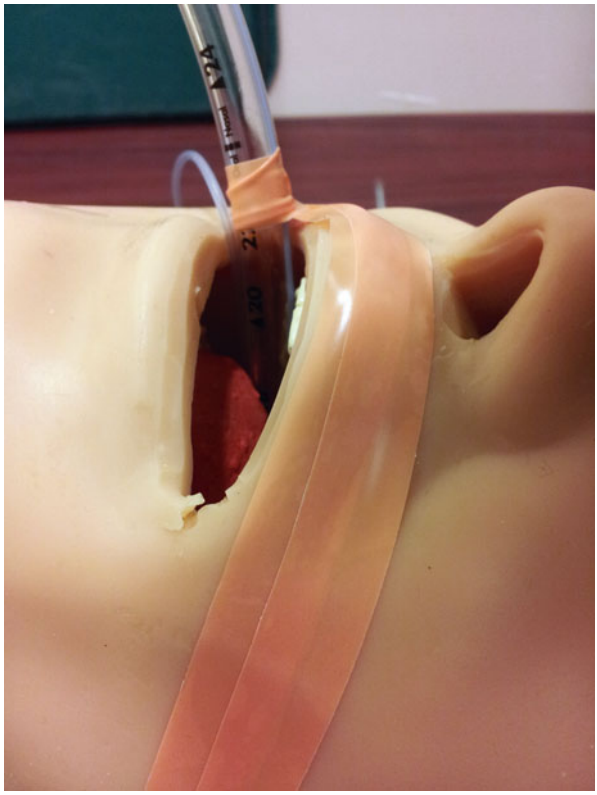


Fig. 6.10 Tape is applied closely to the teeth, and a second piece of tape is applied horizontally over the first

Table 6.4 Basic pharmacokinetics and pharmacodynamics of succinyldicholine

Intubating dose	1.0–1.5 mg/kg
Onset of action	60 s
Duration of action	Recovery of 90 % muscle strength within 9–13 min
Termination of action	Secondary to diffusion away from the NMJ
Elimination	Butyrylcholinesterase rapidly hydrolyzes SDC before and after it reaches the NMJ

Table 6.5 Butyrylcholinesterase deficiency

Type of butyrylcholinesterase	Incidence	Dibucaine number (the percentage by which cholinesterase activity in a serum sample is inhibited by dibucaine)	Response to SDC
Homozygous typical	Normal	70–80	Normal
Heterozygous atypical	1/480	50–60	Lengthened by 50–100 %
Homozygous atypical	1/3,200	20–30	Prolonged by 4–8 h

Table 6.6 Adverse effects of succinyldicholine

Anatomical location	Adverse effect	Key points
Brain	Increased intracranial pressure	Inconsistent and probably not clinically significant
Eyes	Increased intraocular pressure	Intraocular pressure can increase 5–15 mmHg. Though there is little evidence of blindness or extrusion of eye contents with its use, it is generally recommended that SDC not be used in open eye injuries
Masseter	Masseter spasm	A sustained increase in tension that may last for several minutes may be observed. This can be an isolated event or a harbinger of malignant hyperthermia
Heart	Sinus bradycardia, ventricular arrhythmias, nodal arrhythmias	Likely caused by activation of muscarinic receptors. Sinus bradycardia is more common in children but can also be seen in adults, especially after a second dose. Effects can be attenuated or prevented with atropine or glycopyrrolate
Stomach	Increased gastric pressure	While gastric pressure is increased, SDC also increased lower esophageal sphincter (LES) pressure, negating an increase in risk of aspiration of gastric contents unless the LES is incompetent
Muscle	Myalgias	Generalized aches and pains are common 24–48 h after administration, most common in young, ambulatory patients. Precurarization is effective against these
Muscle, systemic	Hyperkalemia	In pathologic states such as major denervation injuries, spinal cord transection, stroke, peripheral denervation, trauma, extensive burns, prolonged immobility, overexpression of Ach receptors on muscle can lead to potassium efflux, increasing serum concentration 0.5–1.0 mEq/mL. Cardiac arrest has occurred. Renal failure does not increase risk for exaggerated response
Systemic	Malignant hyperthermia	Trigger for malignant hyperthermia; should be avoided in patients with known history or family history
Systemic	Anaphylaxis	High incidence of anaphylaxis compared with other anesthesia drugs, 1:5,000–1:10,000

So what were we actually seeing when we thought we identified the main carina immediately distal to our ETT during our initial bronchoscopy? It is likely we mistook the secondary carina separating the right upper lobe and bronchus intermedius for the main carina. A general rule is that while the main carina has a very sharp bifurcation, the secondary carina on the right has a unique broad bifurcation (see Figs. 6.12 and 6.13).

The unique bifurcation of this right secondary carina is a broad enough surface to occlude the beveled orifice of an ETT. We propose that in our case, the ETT was abutting this carina; the positive pressure during inhalation would push the tube back and inhaled air would enter the right lung. However, during passive exhalation,

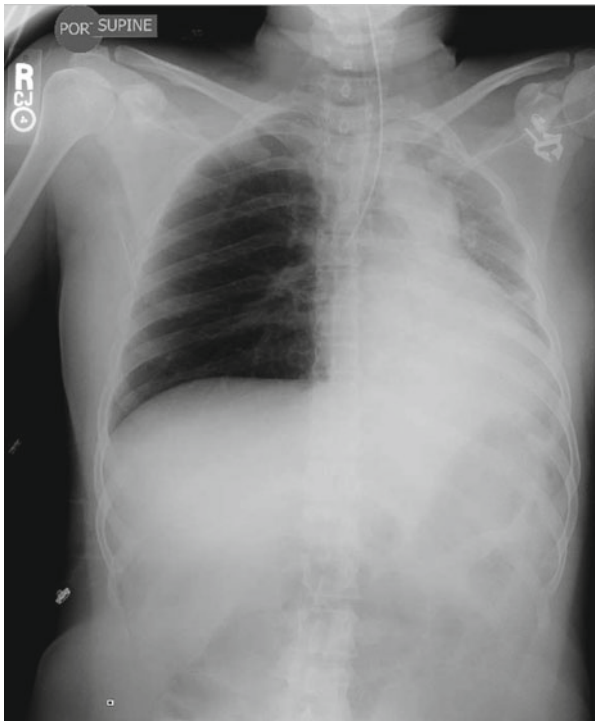


Fig. 6.11 The first intraoperative radiograph clearly shows the ETT traversing the right main bronchus, left lung collapse, and right lung overinflation with mediastinal shift

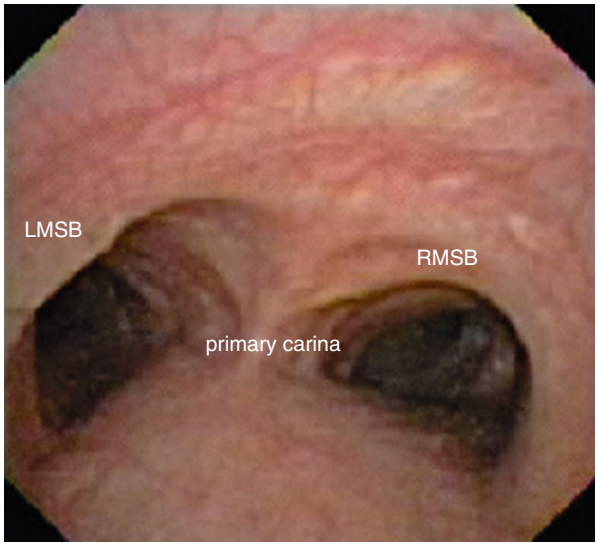


Fig. 6.12 The main carina has a very sharp bifurcation. *LMSB* left mainstem bronchus, *RMSB* right mainstem bronchus

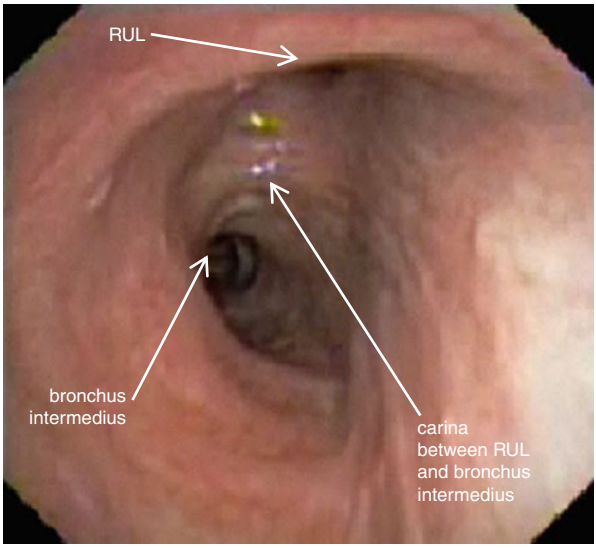


Fig. 6.13 The secondary carina separating the right upper lobe and bronchus intermedius has a very broad bifurcation. *RUL* right upper lobe

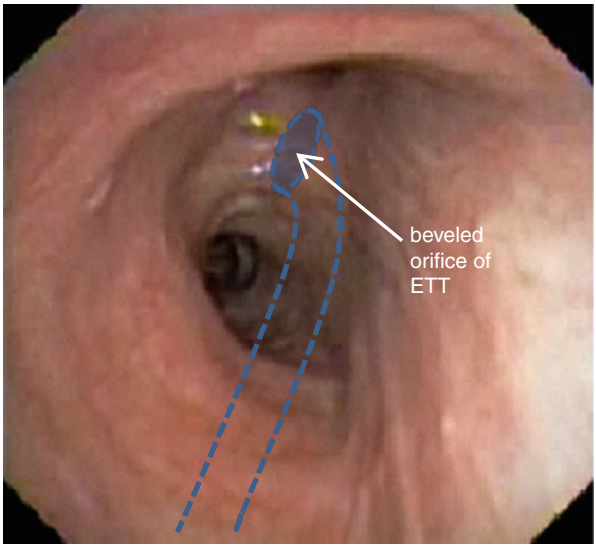


Fig. 6.14 Beveled orifice of ETT occluded by broad secondary carina

the carinal tissue would occlude the beveled orifice and allow egress of a much smaller volume of air. This ball-valve effect ultimately led to overinflation of the right lung and hemodynamic collapse from resulting "tension" physiology (see Fig. 6.14). Withdrawal of the ETT in a total of 5 cm back, to 17 cm at the teeth, brought the tip of the ETT into the trachea, producing clear bilateral breath sounds and normalization of hemodynamics and ventilator pressures. Arterial blood gases and a second intraoperative radiograph confirmed recovery (see Fig. 6.15).

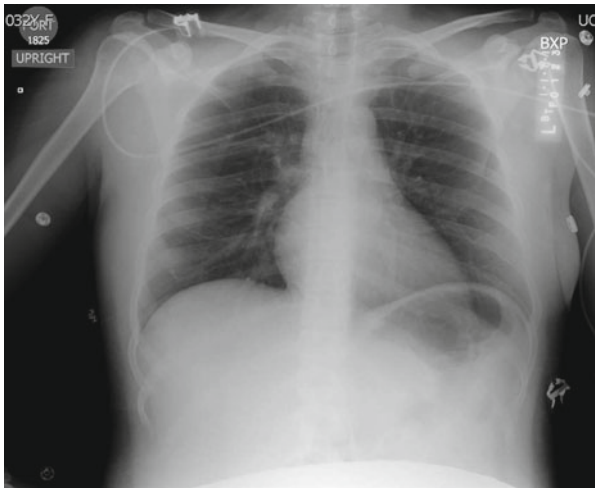


Fig. 6.15 Second radiograph shows reinflation of the left lung and resolution mediastinal shift after withdrawal of the ETT

Recommended Reading

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Chapter 7
Hypoxemia During Tracheostomy

Sun Choe Daly

The patient is a 36-year-old male who presents for a tracheostomy. The patient is 70 in. tall and his BMI is 40. The patient was a victim of a traumatic injury, and his hospital course has been complicated by development of severe adult respiratory distress syndrome requiring aggressive ventilatory support. The decision is made to transport the patient to the operating room from the intensive care unit with bag ventilation via Mapleson circuit (**L-1**).

The case proceeds without complications until the endotracheal tube is advanced and inspired oxygen concentration is decreased to 40 % prior to tracheal incision (**L-2**). The patient develops severe hypoxemia (**L-2**, **L-3**).

Lessons Learned

L-1: Should the patient who is on a ventilator in the ICU be transported to the OR with a transport ventilator?

The answer to the above question depends on the ventilation requirements of the patient. If the patient needs only partial ventilatory support, then a transport ventilator may not be necessary. In cases where the patient is requiring full ventilatory support as indicated by high end-expiratory pressures, high oxygen concentrations for non-hypoxemic PaO_2, and high minute ventilation for normal $PaCO_2$, it will be prudent to have a transport ventilator with a respiratory technician to aid with transport to the operating room (see Table 7.1).

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Table 7.1 Ventilation parameters to consider prior to transport

Parameters	Partial support[a]	Full support[a]
Oxygen concentration to avoid $PaO_2 < 60$ mmHg	<40 %	>60 %
PEEP to avoid $PaO_2 < 60$ mmHg	<10 cm H_2O	>12 cm H_2O
Minute ventilation to avoid $PaCO_2 > 45$ mmHg	<10 L/min	>15 L/min
Ventilation strategy	Volume control, pressure support, IMV, SIMV, standard I:E ratio	APRV, inverse I:E ratio, bilevel ventilation

APRV airway pressure release ventilation, *IMV* synchronized intermittent mechanical ventilation, *SIMV* synchronized intermittent mechanical ventilation, *I:E* inspiratory to expiratory, *PEEP* positive end expiratory pressure

[a]Note the boundary conditions for partial and full support are noncontinuous. For the middle grey zone, clinical judgment is required

L-2: When the surgeon is about to incise the trachea, what are the two things that need to be done?

1. Decrease oxygen concentration to less than 40 % to decrease risk of airway fire if electrocautery is to be used.

   (a) If SpO_2 decreases to less than acceptable levels with decreased inhaled concentration of oxygen, then advise surgeons the patient will not tolerate concentration reduction, and consequently, they must use the clamp and tie method to control bleeding in the field. Increase oxygen concentration as needed to maintain acceptable SpO_2. Under no circumstances should the electrosurgical unit be used with oxygen concentration greater than 50 %.

2. The cuff of the endotracheal tube is usually in the area of the T2–T3 rings which is in the area of the incision. In order to protect the cuff when the surgeon is about to incise the trachea, the endotracheal tube should be advanced 5 cm into the trachea.

   (a) When the endotracheal tube is advanced 5 cm deeper into the trachea, a right mainstem intubation may occur due to:

   The bevel direction of the endotracheal tube
   The larger surface of the right mainstem bronchus
   The shallower angle of the takeoff of the right mainstem bronchus off of the carina (see Fig. 7.1)

L-3: What should be done as the surgeon is about to insert the tracheostomy tube?

The endotracheal tube should be pulled back to the cephalad margin of the incision into the trachea, with the cuff just caudad to the vocal cords so that the endotracheal tube may be pushed back down the trachea in case the surgeon misses putting the

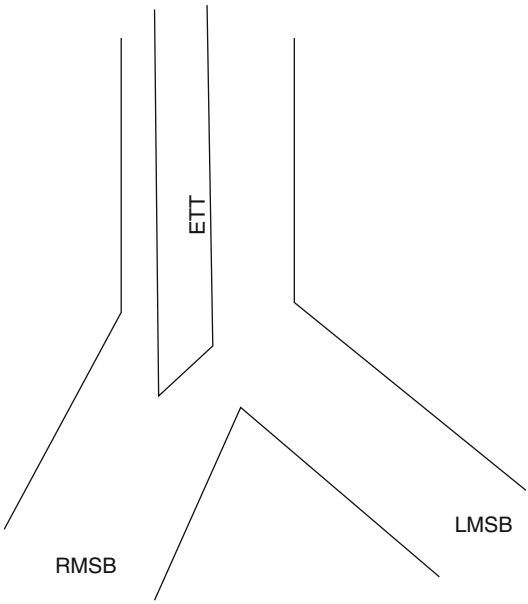

Fig. 7.1 Tendency of endotracheal tube to enter the right mainstem bronchus. *ETT* endotracheal tube, *RMSB* right mainstem bronchus, *LMSB* left mainstem bronchus

tracheostomy tube into the trachea. It is prudent to leave the endotracheal tube past the vocal cords in place until the patient has been transferred back to the intensive care unit in case the newly placed tracheostomy dislodges during transport.

Chapter 8
Anesthetic Depth and Mask Ventilation in the Prone Position

Bahareh Khatibi

The patient was a 26-year-old male (215 lbs, 5′11″) presenting for T11–L1 posterior fusion s/p a T12 burst fracture 2 days prior during a dirt bike accident. The patient was otherwise healthy. Social history was positive for alcohol and tobacco use (1 ppd × 10 years) and occasional marijuana. He denied any other drug use. Airway was favorable and preoperative labs were normal.

The patient was induced on the gurney uneventfully (2 mg versed, 200 mcg fentanyl, 100 mg lidocaine, 200 mg propofol, and 120 mg succinylcholine), intubated (**L-1**), and then flipped prone onto the OSI table. Given that the patient was to have this procedure using intraoperative evoked potentials, the following anesthetic was employed: 50 % nitrous oxide, 0.5 % expired sevoflurane, propofol infusion at 150–200 mcg/kg/min, and fentanyl boluses as needed. No neuromuscular blockade was utilized.

The case proceeded uneventfully and the surgeons began closure of the incision. At this point, propofol was discontinued, fentanyl 100 mcg IV was given, sevoflurane was kept at 0.5 %, and nitrous oxide was increased to 66 % (**L-2, L-3**) in preparation for emergence. Approximately 15 min later, the patient began to move and pushed himself up with his back extended and his head up off the prone view mask (**L-4**). The patient was immediately given 50 mg of propofol and it was noticed that the patient had extubated himself when he lifted his head off the prone view mask (**L-5, L-6**).

The incision was immediately dressed with tegaderms and the patient was flipped supine. He was spontaneously ventilating at this point and vital signs were stable with oxygen saturation of 100 % with face mask. Given that the patient's wound was not fully closed and that the surgeons needed him in the prone position to close the incision, the decision was made to reintubate the patient. Induction proceeded uneventfully (5 mg versed, 100 mg propofol, and 100 mg succinylcholine), and

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after intubation, the patient was flipped prone and the case proceeded. Following closure, the patient was positioned supine and extubated without complication.

The patient was taken to the postanesthesia care unit with no recollection of intraoperative events. He was started on vancomycin and gentamicin immediately postoperatively, and the remainder of the hospital stay was uneventful. The patient was discharged from the hospital on postoperative day 3.

Lessons Learned

L-1: Where is the tip of an endotracheal tube?

(a) Grade I view?

1. 5 cm (tip of ETT to cephalad portion of cuff)
   1 cm (cuff 1 cm below vocal cords)
   +15 cm (distance from lips to vocal cords)
   = 21 cm at the lip

(b) Grade II or III view?

1. 21–22 cm at the lip

(c) In either situation, given that the trachea is 11 cm long, the tip of the ETT should be near the midpoint of the trachea (i.e., approximately 6–7 cm into the trachea).

L-2: What is a MAC?

Calculate the depth of anesthesia in terms of MAC multiples in this case.

(a) MAC is the minimum alveolar concentration, defined as the concentration of inhaled anesthetic at 1 atm that prevents movement to a standard surgical stimulus in 50 % of patients (see Fig. 8.1).

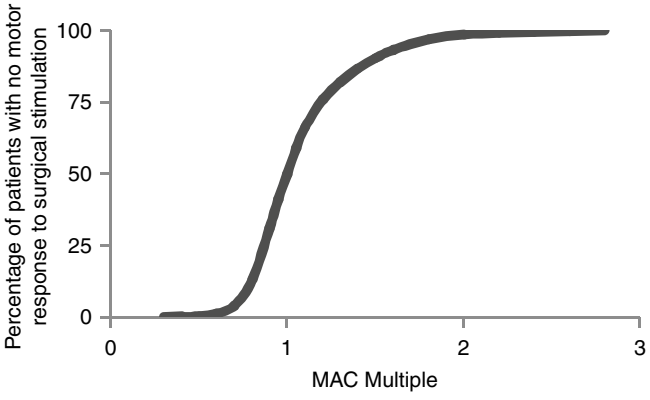

Fig. 8.1 Relationships between MAC multiple of anesthetic provided and the percentage of patient with no motor response to surgical stimulation

(b) $\text{Total MAC} = \text{sum of}\left(\%\,\text{end - tidal anesthetic} / \text{MAC of anesthetic}\right)$
$= \text{MAC multiple of nitrous oxide} + \text{MAC multiple of sevoflurane}$
$= \left(0.66\ \% / 104\ \%\right) + \left(0.5\ \% / 2.1\ \%\right)$
$= 0.63 + 0.24$
$= 0.87\,\text{MAC}$

L-3: How do you monitor depth of anesthesia?

(a) Autonomic responses to stimulation such as hypertension, tachycardia, sweating, and tearing can suggest light anesthesia.
(b) Several numeric indices derived from EEG aim to assess level of sedation. These include spectral edge frequency, median frequency, and bispectral index (BIS). The BIS monitor, for example, reports a single number ranging from 100 (awake) to 0 (isoelectric EEG). The manufacturer recommends a BIS value between 40 and 60 as an appropriate level for general anesthesia.
(c) O_2 (or CO_2) consumption

1. Noninvasive determination of oxygen consumption (VO_2) can be estimated from the analysis of respiratory gases by the equation

$$\begin{aligned} VO_2 &= V_E F_i O_2 - V_E F_e O_2 \\ &= V_E \left(I - E\right) O_2 \end{aligned}$$

L-4: What is the effect of neck flexion on ETT tip position? Neck extension?

(a) Full neck flexion causes the ETT to move inward (distal) 3 cm.
(b) Full neck extension causes the ETT to move outward (proximal) 3 cm.

L-5: How do you ventilate a patient with PPV via a face mask in the prone position?

(a) Turn the patient's head to his/her right. Place your left hand in the usual position for mask ventilation and your right hand on the anesthesia reservoir bag (see Fig. 8.2).

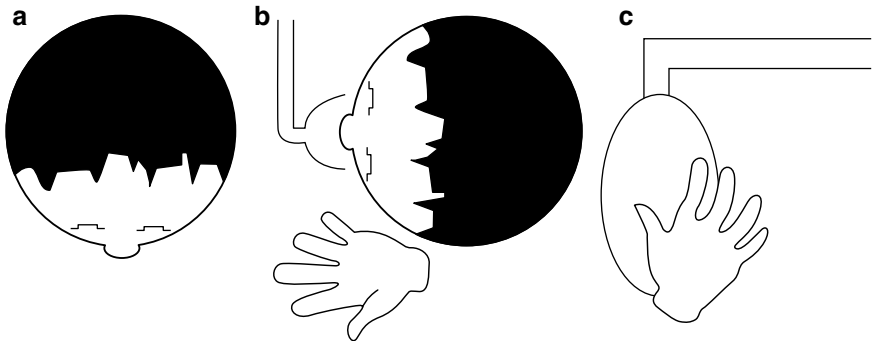

Fig. 8.2 Mask ventilation in the prone position. (**a**) Patient in prone position requiring mask ventilation. (**b**) Turn patient's head to his/her right and use your left hand to secure face mask on patient in the usual position. (**c**) Place right hand on reservoir bag and begin mask ventilation

L-6: Airway emergency + prone position = forceful call for help.
In the case of an airway emergency in the prone position, the call for the gurney to be brought into the OR must be very forceful to get the attention of everyone as well as overcome the disbelief, inertia, and inaction that sometimes accompany emergencies.

Chapter 9
Jet Ventilation Through a Cookgas Airway Exchanger

Erica K. Stary and Creed M. Stary

An otherwise healthy 13-year-old girl (65 kg, 5′5″) involved in a pedestrian vs. auto accident 3 days prior was brought to the operating room (OR) intubated and sedated for placement of a halo orthostasis device for an unstable C2 fracture with anterior listhesis. A request had been made by the pediatric intensive care unit (PICU) team to replace the 7.0-mm internal diameter (ID) cuffed endotracheal tube (ETT) that had been placed in the field by paramedics, because they had intermittent difficulty ventilating her and suspected the cuff was compromised. Other sustained injuries included a closed head injury resulting in bifrontal cerebral contusions and inter-hemispheric subdural hematoma, status post external ventricular drain placement, temporal bone fracture, right tibial plateau fracture, and non-displaced inferior pubic ramus fracture. Her postaccident neurologic exam prior to transport to the OR had been limited by sedation, but she was moving all extremities spontaneously and was following commands, per the PICU attending.

Due to the grave instability of the neck injury, the decision was made to exchange the endotracheal tube after placement of the halo orthostasis. Once brought into the OR, while remaining monitored on the PICU bed, she was placed on the ventilator, kept sedated with sevoflurane, and paralyzed with vecuronium. Vitals signs were stable, not requiring pressors. Vascular access included a left subclavian central venous catheter, several peripheral IVs, and a left femoral arterial line in situ. The placement of the halo orthostasis ensued, with confirmation of reduction of the injury by fluoroscopy.

Following placement of the halo, a second anesthesia attending was called to the room for assistance with the endotracheal tube exchange. Initial paralysis had worn off so she was redosed with vecuronium and disconnected from the ventilator, and a 14-French Cookgas airway exchange catheter was placed through the endotracheal tube (**L-1**, **L-2**). The jet ventilator was attached to the airway exchange catheter, with settings reduced to 15–18 pounds per square inch (PSI), and test ventilation was made

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with the jet. Bilateral excursion of the lungs was observed. At 1,715, the existing ETT was removed over the exchange catheter and a new 7.0-mm ID cuffed ETT was passed over the catheter; however, the tube was unable to be passed into the glottis. Rotational maneuvers were made as well as an attempt at direct laryngoscopy; however, laryngoscopy was impossible with the halo in place. Throughout this time, intermittent estimated 1–1.5-s ventilations through the jet stylet were made, with observation of chest rise and fall, and vitals remained stable. At 1,720, an anesthesia STAT code was called for more assistance, the halo chest plate was removed, and a GlideScope was brought into the room. However, during attempted laryngoscopy with the GlideScope, the patient began to desaturate to the 80s, and she was noted to have subcutaneous air in the chest, neck, and face (**L-3**). At 1,723 an overhead code blue was called, with specific instructions given to a nurse to bring a head and neck surgeon into the room immediately. No view of the glottis was obtained with the GlideScope (**L-4**).

Within seconds of noting subcutaneous air, the blood pressure began to decline rapidly to 60s/40s mmHg, and a wide-complex bradycardia was noted on the monitor. Chest compressions were initiated, defibrillation pads were placed, and 14-gauge needles were immediately placed in the chest bilaterally, with positive excursion of air. Two rounds of epinephrine were administered, with immediate increase in the blood pressure and heart rate. Although all attempts were made to limit jet ventilation at this point, her oxygen saturation (SaO_2) remained in the 80s, and intermittent ventilations with the jet continued.

An ENT surgeon and trauma surgeon arrived, and her neck was prepped for a cricothyrotomy, while bilateral chest tubes were placed. An arterial blood gas around this time indicated pH 7.02, base excess −9 mmol/l, and PaO_2 45 mmHg; 100 meq of bicarb was administered. At 1,730 an airway was secured with a 6.0 cuffed ETT through the cricothyroid membrane, and oxygenation improved shortly thereafter, although SaO_2 remained in the low 90s. Pink frothy sputum was suctioned from the endotracheal tube and saturation improved to mid 90s. Around this time a repeat blood gas showed a pH of 7.39, base excess 2 mmol/l, and PaO2 70 mmHg. A chest x-ray revealed diffuse subcutaneous emphysema.

The front halo plate was replaced, and the ENT surgeons proceeded with placement of a formal tracheostomy. The patient was then transported to the PICU on a propofol infusion with stable blood pressure and heart rate, although oxygenation remained difficult, with SaO_2s in the high 80s to low 90s. A persistent chest tube leak was apparent on the right side at that time. Over the course of the following days, her sedation was weaned and she was noted to be following commands, with plans to return to the OR for open reduction and fixation of her lower extremity fracture.

Lessons Learned

L-1: Exchange of an ETT over an AEC and jet ventilation through an AEC
Table 9.1 illustrates the ideal method of tube exchange and jet ventilation through an AEC.

Table 9.1 Ideal method of tube exchange and jet ventilation using an airway exchange catheter

Parameter[a]	Ideal method	Method used in this case
A. Concomitant laryngoscopy	Yes	Not initially
B. Known depth of insertion of AEC	Yes	No
C. Maintain upper airway patency	Yes	Jaw thrust, but no nasal/oral airways
D. Low pounds per square inch (PSI)	Yes	Yes
E. Short inspiration time (0.5 s)	Yes	No

[a]See text for the importance of each parameter

A. *The Importance of Concomitant Laryngoscopy*
Using direct laryngoscopy while exchanging an endotracheal tube (ETT) over an airway exchange catheter serves to lift supraglottic tissues out of the anesthesiologist's line of vision, and out of the path of the airway exchange catheter and endotracheal tube. This will decrease the risk of failing to be able to pass the ETT through the glottis. Often if resistance is felt when trying to pass an ETT over the AEC, it is because the tip of the ETT is hitting the right vocal cord or arytenoid. By turning the ETT in a counterclockwise manner by 90°, the tip will likely pass through the vocal cords more easily. Direct visualization and lifting of extra tissues will help the success of this process.

B. *The Importance of Knowing the Depth of Insertion of the Airway Exchange Catheter*
Increased depth of insertion of the AEC increases the risk of perforating the tracheobronchial tree and lung parenchyma. Additionally, as the airways become smaller, less room exists around the AEC for air to exit the lungs, leading to hyperinflation, tension pneumothorax, pneumomediastinum, and subcutaneous emphysema. When placing the AEC, matching the centimeter markings on the AEC with those of the ETT and maintaining this depth of insertion will help to assure continued proper depth and to avoid the aforementioned scenario. In an adult, the calibrations on the AEC should never exceed a depth of 26 cm at the patient's teeth.

C. *The Importance of Maintaining a Patent Upper Airway*
When ventilating through an AEC, it is imperative for the upper airway to be kept patent, by employing effective bilateral jaw thrust in combination with oropharyngeal and nasopharyngeal airways (see Fig. 9.1). Whereas the inspired air is forced through the relatively small internal diameter of the airway exchange catheter under high pressure, the expiratory gas must escape under only the relatively low pressure of the elastic recoil of the lungs, and through whatever channel is available. For this reason, as large a channel as possible should be maintained at all times. Without an opportunity for air to exit, over inflation, barotrauma, and eventually pneumothoraces will ensue.

D. *The Importance of Low PSI and Short Inspiration Duration*
To date, tidal volumes through Cookgas airway exchange catheters with varying delivered PSI, and at varying inspiration times, have not been measured. It can been seen from the two figures below that 18 PSI delivered through a medium Sheridan airway exchange catheter at 18 PSI and for 0.5 s led to tidal volumes of about 400 ml (see Fig. 9.2), and that 18 PSI through a 14-gauge IV catheter for

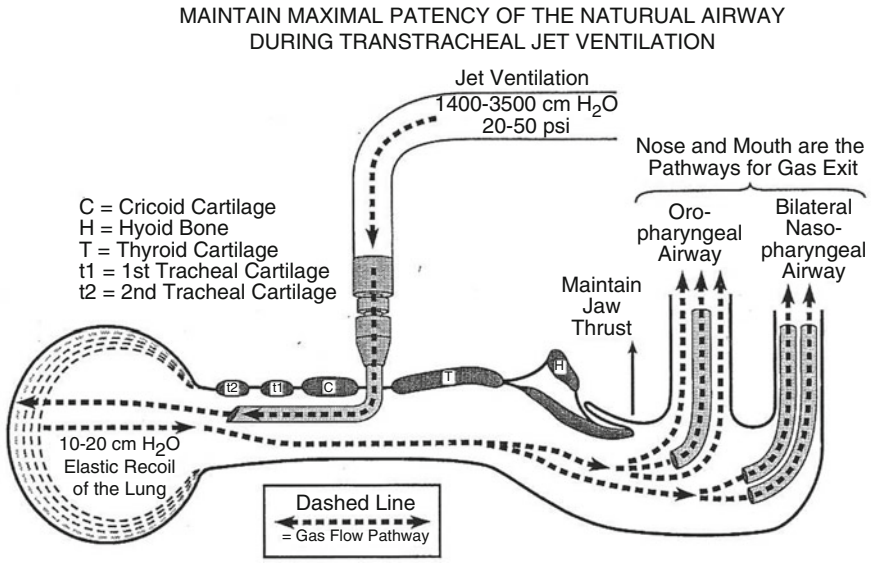

Fig. 9.1 Maximal airway patency must be maintained during transtracheal jet ventilation because the pathway for exhalation of jetted oxygen is the natural airway, and the driving force for exhalation is the relatively very low elastic recoil of the lungs. The same concepts apply to the importance of a patent airway when jet ventilating through an AEC (From Benumof [2]; with permission)

0.5 s led to 800-ml tidal volumes (see Fig. 9.3), at constant lung model compliance. However, a 14-gauge IV catheter is about half the diameter of a medium Cookgas AEC, presumably leading to a relatively smaller measured tidal volume breath than an AEC would deliver, but it is also only about 1/16th the length of a Cookgas AEC, leading to a relatively larger measured tidal volume than the AEC. Similarly, the medium 14-French Cookgas AEC is about 50 mm longer than the Sheridan used for Fig. 9.2 but has about a 0.3-mm larger diameter.

Clearly there are too many known and unknown variables to accurately calculate the tidal volumes administered in the present case. However, at 1–1 ½-s bursts, delivered tidal volumes were likely somewhere between two and three times the 400-ml breaths from the Sheridan catheter (i.e., 800–1,200 ml) and the 800 ml measured through the 14-gauge catheter (i.e., 1,600–2,400-ml breaths). If there was additionally any air trapping due to (1) small space between the outside diameter of AEC and the internal diameter of the tracheobronchial tree or (2) blocking of upper airway patency, then air trapping and barotrauma would have resulted (see Lessons 3C and 3D).

L-2. Ventilation via IV catheter through cricothyroid membrane

See Fig. 9.4 for an illustration of how to place an IV catheter through the cricothyroid membrane and then how to begin to safely perform ventilation through it.

There are four essential rules to be followed when ventilating via IV catheter through a cricothyroid membrane:

1. Freely aspirate air as final step before first jet ventilation.
2. Designate a human hand which has the sole purpose of holding the catheter hub in place at the skin line.

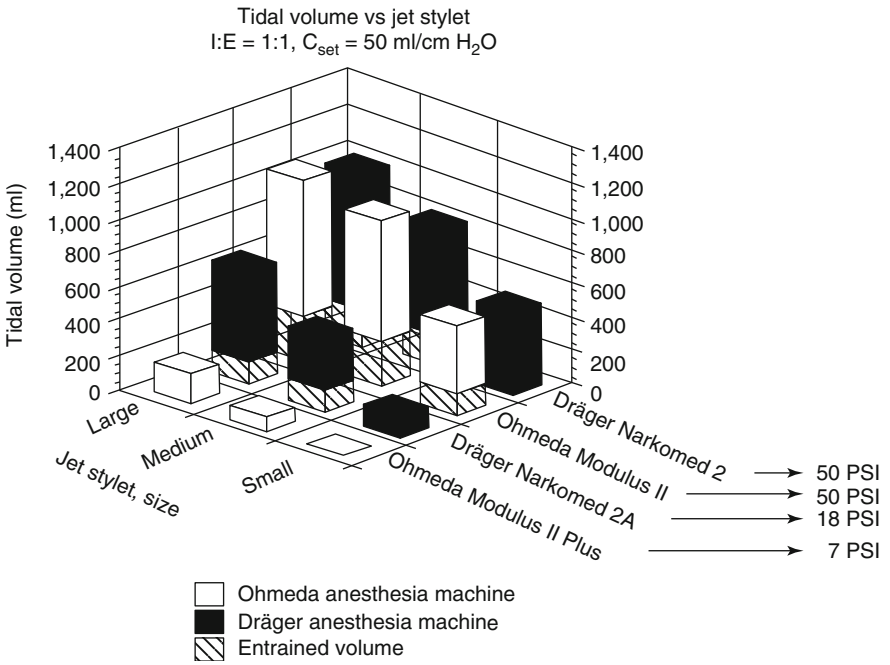

Fig. 9.2 The duration of inspiration for all tidal volumes (V_t) shown above was 0.5 s, through Sheridan Company catheters. The effect of changing jet-stylet size (*x*-axis) on tidal volume (*y*-axis) for all four anesthesia machines (*z*-axis) at compliance (C_{set}) = 50 ml/cm H_2O. As the jet-stylet size increases, tidal volume increases for all four anesthesia machines. Diagonally striped bars represent entrained volume as a portion of total V_t for each experimental condition. Contribution of entrained air to total V_t ranged from 0 to 30 % and accounted for the greatest contribution to total V_t for medium jet stylet/Ohmeda Modulus II anesthesia-machine combination *I*:*E* inspiratory to expiratory ratio. (Adapted from Gaughan et al. [3])

3. Use low PSI (20) and short inspiration times (0.5 s).
4. Keep the upper airway patent, using bilateral jaw thrust in addition to nasopharyngeal and oropharyngeal airways.

L-3: Why subcutaneous emphysema and bilateral pneumothoraces occurred in this case

There were four possibilities as to why subcutaneous emphysema and bilateral pneumothoraces occurred in this case.

A. PSI was at a sufficiently low level in this case, so this is unlikely the cause.
B. Too long of an inspiration time was likely employed, as full rise and fall of patient's chest was seen and breaths were given in approximately 1–1 ½-s bursts (see lesson 1D).
C. Too little space for the exhaled breaths to escape around the exchange catheter, causing breath stacking, may have contributed.

In terms of whether the space outside an AEC but inside an ETT is less than or more than the space inside a 4.0-mm internal diameter ETT, Fig. 9.5 shows all combinations of small, medium, and large Sheridan AEC inside 4.0–9.0 endotracheal tubes.

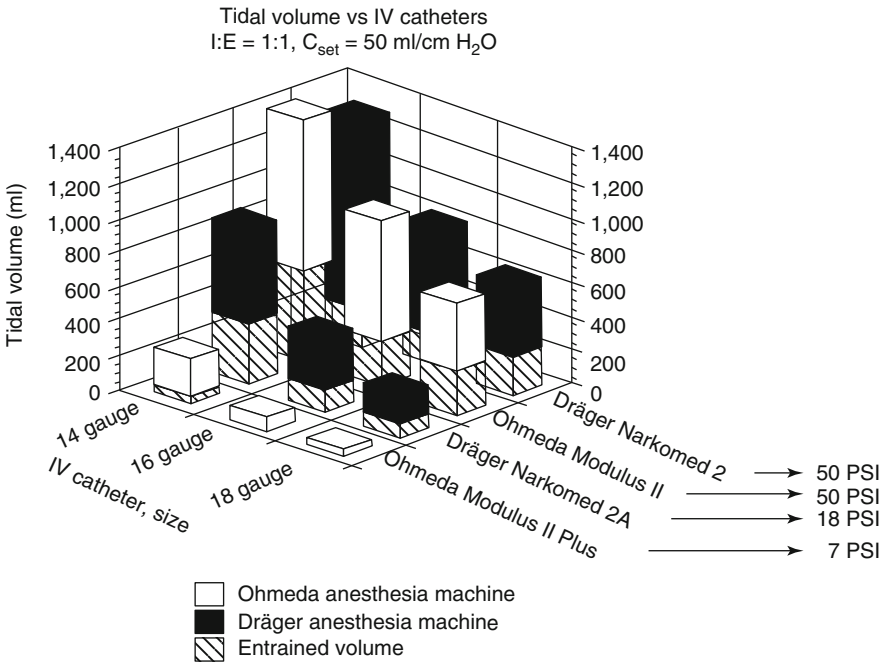

Fig. 9.3 The duration of inspiration for all tidal volumes shown above was 0.5 s. The effect of changing IV catheter size (*x*-axis) on tidal volume (*y*-axis) for all four anesthesia machines (*z*-axis) at C_{set} = 50 ml/cm H_2O. As IV catheter size increases, tidal volume increases for each anesthesia machine. The diagonally striped bars represent entrained volume as a portion of the total V_t for each experimental condition. Contribution of entrained air to total V_t ranged from 0 to 49 %, with greatest contribution total tidal volume using 14-guage IV catheter/Ohmeda Modulus II anesthesia-machine combination. (*IV* intravenous, *I:E* inspiratory-to-expiratory ratio, C_{set} set compliance of mechanical lung model, *PSI* pounds per square inch, V_t tidal volume) (Adapted from Gaughan et al. [3])

In the present case, the ETT was never passed through the glottis. However, the above figure is instructive in knowing in general which combinations of ETTs and AECs are compatible with ventilation. As mentioned above, there may have been a clinically similar issue in which there was inadequate space around the airway exchange catheter within the tracheobronchial tree. In a mechanical lung model, it was found that 4.0–4.5 mm was the effective tracheal diameter that below which a progressive and possibly deleterious increase in end-expiratory lung volume was inevitable, regardless of all variables (upper airway resistance, lung compliance, jet-ventilation time, gas flow rate). "Effective tracheal diameter" was defined as the difference between the internal diameter of the trachea and the outside diameter of the ventilating catheter (Fig. 9.6) [1].

In the present case, the 14-gauge Cookgas AEC has an external diameter slightly smaller than 0.5 cm, so unless it was positioned in a primary bronchi, the effective trachea diameter would have been less than 4.0 mm. This would

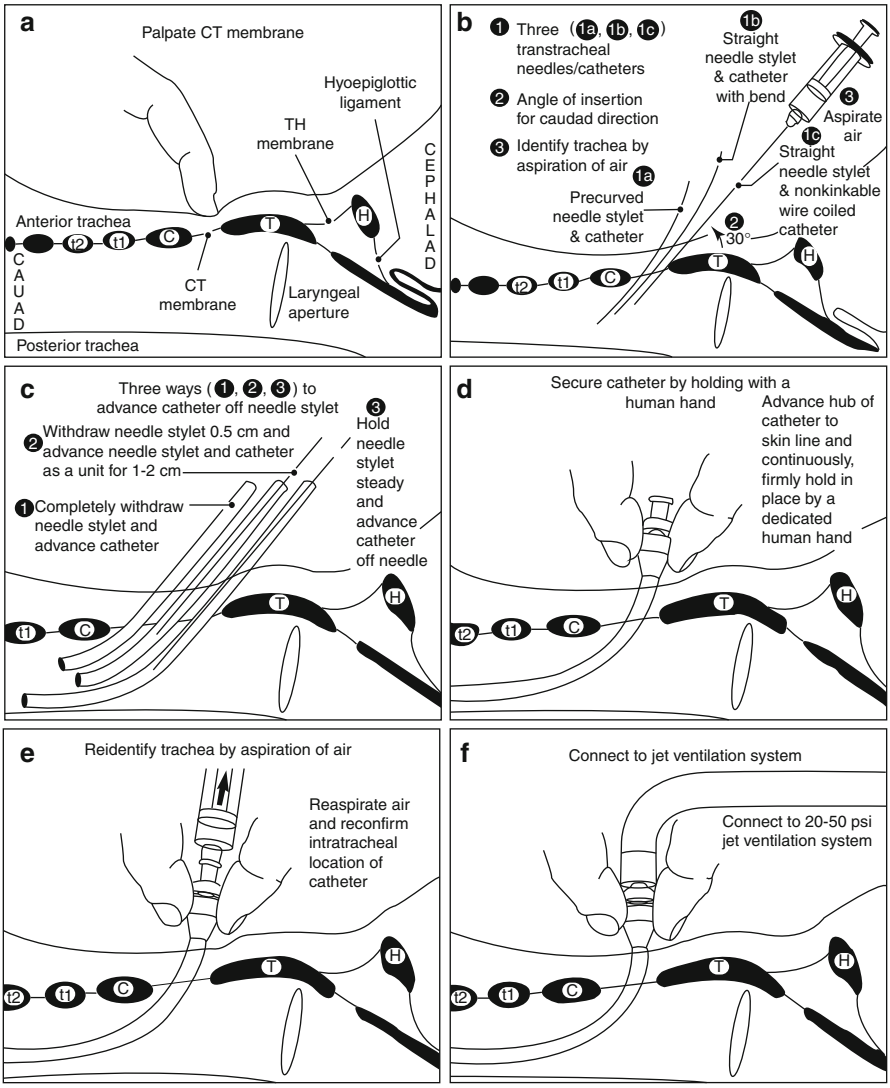

Fig. 9.4 The steps involved in transtracheal ventilation and some alternative options and considerations for some of the steps (**b**, **c**). (**a**) Cricothyroid (CT) is palpated first. (**b**) *1* Transtracheal needle is inserted through the CT membrane. Transtracheal needle/catheter can be *1a* precurved, *1b* straight with distal small-angle bend, or *1c* straight with a wire-coiled nonkinkable catheter. *2* Angle between distal end of the needle, catheter, and the skin (i.e., angle of insertion for caudad direction) should be 30°. *3* Entry into trachea should be confirmed by aspirating air. (**c**) The three ways to advance catheter off needles *1*, *2*, and *3* are explained by labeling. (**d**) Once catheter has been advanced so that the hub is at the skin line, it must be continuously held in place by a human hand (do not try to tape or suture the hub into place). (**e**) Intratracheal location should be reconfirmed by aspirating air. (**f**) Transtracheal jet-ventilation catheter is connected to jet ventilator (*T* thyroid cartilage, *H* hyoid bone, *C* cricoid cartilage, *t1* first tracheal cartilage, *t2* second tracheal cartilage) (Adapted from Benumof [2])

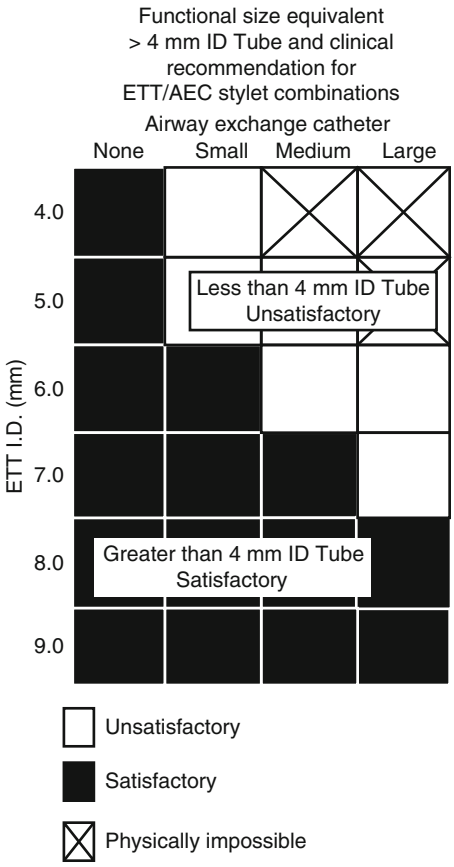

Fig. 9.5 Sheridan airway exchange devices were used to extrapolate the above data (Adapted from Benumof [4])

have led to decreased ability to exhale the jet-ventilated air, increased expiratory lung volumes, and likely dangerously overinflated distal airways.

D. The airway was not sufficiently kept patent. Although jaw thrust was employed, nasal and/or oral airways would have had added benefit.

L-4: Hindsight is 20/20

At this point in the above case, once the GlideScope was inserted and was lifting the supraglottic tissues, it would have been reasonable to attempt to pass the ETT through the glottis again. If successful, the subsequent cricothyrotomy and tracheostomy may have been avoided.

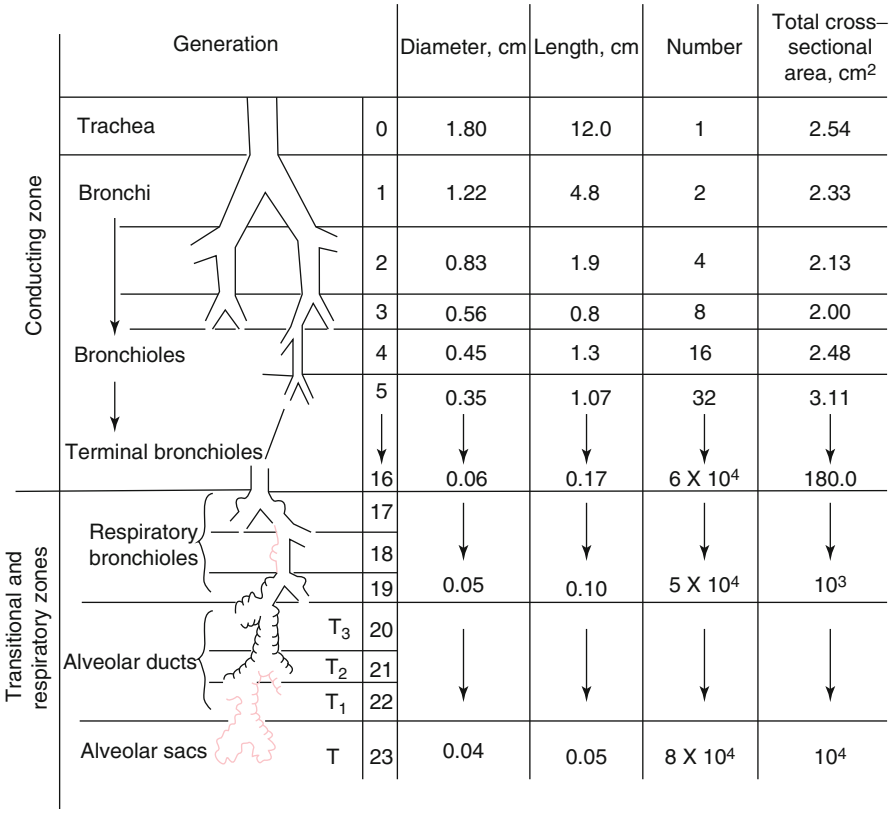

Fig. 9.6 Physical dimensions of the tracheobronchial tree (Adapted from Osborne [5])

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Chapter 10
End of Case Evaluation and Management of a Patient Post Airway Mass Excision

Bahareh Khatibi

The patient was a 48-year-old woman scheduled for excision of a periglottic mass (**L-1**, **L-2**). Preoperative nasal endoscopy (**L-2**) showed a moderately large supraglottic mass but otherwise normal anatomy.

After intravenous induction of general anesthesia, mask ventilation was successful and intubation proceeded without problem with an overall grade 1 view despite moderate obstruction to visualization of the glottis by the mass. A 5.0 ETT was passed without difficulty but was snug (**L-3**). The case proceeded smoothly, and at the end of the surgery, the surgeons were asked specifically if the airway had been improved by the surgery and if there was any significant swelling. They denied any significant swelling and stated that the airway was improved by excision of the mass. During multiple attempts to lighten the anesthesia, the patient had significant coughing, and spontaneous minute ventilation was decreased because of the narrow caliber of the ETT (**L-4**). Anesthesia was deepened and the patient began to breathe regularly. With assisted ventilation to overcome the resistance of the small ETT, minute ventilation was acceptable to maintain normocapnia and SPO_2 was 100 %. Muscle relaxation was documented as fully reversed and the patient was carefully suctioned. A leak test was performed (**L-5**) and there was a leak with expiration but not with inhalation. The pupils were checked and the gaze was conjugate. The decision was made to attempt a deep extubation. Upon extubation the patient had significant obstruction to spontaneous ventilation and placement of an oral airway, and assisted ventilation, while successful in maintaining SPO_2 above 90 %, was quite difficult, requiring two anesthesia providers. Immediate attempted reintubation was unsuccessful. The glottis was easily visualized and there was no significant swelling but a 5.0 ETT would not pass. At this point succinylcholine was given with mild improvement in the ability to ventilate the patient. The ENT surgeons were

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present and were requested to establish a surgical airway. They had significant difficulty with this, and after approximately 5 min, they stated that they could not find the trachea (**L-6**) and asked that a second intubation attempt be made. This time the 5.0 ETT passed with mild resistance and ventilation markedly improved. The surgical airway was then completed and the patient was transported to the SICU in good condition. During the entire episode the SPO_2 only briefly dropped below 89 % during the first reintubation attempt and the BP and HR were stable.

Lessons Learned

L-1: **Anatomy and pathology of the vocal cords**

(a) Relationships

1. The midpoint of the thyroid cartilage is anterior.
2. The C4–5 disc interspace is posterior.

(b) What are the anterior-posterior and lateral dimensions of the vocal cords?

1. The anterior-posterior distance is 18 mm.
2. The lateral distance between the vocal cords is 20 mm posteriorly on abduction.

(c) What composes the vocal cords?

1. The posterior one-third is made up of the vocal cord process of the arytenoids cartilage and the anterior two-thirds is made up of the vocal ligament.

L-2: **Airway evaluation**

(a) On what basis is one justified to make a preoperative diagnosis of a difficult airway?

1. If there is no apparent pathology, one should use the ASA preoperative airway examination to assess the airway (see Table 10.1).
2. If there is pathology, for example, a mass, hematoma, abscess, or laryngeal edema, in addition to the ASA preoperative airway examination, the patient's records and imaging must be reviewed to determine the exact location, size, and composition of the lesion.

(b) An ENT preoperative endoscopy was done which looked reassuring; how does this affect your management?

1. First, it is important to know when the endoscopy was performed.

   Depending on the doubling time of the tumor, the volume of the tumor mass may be significantly increased over a short period of time. For example, the doubling time of malignant lung tumors ranges from 20 to 400 days [1]. The doubling time of benign tumors do not usually fall within this range; they

Table 10.1 ASA preoperative airway examination

Exam	Nonreassuring findings
1. Length of upper incisors	Relatively long
2. Relation of maxillary and mandibular incisors during normal jaw closure	Prominent "overbite" (maxillary incisors anterior to mandibular incisors)
3. Relation of maxillary and mandibular incisors during voluntary protrusion of mandible	Patient cannot bring mandibular incisor anterior to maxillary incisors
4. Interincisor distance	<3 cm
5. Visibility of uvula	Not visible when tongue is protruded with patient in sitting position (e.g., Mallampati class>II)
6. Shape of palate	Highly arched or very narrow
7. Compliance of mandibular space	Stiff, indurated, occupied by mass, or noncompliant
8. Thyromental distance	<3 ordinary finger breadths, <6 cm
9. Length of neck	Short
10. Thickness of neck	Thick
11. Range of motion of head and neck	Patient cannot touch tip of chin to chest or cannot extend neck

Adapted from [1]

have either shorter or longer doubling times [1]. Thus, unless you know the specific tumor growth rate (from serial CT images quantifying tumor volume over a period of time), knowing whether the lesion is benign or malignant or what the airway looked like X days/months ago may not give you a reasonable estimate of the tumor's size on the day of surgery. Error on the safe side and perform an awake intubation if there is any doubt.

2. Even with an endoscopic exam performed on the day of surgery, the airway of a patient under general anesthesia with muscle relaxation in the supine position always looks different than that in an awake, spontaneously ventilating patient. Don't let a reassuring endoscopy fool you!

(c) Determine the patient's exercise tolerance as the final pathway determinant of airway size. As airway diameter decreases below 4.5–5.0 mm, very large increases in expiratory time occur (see Fig. 10.1). This effect will be apparent in the patient's exercise tolerance which can be assessed during the preoperative interview.

L-3: **What is the corresponding outer diameter of a 5.0 endotracheal tube**? **6.0**? **7.0**? **8.0**?

(a) See Table 10.2.

L-4: **Breathing through a 5.0-mm ID ETT**

Breathing through a 5.0-mm ID endotracheal tube at complete rest may be clinically acceptable, but with the addition of general anesthesia, secretions, lung disease, atelectasis, or bronchospasm, a 5.0-mm airway will not be adequate.

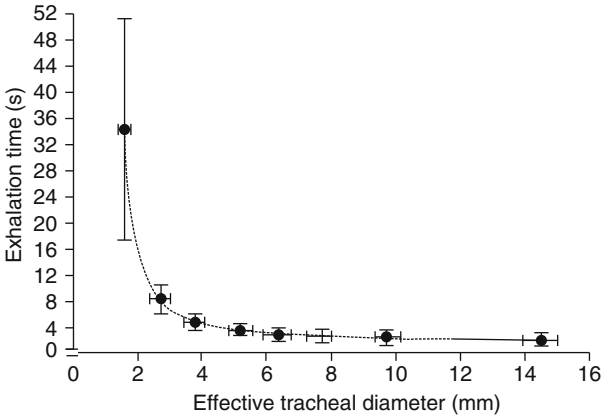

Fig. 10.1 The effect of upper airway resistance on exhalation time (Adapted from [2]; with permission)

Table 10.2 Internal diameter (ID) and outer diameter (OD) of cuffed and uncuffed Mallinckrodt endotracheal tubes

ID (mm)	OD (mm)
5.0	6.9
6.0	8.2
7.0	9.5
8.0	10.8

L-5: **Cuff leak test**
In this case, a cuff leak test was used to assess the internal diameter of the trachea. A recent systematic review of the leak test [3] concluded that a positive leak test ("no leak") should alert the clinician of a high risk of upper airway obstruction following extubation. On the other hand, the detection of a leak has a low predictive value and does not rule out the occurrence of obstruction or the need for reintubation.

L-6: **Shoulder roll for surgical airway**
A crucial (but often overlooked) step when performing a surgical airway is placing a shoulder roll to allow for adequate exposure (extension of the neck) during the procedure.

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Chapter 11
Possible Recurrent Laryngeal Nerve Injury

Sun Choe Daly

The patient is a 42-year-old male who presents for anterior cervical diskectomy and fusion for cervical vertebrae 5–7. The patient denies other significant past medical history. The patient is induced and intubated without complications. During a long and difficult surgery (**L-1**), the surgeon alerts the anesthesiologist that he may have damaged or even severed the recurrent laryngeal nerve (RLN). At the end of the procedure, he requests the anesthesiologist to determine whether there has been any damage to the recurrent laryngeal nerves (**L-2**, **L-3**).

Lessons Learned

L-1: What is the anatomic pathway of the recurrent laryngeal nerve?
On the left side, the recurrent laryngeal nerve hooks under the aorta and ascends in the tracheoesophageal groove. The left recurrent laryngeal nerve then splays out to the larynx under the thyroid cartilage to innervate the larynx.

On the right side, the recurrent laryngeal nerve hooks under the subclavian artery and ascends in the tracheoesophageal groove. The right recurrent laryngeal nerve then splays out to the larynx under the thyroid cartilage to innervate the larynx (see Fig. 11.1).

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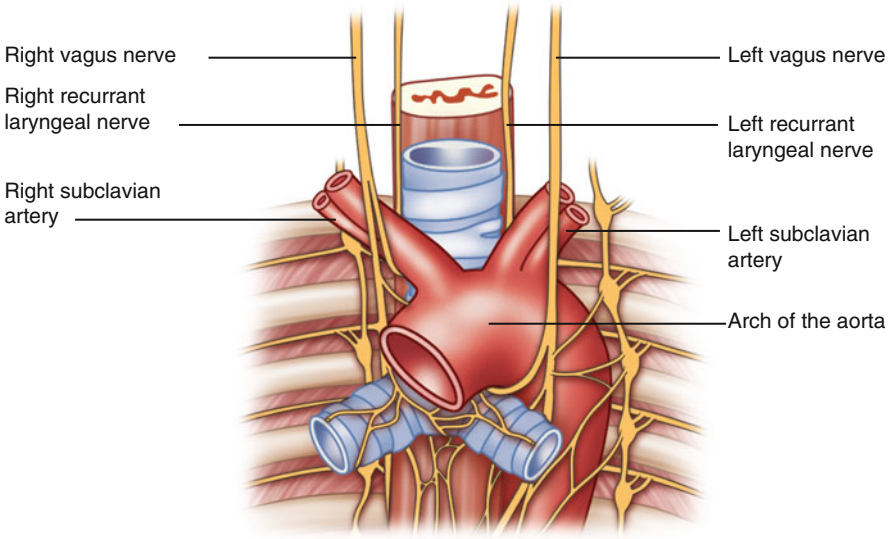

Fig. 11.1 Recurrent laryngeal nerve anatomy

L-2: How do you get an objective clear undisturbed view of the vocal cords during spontaneous ventilation at the end of a case to see if there is an RLN injury?

To get a clear objective view of vocal cord movement while under general anesthesia:

(a) While the patient is under adequate general anesthesia and is paralyzed, change the endotracheal tube to a laryngeal mask airway.
(b) Confirm good fiberoptic view of the vocal cords.
(c) While the patient is still under general anesthesia, reverse muscle relaxant completely.
(d) Allow the $P_{ET}CO_2$ to increase so that the patient begins to breathe spontaneously or will begin to breathe spontaneously as the level of anesthetic is decreased (see step e).
(e) Lighten general anesthesia gradually from stage three towards stage two.
(f) When spontaneous ventilation is well established, the vocal cords will obviously abduct with inspiration and will passively adduct with expiration.

L-3: What do you see when there are different types of RLN injury?

See Fig. 11.2 for various types of RLN injury.

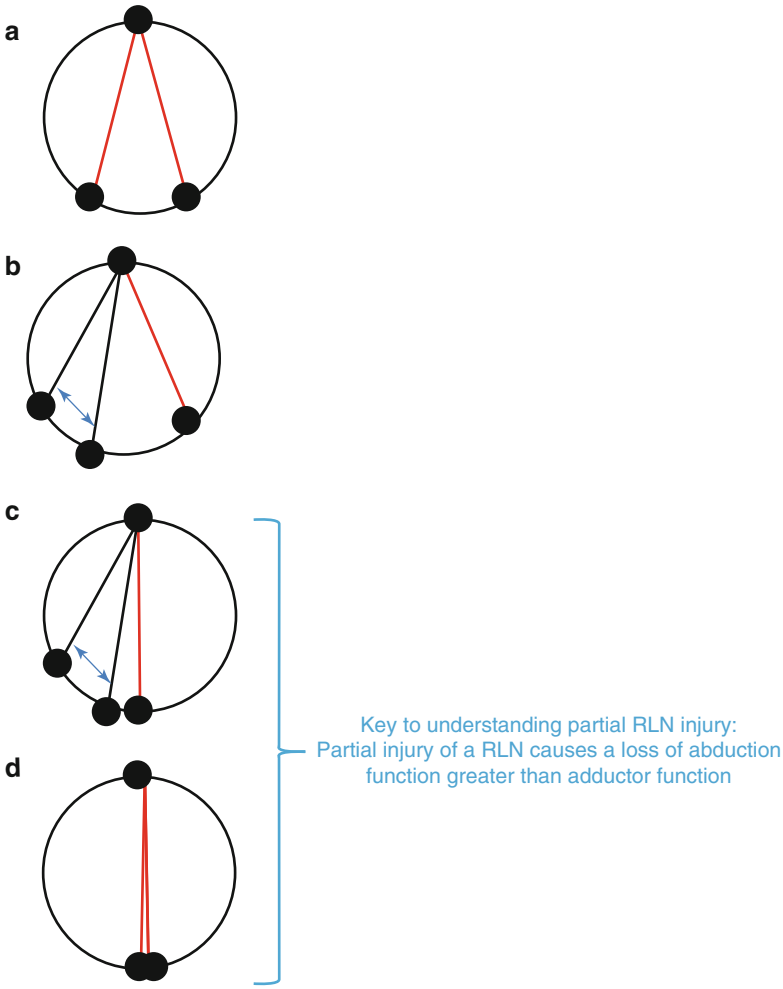

Fig. 11.2 Various types of recurrent laryngeal nerve injury. (**a**) Total transection of bilateral RLN = there is no vocal cord movement, both vocal cord are fixed in the cadaveric 45° position. (**b**) Total transection of unilateral RLN = there is no vocal cord movement on the injured side, and the injured vocal cord is fixed in the cadaveric 45° position. (**c**) Partial injury of unilateral RLN = the injured side is fixed in the adduction towards the midline and does not abduct. The uninjured side abducts and adducts normally. (**d**) Partial injury of bilateral RLN = there is no movement of both vocal cords and both sides are in adduction towards the midline. *Red line* indicates vocal cord with nerve injury. *Black line* indicates vocal cord without nerve injury. The *blue arrows* show movement of uninjured vocal cord

Chapter 12
Obstructive Sleep Apnea and Dead in Bed

Engy T. Said

A 41-year-old man was seen in the Trauma Bay with an open right tibia-fibula fracture, following a rollover accident. He was 5′8″ tall and weighed 230 lb (BMI 35), with no other injuries. Vital signs were stable, and he denied any past medical history to anesthesia providers. Preop labs were normal. He received an uneventful general anesthetic and was taken to the PACU. In speaking with the patient's family, the orthopedics service was informed that the patient had obstructive sleep apnea (OSA) and used a continuous positive airway pressure (CPAP) machine at home (**L-1**, **L-2**). Per nursing staff, the patient was sleeping and snoring shortly after his arrival to his room at about 0400. Per floor nursing records, the patient arrived drowsy but appropriate. He was noted to be "ashy" around 0430 when his nasal cannula had become dislodged and a pulse oximeter spot check revealed 79 % oxygen saturation. The patient complained of pain and was given Dilaudid a half hour later, at 0500. At about 0630, a code blue was called when the patient was found pulseless and apneic. He was resuscitated and transferred to the SICU but was declared brain dead with a negative brain blood flow study the day following his arrest (**L-3**).

Lessons Learned

L-1: **What is OSA**?

There are three different pharynxes (Fig. 12.1a). The nasopharynx is the airspace that is posterior to all of the soft palate (to the tip of the uvula). The oropharynx is the airspace that is posterior to the tip of the uvula, extending to the tip of the

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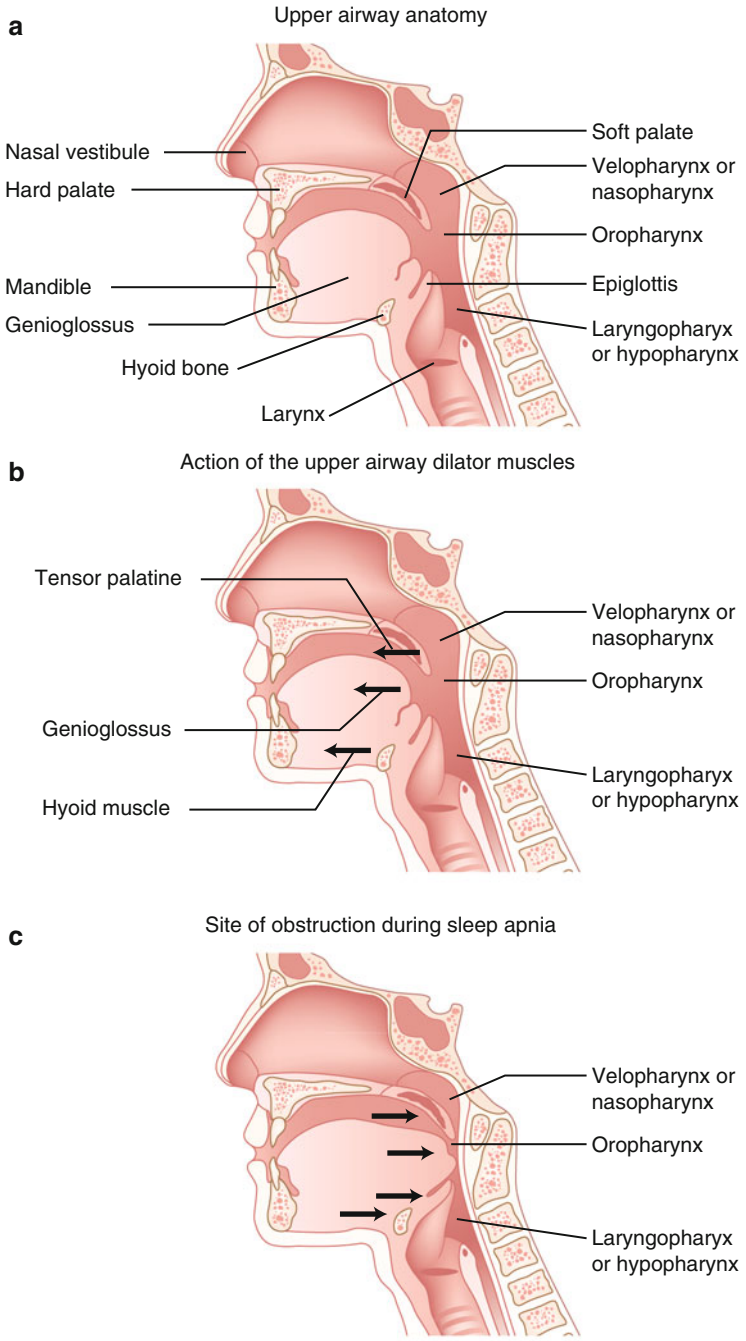

Fig. 12.1 (**a**) Upper airway; (**b**) pharyngeal dilator muscles; (**c**) pharyngeal collapse with pharyngeal muscle relaxation

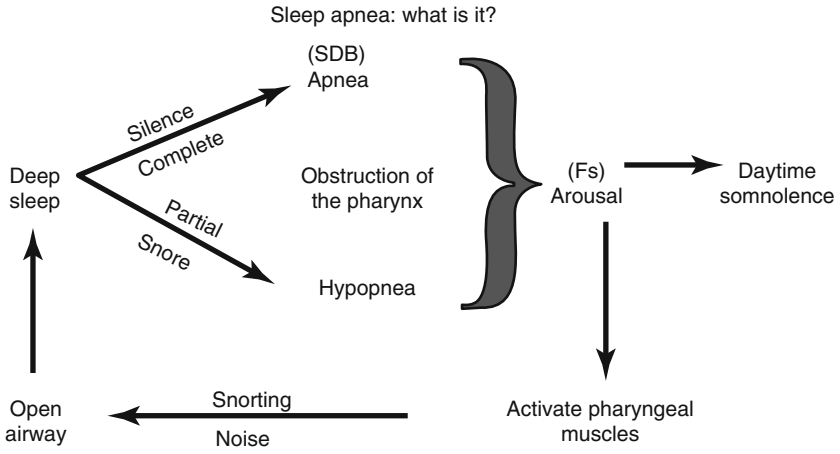

Fig. 12.2 Primary pathophysiology of obstructive sleep apnea (*OSA*). *SDB* sleep-disordered breathing, *FS* fragmented sleep

epiglottis. The laryngopharynx, also known as the hypopharynx, is the airspace that is posterior to the tip of the epiglottis, extending to the vocal cords [1].

Upper airway muscles keep the upper airway open (Fig. 12.1b). The tensor palatine retracts the soft palate away from the posterior pharyngeal wall, thereby maintaining the patency of the retropalatal nasopharynx. The genioglossus muscle moves the tongue anteriorly to open the retroglossal nasopharynx. The geniohyoid, sternohyoid, and thyrohyoid muscles move the epiglottis forward by tensing the hyoepiglottic ligament, thereby enlarging the retroepiglottic laryngopharynx [1, 2].

During deep and restorative sleep, the pharyngeal muscles participate in the loss of muscle tone that occurs throughout the body. If the loss of pharyngeal muscle tone and pharyngeal collapse is partial, but still great enough to cause the inspired air to flutter around the uvula and/or the tongue and/or the epiglottis (Fig. 12.1c), then there will be snoring and hypopnea. If the loss of pharyngeal muscle tone and pharyngeal collapse is great enough to cause complete obstruction, then there will be silence and apnea [3]. Apnea and hypopnea during sleep is called sleep-disordered breathing (Fig. 12.2).

Obese patients are uniquely susceptible to OSA because there is an inverse relationship between pharyngeal area and obesity. When an obese patient goes into deep sleep and there is a given degree of loss of pharyngeal muscle tone and pharyngeal collapse, the greater the amount of intraluminal fat, the greater the pharyngeal obstruction. Other conditions associated with OSA include thick/fat neck, micrognathia and retrognathia, a large tongue, large tonsils, and nasal obstruction [1]. For this reason, if any cause of OSA is known to be present, then the patient should be prompted with questions that would allow the anesthesia providers to rule in or rule out a presumptive clinical diagnosis of OSA.

Table 12.1 Clinical determinants of obstructive sleep apnea (OSA)

OSA feature		Severe	Mild
SDB	Snoring	Loudly, all the time, in all positions	Usually, softly, supine position
	Apnea	Yes, turns blue	No
Arousal S + S		Yes	Occasionally
Daytime somnolence[a]		During all quiet times	Occasionally

SDB sleep-disordered breathing, *S + S* signs and symptoms
[a]See Table 12.3 for further quantification

L-2: **How do you clinically determine the severity of OSA in the absence of a sleep study**?

From Fig. 12.2, three fundamental questions need to be asked in order to make a presumptive clinical diagnosis of OSA: (1) Is there a history or observation of apnea or snoring with hypopnea during sleep (sleep-disordered breathing)? (2) Is there a history or observation of arousal from sleep (extremity movement, turning, vocalization, snorting)? (3) Is there a history or observation of daytime somnolence (easily falls asleep during the quiet times of the day)? Sleep-disordered breathing, arousals, and daytime somnolence are the classic triad of signs and symptoms that make a presumptive clinical diagnosis of OSA [1].

Determining the severity of OSA on the basis of clinical impression is best understood if the issue is polarized between deciding mild versus severe (Table 12.1). In general, a patient with mild OSA is obese, snores in the supine position, has not had definite observed periods of apnea or arousals, and falls asleep during some of the quiet times of the day. Whereas a patient with severe OSA is generally morbidly obese, snores virtually all the time they sleep, has definite periods of apnea with frequent arousals, and falls asleep during most of the quiet times of the day. Moderate severity of OSA is in-between these two extremes [1].

A questionnaire to help anesthesia providers to recognize patients who are at risk for OSA has been developed. It was initially a short four-point questionnaire, named STOP, which was later modified to an eight-point questionnaire, STOP-BANG (Table 12.2). Studies have shown that the STOP-BANG screening tool has 100 % sensitivity in predicting an apnea/hypopnea index (AHI) of greater than 30 events per hour, reflecting severe OSA [4].

The AHI is defined as the number of times that the patient was either apneic (no airflow >10 s) or hypopneic (tidal volume <50 % for >10 s) per hour, and is measured in a formal sleep study. According to the American Academy of Sleep Medicine practice guideline, the severity of OSA is determined by the AHI: 5–15, mild; 15–30, moderate; and >30, severe [5]. Sensitivity refers to the ability of the test to correctly identify the patients with the disease. In other words, a test with 100 % sensitivity identifies 100 % of the patients with the disease. Specificity refers to the ability of the test to correctly identify the patients without the disease. A test with 100 % specificity correctly identifies 100 % of the patients without the disease [6]. Thus, the higher the sensitivity and specificity of a screening test, the better the test.

In patients who have had a formal diagnosis of the severity of their OSA, the STOP questionnaire was found to be more sensitive in detecting the patients with

Table 12.2 The STOP and STOP-BANG questionnaires

S: Do you **S**nore loudly?
T: Do you often feel **T**ired, fatigued, or sleepy during daytime?[a]
O: Has anyone **O**bserved you stop breathing during your sleep?
P: Have you ever been or are you being treated for high blood **P**ressure?
B: Is your **B**ody mass index greater than 35 kg/m^2?
A: Are you over 50 years of **A**ge?
N: Is your **N**eck circumference greater than 40 cm?
G: Are you male [**G**ender]?
High-risk obstructive sleep apnea:
STOP, yes to 2 or more questions
STOP-BANG, yes to 3 or more questions

[a]See Table 12.3 for further quantification

Table 12.3 The Epworth Sleepiness Scale

Use the following scale to choose the most appropriate number for each situation	
0—would *never* doze or sleep	
1—*slight* chance of dozing or sleeping	
2—*moderate* chance of dozing or sleeping	
3—*high* chance of dozing or sleeping	
Situation	**Score**
Sitting and reading	
Watching TV	
Sitting inactive in a public place	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon	
Sitting and talking to someone	
Sitting quietly after lunch (no alcohol)	
In a car, while stopped for a few minutes in traffic	
Total score	
Score results:	
1–6 Congratulations, you are getting enough sleep!	
7–8 Your score is average	
9 and up Very sleepy and should seek medical advice	

Data from [7]

moderate to severe OSA (74.3–79.5 % sensitivity) in comparison to the mild OSA group (65.6 % sensitivity). Adding in the BANG part of the questionnaire into the already present STOP part of the scoring model (Table 12.2) significantly improved the sensitivities to 83.6 % in the AHI mild group, 92.9 % in the moderate AHI group, and 100 % in the severe AHI group [4].

The amount of daytime somnolence (bottom line of Table 12.1, and "T" in STOP questionnaire) can be further quantified by using the Epworth Sleepiness Scale (ESS). ESS is a self-administered eight-question questionnaire that provides a measure of a patient's general level of daytime sleepiness [7] (Table 12.3). This is of paramount importance because daytime somnolence is the end result of the pathophysiology of sleep apnea (Fig. 12.2).

The aforementioned are methods used for the determination of the severity of OSA based on clinical impression or suspicion. However, the gold standard for the diagnosis of OSA and the classification of the severity is a full polysomnography sleep study. This study consists of monitoring the EEG (for stage of sleep and arousal), electrooculogram (for rapid eye movement [REM] vs non-rapid eye movement [NREM] sleep), chest and abdominal pressure and movement (for breathing effort), oral and nasal airflow sensors and capnography (for air movement), noise (for detecting snoring, vocalization, and snorting), submental and extremity electromyography (for pharyngeal muscle activity and extremity movement, respectively), oximetry for SpO_2, and electrocardiogram for heart function [1]. If a patient is found to have OSA, then the study is repeated with a CPAP titration to determine an adequate level of CPAP that leads to a significant decrease in the AHI. This study provides invaluable information to the anesthesiologist about the severity of the OSA by providing the AHI, and the effect of OSA on oxygenation, heart rate and rhythm, as well as the efficacy of the patient's CPAP. For this reason, if an anesthesia provider is to anesthetize a patient with OSA, every effort should be made to obtain the sleep study results. If a patient reports the use of CPAP at home, then they must have had a sleep study in the past.

L-3: **Should a patient with morbid obesity**, **with history of OSA on CPAP at home**, **not be on CPAP and without O_2 or appropriate monitoring**?

The ASA guidelines state that noninvasive pressure ventilation or CPAP should be administered as soon as possible postoperatively, especially to patients with OSA who were receiving it preoperatively [8]. This usually means that for most patients with OSA, the CPAP should be applied towards the end of the PACU stay. In some cases, waiting for the patient to be transferred to the floor to administer CPAP poses unnecessary risk [1]. The guidelines also point out that CPAP compliance is increased if the patients bring their own equipment to the hospital [8].

Furthermore, the guidelines states that "continuous SpO_2 [monitoring] in an ICU, step-down unit or by telemetry or by a dedicated professional observer in a private room, reduces the likelihood of perioperative complications among patients who they believe are at an increased perioperative risk from OSA" [8]. Patients with moderate to severe OSA, with painful respirations necessitating narcotics, are especially at increased risk and would definitely require continuous monitoring. Likewise, "continuous bedside SpO_2 without continuous [standby human] observation does not provide the same level of safety" [8].

As for supplemental oxygen, according to the guidelines, it should be administered when necessary in order to maintain "acceptable" SpO_2 [1, 8].

The ASA guidelines on perioperative management of the patient with OSA make other postoperative care recommendations. First, in the PACU setting, the guidelines state that "patients with OSA should be monitored for a median of 3 h longer than their non-OSA counterparts before discharge from the facility" and that monitoring patients with OSA who have had an observed respiratory event should continue for up to 7 h after the last episode of airway obstruction or hypoxemia while breathing room air in an unstimulating environment [1, 8, 9].

Second, regarding postoperative pain management, it is generally agreed upon that less opioid is better for respiratory function. Other considerations include (1) the utility of postoperative regional anesthesia in decreasing adverse outcomes in comparison to systemic opioids. (2) Exclusion of opioids even in neuraxial analgesia reduces risk for OSA patients. (3) Nonsteroidal anti-inflammatory drugs, when indicated, can result in less opioid use and thus decrease adverse outcome. (4) Avoidance of basal patient-controlled analgesia rates as a basal rate generally results in an increased incidence of hypoxemia [1, 8].

The University of California San Diego Medical Center has, since the case described in this chapter, adopted the STOP questionnaire alone as the initial preoperative screening tool for OSA, based on its simplicity of use and satisfactory sensitivity and specificity profile [9], as discussed in (**L-2**). It should be noted that adding BANG (BMI > 35 kg/m^2], age >50 years, neck circumference >40 cm, and male gender) to the STOP (i.e., using STOP-BANG) would greatly increase the number of false-positives because many patients have the BANG characteristics but not OSA. The patients, who are found high risk for OSA based on the STOP questionnaire, are then flagged on the operating room schedule for further perioperative management and appropriate monitoring, according to ASA guidelines and recommendations [9].

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Chapter 13
Bi-level Positive Airway Pressure, Decreased Sensorium, Aspiration, and Capnography

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Our anesthesia team was paged by the medical intensive care unit (MICU) resident for an emergent intubation of a 55-year-old male with acute respiratory distress secondary to sepsis. He is 74 in. tall and weighs 130 kg, with a BMI of 37. This is his hospital day #1, and his past medical history is significant for an aortic valve replacement, diabetes mellitus, colon cancer status post a partial colectomy, chronic osteomyelitis, and EtOH abuse. He presented with hypotension, respiratory distress, acute renal failure, and a non-ST elevation myocardial infarction (NSTEMI). Since admission, his respiratory status had continued to decline, and by the time our service was consulted, he had been on BiPAP for at least 8 h. On arrival to bedside, he was found obtunded with a Glasgow Coma Scale (GCS) of 8 on BiPAP with oxygen saturations in the low 70 % s (**L-1**). He was hypotensive, and the MICU team was starting Levophed. The patient was then ventilated with a Mapleson circuit via facemask, an oral airway was placed, and cricoid pressure was applied. Despite two-person hand ventilation, his oxygen saturation remained low around 80 %. He was then sedated with 20 mg of etomidate and 100 mg of rocuronium. A direct laryngoscopy with a MAC4 blade yielded a view of copious amounts of brown emesis obstructing the airway. Once suctioned, a grade II view was obtained and an 8.0 ETT was easily passed in the midline just anterior to the corniculate cartilages. No etCO_2 was detected with an Easy Cap, the patient was difficult to ventilate, and brown emesis was then coming out of the ETT (**L-2**). At the same time, it was noted that the patient was in ventricular fibrillation, a code blue was called, and chest compressions were started. ETT placement through the vocal cords was then confirmed with a GlideScope. CPR lasted a total of 10 min and during this time, the patient was given epinephrine × 3 mg, vasopressin, and two

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rounds of defibrillation. An amiodarone drip was started. During CPR the patient remained difficult to ventilate and brown emesis was continuously suctioned. Once the patient had a pulse, $etCO_2$ was detected and bilateral breath sounds were appreciated. However, the patient remained difficult to ventilate, with oxygen saturations remaining in the low 80s (**L-3**). The pulmonary team was then consulted for an emergent bronchoscopy, which revealed copious quantities of dark gastric material in the pharynx and supraglottic region in addition to all segments of the right and left lungs (**L-4**). Furthermore, a "plastic blue object containing a metallic object" was found in the pharynx.

L-1: **What is BiPAP**? **BiPAP in a patient with a GCS score of 8**. **With the buildup of air in the stomach and the finding of low oxygen saturation**, **aspiration has most likely occurred**. **Explain why and how does aspiration lower oxygen saturation**?

Noninvasive positive pressure ventilation (NIPPV) is often used to provide ventilatory support, without an endotracheal tube, to patients in respiratory distress. Pressurization of the airway serves three purposes: (1) to reduce the work of breathing, (2) to reduce atelectasis and improve oxygenation, and thereby (3) to reduce pulmonary vascular resistance and right ventricular afterload [1]. The increase in airway and alveolar pressure supports the patency of the airway, prevents alveolar collapse/atelectasis, maintains functional residual capacity, and thereby decreases the work of breathing [2].

There are two modes of application of NIPPV, one with continuous positive airway pressure (CPAP) and the second with bi-level positive airway pressure (BiPAP), as demonstrated in Fig. 13.1. During spontaneous ventilation, the inspiratory descent of the diaphragm causes negative airway and alveolar pressures relative to ambient pressure (defined as 0 cm H_2O), which pulls air into the lungs. Conversely, the elastic recoil of the chest wall and lungs causes positive airway and alveolar pressures which pushes air out the lungs (Fig. 13.1a). CPAP is constant positive airway pressure that is applied throughout both the inspiratory and expiratory phases of ventilation. CPAP essentially raises the baseline pressure from which the inspiratory descent of the diaphragm causes a relatively negative pressure (Fig. 13.1b). BiPAP provides two levels of pressure: inspiratory positive airway pressure (IPAP) and a lower expiratory positive airway pressure (EPAP) for easier exhalation. Unlike spontaneous ventilation and CPAP, BiPAP has a higher pressure on inspiration than on exhalation, but both pressures are above ambient. For example, a BiPAP setting of 10/5 cm H_2O implies an IPAP of 10 cm H_2O and an EPAP of 5 cm H_2O (Fig. 13.1c). BiPAP is essentially equivalent to pressure support ventilation (PSV) plus CPAP, where IPAP = PSV, and EPAP = CPAP.

Table 13.1 outlines the major advantages and disadvantages of NIPPV in comparison to standard tracheal intubation and mechanical ventilation [3]. One of the "disadvantages" listed is that NIPPV requires an awake and cooperative patient with adequate reflexes to protect his or her airway from aspiration. Likewise, gastric distention seems to be an inevitable outcome when using high

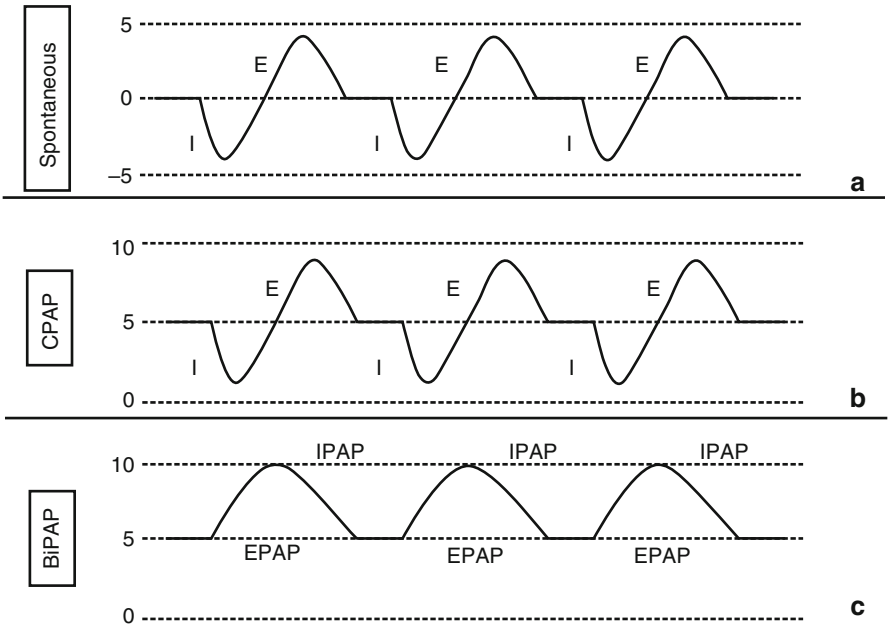

Fig. 13.1 Alveolar pressure curves with (**a**) spontaneous ventilation, on inspiration (*I*) there is negative pressure relative to ambient pressure (0 cm H_2O). On exhalation (*E*), there is a return to ambient pressure. (**b**) *CPAP* (continuous positive airway pressure) of 5 cm H_2O and (**c**) *BiPAP* (bi-level positive airway pressure) of 10/5 cm H_2O. Inspiratory positive airway pressure (*IPAP*) = 10 cm H_2O and expiratory positive airway pressure = 5 cm H_2O

Table 13.1 Advantages and disadvantages of noninvasive positive pressure ventilation (NIPPV) in comparison to invasive modes of ventilation

Advantages	Disadvantages
Reduces morbidity associated with endotracheal intubation and allows the patient to remain awake	Lack of patient compliance secondary to discomfort from tight-fighting mask. Also poorly tolerated by claustrophobic patient
Shorter ICU stay, by avoiding intubation especially in patients with acute COPD exacerbations, pneumonia, congestive heart failure, and postsurgical respiratory failure	No airway protection, therefore the patient must be awake enough and have an adequate cough and gag reflex to protect their airway
	Gastric distention from swallowing air
	Inability to deliver enteral nutrition during ventilation
	Pressure necrosis with prolonged use because of the tight-fitting mask

inspiratory pressure. Our patient's GCS score of 8 places him in the category of severe neurologic injury (Table 13.2), and he is by no means awake enough to protect his own airway, much less in the setting of positive pressure ventilation.

Table 13.2 Glasgow Coma Scale

Category/ system of response	Response score					
	1	2	3	4	5	6
Eye opening	None	To pain	To speech	Spontaneous	N/A	N/A
Speech/ verbal	None	Incomprehensible sounds	Inappropriate words	Disoriented	Oriented × 3[a]	N/A
Motor	None	Decerebrate posturing[b]	Decorticate posturing[c]	Withdraws to pain	Localizes pain	Follows commands

A score of 13–15 indicates minor neurologic injury, 9–12 moderate injury, 5–8 severe injury, and 3–4 indicates very severe neurologic injury [4]

[a]Oriented × 3 = to person, place, and date

[b]Decerebrate posturing = extension to painful stimulus

[c]Decorticate posturing = flexion to painful stimulus

Thus in this case,

Obtunded patient + BiPAP × 8 h → air in the stomach → Increase in intragastric pressure → movement of gastric contents to oropharynx and laryngopharynx → and then into the trachea because of the open larynx (BiPAP = NIPPV)

Aspiration can be defined as the inhalation of material into the airway, which can sometimes lead to significant tissue damage. This material can consist of nonacidic particulate matter, acidic gastric contents, or both. The lower esophageal sphincter (LES) functions as a valve in order to prevent the reflux of gastric contents. In cases where the LES pressure is reduced, or when the intragastric pressure is increased, aspiration becomes more likely to occur. Examples of increased intragastric pressure include intestinal obstruction, a non-fasted patient, delayed gastric emptying, or increased air in the stomach from positive pressure ventilation, as this patient had. Aspiration of non-neutralized acidic gastric contents causes direct chemical damage to the tracheobronchial tree and alveolar parenchyma, leading to airway and alveolar inflammation and edema as well as interstitial edema. Studies have demonstrated that the severity of injury increases with the volume of aspirate as well as a decrease in pH [4]. Particulate matter from the stomach can lead to obstructions in the tracheobronchial tree causing distal atelectasis. In both scenarios, a second proinflammatory stage, which occurs within 2–3 h and is mediated by cytokines which then recruit neutrophils, is crucial to the development of aspiration pneumonitis [5, 6]. Once in the lungs, neutrophils primarily cause lung injury via the release of oxygen radicals and proteases (Fig. 13.2). This inflammatory reaction, or aspiration pneumonitis, in turn reduces host defense against infection, increasing the risk of bacterial colonization and superinfection [5]. For this reason, bacterial infection does not play a significant role in the early stages of acute lung injury following aspiration of gastric contents [5]. Clinically, these patients can be either asymptomatic or present with bronchospasm, wheezes, cyanosis, and respiratory insuffiency [6].

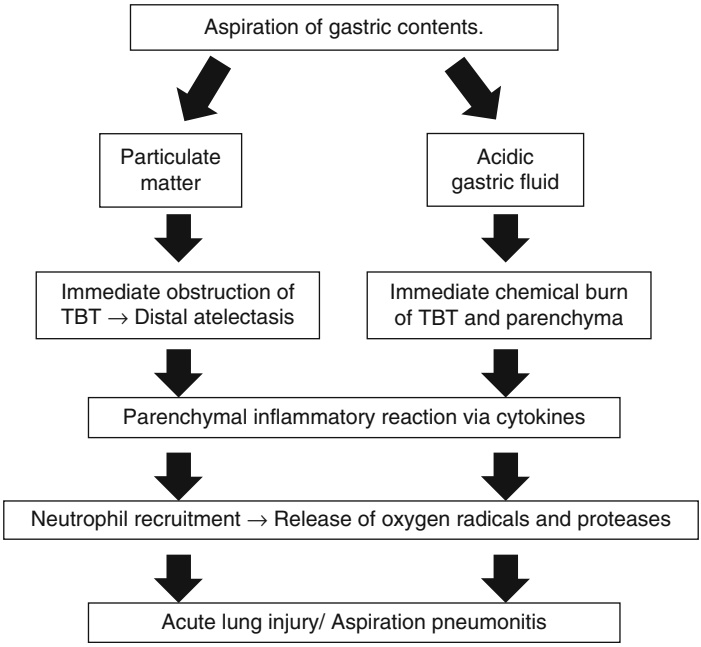

Fig. 13.2 Pathophysiology of aspiration of gastric contents. *TBT* tracheobronchial tree

Table 13.3 Easy Cap color changes based in response to varying $ETCO_2$ concentrations [9]. These values slightly vary based on manufacturer. Below are the Easy Cap II values, as it is used at our institution

Purple	Tan	Yellow
0.03–0.5 %	0.5–2.0 %	>2.0 %

The patient described in this case was unfortunately a perfect setup for aspiration leading to significant lung injury. His neurologic status in conjunction with increased intragastric pressure secondary to positive pressure ventilation for 8 h provides a good explanation for the dark gastric contents suctioned throughout laryngoscopy and tracheal intubation and found on bronchoscopy. The meaning and importance of the finding of a mysterious "plastic blue object containing a metallic object" in the pharynx is unknown.

L-2: **What are the sensitivity and specificity rates of an Easy Cap**? **What factors would lead to false-positive and false-negative findings**?

An Easy Cap is a colorimetric $etCO_2$ monitor with an indicator that changes in color, from purple to tan to yellow, in response to $etCO_2$ concentration (Table13.3).

Sensitivity refers to the ability of the test to correctly identify the patients with the disease or in this case a proper endotracheal intubation. In other words, a monitor with 100 % sensitivity identifies 100 % of the patients with endotracheal

Table 13.4 Factors that would lead to false-positive (inaccurate detection of CO_2 when ETT is not in the trachea) and false-negative (lack of CO_2 with successful airway intubation) findings by colorimetric CO_2 detection device [9]

False positive	False negative
When large amount of expired gas is forced into esophagus during bag-mask ventilation	Cardiac arrest
Recent ingestion of carbonated beverages or antacids	Improper ETT cuff inflation postintubation
Permanent yellow (positive) color change secondary to gastric fluid or drugs	ETT kinked
	ETT disconnect
	ETT position malfunction (distal end abutted against mass or deviated tracheal wall)
	ETT filled with fluid (blood, edema, pus, vomit)
	Short shelf half-life, 5 h once opened and 15 min in high humidity

intubation. Whereas specificity refers to the ability of the test to correctly identify the patients without the disease, or in this case a non-tracheal intubation [7]. Ideally, one would like that the Easy Cap monitor would have 100 % sensitivity, that the monitor would reliably detect endotracheal intubation in 100 % of the patients in whom proper endotracheal intubation was achieved.

In a multicenter study to determine the efficacy of colorimetric etCO_2 monitors, it was found that in 83 intubated patients who were not in cardiopulmonary arrest, the colorimetric etCO_2 device was 100 % sensitive and 100 % specific (with the ETT cuff inflated) in detecting proper endotracheal tube placement. Whereas in 144 intubated patients, who were in cardiopulmonary arrest, the colorimetric etCO_2 device was 69 % sensitive and 100 % specific in detecting proper endotracheal tube placement [8]. The reason for the drastic difference in sensitivity is that during cardiac arrest, pulmonary blood flow may be so low that there is insufficient expired CO_2 to be correctly identified by colorimeter device.

Table 13.4 outlines some of the causes that would lead to false-positive (inaccurate detection of CO_2 when ETT is not in the trachea) and false-negative (lack of CO_2 with successful airway intubation) findings by colorimetric CO_2 detection device [9]. There is also report of the Easy Cap having permanent color change to yellow with exposure to and contact with gastric contents. For this reason, it is imperative that one watches for a color change with each breath. Although the package insert of the Easy Cap alerts of this permanent color change with gastric contents or drugs, most providers are not aware of it [10].

As of 2010, the revised guidelines of Advanced Cardiac Life Support (ACLS) recommend the use of quantitative waveform capnography not only for confirmation of tracheal tube placement but also to monitor the effectiveness of chest compressions. Likewise, according to the American Heart Association (AHA), continuous waveform capnography along with clinical assessment is the most reliable method of confirming and monitoring correct placement of an ETT.

Table 13.5 Advantages of an esophageal detector device (EDD) [12]

Mechanism of action is mechanical, not chemical
Easy to use: reliably used by paramedical staff and non-anesthesia personnel
Easy assembly, inexpensive, portable, and reusable
Nonelectronic, so no electricity supply is required
Rapid test results
Highly reliable including during cardiac arrests

Continuous waveform capnography directs CPR for optimum chest compressions and allows for earlier detection of return of spontaneous circulation (ROSC) during chest compressions [11]. Based on these guidelines, we would project that in the near future, every code cart would have a mounted capnography unit ready for use in emergency situations.

L-3: **What is an esophageal detector device** (**EDD**)? **Would an EDD have worked in this case**?

An esophageal detector device (EDD), despite its name implication, is designed to detect both esophageal and tracheal intubations. It is designed to aspirate air (with negative pressure) from the ETT and relies on the fact that the trachea is rigid and would not collapse when negative pressure is applied, indicating a tracheal intubation. Conversely, the esophagus is collapsible with exposure to negative pressure, indicating esophageal intubation [12]. There are two types of EDD:

1. *Syringe type*: The device includes a 60 mL catheter tip syringe attached to a right-angled ETT connector. Once attached to the ETT, easy aspiration of 30 mL of air indicates a tracheal intubation. On the other hand, if resistance is encountered, as in esophageal intubation, when the plunger is released it usually rebounds to its original position [12].
2. *Bulb type*: A bulb is squeezed then attached to the ETT. Passive reinflation indicates a tracheal intubation, while failure to reinflate (secondary to a collapsed esophagus) would indicate an esophageal intubation [12].

There are several advantages of the EDD, as listed in Table 13.5. Aside from simplicity and portability, the greatest advantage of the EDD is that unlike the colorimetric CO_2 device, the mechanism of action is mechanical and does not depend on CO_2 being present in the exhaled gas. This can be especially useful in situations of cardiac arrest. In a study by Bozeman et al., among 37 patients in the cardiac arrest group, the EDD correctly indentified tracheal intubation in all 37 patients (100 % sensitivity), in comparison to $ETCO_2$ monitor (a spectrographic qualitative monitor), which correctly identified tracheal intubation in only 26 of the 37 patients (70 % sensitivity) [13].

As with colorimetric CO_2 detectors, there are certain situations that can lead to a false-positive result, where the ETT is in the esophagus but the EDD suggests that the ETT is in the trachea, by the ability to aspirate air with the syringe or the bulb

Table 13.6 Causes of false results with the esophageal detector device (EDD) [12]

False-positive result[a]
1. Regurgitation of gas from the stomach
2. Esophageal distention with gas
3. EDD is not airtight
False-negative result[b]
1. Fluid occluding the ETT (blood, edema, pus, vomit)
2. Occlusion of the end of an ETT by the tracheal wall or mass
3. Bronchial intubation
4. Tracheal compression
5. Obese patient
6. Chronic obstructive pulmonary disease

[a]*False positive*: ETT in esophagus and able to aspirate air with the syringe, or the bulb refills, suggesting tracheal intubation
[b]*False negative*: ETT in trachea and unable to aspirate air with the syringe, or the bulb does not refill, suggesting esophageal intubation

refills (Table 13.6). This is the case if the EDD is not airtight or if there is esophageal distention with gas or regurgitation of gas from the stomach. Similarly there are cases of false-negative results, where the ETT is in the trachea but there is resistance to air aspiration with the syringe or the bulb does not refill, suggesting esophageal intubation (Table 13.6). These include distal ETT obstructions such as secretions or occlusion by the tracheal wall.

In this case, an Easy Cap failed to detect a properly placed ETT, once immediately after intubation due to dark emesis occluding the ETT and a second time during cardiac arrest. An EDD would have likely failed in the first case, but may have been successful once the emesis was cleared from the ETT, during the cardiac arrest period.

Every single major study evaluating the efficacy of continuous waveform capnography in comparison to other methods of CO_2 detection, such as colorimetric device or EDD, demonstrated the superiority of waveform capnography in arrest as well as non-arrest population. A study by Knapp et al. revealed continuous capnography to have a 0 % error rate on non-arrest ICU patients, clearly performing better than auscultation, EDD, or a lighted stylet [9, 14]. Likewise, a study by Singh et al. revealed that continuous capnography is superior to the semiquantitative colorimetric device in an outside-of-hospital setting in both arrest and non-arrest patients [9, 15]. Based on these findings, the AHA ACLS guidelines were changed to incorporate continuous capnography monitoring, as discussed in (**L-2**).

L-4: **Diagram of the lobar bronchial and segmental orifices**

Figure 13.3 demonstrates a schematic diagram of the major bifurcations of the tracheobronchial tree. The trachea has cartilaginous rings that run anteriorly (from 8 to 4 o'clock [with 12 o'clock being most anterior]) down to the carina. The right main bronchus is more vertical in comparison to the left, and the right upper lobe (RUL) can be identified within 1–2 cm of the primary bifurcation into the mainstem bronchi. The RUL then trifurcates into the 1st, 2nd, and 3rd segments. Past the RUL,

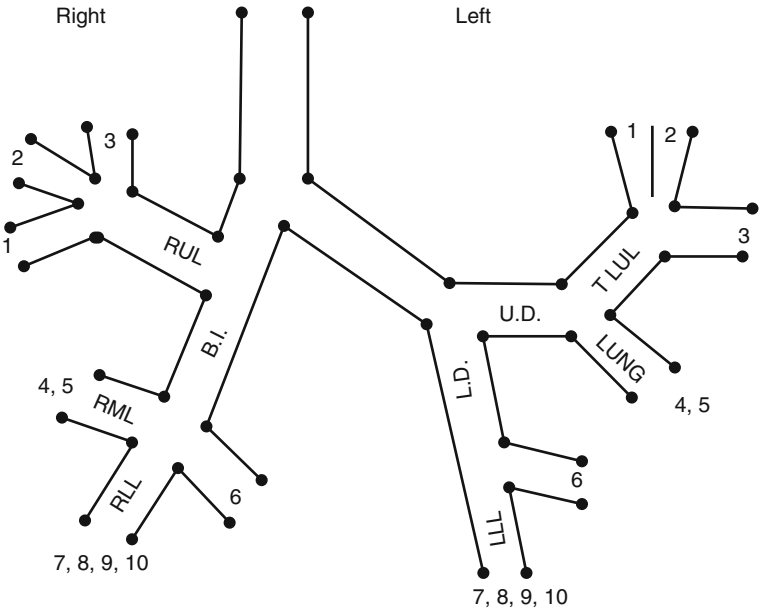

Fig. 13.3 Schematic diagram of the tracheobronchial tree (*TBT*) anatomy. *RUL* right upper lobe, *B.I.* bronchus intermedius, *RML* right middle lobe, *RLL* right lower lobe, *U.D.* upper division, *L.D.* lower division, *T LUL* true left upper lobe, *LING* lingula, *LLL* left lower lobe, *1–10* segmental orifices

from the right bronchus intermedius (BI), a second trifurcation can be visualized which consists of the right middle lobe (RML, segments 4 and 5), segment 6, and the right lower lobe (RLL, segments 7, 8, 9, and 10). The left main bronchus lies more horizontal than the right main bronchus; it is longer and divides into the upper and lower divisions (UD, LD). The UD divides into the true left upper lobe (T LUL) and the lingual (LING). The LD divides into the left lower lobe (LLL) and segment 6.

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Chapter 14
Perioperative Management of a Patient Previously Treated with Bleomycin Undergoing Thoracic Surgery

Pariza Rahman

A 23-year-old male was scheduled for thoracolumbar dissection for a large retroperitoneal mass. He originally presented with flank pain which led to discovery of the mass. The patient had just completed a course of chemotherapy which included bleomycin (**L-1 to L-8**) for the working diagnosis of germ cell tumor with metastasis to retroperitoneal lymph nodes. He was an otherwise healthy male, weighing 120 lb, 5′4″ height. The mass involved the vasculature of the left kidney, abutting up against both the inferior vena cava (IVC) and aorta. Preoperative thoracic epidural was not placed; an arterial line and right internal jugular central venous pressure (CVP) line were placed (**L-9**, **L-10**, **L-11**). His anesthetic care included premedications, routine monitoring, preoxygenation with 100 % oxygen for approximately 3 min followed by induction with fentanyl, propofol, and vecuronium. As soon as easy mask ventilation was achieved, the fraction of inspired oxygen (F_IO_2) was reduced to 50 %. Endotracheal intubation with a single lumen tube (**L-12**, **L-13**) was uneventful, postintubation F_IO_2 was lowered to 28 % (**L-14**, **L-15**). His postinduction CVP was 9 cm H_2O, which was maintained between 6 and 10 cm H_2O intraoperatively with crystalloids and colloid infusion. Postoperatively, he remained intubated for 11 h on F_IO_2 of 21 % (**L-14**, **L-15**). He was extubated the following day (POD #1) to room air. His postoperative course was complicated by pancreatitis; however, no pulmonary complication was noted related to bleomycin.

Lessons Learned

L-1: **What is the mechanism of action of bleomycin**?
Bleomycin is an antitumor antibiotic that was isolated from a strain of *Streptomyces verticillus* in 1966. It has been used successfully to treat a variety of malignancies,

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including squamous cell carcinoma of the head and neck, cervix, and esophagus; germ cell tumors; and both Hodgkin and non-Hodgkin lymphoma.

The antineoplastic effect of bleomycin is unique among anticancer agents and is thought to involve the production of single- and double-strand breaks in DNA (scission) by a complex of bleomycin, ferrous ions, and molecular oxygen. Bleomycin binds to DNA causing a DNA strand break. Bleomycin is inactivated in vivo by the enzyme bleomycin hydrolase, a cytosolic aminopeptidase that has lower activity in the skin and lungs [1, 2].

L-2: **Describe the pathogenesis of bleomycin-induced pneumonitis**

Pathogenesis

The mechanism of bleomycin-induced lung injury is not entirely clear but likely includes components of oxidative damage, relative deficiency of the deactivating enzyme bleomycin hydrolase, genetic susceptibility, and elaboration of inflammatory cytokines [3].

Bleomycin hydrolase, an enzyme that degrades bleomycin, is active in all tissues with the exception of the skin and the lung, which may account for the specific toxicity of the drug to these organs.

The acute pulmonary toxicity of bleomycin has been attributed to DNA strand scission with resulting chromosomal injury.

The chronic fibrotic response to bleomycin-induced injury is associated with an acquired loss of bleomycin hydrolase activity and is mediated by an immunologic mechanism, characterized by the migration of alveolar macrophages in the lung and the release of proinflammatory mediators that eventually result in the development of pulmonary fibrosis [1]. Bleomycin receptors have been identified on the surface of rat alveolar macrophages (AMs), suggesting that activation might be mediated through a second messenger.

L-3: **What is the incidence of bleomycin-induced pulmonary fibrosis**?

The incidence of bleomycin-induced pulmonary fibrosis is largely dependent on cumulative drug dose. In patients exposed to a total of 270 units or less (one unit = 1 mg), high-grade lung toxicity is seen in 0–2 %, while rates among patients receiving doses of 360 units or more range from 6 to 18 %. In trials of standard-dose chemotherapy, for germ cell tumors (three or four cycles, which contain a cumulative bleomycin dose of 270 or 360 units), fatal pulmonary toxicity rates have been in the range of 0–1 % and 0–3 %, respectively [1, 2].

L-4: **What are the risk factors for bleomycin-induced pulmonary fibrosis**?

Age, cumulative drug dose, renal function, the severity of the underlying malignancy at presentation, and also concomitant use of oxygen, radiation therapy, other chemotherapeutic agents, and hematopoietic colony-stimulating factors may all influence the risk of developing bleomycin-induced pneumonitis [1, 2].

(a) Age

The risk of bleomycin-induced lung toxicity appears higher in older patients:

- Age over 40 was associated with a 2.3-fold higher risk of pulmonary complications.

(b) Dose
Cumulative doses of more than 400 units are associated with higher rates of pulmonary toxicity. Although high-grade lung injury is very rare with cumulative doses under 400 units, injury can occur at doses less than 50 units. Rapid intravenous infusion may also increase the risk of toxicity [1].

(c) Other chemotherapy drugs—In the treatment of testicular germ cell tumors, bleomycin is administered in conjunction with other chemotherapy agents. Concomitant use of cisplatin or gemcitabine with bleomycin may increase the risk of pulmonary toxicity.

(d) High fraction of inspired oxygen—The evidence that oxygen exposure may increase the risk of pulmonary toxicity in humans is largely anecdotal.

Acute respiratory failure from adult respiratory distress syndrome has been reported following general anesthesia in patients previously treated with bleomycin [4]. Following the death of five bleomycin-treated patients from postoperative pulmonary complications at Memorial Sloan-Kettering Cancer Center in the 1970s, a new intraoperative protocol was developed in which oxygen exposure was minimized and intravenous fluid replacement was judiciously administered. With the adoption of this protocol, none of the subsequent 12 patients who underwent postbleomycin surgery for metastatic germ cell tumors developed pneumonitis or died from postoperative pulmonary complications [5].

Not all of the data regarding the effect of oxygen on bleomycin lung injury are consistent, however. A review of 77 patients undergoing major surgery following bleomycin-containing chemotherapy failed to demonstrate a correlation between perioperative oxygen restriction and either postoperative pulmonary morbidity or survival [6]. These authors suggested that careful fluid management during surgery was more important than oxygen restriction.

Nonetheless, the anecdotal data in humans, combined with the animal data, have had a dramatic impact on clinical practice and have led to widespread recommendations for lifelong avoidance of high concentrations of supplemental oxygen in patients previously exposed to bleomycin unless necessary to maintain an adequate arterial oxygen saturation.

(e) Radiation Therapy
Thoracic irradiation increases the risk of bleomycin lung toxicity, whether it is administered prior to or simultaneously with bleomycin. It is unclear whether a long interval between irradiation and administration of bleomycin eliminates the increased risk of lung injury. However, preliminary evidence from a study of 15 patients with advanced-stage Hodgkin lymphoma suggests that the risk of pulmonary toxicity during consolidative irradiation is low when there is an interval of at least 4 weeks between chemotherapy and irradiation [3].

(f) Renal Insufficiency
Renal insufficiency is a risk factor for bleomycin toxicity. This is not surprising since more than 80 % of the drug is rapidly eliminated by the kidney in normal individuals [3].

(g) Colony-Stimulating Factors
Concomitant treatment with granulocyte colony-stimulating factor (G-CSF) (filgrastim) was identified as a possible risk factor for the development of bleomycin-induced lung injury in animal studies. However, data in humans are conflicting as to whether G-CSF increases the risk of bleomycin-induced pulmonary toxicity [7].

Regardless, many clinicians avoid using G-CSF in regimens containing bleomycin, particularly ABVD (doxorubicin, bleomycin, vinblastine, and dacarbazine).

L-5: **What is the clinical presentation of bleomycin-induced pulmonary toxicity**?
Symptoms and physical signs associated with bleomycin-induced lung injury are nonspecific. However, the time course of the onset of clinical manifestations may suggest a particular type of lung injury, as described below:

- *Bleomycin-induced interstitial lung disease*: usually develop subacutely between 1 and 6 months after bleomycin treatment but may occur during therapy or more than 6 months following the administration of bleomycin. Symptoms and signs include nonproductive cough, dyspnea, pleuritic or substernal chest pain, fever, tachypnea, auscultatory crackles, lung restriction, and hypoxemia.
- *Bleomycin-induced hypersensitivity pneumonitis*: may present with more rapidly progressive symptoms.

An acute chest pain syndrome occurs in approximately 1 % of patients during infusion of bleomycin, but does not predict the development of pulmonary fibrosis.

L-6: **Preoperative/clinical evaluation of bleomycin-induced pulmonary toxicity**

Evaluation of patients with suspected bleomycin-induced lung injury typically includes a complete blood count with differential, radiographic imaging, and pulmonary function testing. Many patients will also undergo bronchoscopy with bronchoalveolar lavage to rule out infection or malignancy. Lung biopsy is typically reserved for patients in whom the diagnosis remains unclear after initial testing.

L-7: **What are the high-resolution CT chest findings in patients with bleomycin-induced pulmonary toxicity**?
HRCT is generally not used as a screening tool for bleomycin-induced lung injury, although HRCT is more sensitive than chest radiograph in identifying lung abnormalities in bleomycin-exposed patients. HRCT patterns associated with bleomycin toxicity usually reflect the underlying histopathology:

- *Diffuse alveolar damage* is usually associated with airspace consolidation or ground glass opacities in dependent locations.
- *End-stage fibrosis* presents with extensive reticular markings at the lung periphery, traction bronchiectasis, and honeycombing.
- *Nonspecific interstitial pneumonia* presents with ground glass opacities, increased reticular markings in a subpleural location, and bronchiolectasis.

- *Organizing pneumonia* presents with ground glass opacities in a bilateral but asymmetric pattern or by airspace consolidation in a subpleural or peribronchial distribution. In the setting of bleomycin toxicity, organizing pneumonia may rarely present as one or more nodular densities that may mimic tumor metastases. Often, the abnormalities are in a subpleural location.
- *Hypersensitivity pneumonitis* presents with diffuse, bilateral ground glass opacities and/or centrilobular nodules.

L-8: **What is the role of PFT in bleomycin-induced pulmonary toxicity**?
The National Comprehensive Cancer Network (NCCN) guideline recommends a baseline pulmonary function test (PFT) before starting bleomycin.

Subsequently, PFTs are usually obtained only to evaluate dyspnea, cough, crackles on chest examination, or an abnormal chest radiograph. The majority of patients treated with bleomycin will have a decrease in their diffusing capacity for carbon monoxide (DLCO), and those with significant pulmonary toxicity will have a decrease in lung volumes (e.g., forced vital capacity (FVC) and total lung capacity (TLC)). However, only a small percentage of patients exposed to bleomycin develop clinical signs or symptoms of lung toxicity.

DLCO initially appeared to be a useful test to screen for early pulmonary toxicity; however, subsequent data has questioned its use as a screening test. In early clinical trials, decreases in lung volumes and DLCO appeared to precede the development of severe bleomycin lung damage, and a decline in DLCO appeared to be the earliest and most sensitive indicator of subclinical lung injury. Subsequently, it has become apparent that PFTs, including DLCO, are neither sensitive nor specific for bleomycin lung toxicity, and many have questioned the clinical significance of these changes [1, 2].

A particular concern is that routine screening for pulmonary toxicity with DLCO results in many false-positive results, which may lead to the premature and unnecessary discontinuation of bleomycin. The clinical consequences include inferior cancer control and/or toxicity due to the substitution of an alternative, more toxic agent for the bleomycin.

L-9: **What is the role of TEA in postthoracotomy pain**?
Presently TEA is an important component of the perioperative care after major thoracic and abdominal surgery [8], providing very effective analgesia and facilitating early tracheal extubation. Indeed, lumbar epidural analgesia has gradually been replaced by thoracic epidural analgesia for thoracic surgery. The majority of thoracotomies done in US-teaching hospitals today receive a thoracic epidural between T3 and T8 with infusion of bupivacaine and fentanyl or hydromorphone.

Routine use of neuraxial analgesia for the postthoracotomy patient is a relatively recent concept. Numerous clinical studies have been published regarding the use of epidural techniques in patients undergoing thoracotomies [8–11]. Earlier studies [12–14] focused on the ability of these techniques to attenuate stress response (as assessed via a wide variety of mediators in the blood) and/or induce thoracic cardiac sympathectomy (as assessed via various hemodynamic changes); since 2000, studies have more appropriately focused on the ability of these techniques to affect

clinical outcomes (morbidity and mortality). A meta-analysis of respiratory complications after various types of surgery has shown that epidural techniques reduce the incidence of respiratory complications [15]. Beyond its analgesic properties, TEA also affects the postoperative neurohumoral stress response (decreased), cardiovascular pathophysiology, and intestinal dysfunction.

The evolution of TEA as a routine treatment has been the result of advancement in technology and knowledge [8]. The technology required was development of portable, safe, and reliable infusion pumps, which did not delay postoperative ambulation. The knowledge required was as follows:

1. First, the importance of synergy between local anesthetics and opioids for segmental thoracic analgesia was not appreciated originally. The combination of local anesthetics and opioids provides better epidural analgesia at lower doses than either drug alone.
2. Second, previous concerns for patient's risk due to postoperative neuraxial analgesia causing respiratory depression and hemodynamic instability were largely due to use of bolus injection techniques. Use of low-dose continuous epidural infusion has reduced the risk and has excellent safety record [8]. Studies have demonstrated that the risk of accidental dural puncture (1 %) was lower than lumbar epidurals and noted no permanent neurological damage.
3. Third, the use of the paramedian approach to the epidural space in the mid-thoracic level has improved the success rate for clinicians performing TEA.

L-10: **What are the cardiovascular effects of TEA**?

High TEA has the potential for blocking cardiac afferent and efferent fibers, which originate from the first to fifth thoracic level ($T_{1–5}$) [10]. TEA affecting $T_{1–5}$ segments produce sensory blockade, motor blockade (depending on concentration), and blockade of the cardiac sympathetic fibers. Blockade of $T_{1–5}$ results in the opposite effects of stimulations shown in Fig. 14.1, namely, decreased heart rate, decreased inotropy (contractility), and decreased myocardial VO_2.

Indeed, in patients with coronary artery disease, it has been reported that TEA leads to a reduction in heart rate, cardiac output, and systemic vascular resistance, and it may therefore decrease myocardial oxygen demand. Although oxygen supply demand ratio (due to decreased myocardial oxygen consumption and contractility) to the ischemic myocardium is improved, total coronary blood flow is unaltered. The effect of TEA on left ventricular (LV) contractility has been variable.

L-11: **What are the effects of TEA on GI surgery**?

Intestinal Perfusion

TEA improves intestinal perfusion, provided normal hemodynamics is maintained. In patients undergoing esophagectomy, TEA with continuous infusion (without bolus) of bupivacaine increases mucosal blood flow compared with control group.

Intestinal Motility

TEA improves postoperative ileus. The faster return of intestinal motility is suggested to be due to adequate pain control, reduced opioid use, and sympathetic block.

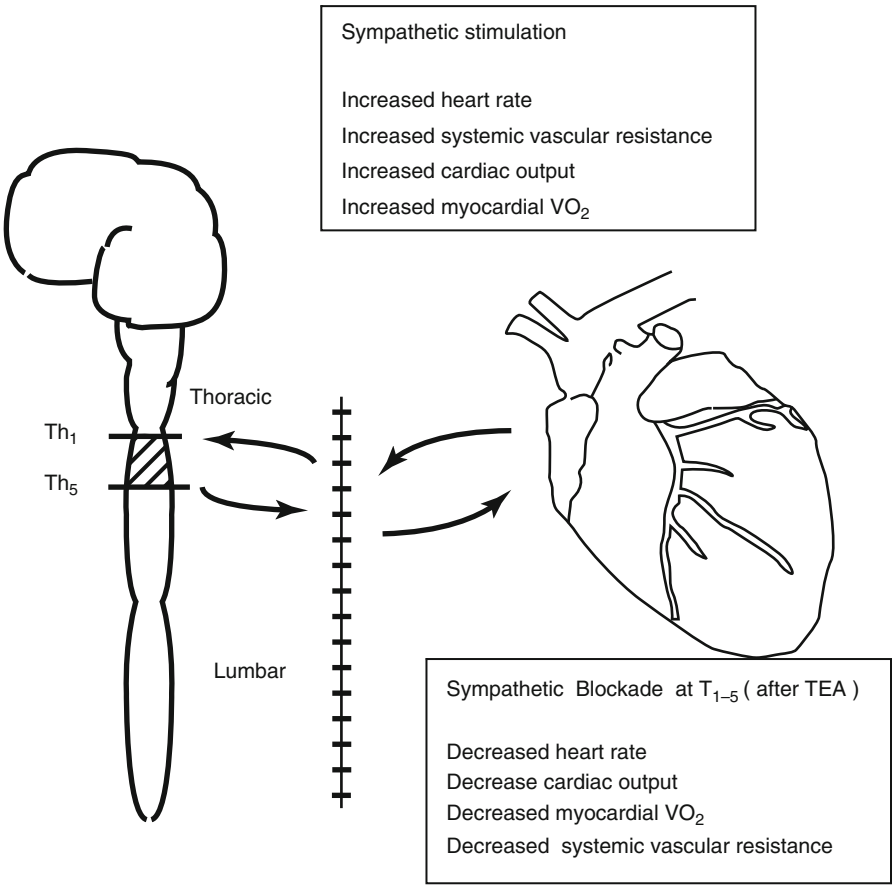

Fig. 14.1 Schematic illustration of the cardiac sympathetic innervation. Sympathetic stimulation in the thoracic region (T_{1-5}) leads to an increase in heart rate, blood pressure, systemic vascular resistance, and cardiac output. Blockade of T_{1-5} results in opposite effects

Anastomotic Perfusion and Patency

The effect of TEA on anastomotic perfusion and healing of anastomosis has been encouraging. There are recent convincing data that TEA reduced risk of anastomotic leak by 70 % [11].

L-12: **Could this case be managed with a DLT and one-lung ventilation**?

Patients undergoing one-lung ventilation have a high risk of developing hypoxemia. Inspired oxygen concentrations (F_IO_2) 1.0 are, therefore, commonly employed during one-lung ventilation to minimize this risk. If hypoxemia does develop during one-lung ventilation, then 5–10 cm H_2O CPAP with 100 % oxygen applied to the nonventilated lung is an effective treatment.

If a bleomycin-treated patient, or any other patient with pulmonary disease, was ventilated with less than 30 % oxygen during one-lung ventilation, then the risk of hypoxemia is high. In a bleomycin-treated patient undergoing OLV, the use of

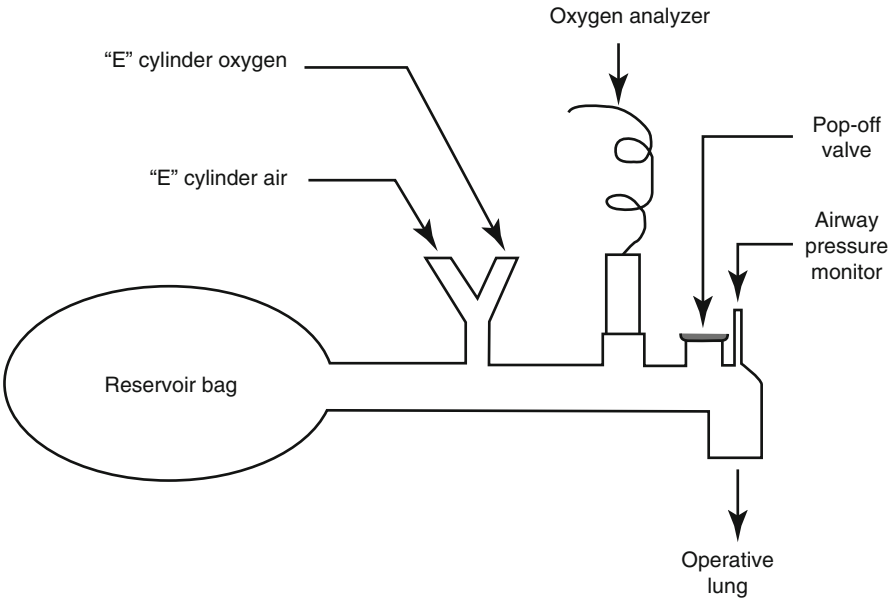

Fig. 14.2 Schematic of the variable FIO operative lung, CPAP system. Oxygen and room-air tanks are connected by a Y-piece leading to an FIO analyzer; the FIO_2 analyzer is connected in series to a CPAP device consisting of a reservoir bag with a pressure-relief valve and a pressure manometer

nonventilated lung CPAP with an FiO_2, of 0.3 to allow a ventilated lung FiO_2,=0.30 has been suggested [16] to maintain adequate PaO_2.

However, during OLV in lateral decubitus position, CPAP may not be required (because of relatively diminished blood flow to nonventilated lung). This author has reported bleomycin-treated patient undergoing OLV during en bloc esophagectomy who did not require CPAP to nonventilated lung and had no postoperative pulmonary complications.

L-13: **How is a CPAP device used to maintain variable F_IO_2**?
Special equipment (described herein) is necessary to administer CPAP with a variable F_IO_2 to the nonventilated lung.

The CPAP apparatus consists of a 100 % oxygen tank and a room air tank, both with flow meters, joined by a Y-piece, and in series with a rapidly responding F_IO_2 analyzer, a pressure manometer, and a reservoir bag with a Heidebrink pressure-relief valve (Fig. 14.2). This system allowed the administration of a F_IO_2, between 0.21 (no flow from the oxygen tank) and 1.0 (no flow from the room air tank) and any level of CPAP [16].

L-14: **What is the role of supplemental O_2 during perioperative management**? **Is hyperoxia (F_IO_2 >30 %) in a patient previously treated with bleomycin safe**?
Although the evidence is largely anecdotal and inconsistent [4–6], exposure to high inspired oxygen concentrations, even many years following exposure to bleomycin,

may increase the risk for pulmonary toxicity. For patients with prior bleomycin exposure who have hypoxemia, supplemental oxygen is administered sparingly to achieve PaO_2 >70 mmHg and SaO_2 >90 %. The priority is to maintain adequate oxygen saturation, even if that requires a high F_IO_2.

L-15: **Is hyperoxia exposure (FIO_2 ≥30 %) in a patient previously treated with bleomycin safe**?

The relationship between increased but normally nontoxic concentration of oxygen in bleomycin-treated patients and pulmonary fibrosis is controversial.

Goldiner et al. [4] reported in 1979 five consecutive patients undergoing retroperitoneal node dissection or wedge resections of the lung 6–12 months after receiving bleomycin. Average total doses of bleomycin were 426 mg with an average FIO_2 of 39 % and a duration of 5.9 h. These five patients subsequently developed adult respiratory distress syndrome (ARDS) at 3–5 days after surgery and died of respiratory failure. The subsequent 12 patients Goldiner and Schweizer [5] studied with similar medical profiles were maintained on FIO_2 25 %. These patients had no reported respiratory complications. The Goldiner et al. [4] articles are considered the landmark articles supporting the use of low FIO_2.

However, other authors [15] have documented no respiratory complication following high FIO_2 ≥0.3, with and without prior abnormal diffusion capacity. It was suggested that perioperative oxygen restriction in patient treated with bleomycin is not necessary [15] and that intravenous fluid management appears to be the most significant factor affecting postoperative pulmonary morbidities and overall clinical outcome. Avoiding hyperoxia during surgery while respecting safety rules for preoxygenation with 100 % oxygen during induction in patients who underwent prior treatment with bleomycin seems to be a safe approach without negative effects on pulmonary functions.

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Chapter 15
Intraoperative Airway Fire

Joseph Soo

History

A 37-year-old otherwise healthy female presented to the University of California San Diego (UCSD) Neurosurgery Clinic with a chief complaint of neck and right arm pain and right triceps weakness. Her symptoms began while working out approximately 1 month prior when she felt an acute onset of weakness and pain in the right upper extremity. MRI showed a large disk extrusion at C6–7 with compression of the C7 root. The physical exam revealed 3/5 weakness of the right triceps. She was scheduled for C6–7 anterior cervical discectomy with fusion and plating to repair her C6–7 disk protrusion.

Past Medical and Surgical History

No prior medical or surgical history. She is currently taking Percocet and Flexeril to alleviate her pain.

Physical Exam

Her physical exam is unremarkable. She is overweight with a BMI of 29 (height 5′6″, weight 177 lb). The airway exam was also unremarkable, with normal neck range of motion despite her C6–7 disk abnormality.

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Laboratory Data

A complete blood count was obtained, and the results were all within normal limits.

Description of Events

The case was started in the usual fashion: preoxygenation, IV induction with fentanyl 200 μg, lidocaine 100 mg, propofol 150 mg, and rocuronium 50 mg. Easy mask ventilation, grade II view of the vocal cords with a MAC-3 laryngoscope followed by atraumatic placement of a 7.0 mm inner diameter endotracheal tube (ETT). The ventilator was switched on with the following settings: tidal volume of 700 mL, rate of 8 breaths/min, I:E ratio of 1:2, and FiO_2 ~50 % (2 % sevoflurane and 50 % nitrous oxide). The surgical field was prepped and draped, which resulted in the head being completely covered and not readily accessible to the anesthesiology team.

The surgery proceeded uneventfully until the ventilator alarm sounded as a result of inadequate tidal volume. Upon an immediate survey it was noted that the ventilator bellow was completely down and the $etCO_2$ tracing was zero on the capnograph (**L-1**). The first response was to check the breathing circuit for disconnection at the ventilator and endotracheal tube; however, none was found. Secondly, the ventilator was switched off and manual ventilation attempted, also without success as the bag completely deflated despite the pop-off valve being closed and there was adequate fresh gas flow (10 L/min). Next, the ETT cuff pressure was evaluated by manually palpating the ETT balloon, which revealed that the ETT cuff was completely deflated. Of note, upon reinflating the cuff, the cuff would immediately lose pressure. At this time the surgeon was notified of our inability to ventilate the patient. He then mentioned that shortly before (~20–30 s) he noticed a larger than usual spark in the surgical field when electrocautery was used (**L-2**).

At this time the surgeon was notified that the most likely scenario of our inability to ventilate the patient was that the ETT cuff had been punctured by fire, which likely was also associated with a tracheal injury (**L-3**). He was notified that the ETT needed to be immediately replaced (**L-4, L-5**).

The circuit was manually disconnected from the anesthesia machine, and the surgeon was asked to stop using electrocautery until the ETT was replaced. Because the patient's head was inaccessible, we changed the presumably damaged 7.0-mm ID ETT for a new one of the same size over a bougie (Fig. 15.1) (**L-6**). During the change out, we maintained the patient's oxygen saturation above 95 %. The old ETT was noted to have blood on it, and the cuff had a large leak when tested underwater.

At that time, ENT was intraoperatively consulted STAT; however, because the patient was very stable, they suggested that neurosurgery finish the original procedure prior to examining the trachea. Surgery was completed without additional problems.

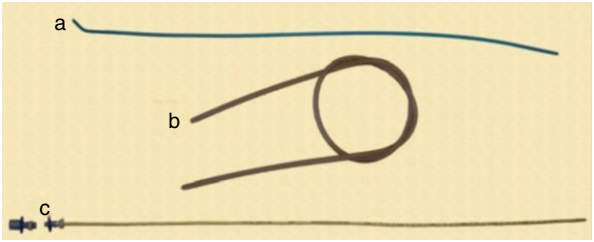

Fig. 15.1 Devices available to achieve ETT exchanges. (**a**) Bougie introducer (SunMed, Largo, FL), (**b**) Sheridan Tracheal Tube Exchanger (Teleflex Medical, Research Triangle Park, NC), and (**c**) Cook Airway Exchange Catheter (Cook Inc., Bloomington, IN). (**a**) is the most rigid of them, and it has a bend at the tip. It was designed to aid in difficult laryngoscopies where inserting the ETT proves to be difficult. (**b**) is the most flexible, allowing for exchanging the ETT with minimal injury to the trachea. Either end can be adapted for jet ventilation. (**c**) is similar to (**b**), but adaptors for both jet ventilation and conventional ventilation from the breathing circuit are provided in the package

Upon evaluation by ENT, they found a 3-cm anterior laceration of the anterior tracheal wall involving the second, third, and fourth tracheal rings, along with a 2.5-cm injury to the posterior wall of the trachea and the anterior aspect of the esophagus. ENT decided that the best approach would be to place a size 8-cuffed Shiley tracheostomy tube through the anterior defect and allow the trachea to heal over the next 5 days. Flexible bronchoscopy showed some blood in the left main stem bronchus, hyperemic mucosa of the trachea, and the main stem bronchi, but no areas of eschar or necrosis. The patient was placed on tube feedings to allow the esophagus to heal. After 10 days in the hospital, without any respiratory distress issues, the patient was discharged to home with a tracheostomy tube in place. After another week the tracheostomy tube was removed, and she continued to improve. After 7 months of continuous improvement, she is only left with an unsightly neck scar, which she is to have revised at some point in the future.

Lessons Learned

L-1: Differential diagnosis
Our differential diagnosis at this point included (a) respiratory circuit disconnection, (b) large leak around the ETT, and (c) scavenger suction set too high.

L-2: Causes for a large spark in the surgical field
Causes for a large spark in the surgical field include (a) high oxygen concentration in the surgical field; (b) nitrous oxide (N_2O) present in the surgical field (**L-3**); and (c) a route for the fresh gases from the anesthesia machine to escape into the surgical field, i.e., tracheal wall injury due to surgery, allowing fresh gas from the breathing circuit to escape into the surgical field.

L-3: Airway fires

Airway fires are potentially catastrophic emergencies where quick action on the part of the provider is crucial to the outcome. Although the occurrence of airway fires is likely underreported, it is estimated that about 100 to 200 such events occur yearly in the United States. About 20 % of these cases result in serious injury to the patient, causing one to two deaths per year [1]. For a fire to occur, three components are needed: fuel (ETT), oxidant (oxygen), and ignition (spark). Nitrous oxide (N_2O) at room temperature is inert and has few reactions, but at elevated temperatures, its reactivity increases. In fact, in the presence of a heated catalyst, N_2O will decompose exothermically into nitrogen and oxygen, according to the following equation:

$$N_2O \overset{spark\ \ catalyst}{\rightarrow} N_2 + \frac{1}{2}O_2 + energy$$

From this equation it becomes evident that if nitrous oxide is present in the field, a spark from the electrocautery could provide the catalyst for the reaction to continue to occur and provide oxygen, which propagates the fire.

L-4: What to do in an airway fire

Protocols have been developed to quickly manage operating room airway fires [2]. Such algorithms call for simultaneous action on the part of the surgeon and anesthesiologist. The protocol consists of (a) cease ventilation and turn off fresh gas flow; (b) extinguish flames with saline solution; (c) remove ETT; (d) ventilate the patient with a facemask and 100 % oxygen after all the flame has been extinguished, then resecure the airway; and (e) examine the airway for burns and foreign objects, such as ETT remnants.

L-5: Esophageal packing

It has been suggested that in the case of a difficult airway, it may be too risky to remove the existing ETT once it has been extinguished [3]. In such cases, the oropharynx can be packed with saline-soaked gauze. Although we could have attempted to pack the oropharynx with wet gauze, to attempt to minimize the leak, in our case this would not have offered even a temporary solution, since there was a large tear in the wall of the trachea and most of the fresh airway gas was escaping through it. However this is something to keep in mind, if re-intubating the patient is difficult.

L-6: Changing an ETT over a stylet

Sometimes this procedure can be difficult. The passage of the ETT can be obstructed, especially if the patient is not paralyzed. Maneuvers that can make this procedure more straightforward are (a) lifting the tongue with a laryngoscope, to allow easier passage of the ETT through the oropharynx; (b) if the ETT is "caught" on the vocal cords, gently turning the ETT counterclockwise while advancing would allow the bevel to spread the vocal cords apart; (c) lubricating the inside of the ETT to allow for easy sliding over the intubating stylet; and (d) using a commercial airway exchange catheter (AEC) (Cook Medical Inc., Bloomington, IN). The hollow AEC allows for jet ventilation in case the new ETT will not follow the AEC into the trachea.

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Chapter 16
Obesity Hypoventilation Syndrome

Daniel Fox

The on-call anesthesia team was paged by the medical intensive care unit (ICU) team for an urgent intubation. The patient was a 29-year-old morbidly obese male. His history was notable for multiple prior admissions for obesity-related complications as well as a prior intubation for obesity hypoventilation syndrome (OHS) (**L-1**). The indications for intubation were worsening subjective shortness of breath and hypercarbic respiratory failure. The most recent arterial blood gas (ABG) showed a pH of 7.29 and a $PaCO_2$ of 83 mmHg (**L-2**, **L-3**). On initial physical exam, the patient was clearly morbidly obese, sitting up in bed with bilevel positive airway pressure (BiPAP) ventilator support (**L-4**). Vitals were blood pressure (BP) 160/100 mmHg, pulse 100 beats per minute (bpm), respiratory rate 20 respirations per minute, and O_2 saturation 95 %. Airway exam revealed a class III Mallampati airway, full range of motion of the neck, greater than 5 cm hyoid-mental distance, and intact dentition. The patient was noted to be anxious but was alert and oriented and able to follow commands appropriately.

The anesthesia team on call opted for an awake fiber optic intubation (**L-5**) given their concern for a potentially difficult airway and his relatively stable condition and ability to cooperate. The procedure was fully explained to the patient, stressing the importance of his cooperation. 0.2 mg of glycopyrrolate was given intravenously. His airway was subsequently topicalized with a "lidocaine lollipop" and atomized lidocaine. After topicalization the fiberoptic bronchoscope was placed through the oral cavity, the vocal cords were easily visualized, and a 7.0 mm inner diameter endotracheal tube was successfully advanced over the bronchoscope.

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Lessons Learned

L-1: What is "obesity hypoventilation syndrome"? How is it diagnosed and how is it differentiated from obstructive sleep apnea?

Obesity hypoventilation syndrome (OHS) is commonly defined as the constellation of obesity (body mass index [BMI] ≥30 kg/m^2), daytime awake hypoventilation manifesting as arterial hypercapnia (partial pressure of arterial carbon dioxide ≥45 mmHg at sea level), and sleep-disordered breathing without an alternative explanation for hypoventilation such as a neuromuscular, mechanical, or metabolic disturbance [1]. Hypoxemia (partial pressure of arterial oxygen <70 mmHg at sea level) is also observed and may be added to the definition of OHS although this is not always included [2]. While not a defining characteristic, elevated serum bicarbonate is also seen in OHS due to metabolic compensation for chronic respiratory acidosis.

The primary point of differentiation between OHS and obstructive sleep apnea (OSA) is awake hypercapnia [1]. By definition, those with OSA will have normal awake $PaCO_2$ levels, while those with OHS will have an elevated awake $PaCO_2$ (≥45 mmHg). Obesity can also be a point of differentiation, as individuals with OSA are not always obese, but those with OHS are obese by definition [1, 3]. Of note the vast majority (nearly 90 %) of patients with OHS has coexisting OSA as well, but OHS can exist without the diagnosis of OSA [4].

L-2: Why would an obese patient reset their resting $PaCO_2$ level?

Awake hypercapnia is one of the defining features of OHS and is used to distinguish OHS from an obese patient with OSA, but the exact pathogenesis of chronic daytime hypoventilation and its resultant hypercapnia remains unclear. It is known that the hypercapnia is entirely due to hypoventilation because a short course of noninvasive positive pressure therapy improves hypercapnia without a concomitant change in BMI, CO_2 production, or dead space volume [5]. There are currently three leading hypotheses: (a) excessive load on the respiratory system due to obesity, (b) leptin resistance causing central hypoventilation, and (c) impaired compensatory response to acute hypercapnia in patients with sleep-disordered breathing [1, 2] (See Fig. 16.1):

(a) The hypothesis that excessive load on the respiratory system leads to awake hypercapnia postulates that OHS patients are unable to ventilate adequately given their poor respiratory mechanics. This leads to increased work of breathing and hypoventilation due to fatigue and weakness of the respiratory musculature. The excessive load on the respiratory system has multiple possible causes. If, and to what extent, each possible cause is responsible for awake hypercapnia remains unclear [2]. Upper airway resistance has been noted to be elevated in patients with OHS compared to equally obese patients with eucapnic OSA [6]. Lung compliance and chest wall compliance are both diminished compared to equally obese control groups while lung resistance is increased [7]. Poor lung compliance and increased lung resistance can be explained by the lower functional residual capacity of patients with OHS. Spirometric studies are

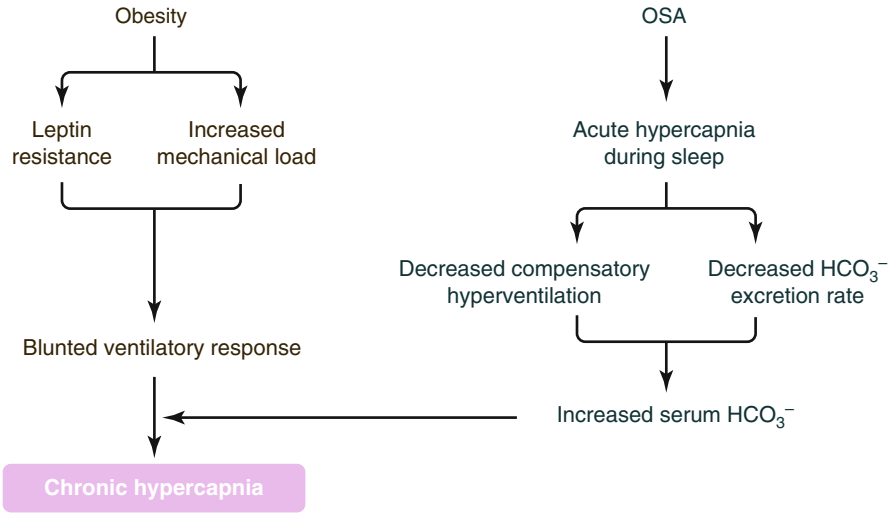

Fig. 16.1 Possible mechanisms for the development of awake hypercapnia in obesity hypoventilation syndrome. *OSA* obstructive sleep apnea, *HCO_3^-* serum bicarbonate

consistent with a restrictive pattern with low forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV_1) and normal FEV_1/FVC ratio. Although the exact cause of excessive load on the respiratory system remains unclear, it has been shown that patients with OHS have a threefold increase in their work of breathing, and it has been shown that morbidly obese patients dedicate 15 % of their total oxygen consumption to the work of breathing, compared to only 3 % in nonobese patients [7, 8].

(b) Leptin resistance may also help to explain awake hypercapnia in OHS. Leptin is a hormone produced by adipocytes which functions to regulate appetite and energy expenditure. Leptin also acts to stimulate ventilation [9]. Obesity leads to increased CO_2 production. Typically as obesity increases, excess adipose tissue accumulates and leptin levels increase, causing increased minute ventilation, and eucapnia is maintained in obese patients despite an increased CO_2 load. In contrast, patients with OHS have higher leptin levels compared to eucapnic patients with OSA matched for percent body fat and apnea/hypopnea index (AHI), and their serum leptin levels fall after treatment with positive airway pressure (PAP) therapy [10, 11]. These results suggest that patients with OHS may be resistant to leptin and that resistance may contribute to the hypoventilation seen in OHS.

(c) Impaired compensation for acute hypercapnia in sleep-disordered breathing may also contribute to awake hypercapnia. Patients with OSA, by far the most common type of sleep-disordered breathing seen with OHS, experience periods of apnea, resulting in acute episodes of hypercapnia. Patients with OSA but not OHS are able to maintain carbon dioxide homoeostasis via hyperventilation between apneic periods as well as by renal bicarbonate retention [12, 13]. When

compared to eucapnic patients, patients with OHS show a reduced duration of ventilation between apneic periods likely due to gradual adaptation of chemoreceptors from mild elevation of serum bicarbonate [1, 14]. A transition from acute to chronic daytime hypercapnia may be explained by the kidney's inability to completely excrete the small amount of retained bicarbonate. A mathematical model has been constructed and showed that when both carbon dioxide response and rate of renal bicarbonate excretion were abnormally low, awake hypercapnia developed over multiple days [13].

L-3: The patient's pre-intubation ABG showed a pH of 7.29 and a $PaCO_2$ of 83 mmHg. What can be said of this patient's risk for OHS based just on this ABG?

While a diagnosis of OHS cannot be made solely on the bases of the above ABG, one's clinical suspicion can certainly be peaked. The ABG shows a respiratory acidosis. If we treat this as acute respiratory acidosis, one expects the pH to fall 0.08 units for every 10 mmHg increase in $PaCO_2$. Based on this assumption one would expect a pH of approximately 7.1. Since the pH was actually 7.29, there must be concomitant partial metabolic compensation. Knowing that for every 0.1 units the pH is returned towards normal and the bicarbonate and base excess (BE) both increase by 7 mEq/L, one can calculate the base excess and bicarbonate values. Since the pH was restored 0.2 units towards normal, the bicarbonate and BE both had to increase by 14–40 mEq/L and 14 mEq/L, respectively. Through these calculations one can see that at the very least this patient has an element of chronic respiratory insufficiency causing a chronic respiratory acidosis with partial metabolic compensation and could certainly be suffering from OHS (although other causes of hypoventilation must first be ruled out before a diagnosis of OHS can be made).

L-4: What is BiPAP and is it useful in patients with OHS?

BiPAP is defined as bilevel positive airway pressure and is a mode of noninvasive ventilation. It is essentially a combination of CPAP with pressure support for each sensed breath. Although it was insufficient in the case presented, positive airway pressure (PAP) therapy is considered the first-line therapy for the treatment of OHS (other treatment modalities include supplemental oxygen, weight reduction surgery, and pharmacologic respiratory stimulants). Short-term PAP therapy ($\leq$3 weeks) has been shown to improve gas exchange and sleep-disordered breathing. Long-term PAP therapy ($\geq$4 weeks) improves lung volumes and central respiratory drive to CO_2 and lowers mortality [1].

L-5: What are the components of preparation for an awake fiberoptic intubation?

A methodical practiced approach to an awake fiberoptic intubation will help to ensure the greatest possibility of success. The main components of a successful fiberoptic intubation are psychological buy-in, antisialagogue, adequate topicalization, proper patient and operator positioning, and sedation (see Table 16.1). Of primary importance is obtaining psychological buy-in from the patient. They must fully understand why the procedure must be done and the potential life and death

Table 16.1 Components of an awake fiberoptic intubation

Psychological buy-in
Use of an antisialagogue
Adequate topicalization
Proper patient and operator positioning
Nerve blocks
Adequate sedation

nature of the procedure should be emphasized. After securing psychological buy-in, an antisialagogue, such as glycopyrrolate, should be given to help dry out the airways. After the antisialagogue has had time to act and the airways are dry, one should begin topicalizing the airway. Prior to topicalization sedation is often given provided that patient safety is not compromised. Multiple drugs can be used to accomplish this including remifentanil, fentanyl, midazolam, dexmedetomidine, and ketamine. The choice of agent is often less important than careful titration to avoid oversedation. Thought must also be given to the best position for the procedure. The sitting position is typically best with obese patients as it helps to keep the tongue off of the posterior pharynx and decreases the pressure of abdominal contents on the diaphragm. Looking at the case presented, the methods used appear reasonable, adequately addressing the main components of an awake fiberoptic intubation. Psychological buy-in was obtained, an antisialagogue was given prior to topicalization, and adequate topicalization was achieved. A "lidocaine lollipop" consisting of lidocaine ointment on a tongue blade was used initially to begin topicalization. The patient was instructed to rest the lidocaine lollipop on the back of the tongue, and as time passed the patient was encouraged to advance the tongue blade farther into the oropharynx right up to the point of gagging. After the tongue blade can be placed on the posterior tongue without eliciting a gag reflex, further topicalization is done with atomized lidocaine. Four percent lidocaine was placed in an atomizer and the patient was instructed to open his mouth, and the atomizer was advanced into the oropharynx and sprayed towards the posterior oropharynx; while spraying the patient was instructed to pant to facilitate movement of the lidocaine towards the larynx and vocal cords. To minimize systemic local anesthetic uptake, oral suction was used between bouts of atomized lidocaine to remove any oral secretions and excess lidocaine. The procedure is repeated until the patient can tolerate the atomizer being placed past the posterior tongue. Of note, careful attention must be given to the amount of local anesthetic used so as not to cause local anesthetic toxicity. To judge adequacy of topicalization, an oral airway that allows passage of the fiberoptic bronchoscope (FOB) and endotracheal tube (ETT) (such as a Berman airway) with lidocaine paste on the posterior aspect was placed into the oropharynx. If the patient tolerates this, the FOB is passed through the oral airway. Once a view of the cords was established, 4 % lidocaine was sprayed onto the cords via the FOB after warning the patient that they will likely cough. Once completed, the FOB can be advanced through the vocal cords and the ETT slid over the FOB into the trachea. Proper placement of the ETT is confirmed via direct visualization with the FOB, capnography, and auscultation.

Nerve blocks can also be used to anesthetize the oropharynx. Cranial nerves IX and X must both be blocked. The glossopharyngeal nerve (cranial nerve IX) innervates the pharynx and posterior third of the tongue, while the vagus (cranial nerve X) innervates above and below the vocal cords via its branches: the superior laryngeal nerve (SLN) and the recurrent laryngeal nerve (RLN). The SLN innervates the arytenoids, epiglottis, and sensation above the cords, while the RLN provides sensory innervation below the cords. Of note, this is often quite difficult in obese patients because their anatomy can be unfavorable making identification of landmarks difficult and blocks potentially unreliable.

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Part II
Circulation-Related Cases

Chapter 17
Hemorrhage During Endovascular Repair of Thoracic Aorta

Michael Bronson

The patient is a 62-year-old male scheduled to undergo a thoracic aorta endograft in the interventional radiology (IR) suite. He is 5′7″ tall and weighs 74 kg with a BMI of 25.5. He has a past medical history of hypertension, rectal cancer with possible metastases to his liver, and a newly diagnosed descending thoracic aorta aneurysm found during work-up for his rectal cancer. He denies any chest pain or shortness of breath with exertion. He has a 40-pack-year smoking history; however, he quit several months ago. He has no past surgical history. Current medications include enalapril, amlodipine, oxycodone, and lansoprazole. He has no drug allergies. Laboratory analysis showed electrolytes and liver function tests within normal limits. His starting HCT was 29.2 %. EKG showed normal sinus rhythm. Airway exam was significant for a class II Mallampati score, >6 cm thyromental distance, full range of motion of his neck including excellent extension, good dentition, and the ability to prognath.

Before the start of the case, it was discussed with the surgeons the details of their procedure and what sort of difficulties they may face (**L-1**, **L-2**). The surgeon initially said that if anything went wrong with the case, "it would be so catastrophic that there would be nothing we could do to fix the problem." However, the surgeon reassured the anesthesia team that this was a simple procedure and that he had little cause for concern (**L-3**). The patient was brought to the IR suite and preoxygenated while routine monitors were placed (**L-4**, **L-5**). Induction proceeded smoothly with versed, fentanyl, propofol, and rocuronium. A grade 1 view was obtained on direct laryngoscopy and the endotracheal tube was placed easily without complication (**L-6**). After securing the endotracheal tube, a 16-g peripheral IV was placed to supplement the 18-g IV on the opposite extremity. An inhaled anesthetic was initiated and the case proceeded normally in the initial stages of the procedure.

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Approximately 2.5 h into the procedure, the surgeons began to remove the endovascular sheath from the groin area; it appeared to the anesthesia provider that the device was giving the surgeons some difficulty with removal. At the same time a blood pressure cuff reading of 85/50 mmHg was noticed on monitors, which was an abrupt change from his previously stable 110s/60s mmHg. Simultaneously it was noticed that the patient had a very abrupt drop in $etCO_2$ from 35 to 25 mmHg within the space of a breath or two. Within two more breaths the $etCO_2$ was 22 mmHg (**L-7**). The surgeons were immediately notified of the acute change in vital signs, and both IVs were opened wide. They asked the anesthesia provider to ensure the endotracheal tube was still correctly positioned, and this was confirmed within several seconds. The surgeons quickly used fluoroscopy with contrast to confirm brisk extravasation from the iliac artery. Two units of vasopressin were given at this time, and extra help was called for in addition to a rapid blood infuser and blood products. A cordis kit was also requested, but the IR fellow said he could quickly place a cordis in the femoral vein while the anesthesia provider took care of the patient in all other respects.

The next cuff cycle showed a blood pressure of high 30s/20s mmHg. An additional two units of vasopressin was given, and fluids were squeezed in as fast as possible while awaiting blood products. A STAT consult was placed to vascular surgery. Within several minutes from the initial call for help, several attendings arrived, as did the anesthesia technicians with a level 1 infuser. The infuser was connected to the right femoral vein cordis that had been placed, and an arterial line was placed by one of the anesthesia attendings. The next blood pressure reading was 110/50 mmHg. At this point blood products arrived from the blood bank, and four units of PRBCs were checked and given rapidly. The surgeons were able to obtain hemostasis very quickly, and it was believed that the bleeding and hemodynamics were under control.

The rest of the case proceeded well from a hemodynamic standpoint. In total, 5 L of crystalloid, 500 mL of albumin, nine units of PRBCs, eight units of FFP, two units of platelets, and one unit of cryoprecipitate were given. At the end of the case, the patient did not have good distal lower extremity pulses, so endovascular clot removal was attempted without success. The patient was then taken to the operating room for thrombectomy. Following the case he was left intubated and transported to the SICU. The next morning he was extubated without complication and no neurological deficits were noted.

Lessons Learned

L-1: **What are the details of the procedure to place a thoracic aorta endograft**?

Table 17.1 and Fig. 17.1 outline the basic steps for placing a thoracic aorta endograft [1]. This procedure is usually performed by a vascular surgeon, interventional radiologist, or cardiologist and can take place in the cath lab, in the

Table 17.1 Basic steps for placement of thoracic aorta endograft

1. Patient is placed either in supine or in slight right lateral decubitus position. The left side of thorax is sometimes prepped in anticipation of possible conversion to thoracotomy
2. In surgical cutdown to the common femoral or common iliac artery, the artery is punctured and a guidewire is threaded. A pigtail catheter is inserted over the guidewire into the thoracic aorta and an aortagram is performed
3. The patient is anticoagulated with 100 units/kg of heparin
4. A long stiff guidewire is then placed into the artery, and a 24-French sheath and dilator are inserted over the guidewire until the tip of the sheath is proximal to the proximal end of the aneurysm
5. The guidewire and dilator are removed, and the stent-graft is then placed through the sheath
6. Prior to deployment of the stent, the surgeon may request that the BP be decreased to MAPs of 50–60 mmHg, usually with sodium nitroprusside, to prevent inadvertent downstream deployment of the graft
7. The sheath is rapidly removed and the stent deploys
8. Heparin is reversed with protamine after sheath removal and arteriotomy is repaired

See Fig. 17.1

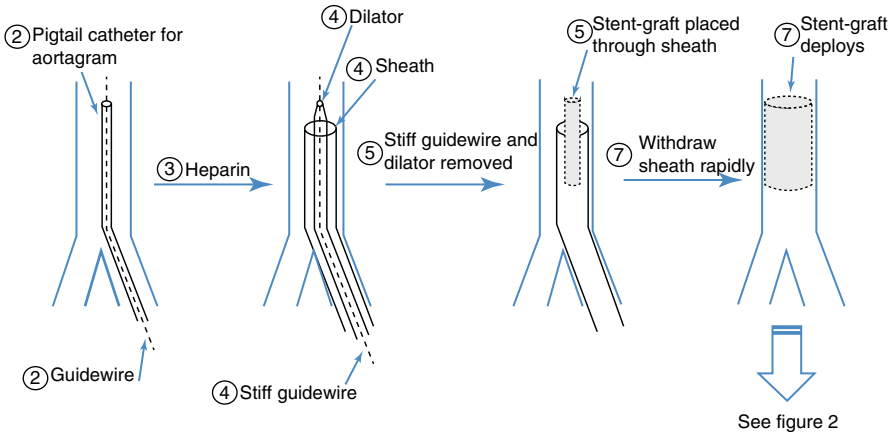

Fig. 17.1 Basic steps for placement of thoracic aorta endograft

interventional radiology suite, or in the operating room. The anesthesia for this procedure is usually general, although it has been done under regional anesthesia as well as local anesthesia with moderate sedation [2]. The blood loss during the case is usually very minimal; however, massive hemorrhage can occur. The rate of conversion to open thoracotomy has been reported to occur in up to 2.9 % of cases [3].

L-2: **What are the prerequisites for doing this procedure**?

During work-up for this procedure, the patient undergoes a contrast spiral CT scan of the thorax and a thoracic aortography to best assess the dimensions of the aneurysm [1]. Adequate distance of normal vessel at the proximal and distal necks of the

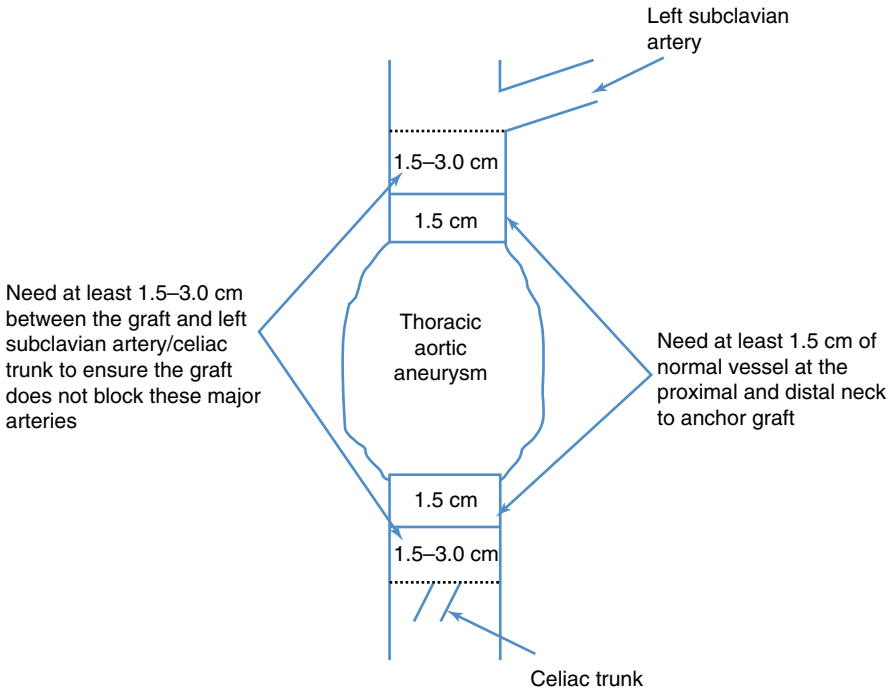

Fig. 17.2 Prerequisites for performing thoracic aorta endograft

aneurysm is essential to anchor the graft (Fig. 17.2). The stent must be placed at a location so as to not occlude major arteries (left subclavian, celiac trunk, mesenteric, renal, and iliac arteries). In addition, the length of the stent is kept to a minimum to decrease the involvement of intercostal arteries.

L-3: **Should the patient have a blood type and cross completed prior to undergoing this procedure**?
Estimated blood loss during thoracic aorta endograft placement is usually minimal. However, in the event of an aortic rupture, bleeding can be quite massive. Damage to vessels in the groin used for access can also be a site of substantial blood loss. Given the potential for high volume losses, patients undergoing this procedure should have a blood type and cross for at least two units of PRBCs prior to the start of the case.

L-4: **Should an arterial line be placed preoperatively**?
Table 17.2 lists indications for arterial line placement. Specifically for this procedure, rapid detection of complications is essential since catastrophic hemorrhage can occur without warning during any part of this procedure. Consequently, arterial line placement should be performed preoperatively. Also, planned intervention with sodium nitroprusside during graft deployment requires immediate feedback on hemodynamic response to maintain tight control of blood pressure with a goal MAP of 50–60 mmHg. It is recommended that the arterial line be placed in the right radial artery as the left brachial artery is sometimes used for access by the surgeons to perform aortic angiography [1].

Table 17.2 Indications for arterial line placement

1. Continuous hemodynamic monitoring:
a. Anticipated hemodynamic perturbations
b. Planned pharmacologic manipulation
2. Frequent blood sampling
3. Unable to perform noninvasive BP monitoring
4. Means to assess stroke volume variation and fluid responsiveness

Table 17.3 Indications for central line placement

1. Monitoring of CVP
2. Pulmonary artery catheter placement
3. Conduit for rapid fluid/product administration
4. Drug administration
5. Inadequate peripheral venous access
6. Need for repeated blood sampling
7. Aspiration of air emboli
8. Transvenous cardiac pacing
9. Temporary hemodialysis

L-5: **Should a central line be placed preoperatively**?
Table 17.3 lists indications for central line placement. Ensuring adequate intravenous access in a case with the potential for massive transfusion should be part of the preoperative preparation for thoracic aorta endograft; thus, placement of a central line should be strongly considered. Central access would provide a means of fluid resuscitation in addition to providing the anesthesiologist the ability to rapidly control hemodynamics during graft deployment or in the case that pressor support is needed.

L-6: **In view of an up to a 2.9 % rate of conversion to thoracotomy**, **what are the considerations for airway management**?
The conversion from an endovascular procedure to an open thoracotomy would require the initiation of single-lung ventilation to optimize surgical exposure. Delaying surgical correction while an airway device is exchanged to achieve single-lung ventilation could be devastating for the patient, so the preoperative plan for airway management should focus on this important aspect. The three main options for airway management for this type of case are:

1. Placing an endotracheal tube large enough to accommodate a bronchial blocker if needed
2. Placing a Univent tube
3. Placing a double-lumen tube

Having a fiberoptic bronchoscope immediately available is vital regardless of which type of airway device is chosen. If a double-lumen tube is used for the case and the plan is for the patient to remain intubated postoperatively, this should be exchanged for a single-lumen endotracheal tube prior to transport to the ICU.

L-7: **What is the differential diagnosis for an acute drop in $P_{ET}CO_2$ with a constant minute ventilation and no change in peak inspiratory pressure**?

The $P_{ET}CO_2$ is most fundamentally determined by the $\frac{V_A}{Q_t}$ ratio.

An $\uparrow$ in $\frac{V_A}{Q_t} \rightarrow \downarrow P_{ET}CO_2$

A $\downarrow$ in $\frac{V_A}{Q_t} \rightarrow \uparrow P_{ET}CO_2$

Assuming no inadvertent increase in minute ventilation via an increase in tidal volume and/or respiratory rate, the differential diagnosis can be broken down into four main categories: decreased cardiac output, increased dead space ventilation, decreased elimination of CO_2, and a sampling line leak causing falsely low readings (Fig. 17.3).

First, a decrease in cardiac output leads to decreased perfusion of the lungs, causing a decrease in the denominator in the ratio of *V*/*Q*. Maintained ventilation in the face of lowered perfusion of the lungs will increase this ratio, leading to a lower

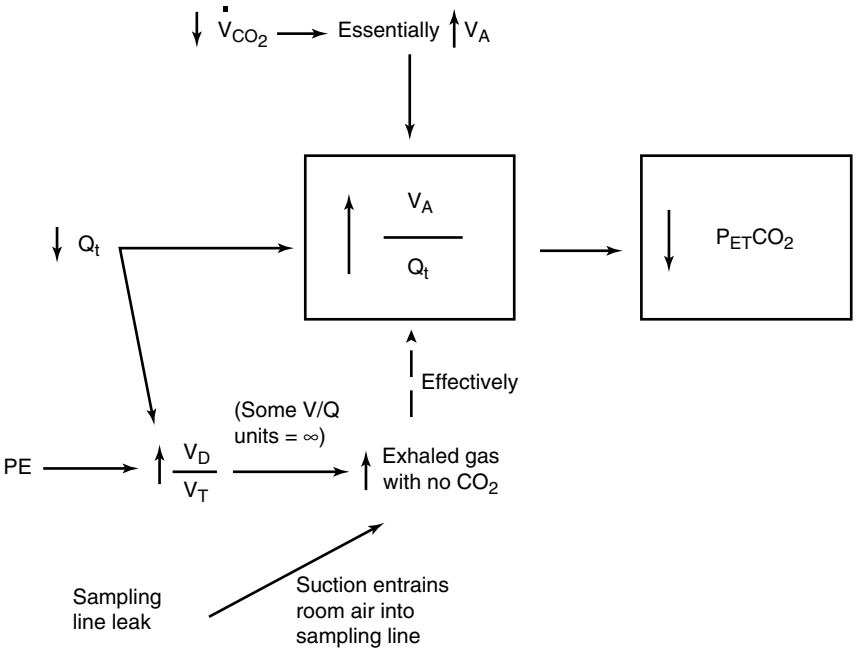

Fig. 17.3 Differential diagnosis of an abrupt decrease in $P_{ET}CO_2$ with a constant minute ventilation and no change in peak inspiratory pressures. $\dot{V}_{CO_2}$ production of carbon dioxide, V_A alveolar ventilation, Q_t cardiac output, *PE* pulmonary embolus, V_D dead space ventilation, V_T tidal volume, $P_{ET}CO_2$ partial pressure of end-tidal carbon dioxide

partial pressure of end-tidal carbon dioxide. Causes of decreased cardiac output are cardiogenic shock, hemorrhagic shock, and cardiopulmonary arrest.

Second, an increase in dead space ventilation with constant minute ventilation (tidal volume x respiratory rate) increases the amount of exhaled gas that does not contain carbon dioxide. This effectively increases the *V*/*Q* ratio as some lung segments now have a *V*/*Q* ratio equal to infinity. In addition to lower cardiac output states, embolic phenomenon to the pulmonary vasculature increases dead space ventilation.

Third, decreased production of carbon dioxide with a constant minute ventilation essentially increases V_A and the V_A/Q_t ratio and decreases $P_{ET}CO_2$. Potential causes during anesthesia of an acute or subacute decreased production of carbon dioxide are deepening the anesthetic (either intravenous or inhalational) or hypothermia.

Fourth, a leak in the sampling line will cause falsely low reading on the CO_2 monitor. The monitor uses a continuous suction technique to ensure adequate sampling, and a leak in the sampling line will lead to entrainment of room air with no CO_2 content. This room air will mix with the patient's exhaled gas within the sampling tubing, essentially diluting the actual partial pressure of CO_2.

Almost certainly in this case, the cause of the decrease in $P_{ET}CO_2$ was caused by a decrease in cardiac output from massive hemorrhage.

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Chapter 18
Pacemakers and Automatic Implantable Cardioverter Defibrillators

Bahareh Khatibi

A 72-year-old 75 kg and 70 in. tall man presented for TURP (**L-1**, **L-2**). His past medical history included hypertension and automatic implantable cardioverter defibrillator (AICD) placement (**L-3**, **L-4**, **L-5**) 1 year ago for inducible ventricular tachycardia during electrophysiologic testing. Adenosine MIBI reveals mild left ventricular dilation with normal systolic function. Electrocardiogram shows NSR with a heart rate of 65 bpm. Current medication included atenolol and ramipril.

A spinal anesthetic was performed with bupivacaine with a resultant sensory block below T9–10. The patient was then placed in the lithotomy position. Following the spinal anesthetic, the blood pressure was 110–120/60–70 mmHg with a heart rate of 65 bpm. Approximately 20 min into the procedure, the blood pressure decreased to 90–100/40–50 mmHg and a heart rate of 50–60 bpm was observed. The patient was given two 100-mcg boluses of phenylephrine (**L-6**). After an additional 10 min, the blood pressure increased to 140–160/70–90 mmHg and the heart rate increased to 115–125 bpm. Monitors showed paced tachycardia with no significant ST changes (**L-7**). The patient denied pain or dyspnea but did complain of a burning sensation on his face. ABG obtained 20 min later showed Na 125, K 4.3, and Mg 1.6. Hypertonic saline infusion was started and Mg 2 g was given in the PACU. Heart rate and rhythm converted to normal and paced in the PACU approximately 1 h after onset of tachycardia. The AICD manufacturer's representative found no mode switch episodes on the counters since the mode switch was programmed with tachyarrhythmia detect rate of 176 bpm.

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Lessons Learned

L-1: **What is TURP syndrome**? **How is it treated**?

(a) TURP syndrome is a complication of TURP that arises from intravascular absorption of large volumes of irrigation fluid during the procedure. During the resection, the bladder is continuously irrigated via a cystoscope to aid visualization while removing blood and resected material. The irrigation fluid, which is a nonelectrolyte fluid containing glycine, sorbitol, or mannitol, may be absorbed via the prostatic venous plexuses. This can cause volume overload, hyponatremia, and hypo-osmolality. TURP syndrome usually manifests with cardiovascular and neurologic signs and symptoms (see burning face later in the case).
(b) Treatment consists of termination of the surgery, as well as diuretics and hypertonic saline as needed for relief of symptoms or severe hyponatremia.

L-2: **How do you diagnose bladder perforation**?

(a) Bladder perforation may occur during TURP. Diagnosis is difficult because signs and symptoms are nonspecific and may be obscured by anesthesia. Symptoms include abdominal pain, respiratory compromise, and a tense abdomen. Diagnosis can be confirmed with a cystogram.

L-3: **What is proper pacemaker/AICD nomenclature**?

(a) See Table 18.1.

L-4: **What are some special considerations for patients with pacemakers undergoing surgery**? **What information should you know about a patient with a pacemaker preoperatively**? **What does a magnet do**?

(a) Electrical interference produced by electrocautery can be sensed by a pacemaker and interpreted as myocardial activity. This can cause the pacemaker to inhibit the pacemaker generator, or if the activity is interpreted as ventricular fibrillation or ventricular tachycardia, this can cause the ICD to provide a shock to "defibrillate" the patient, which can produce asystole or lead to pacemaker dysfunction.

Table 18.1 Pacemaker nomenclature

First position	Second position	Third position	Fourth position	Fifth position
Chamber paced	Chamber sensed	Response to sensing	Rate modulation	Multisite pacing
O = none	O = none	O = none	O = none	O = none
A = atrium	A = atrium	T = triggered	R = rate modulated	A = atrium
V = ventricle	V = ventricle	I = inhibited		V = ventricle
D = dual	D = dual	D = dual (triggered and inhibited)		D = dual (pacing and shock)

Data from [1]

(b) Prior to elective surgery, it is important to know the pacemaker manufacturer, model number, serial number, date of implantation, and response to magnet placement. The reason for implantation and the patient's degree of pacemaker dependency are also vital. In addition, it is important to review a recent interrogation that confirms correct functioning and adequate battery life
(c) Although a magnet converts some pacemakers to a nonsensing asynchronous mode, this is not always the case. The effect of placing a magnet on a pacemaker should be determined preoperatively.

L-5: **Where should the grounding pad be placed in a patient with a pacemaker**? **What type of polarity should an electrosurgical unit probe have to minimize interference with a pacemaker**?

(a) The grounding pad should be placed as far as possible from the pulse generator to minimize detection of electrocautery. Its position should also minimize current flow through the heart.
(b) Bipolar electrodes are preferable because they confine current propagation to a few millimeters.

L-6: **Phenylephrine**
Despite phenylephrine theoretically having the advantage of not inducing tachycardia (compared to ephedrine), a tachycardia occurred anyway.

L-7: **What causes pacemaker-mediated tachycardia**?

(a) Pacemaker-mediated tachycardia (PMT) describes any condition in which a pacemaker paces the ventricles at an inappropriately fast rate. This can be due to several factors including:

    1. A rate response setting that is too sensitive
    2. Tracking of atrial noise (such as electromagnetic interference)
    3. Inappropriate pacemaker manipulation with rate response on
    4. Tracking of an atrial tachyarrhythmia related to upper rate settings
    5. Classically, a reentrant tachycardia in patients with dual-chamber pacemakers [2, 3]

(b) We do not know why the pacemaker doubled its rate intraoperatively or why it halved its rate in the PACU (i.e., all rate changes were paced by the pacemaker). Possible explanations include the rate modulation function, if present, that was not turned off preoperatively, electrocautery interference, and the electrolyte changes associated with the TURP syndrome.

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Chapter 19
Acute Myocardial Infarction During Laparoscopic Surgery

Geoffrey Langham

A 67-year-old man, 77 kg and 67 in. tall, was brought to the operating room for laparoscopic cholecystectomy for symptomatic cholelithiasis. The past medical history was significant for type 2 diabetes mellitus, controlled with oral hypoglycemic drugs, and gastroesophageal reflux disease, controlled with a proton pump inhibitor. The patient walked 2 miles every other day with no chest pain or shortness of breath. The patient had a 15 pack-year history of smoking but had quit 35 years prior. Medications included glyburide and lansoprazole. The preoperative electrocardiogram (ECG) revealed sinus bradycardia at a rate of 55 beats/min, with normal ST and T wave morphology. Last oral intake was 10 h prior to arriving in the preoperative area.

The patient was taken to the operating room, and after application of standard monitors and preoxygenation, induction of anesthesia was performed with fentanyl, lidocaine, propofol, and vecuronium, and laryngoscopy and tracheal intubation proceeded uneventfully. Surgery began, and pneumoperitoneum was established with CO_2 insufflation at 15 mmHg (**L-1**).

As the gallbladder was being mobilized and taken down from the liver bed, acute elevation of the ST segments were noted on lead V of the ECG (**L-2**). Over the next 4 min, the ST segment elevation progressed to 8 mm. The patient's heart rate and mean arterial pressure remained normal and unchanged at 55–60 bpm and 85–90 mmHg, respectively. Sublingual nitroglycerin spray was administered and repeated 5 and 10 min later with no change in ST segment morphology or vital signs. Nitroglycerin paste was applied to the chest wall, and IV diltiazem was given once, with no change in ST segment morphology or vital signs.

By this point, the surgeons had removed the gall bladder, ceased pneumoperitoneum, and closed the surgical incisions. A 12-lead ECG was performed in the operating room and revealed 7 mm ST segment elevation in leads I, aVL, and V_2–V_5,

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with ST segment depression in leads III and aVF. A nitroglycerin infusion was started while the patient emerged from anesthesia, was extubated, and was taken to the PACU. In the PACU, the cardiology service was consulted, aspirin by mouth was administered, and cardiac enzymes were drawn. The patient denied chest pain or pressure. A bedside transthoracic echocardiogram revealed akinesis of the left ventricular apex and hypokinesis of the left ventricular lateral wall.

The patient was taken emergently for left heart catheterization, where a 90 % occlusive thrombus was found in the left anterior descending coronary artery (**L-3**). Angioplasty was performed but no coronary stent was placed. The patient tolerated the procedure well and was discharged to home on POD #6.

Lessons Learned

L-1: What are the pathophysiologic effects of laparoscopy on the circulatory and respiratory systems?

The numerous effects of laparoscopy can be best understood by considering the three basic derangements associated with laparoscopic surgery: increased intra-abdominal pressure (IAP), CO_2 insufflation, and extremes of patient position. The descriptions below are depicted in Fig. 19.1.

Intra-abdominal pressures commonly used for laparoscopy range from 12 to 15 mmHg and have multiple effects on the circulatory and respiratory systems. First, venous return to the heart is impaired as the IAP compresses the inferior vena cava and increases resistance to venous blood flow from the lower extremities to the heart [1]. The decreased venous return reduces stroke volume and cardiac output. Second, increased IAP raises systemic vascular resistance (SVR); the mechanism is incompletely understood but thought to involve compression of the aorta or intra-abdominal resistance arterioles [2]. Third, distension of the abdominal wall and viscera, especially in patients with high vagal tone and young women, predisposes to vagally mediated reflexes such as bradycardia and bronchospasm [3]. Fourth, increased IAP displaces the diaphragm in a cephalad direction, which reduces functional residual capacity (FRC) and predisposes to $\dot{V}/\dot{Q}$ mismatching. Fifth, increased IAP also displaces the carina cephalad, which predisposes to inadvertent mainstem bronchial intubation [4]. Sixth, increased IAP reduces thoracic static compliance by 30–50 % [5]. Seventh, the gas used for pneumoperitoneum can dissect into the pleural, pericardial, mediastinal, and subcutaneous spaces, especially if the patient has an embryologically defective aortic or esophageal hiatus or is undergoing a surgery with extraperitoneal insufflation, e.g., inguinal hernia repair [6].

The choice of CO_2 as the insufflating gas also affects the circulatory and respiratory systems. The eighth effect of laparoscopy is the direct absorption of CO_2 into tissues and the circulation. This absorption raises the venous pCO_2 and in turn the arterial pCO_2, which usually necessitates an increase in minute ventilation to restore normocapnia. Ninth, CO_2 embolism is a rare but potentially fatal event and occurs most commonly at the initiation of insufflation if the insufflating needle or trocar

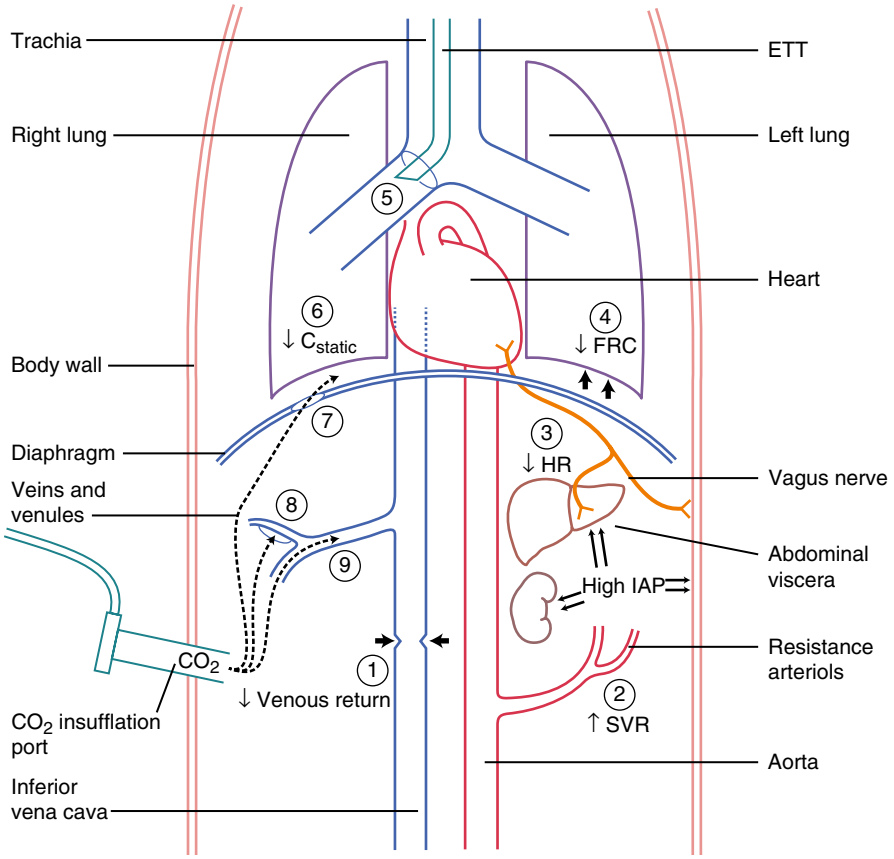


Fig. 19.1 Effects of laparoscopy on the circulatory and respiratory systems. Effects of elevated intra-abdominal pressure (*IAP*) and CO_2 insufflation in the thorax and abdomen are numbered in the order described in the text, starting at the bottom of the diagram and proceeding counterclockwise. *1* Decreased venous return; see also Fig. 19.2. *2* increased systemic vascular resistance (*SVR*). *3* Predisposition to arrhythmias, especially bradyarrhythmias. *4* Decreased functional residual capacity (*FRC*). *5* Predisposition to mainstem bronchial intubation. *6* Decreased thoracic static compliance (C_{static}). *7* CO_2 dissection into anatomic planes of the chest. *8* Systemic CO_2 absorption. *9* CO_2 embolism. *ETT* endotracheal tube

has been inadvertently placed into a vascular structure [7, 8]. CO_2 embolism generally has a better outcome than an air embolism of a similar volume, owing to the higher solubility in blood of CO_2 than air (79 % nitrogen/21 % oxygen) and since CO_2 embolism is responsive to increased ventilation.

It is important for the clinician to note that the effects of increased IAP on the circulatory system are dose dependent, as shown in a 1978 study in which Diamant et al. applied stepwise increases in pneumoperitoneum to anesthetized dogs [9]. The dose dependency of IAP and the magnitude of the effect of high IAP on hemodynamics are quite striking, as shown in Fig. 19.2. In addition, this study showed that

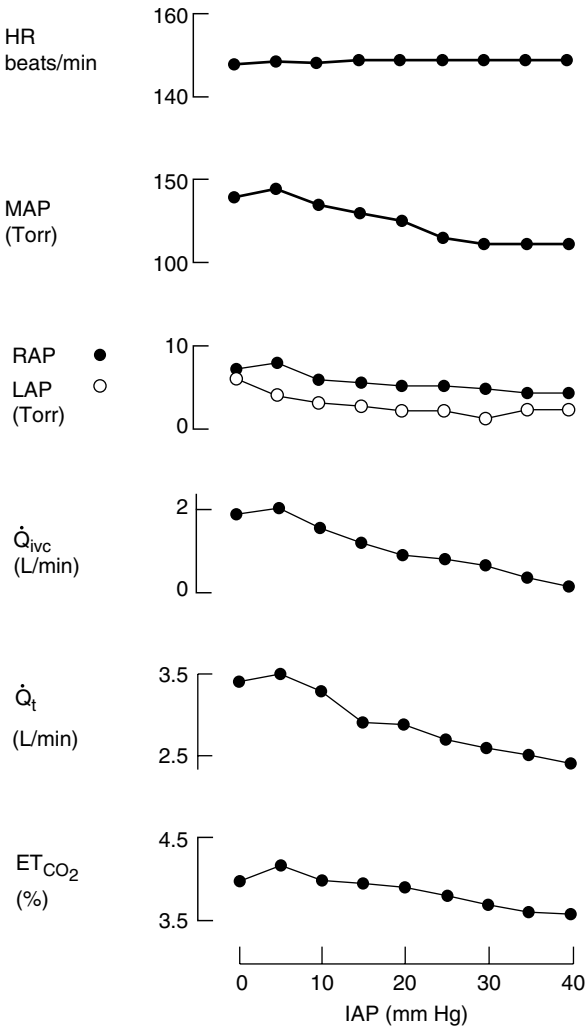


Fig. 19.2 Hemodynamic responses to increased intra-abdominal pressure caused by CO_2 pneumoperitoneum in an anesthetized dog [9]. Since minute ventilation was held constant during CO_2 insufflation, the end-tidal CO_2 decrease is a result of decreased cardiac output. *HR* heart rate, *MAP* mean arterial pressure, *RAP* right atrial pressure, *LAP* left atrial pressure, $\dot{Q}_{ivc}$ inferior vena cava blood flow, $\dot{Q}_t$ cardiac output, ET_{CO_2} end-tidal CO_2, *IAP* intra-abdominal pressure

the negative hemodynamic effects of increased IAP were magnified by volatile anesthetic administration and hypovolemia.

Laparoscopic surgery frequently requires extremes of patient position such as steep Trendelenburg or side-to-side tilt in order to facilitate surgical exposure. The reader is directed elsewhere for a thorough discussion of the effects of such positions on the circulatory and respiratory systems [10].

L-2: How is acute ST segment elevation interpreted in this situation?

The differential diagnosis of ST segment elevation is relatively short. ST-elevation myocardial infarction (STEMI), usually in the setting of complete coronary occlusion, is the most important cause of ST elevation because it has significant associated morbidity and mortality; however, it is not the most common cause. In a retrospective

study of patients presenting to the emergency department with chest pain and having ST elevation on the ECG, STEMI was the etiology in only 15 % of patients [11]. Left ventricular hypertrophy (25 %), bundle branch blocks (20 %), early repolarization (12 %), pericarditis (1 %), and ventricular-paced rhythm (1 %) were among the remaining etiologies of ST segment abnormality. Even among a carefully selected population, i.e., patients referred for primary percutaneous coronary intervention for suspected STEMI, various authors have found a 1–13 % incidence of nonischemic etiology for ST elevation [12, 13]. Clearly, STEMI is not the most common cause of ST elevation in the settings studied above, but the design of these studies and the chronicity of the more common diagnoses, e.g., left ventricular hypertrophy, unfortunately limit the usefulness of these data for anesthesiologists.

An important tool to differentiate ischemic from nonischemic causes of ST elevation is the presence or absence of "reciprocal changes," that is, ST segment depression in leads representing a coronary artery distribution distinct from those showing ST elevation. The classic example is an acute inferior wall myocardial infarction (MI) associated with ST elevations in the inferior leads and reciprocal ST depressions in the precordial leads. The mechanism of reciprocal changes is not fully understood, but such changes do not appear to represent subendocardial ischemia in the territory showing ST depression. Reciprocal changes in the setting of ST elevation are valuable because they are strongly associated with acute MI and provide a positive predictive value for STEMI greater than 90 %, thus making STEMI the leading diagnosis. Reciprocal changes also identify patients at higher risk of poor outcomes, which can in turn justify aggressive therapy.

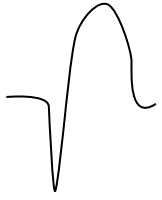
As for this case, the hyperacuity of the ST elevation, the change from a previously normal baseline, the presence of reciprocal changes, and the physiologically intrusive nature of laparoscopic surgery (as in Fig. 19.1) and general anesthesia all contribute to bring STEMI to the top of the differential diagnosis. Table 19.1 describes etiologies for hyperacute ST elevation pertinent to this case. Additional diagnoses worthy of mention here include subarachnoid hemorrhage and intrinsic gallbladder disease, both of which have been associated with ST depression and T wave abnormalities, but not with ST elevation perse.

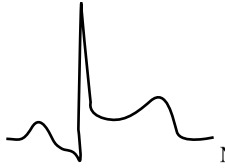
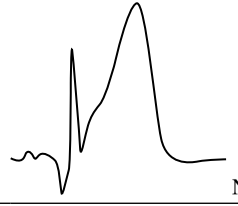
L-3: Why did this coronary artery become occluded at this time?

The two major mechanisms leading to MI are (1) transmural ischemia resulting from complete coronary artery occlusion and (2) subendocardial ischemia resulting from myocardial oxygen supply-demand imbalance. Historically, perioperative MI was most commonly observed on the third postoperative day after the detection of pathologic Q waves on the ECG and attributed to the first mechanism described above: complete coronary occlusion causing transmural ischemia [16].

However, our understanding of perioperative MI has shifted as a result of increased awareness of this clinical entity and improved diagnostic techniques, including serum cardiac enzymes and echocardiography. Currently, it is thought that many perioperative MIs occur in the first 24–48 h postoperatively and share the second mechanism described above: subendocardial ischemia related to myocardial oxygen supply-demand imbalance. Figure 19.3 shows the factors contributing to the

Table 19.1 Causes of acute ST segment elevation pertinent to this case

Cause	Mechanism	Distinguishing factors	Characteristic ECG
STEMI [14]	Coronary artery occlusion: plaque rupture, vasospasm, or embolism	1 mm elevation in at least two contiguous ECG leads ST elevation either convex upward or flat	 Note convex-upward nature of ST segment
		Reciprocal changes may be present Pathologic Q waves may be present	 Note pathologic Q wave and "tombstone" appearance of ST complex
Early repolarization [15]	Genetic variant with abnormal ventricular repolarization	ST segment concave upward Elevation in multiple leads Height of ST elevation may increase with sympathetic nervous system activity Reciprocal changes must be absent	 Note concave-upward nature of ST segment

Pericarditis or myocarditis	Epicardial or myocardial injury with repolarization abnormality	Elevation in many or all leads ST segment concave upward Reciprocal changes must be absent	 Note concave-upward nature of ST segment
Hyperkalemia	Tall, peaked T wave "pulls" ST segment above baseline	ST segment concave or flat Other ECG findings of hyperkalemia (PR interval prolongation, QRS complex widening)	 Note concave-upward nature of ST segment

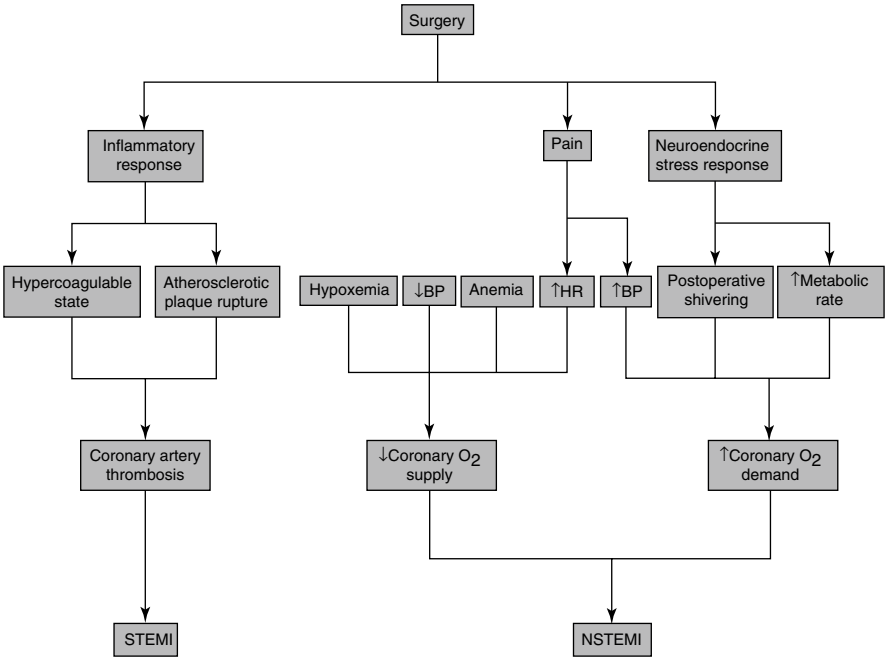


Fig. 19.3 Factors contributing to the development of perioperative myocardial infarction (Adapted from Akhtar [16]). *BP* blood pressure, *HR* heart rate, *STEMI* ST-elevation myocardial infarction, *NSTEMI* non-ST-elevation myocardial infarction

development of perioperative MI [16], while Fig. 19.4 delineates in greater detail and completeness the determinants of myocardial oxygen supply and demand [17]. Perhaps as a result of this shift in diagnostic approach and early recognition, mortality rates associated with perioperative MI have declined to 10–15 % from earlier estimates as high as 50 %.

This patient developed acute myocardial infarction as a result of near-complete coronary artery occlusion, likely due to atherosclerotic plaque rupture and thrombosis. This case of perioperative MI is interesting in that it was "classic" in the sense of involving acute coronary occlusion but also unusual in that it occurred during the intraoperative period.

The patient's normal cardiopulmonary functional capacity, history of diabetes mellitus, and intermediate-risk surgery do not a priori suggest a patient at high risk of perioperative MI. And, in fact, it appears that none of the nine circulatory and respiratory effects of laparoscopy described above was the direct etiology of the MI in this case. Nonetheless, this case is a reminder of several clinical pearls regarding coronary artery disease (CAD) in the perioperative period. First, subclinical CAD is present in many of our patients; the patient and clinician are unaware of the disease because nonobstructive atherosclerotic coronary lesions do not cause symptoms. Second, such non-flow-limiting plaques, with a thin fibrous cap, without calcification, and typically causing less than 50 % stenosis, are the plaques that tend to

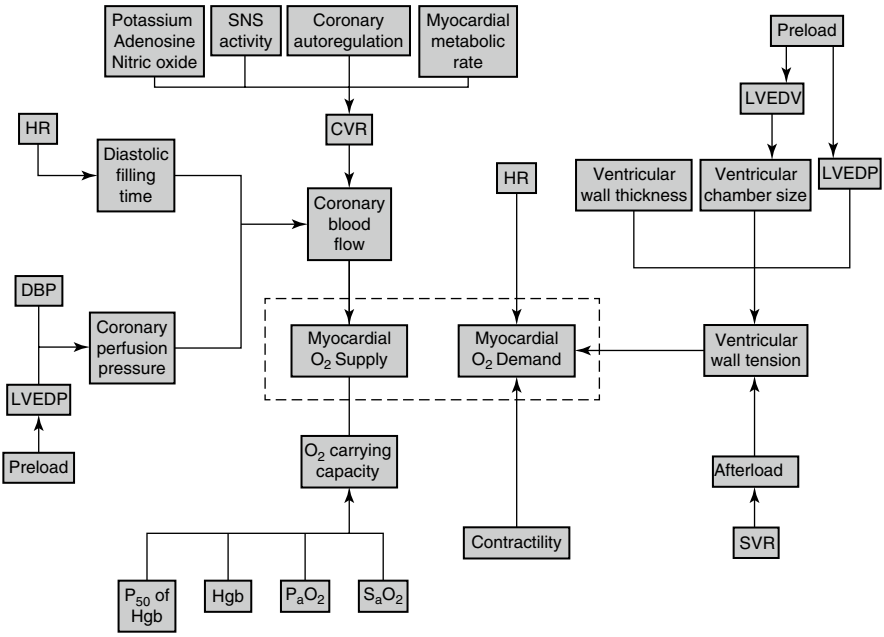


Fig. 19.4 Determinants of myocardial oxygen supply and demand. Note that heart rate (*HR*) and preload each contribute in several ways. *SNS* sympathetic nervous system, *CVR* coronary vascular resistance, *DBP* diastolic blood pressure, *LVEDP* left ventricular end-diastolic pressure, *Hgb* hemoglobin, P_aO_2 arterial partial pressure of oxygen, S_aO_2 arterial oxyhemoglobin saturation, *LVEDV* left ventricular end-diastolic volume, *SVR* systemic vascular resistance (Adapted from Benumof [17])

rupture and cause acute MI, especially in the setting of systemic inflammation [18]. Last, patients with nonobstructive CAD rarely have well-developed collateral vessels and, in the setting of acute coronary occlusion, may suffer massive myocardial infarction and necrosis with resultant heart failure or shock.

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Chapter 20
Sickle Cell and Preeclampsia

Bahareh Khatibi

The patient was a 32-year-old, 26-week pregnant female admitted to the CCU for sickle-cell SC disease (**L-1**, **L-2**) with acute chest syndrome (**L-3**) and severe preeclampsia (**L-4**, **L-5**). On hospital day 5, the patient complained of contractions and difficulty breathing. The obstetrics team evaluated the patient and requested that the anesthesia team stand by during the stat delivery.

Upon the anesthesia team's arrival to the CCU, the patient (117 kg) appeared to be in respiratory failure with an SaO_2 of 83 % on 10 L/min of oxygen via non-rebreather mask, blood pressure of 227/156 mmHg, and heart rate of 120 bpm. 10 L/min of oxygen via mask and Mapleson circuit was applied. Routine monitors were placed and the patient was transported to the labor and delivery department. Midazolam 5 mg IV and fentanyl 100 μg IV were given by titration. The baby was delivered within 15 min. IV Pitocin was started per OB request and the OB team started to repair perineal lacerations.

After 20 min, the patient began having worsening respiratory distress. The decision was made to intubate the patient. Rapid sequence induction with propofol 150 mg IV, fentanyl 300 μg IV, and succinylcholine 120 mg IV and cricoid pressure were performed, and the patient was intubated with a 7.0-mm ID ETT without complication. Mechanical ventilation was set at TV 1,000 mL, PEEP 8 cm H_20, and respiratory rate 8 breaths/min, with PIP 48 cm H_2O. It was noted that straw-colored froth was coming out of the ETT (**L-6**). Vital signs were BP 103/60 mmHg, HR 110 bpm, $etCO_2$ 38 mmHg, and SaO_2 100 % on FiO_2 of 100 %. Arterial line was placed and blood gas showed 7.27 /50/101/-5/22, Na 137, K 4.2, Glu 190, Ca 0.95, and Hct 32. The patient was given fentanyl, midazolam, rocuronium, and furosemide 20 mg IV.

The OB team finished the repair of lacerations and noted a blood loss of 300 mL. A right internal jugular (IJ) 9 F cordis was placed and a pulmonary arterial catheter was placed. Wedge pressure was 24 mmHg. At this point, the patient's vital signs

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were BP 120/65 mmHg, HR 120 bpm, and SaO_2 100 %. The chest x-ray that was obtained confirmed right IJ central line with no evidence of pneumothorax and bilateral total lung opacities.

Forty-five minutes later, the patient's vital signs were BP 75/55 mmHg, HR 128 bpm, and SaO_2 100 %. Pitocin was turned off and phenylephrine was given. Transesophageal echocardiography (TEE) was placed and showed mildly dilated right ventricle (RV) and left ventricle (LV), poor systolic function, and trivial pericardial effusion. Dobutamine was started at 5 μg/kg/min. Calcium was repleted and furosemide 20 units IV was given for anuria.

The patient was given midazolam 2 mg IV, fentanyl 100 μg IV, and rocuronium 50 mg IV for transport to the CCU. Upon arrival the patient had made 45 mL of urine, and her vital signs were BP 135/90 mmHg, HR 133 bpm, and SaO_2 100 %.

Lessons Learned

L-1: Discuss the types of sickle-cell disease. What are the causes of sickle-cell disease? What are the signs and symptoms of patients with sickle-cell disease?

(a) Sickle-cell diseases are congenital hemoglobinopathies. The term "sickle-cell disease" includes all manifestations of abnormal homozygous sickle-cell (hemoglobin S [HbS]) levels, including homozygous sickle-cell disease (HbSS) and mixed heterozygous hemoglobinopathies (HbS/β-thalassemia, HbSC disease, and other combinations). See Table 20.1 [1].

1. Sickle S hemoglobin

   HbS differs from the normal Hb A in the substitution of a valine for a glutamic acid in the beta-globin subunit.

2. Sickle C hemoglobin

   HbC causes the red blood cell (RBC) to lose water via enhanced activity of the potassium-chloride pump, producing mild to moderate hemolytic anemia.

   The dehydration caused by HbC increases the concentration of HbS within the RBC, exacerbating its insolubility and tendency to polymerize.

3. Sickle β-thalassemia hemoglobin

   Normal hemoglobin is made up of two α- and two β-globin chains. β-thalassemias result from insufficient (β+) or absent (β0) production of β-globin chains.

4. Unstable hemoglobins

   More than 100 unstable hemoglobin variant have been documented; most are associated with only minimal clinical manifestations, but severe anemia and hemoglobin-induced renal injury are possible [2].

Table 20.1 Diagnoses, genotype, and cardinal symptoms of sickle-hemoglobin disorders

Diagnosis	Genotype	RBC studies	Symptoms
Sickle-cell anemia	HbSS	Hb 6–9 g/dL	Sickle-cell crises/pain crises
		Normochromic sickle cells	Acute organ syndromes
		Positive hemolysis parameters	Chronic hemolytic anemia
Sickle-cell trait	HbAS	Normal	No apparent illness
HbSC disease	HbSC	Hb 10–13 g/dL	Weak symptoms of sickle-cell disease
		Target cells Microcytosis	Chronic hemolytic anemia
Sickle-cell β+−thalassemia	HbS and β+−thalassemia	Hb 9–12 g/dL Hypochromia, microcytosis	Variable, mild sickle-cell disease
Sickle-cell β0-thalassemia	HbS and β0-thalassemia	Hb 6–10 g/dL Hypochromia, microcytosis	Severe sickle-cell disease

Adapted from [1]

L-2: Why do RBCs sickle in susceptible patients?

(a) RBCs sickle in the setting of hypoxia, hypothermia, low-flow states, and acidosis. When these clinical conditions occur, Hb S becomes deoxygenated. Deoxygenated HbS, being 50 times less soluble than deoxygenated Hb A, aggregates into long stacks called tactoids. Tactoids alter the shape, function, and fluidity of the RBC [3].

(b) Avoidance of hypoxia, hypercarbia, hypothermia, low-flow states, and acidosis is crucial in patients with sickle-cell disease.

L-3: What is acute chest syndrome? What is the pathophysiology?

(a) Acute chest syndrome (ACS) is an acute pulmonary illness characterized by a new pulmonary infiltrate involving at least one complete lung segment and at least one of the following: chest pain, fever >38.5 °C, tachypnea, wheezing, or cough. The pathogenesis of ACS includes infection, pulmonary vascular occlusion by sickled erythrocytes, and fat from bone marrow that was infarcted during acute pain episodes. Conditions that can precipitate ACS include general anesthesia, surgery, and bronchospasm from asthma—most likely because these primary conditions can induce the secondary changes listed in 2b. Treatment requires focused attention of oxygenation, ventilation, temperature, adequate analgesia, and frequent blood transfusion to correct anemia, tissue perfusion, and improve oxygenation [4].

L-4: What is preeclampsia?

(a) Preeclampsia is a syndrome that usually occurs after 20 weeks of gestation (or earlier in the case of trophoblastic diseases such as hydatidiform mole or hydrops) and is diagnosed by systolic blood pressure >140 mmHg or diastolic blood pressure >90 mmHg and proteinuria defined as urinary excretion of >0.3 g in a 24-h collection.

L-5: What is the management of preeclampsia?

(a) There is little evidence to suggest that any therapy alters the underlying pathophysiology of preeclampsia. Therapeutic efforts may slow the progression of the disorder and permit continuation of the pregnancy.
(b) Fetal surveillance is indicated for women with preeclampsia. Fetal movement counts and the biophysical profile (consisting of a non-stress test and the ultrasonographic assessment of the fetus and amniotic fluid volume) constitute the most common fetal surveillance techniques.
(c) Antihypertensives, including hydralazine 5–10 mg IV and labetalol 20 mg IV, are indicated when the diastolic blood pressure is >105–110 mmHg. The goal of hypertension control is to lower blood pressure to prevent cerebrovascular events while maintaining uteroplacental blood flow.
(d) Magnesium sulfate is the first-line treatment for the prevention of eclamptic seizures.
(e) Delivery is the only definitive treatment for preeclampsia [5].

L-6: What is the differential diagnosis of pulmonary edema?

(a) Cardiogenic.
(b) Fluid overload.
(c) Neurogenic.
(d) Acute lung injury.
(e) Pneumonia.
(f) Negative pressure pulmonary edema.
(g) Hypoproteinemia.
(h) High altitude.
(i) Preeclampsia.

In this case, the pulmonary edema observed was most likely a combination of (d) acute lung injury and (i) preeclampsia.

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Chapter 21
Dysrhythmias in a Patient with Crohn's Disease

Geoffrey Langham

A 48-year-old man, 70 kg and 71 in. tall, with a 30-year history of Crohn's disease (CD), was brought to the operating room for exploratory laparotomy and lysis of adhesions with release of a chronic small bowel stricture. The stricture had been causing daily postprandial emesis. The patient previously had two bowel resections related to his CD, resulting in short bowel syndrome (**L-1**). The patient demonstrated a normal cardiopulmonary functional capacity and could achieve four metabolic equivalents without symptoms. The past medical history was also significant for chronic opioid-dependent low back pain, for which the patient took methadone 90 mg by mouth twice daily. Preoperative laboratory assessment was notable for serum sodium concentration of 133 mEq/L and potassium concentration ($[K^+]$) of 3.2 mEq/L (**L-2**, **L-3**). The patient took a bowel preparation by mouth the night before the operation.

After transfer to the operating room, uneventful rapid-sequence induction of anesthesia and placement of an endotracheal tube, a second peripheral IV line, and an arterial line was performed. An ABG that was taken at 11:30, 15 min prior to surgical incision, is shown in Table 21.1.

One hour into the operation, the surgeon encountered about 400 mL of acute blood loss, which was controlled by the surgeon and treated with two units of packed RBCs and 1 L of hetastarch solution. Vital signs remained within normal limits, with HR 70–80 bpm, MAP greater than 75 mmHg, and temperature 36.5 °C. A posttransfusion ABG is shown in Table 21.1 (**L-4**).

One hour later, the ECG showed flattening of T waves seen in leads II and V, which progressed to deep T wave inversions in the inferior leads. Within 5 min, the patient developed frequent bizarre wide-complex beats and T wave inversions in all leads, as shown in Fig. 21.1. These beats were diagnosed as multifocal premature ventricular contractions (PVCs). These beats became more frequent and progressed

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Table 21.1 Arterial blood gas data

Event	Time	pH	pCO_2, mm Hg	pO_2, mm Hg	Base excess, mmol/L	Hematocrit, %	$[K^+]$, mEq/L	Ionized Ca^{2+}, mmol/L
Pre-incision	11:30	7.46	43	154	5.7	28	3.0	1.00
Posttransfusion	13:00	7.39	49	200	2.8	31	3.5	1.06
PVC recurrence	14:15	7.43	45	221	4.8	30	3.3	1.06

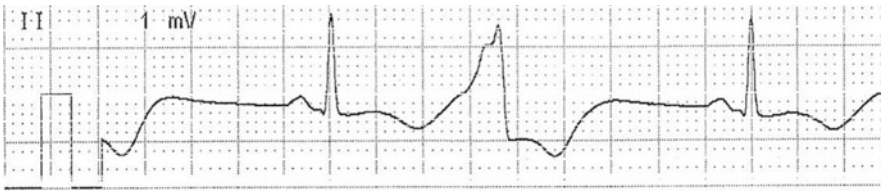

Fig. 21.1 Intraoperative electrocardiogram. Deep, inverted T waves and a wide-complex premature ventricular contraction are seen in lead II

to bigeminy and trigeminy. Despite the frequent PVCs, the HR remained 55–70 bpm and the MAP remained greater than 80 mmHg.

Lidocaine 100 mg IV was given, the PVCs quickly resolved, normal sinus rhythm was restored, and the T wave inversions became more shallow. After 15 min, T wave inversions deepened further, and frequent PVCs recurred and were again successfully treated with lidocaine 100 mg IV. An ABG during PVC recurrence is shown in Table 21.1.

Myocardial ischemia was considered in the differential diagnosis. Empirically, a nitroglycerin infusion was started and a third unit of RBCs was transfused. A transesophageal echocardiogram examination was performed and revealed no regional wall motion abnormalities and normal ventricular filling.

T wave inversions and PVCs again recurred, and this time, amiodarone, 150 mg IV over 10 min, was given, and an infusion at 1 mg/min was started (**L-5**). Chemistry panel sent at this time revealed serum sodium 138 mEq/L $[K^+]$, 3.5 mEq/L, and magnesium 1.3 mg/dL (normal range, 1.8–3.5 mg/dL).

A diagnosis of hypomagnesemia was made, and a magnesium sulfate infusion was started (**L-6**, **L-7**). Cardiac enzymes were sent at this time, which returned within normal limits. The T wave inversions remained, but the PVCs resolved on the amiodarone, magnesium, and nitroglycerin infusions. The surgery concluded uneventfully, and the patient was taken to the intensive care unit intubated and sedated. The patient was weaned from the nitroglycerin and amiodarone infusions, started on intermittent IV magnesium supplementation, and extubated on POD #1.

Lessons Learned

L-1: **What is the pathophysiology of Crohn's disease**? **What is short bowel syndrome**?

Crohn's disease is an inflammatory bowel disease characterized by T-cell-mediated inflammation of bowel mucosa, serosa, and mesentery. The inflammation may occur anywhere along the gastrointestinal (GI) tract and is classically "segmental,"

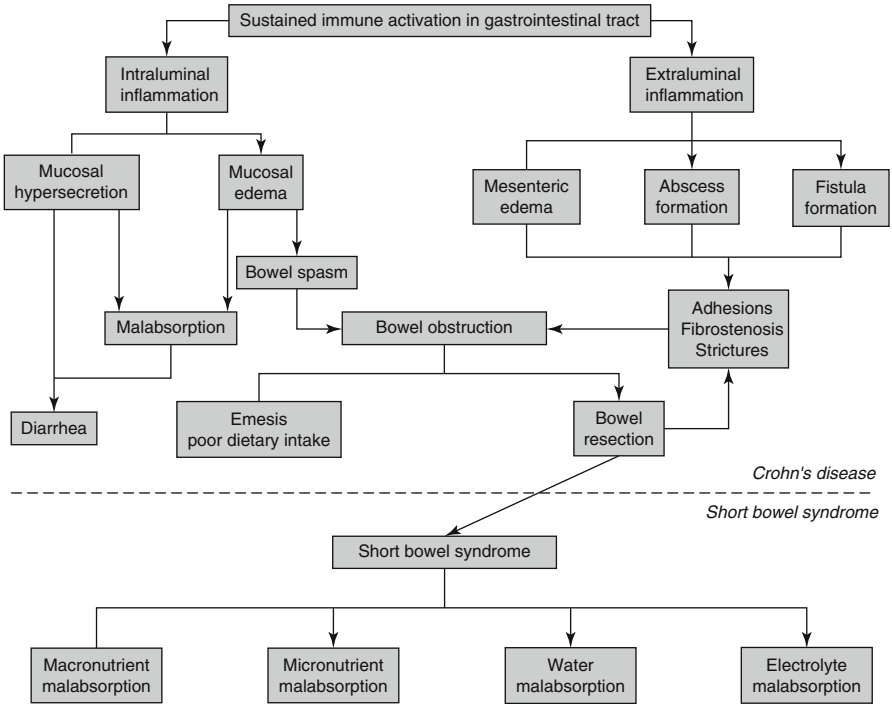

Fig. 21.2 Pathophysiology of Crohn's disease and short bowel syndrome. Clinical features shown above the dashed line are found in Crohn's disease, which can lead to the short bowel syndrome, the features of which are shown below the dashed line

i.e., lesions are separated by sections of healthy GI mucosa. Inflammation often extends transmurally, from gut mucosa to mesentery. The clinical manifestations of CD are attributed to chronic inflammation and its sequelae: bowel edema, malabsorption, stricture, obstruction, surgical resection, and short bowel syndrome [1, 2]. See Fig. 21.2.

Short bowel syndrome is the constellation of signs and symptoms associated with malabsorption in the context of extensive bowel resection or radiation enteritis. The most common cause is extensive surgical resection such as in CD. Loss of adequate intestinal absorptive surface leads to malabsorption of some or all components of dietary intake, which are broadly classified as macronutrients (carbohydrates, fats, amino acids), micronutrients (vitamins, iron), electrolytes, and water. Specific deficiencies are shown in Table 21.2.

L-2: **What are the preoperative and intraoperative considerations of Crohn's disease**?

Patients with CD may have deficiencies of any substance that a healthy GI tract would absorb, especially if the short bowel syndrome is present. The substances in question and their clinical sequelae are shown in Table 21.2.

Electrolyte deficiencies are typically expressed as abnormalities of skeletal muscle function, nervous tissue conduction, and cardiac rhythm. In patients with CD,

Table 21.2 Selected nutritional deficiencies in Crohn's disease and short bowel syndrome, sequelae, and treatment

Deficiency	Clinical sequelae	Treatment
Water	Dehydration (hypovolemia) Hyponatremia or hypernatremia	IV fluids to restore osmolarity and volume
Potassium	Hypokalemia with dysrhythmias or weakness	Potassium supplementation
Magnesium	Hypomagnesemia with dysrhythmias or muscle cramping	Magnesium supplementation
Calcium	Hypocalcemia with tetany or dysrhythmias	Calcium supplementation
Iron	Anemia	Iron supplementation
Water-soluble vitamins	Niacin, B_{12} deficiency	Niacin or B_{12} supplementation
Fat-soluble vitamins	Vitamin K deficiency with coagulopathy Vitamin D deficiency	Vitamin K supplementation Coagulation factor replacement Vitamin D supplementation
Protein	Hypoalbuminemia	Albumin supplementation

a careful history and physical examination should be performed to assess the presence of disturbances in these systems, followed by laboratory evaluation of sodium, potassium, calcium, magnesium, and phosphate levels.

Notably, many patients with CD take glucocorticoids as part of a chronic immunosuppressive regimen. Glucocorticoid use predisposes to preoperative hypertension, insulin resistance, and electrolyte abnormalities, as well as perioperative adrenal insufficiency.

L-3: **How should hypokalemia be corrected intraoperatively**? **Should this patient receive IV fluids supplemented with potassium**, **e.g.**, **normal saline with 20 mEq/L potassium chloride**?

Intraoperative potassium (K^+) supplementation should always proceed carefully and after consideration of the risks and benefits. Truly life-threatening hypokalemia is fortunately rare, and accordingly the clinician's attention in correcting hypokalemia should be directed toward symptomatic cases only (e.g., PVCs) and toward reducing the risk of acute K^+ overdose, which could lead to life-threatening hyperkalemia. The practice of syringe bolusing K^+ no longer has a role outside of cardiac surgery.

As such, intraoperative K^+ infusions should always be given slowly using an infusion pump and no more quickly than 20 mEq/h.

Adding potassium to 1-L bags of normal saline or lactated Ringer's may create a chance of infusion much more quickly than 20 mEq/h if those fluids are bolused to correct hypovolemia. A burette system with a 100-mL chamber, as used commonly in pediatric anesthesia, represents a "middle ground" of control between free-flowing IV fluids and a tightly controlled infusion pump but still carries the risk of rapid potassium infusion.

Table 21.3 Potassium concentration in packed red blood cells at 0, 7, 14, 21, and 42 days of storage

Day	0	7	14	21	42
$[K^+]$, mEq/L	2.2 ± 0.4	14.4 ± 0.8	22.8 ± 2.5	29.9 ± 2.9	44.2 ± 3.5

Table 21.4 Causes of premature ventricular contractions

Arterial hypoxemia	Mechanical irritation (intravascular catheters, surgical manipulation)
Myocardial ischemia	Digitalis toxicity
Hypokalemia	Caffeine
Hypomagnesemia	Cocaine
Sympathetic stimulation	
Catecholamine administration	

L-4: **How does the potassium concentration in a unit of packed RBCs change with duration of storage**? **What is the potassium "dose" in a unit of packed RBCs**?

It is well known that cold storage of RBCs results in hemolysis of the RBCs and release of K^+ into the extracellular fluid in the unit. One recent study measured the K^+ concentration in banked RBCs at various intervals during storage [3] (Table 21.3).

Thus, a typical unit of RBCs, with a volume of 300 mL and a storage time of 14–21 days, represents a potassium dose of approximately 8 mEq. Although this is a significant dose, it is uncommon for this dose to have clinical sequelae outside of a scenario involving rapid or massive transfusion.

L-5: **What is the treatment of intraoperative PVCs**?

PVCs are relatively common during anesthesia. Treatment is guided by the presence or absence of hemodynamic instability, which is a consequence of the decreased stroke volume generated by a premature ventricular beat. If there are more than 6 PVCs/min, if the PVCs are repetitive, or if short runs of ventricular tachycardia occur, treatment is indicated. A differential diagnosis [4] for the cause of the PVCs must be considered concomitantly and is shown in Table 21.4.

Lidocaine 1–1.5 mg/kg IV is the most common treatment for intraoperative PVCs and is a reasonable initial treatment given its limited side effect profile. β-blockers are the drugs most likely to succeed in suppressing PVCs; common choices are metoprolol 2.5–5 mg IV up to 15 mg or esmolol 0.5–1.0 mg/kg bolus, followed by infusion. As in this case, amiodarone, 150 mg IV over 10 min, is a reasonable second- or third-line treatment.

L-6: **What are the characteristic ECG changes with hypokalemia and hypomagnesemia**?

Potassium and magnesium are critical to normal myocardial conduction and contractility. Potassium is largely responsible for maintaining cellular transmembrane

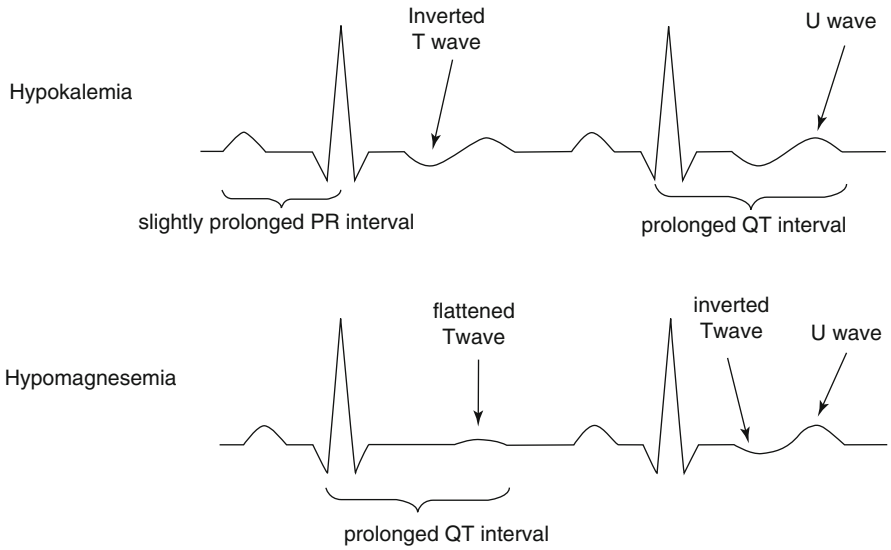

Fig. 21.3 Characteristic electrocardiogram findings in hypokalemia and hypomagnesemia

potential, while magnesium is a cofactor for the Na^+-K^+-ATPase and numerous other enzymes involved with conduction and contraction.

In hypokalemia, there may be flattened or inverted T waves, U waves, ST segment depression, or QT interval prolongation. Any of these may contribute to ventricular dysrhythmias including PVCs, ventricular tachycardia, or ventricular fibrillation [5].

In hypomagnesemia, there may be flattened T waves, U waves, or QT interval prolongation. Any of these may contribute to atrial or ventricular dysrhythmias including premature atrial contractions, atrial fibrillation or flutter, PVCs, or ventricular tachycardia (especially torsades de pointes) [6] (Fig. 21.3).

The ECG findings in hypokalemia and hypomagnesemia share many similarities, a point that underscores the importance of investigating abnormalities of potassium, magnesium, and calcium levels any time an abnormality of one of them is detected or suspected.

L-7: **How is magnesium infusion dosed**?

For urgent intraoperative correction of hypomagnesemia, IV magnesium sulfate is the formulation of choice. A typical loading dose is 2–3 g over 10 min followed by 1–2 g/h. Intermittent bolus dosing may not be as effective as infusion because retention of magnesium after bolus dosing is limited by ongoing renal filtration and excretion [6].

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Chapter 22
Hematologic Disorders: Hemophilia and Disseminated Intravascular Coagulation

Bahareh Khatibi

The patient was a 52-year-old male with hemophilia B (**L-1**), hepatitis C, and history of alcohol abuse who was in the intensive care unit (ICU) following multiple surgeries for a right thigh abscess, each resulting in significant blood loss. The patient had been told that an amputation may be necessary to prevent further complications of blood loss, disseminated intravascular coagulation (DIC) (**L-2**), and sepsis (**L-4**). The patient refused and stated he would rather die than lose his leg. The patient developed fulminant (DIC) and began receiving heparin therapy (**L-3**) to decrease the likelihood of thrombogenesis and end-organ dysfunction. His blood pressure in the ICU had been labile and blood products were required frequently.

On postoperative day 2 after a right thigh abscess incision and drainage, the patient's heart rate (HR) increased from approximately 110 to 150 beats per minute (bpm) over 30 min and mean arterial pressure (MAP) was 116 mmHg. The calculated fluid balance was +4 L. The patient appeared agitated. The hematocrit (HCT) was 35 after receiving two units of packed red blood cells (PRBCs). Propofol infusion was started. The HR remained unchanged, so metoprolol 2 mg IV was given. The HR came down to 120 bpm but the MAP simultaneously decreased from approximately 110 to 50 mm Hg. Propofol was stopped and phenylephrine infusion was started, which increased the MAP to 70 mmHg. Over the next 2 h, the patient had no urine output. The HCT was checked again and was found to be 25. Two units of PRBCs were transfused and urine output improved. Blood draw 1 h later showed HCT of 21. Three units of PRBCs and two units of fresh frozen plasma (FFP) were transfused. The MAP increased to 100 mmHg and phenylephrine was weaned off. One hour later the HCT was 26. An additional three units of PRBCs and two units of FFP were given.

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The patient had bedside surgery on the right thigh later that morning. The MAP was in the 95 mmHg, the HR was in the 110 bpm, and the HCT was 31. Throughout the morning the partial thromboplastin time (PTT) was in the target range of 40–60 s.

Lessons Learned

L-1. What is hemophilia A and B?

(a) Hemophilia is a defect in the propagation phase of coagulation and is associated with a prolonged activated partial thromboplastin time and normal prothrombin time:

(i) Hemophilia A (congenital factor VIII deficiency) is caused by a mutation in the factor VIII gene.
(ii) Hemophilia B (congenital factor IX deficiency) is caused by a mutation in the factor IX gene.
(iii) In general, clinical severity of hemophilia is correlated with factor activity level:

1. Severe hemophiliacs have factor activity levels <1 % of normal. These patients are usually diagnosed in childhood because of frequent, spontaneous bleeding into joints, muscles, and organs.
2. Moderate disease is seen with factor activity levels 1–5 % of normal. These patients are at increased risk of hemorrhage with surgery or trauma but have fewer spontaneous hemarthroses or hematomas.
3. Mild hemophiliacs have factor activity levels >5 % of normal. These patients may go undiagnosed well into adult life but are at risk for excessive bleeding with major surgical procedures [1].

L-2. What are the most likely causes of DIC in this patient?

(a) The extensive tissue damage from the patient's thigh abscess and his multiple recent surgeries are a likely cause of DIC. In addition, endotoxins such as those released from gram-negative infections can cause DIC.

L-3. Describe the basis for heparin use in the management of DIC

(a) Heparin binds to and activates antithrombin III which then inactivates thrombin and other clotting factors, leading to decreased coagulation. Heparin therapy must be used cautiously in patients with DIC given that these patients experience a consumption of clotting factors which increases their propensity to bleed.

L-4. What are the definitions of infection, bacteremia, SIRS, sepsis, severe sepsis, septic shock and multiorgan dysfunction syndrome?

(a) See Table 22.1.

Table 22.1 Definitions

Infection	Microbial phenomenon characterized by an inflammatory response to the presence of microorganisms or the invasion of normally sterile host tissue by those organisms
Bacteremia	The presence of viable bacteria in the blood
SIRS (systemic inflammatory response syndrome)	The systemic inflammatory response to a variety of severe clinical results. The response is manifested by two or more of the following conditions: 1. T >38°C or <36°C 2. HR >90 bpm 3. RR >20 breaths/min or Pa CO_2 <32 mmHg 4. WBC >12,000 cells/mm^3, <4,000 cells/mm^3, or >10 % immature (band) forms
Sepsis	The systemic response to infection. The systemic response is manifested by two or more of the following condition as a result of infection: 1. T >38°C or <36°C 2. HR >90 bpm 3. RR >20 breaths/min or Pa CO_2 <32 mmHg 4. WBC >12,000 cells/mm^3, <4,000 cells/mm^3, or >10 % immature (band) forms
Severe sepsis	Sepsis associated with organ dysfunction, hypoperfusion, or hypotension. Hypoperfusion and perfusion abnormalities may include, but are not limited to, lactic acidosis, oliguria, or an acute alteration in mental status
Septic shock	Sepsis with hypotension, despite adequate fluid resuscitation, along with the presence of perfusion abnormalities that may include, but are not limited to, lactic acidosis, oliguria, or an acute alteration in mental status. Patients who are on inotropic or vasopressor agents may not be hypotensive at the time that perfusion abnormalities are measured
Multiple organ dysfunction syndrome	Presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention

Adapted from [2]

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Chapter 23
Blood Transfusion and the Jehovah's Witness Patient

Ankur P. Patel

The patient is a 38-year-old female, G4 P3003, who is a Jehovah's Witness (**L-1**). She was admitted to L&D with spontaneous rupture of membranes and a term pregnancy complicated by a fetus which is large for gestational age and thrombocytopenia (111,000) and was being worked up for the possibility of developing preeclampsia.

The patient was started on an oxytocin infusion to augment her labor. While on oxytocin, her blood pressures were noted to be rising. The patient was ruled out for preeclampsia with urinalysis, liver function tests, and platelet count. At 7:38 a.m., it was noted the fetal heart rate tracing had minimal variability. There were also noted to be early and variable decelerations on the tocodynamometer. This diminished variability continued and Pitocin was subsequently stopped (**L-2**). At this point, the patient was in active phase of labor and was beginning to experience pain.

An anesthesiologist was called to evaluate the patient for epidural placement at 12:39 p.m. Her past medical history was insignificant; she took no medications and had three children at home, who were all vaginally delivered without incident.

Her vitals were 68 kg, 5′2″; BP 150/90 mmHg; HR 63 bpm; temp 98.2 F; respiratory rate 33 per minute; and SpO_2 99 % on room air. She had a Mallampati Class II airway, with 5-cm thyromental distance, intact dentition, and the ability to prognath.

After review of her chart, the following statement was recorded in a progress note from the same day:

"JW: refusal signed and scanned in media and in chart. Re-confirmed upon admission. Refuses all." This discussion had taken place between the primary team and was not reiterated with the consulting anesthesiologist upon interview prior to epidural placement, since it was documented in the chart (**L-3**).

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A lumbar epidural was placed without incident and was connected to the patient-controlled epidural anesthesia pump. The pump was programmed to run 0.1 % bupivacaine with 2 mcg of fentanyl/cc at 10 cc/h with q 20-min availability of a patient-controlled 5 cc bolus.

At 1:24 p.m., the anesthesiologist was paged "stat section." Upon arrival to the operating room, the patient was already on the table wearing a simple face mask flowing 10 L/min of oxygen, and her abdomen was prepped for surgical incision with gowned and gloved obstetricians standing by (**L-4**). Standard ASA monitors were applied, the patient was connected to the circle system for preoxygenation, and the attending anesthesiologist was immediately notified. An RSI induction was performed with 200 mg propofol and 100 mg succinylcholine with cricoid pressure after five vital capacity breaths. A Macintosh 3 blade was used to achieve a grade I view, and 6.0-mm-internal-diameter ETT was placed at 18 cm at the teeth with subsequent bilateral breath sounds and positive end-tidal CO_2. General inhalational anesthesia was maintained with 50 % inspired oxygen concentration, 50 % nitrous oxide, and 1 % end-tidal sevoflurane. An orogastric tube, a bite block, and a temp probe were placed.

The fetus was delivered and oxytocin 40 units/1 L was given through the existing 18-gauge peripheral IV. After observing the uterus over the drapes, it was noted that there was no tone and that bleeding was ongoing. A 16-gauge peripheral IV was placed into the LUE in a separate vein, and a left radial 20-gauge arterial line was placed.

The patient was kept hemodynamically stable with infusion of 6 L of crystalloid 1 L of Hespan, a phenylephrine drip at 100 mcg/min, and intermittent boluses of vasopressin and phenylephrine. Blood gasses revealed a pH of 7.23 with hemoglobin and hematocrit values of 4.5 g/dL and 13 %, respectively, with a base deficit of 9.8 (**L-5**). Initial coagulation labs revealed an INR of 1.1, PTT of 32.5, and fibrinogen of 257 at 1:55 p.m.

This was communicated to the obstetricians, at which point they asked if the patient should be taken to IR for balloon placement into the uterine arteries to stop the hemorrhage. Also, they inquired if a cell saver should be brought into the room. The anesthesiology team informed them that these maneuvers would be futile since time was becoming a factor.

After 2+ L of blood loss and continued uterine atony, intra-myometrial oxytocin (10 units), Hemabate, and rectal misoprostol were administered. This was unsuccessful in improving uterine tone. A B-Lynch suture was performed in the uterus. Tone still did not improve and there was ongoing hemorrhage so the decision to perform a hysterectomy was made. At around 3:00 p.m., the attending anesthesiologist went to speak with the father of the patient's child. He told the father that if the patient does not receive blood and other products, she will likely expire. The decision for massive transfusion protocol was made by the father of the child and carried out. The obstetricians were able to slow down the bleeding by isolating and clamping the uterine vessels. They then packed the wound and allowed the anesthesiologists to resuscitate the patient with bank blood before completing the hysterectomy.

A total of 8 units of RBC, 7 of FFP, and 3 of platelets were given, after which the vasopressor requirements disappeared. The uterus was removed and the wound was closed. During closure of the surgical wound, the nurse informed the anesthesiologist that the father of the baby who gave consent for transfusion was not married to the patient.

At 4:00 p.m., repeat coagulation labs showed an INR of 1.3, a PTT of 41.9, and a fibrinogen of 125.

Final blood gas revealed a pH of 7.44, hemoglobin of 9.8 g/dL, and hematocrit of 29 % with a base deficit of 2.3.

The patient was taken to the ICU intubated. On POD 1 the patient's sedation was held and she was following commands with intact mentation. She then consented to further blood transfusion. She was subsequently resuscitated in the ICU and discharged home when stable with her new child several days later.

This case certainly highlights the importance of discussing transfusion goals with a Jehovah's Witness patient prior to emergent events and reminds us to anticipate these emergent events in seemingly healthy patients.

Lessons Learned

L-1: **What is a Jehovah's Witness**?

Jehovah's Witnesses are members of a sect of Christianity who strictly and fundamentally believe that the Bible is the literal word of the Christian god, whose name is Jehovah. In this religion, the only way to achieve salvation and eternal life is to follow the directions outlined in the Bible. Germaine to this case discussion is the administration of blood and blood products. According to the Jehovah's Witness belief, the Bible forbids taking blood into the body through the mouth or veins [1]. There are three scriptures in the Bible that are referenced as the basis for this belief:

> Genesis. 9:3, 4
>
> 4 Only flesh with its soul—its blood—YOU must not eat. 5 And, besides that, YOUR blood of YOUR souls shall I ask back. From the hand of every living creature shall I ask it back; and from the hand of man, from the hand of each one who is his brother, shall I ask back the soul of man.

> Leviticus. 17:14
>
> 10 "'As for any man of the house of Israel or some alien resident who is residing as an alien in YOUR midst who eats any sort of blood, I shall certainly set my face against the soul that is eating the blood, and I shall indeed cut him off from among his people. 11 For the soul of the flesh is in the blood, and I myself have put it upon the altar for YOU to make atonement for YOUR souls, because it is the blood that makes atonement by the soul [in it]. 12 That is why I have said to the sons of Israel: "No soul of YOU must eat blood and no alien resident who is residing as an alien in YOUR midst should eat blood."
>
> 13 "'As for any man of the sons of Israel or some alien resident who is residing as an alien in YOUR midst who in hunting catches a wild beast or a fowl that may be eaten, he must in that case pour its blood out and cover it with dust. 14 For the soul of every sort of flesh is its blood by the soul in it. Consequently I said to the sons of Israel: "YOU must not eat the blood of any sort of flesh, because the soul of every sort of flesh is its blood. Anyone

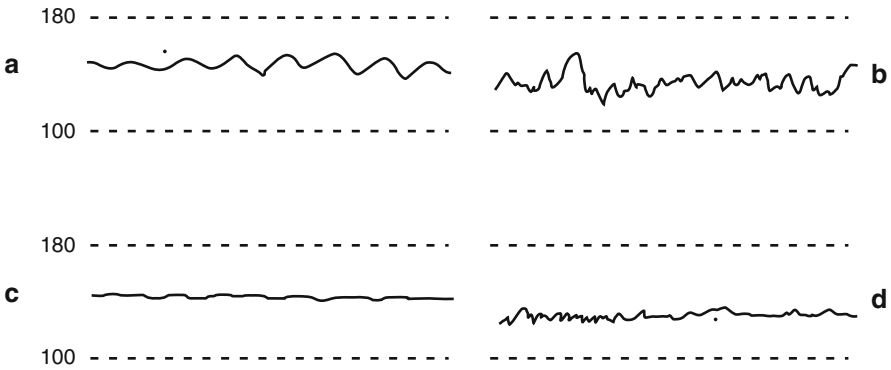

Fig. 23.1 Components of heart rate variability from internal monitor. (**a**) Long-term variability without short-term variability. (**b**) Long- and short-term variability. (**c**) No long- or short-term variability. (**d**) Short-term variability without long-term variability (Adapted from Zanini et al. [7])

> eating it will be cut off." 15 As for any soul that eats a body [already] dead or something torn by a wild beast, whether a native or an alien resident, he must in that case wash his garments and bathe in water and be unclean until the evening; and he must be clean. 16 But if he will not wash them and will not bathe his flesh, he must then answer for his error.'"
> Acts 15:28, 29
> 28 For the holy spirit and we ourselves have favored…abstaining from things sacrificed to idols and from blood and from things strangled and from fornication.

According to these references, if a member of this religion is in violation of these laws, they will be "cut off from among his people," which is meant to mean they will not be granted salvation and eternal life.

Simply put, transfusing a Jehovah's Witness patient with blood will deprive them of an eternal life of salvation and grace, and that is an unimaginably high price to pay for a mistake made by the patient or a healing doctor caring for the patient.

L-2. What is the differential diagnosis of lack of variability in fetal heart rate?
The fetal heart rate is considered to have variability in two forms: beat-to-beat and long-term variability. In beat-to-beat variability, there are changes in the fetal heart rate over two or three beats which is thought to result from normal parasympathetic tone in the fetus. Long-term variability is seen on a fetal heart rate tracing as sine waves occurring three to six times per minute and changing by at least 6 bpm [2]. The differential diagnosis of loss of variability includes fetal hypoxia, CNS disease, medication effects (i.e., opiates, atropine), and fetal sleep state (Fig. 23.1).

L-3. What should the anesthesiologist discuss with a Jehovah's Witness patient regarding transfusion?
For the anesthesiologist, it is important to have a discussion with the patient regarding their beliefs regarding blood transfusion. Some Jehovah's Witnesses will accept albumin, cell saver, blood, or other products depending on their personal beliefs and preferences. Others will only accept crystalloid or colloid from nonliving sources (i.e., Hespan). When having this discussion, it's important to talk with the patient

alone and without other family or clergy members present, since this may coerce the patient into accepting or refusing transfusions.

There are three types of Jehovah's Witnesses for anesthesiologists to concern themselves with:

1. The patient is a competent adult capable of consent/non-consent.
2. Parents of children who are Jehovah's Witnesses cannot martyr or make martyrs of their children.

   If the patient is a minor, a court order can be obtained in every local in the United States to give transfusions in cases where life-threatening hemorrhage is anticipated or is occurring.
3. The patient is pregnant. The law is less clear about transfusing a pregnant patient for the purpose of fetal resuscitation; the fetus cannot obviously consent.

If it has already been documented in the medical record that other physicians have come to an agreement with the patient pertaining to decisions regarding transfusion of blood/blood products, it is prudent for the new incoming members of the care team to individually reaffirm the previous agreement, even if the discussion is repetitive.

L-4: **Decreasing the need for transfusion in the Jehovah's Witness patient**

A. Preoperatively

1. Iron supplementation. If a patient is presenting for an elective case, the patient may be started on iron supplementation (how much and how long prior to case).
2. Erythropoietin may also be administered. Both iron and erythropoietin will assist the patient in making endogenous hemoglobin, which will confer oxygen-carrying capacity for the patient. Recall the arterial content of oxygen is defined by the equation:

$$CaO_2 = 1.34 \times \text{hemoglobin concentration} \times SaO_2 + (0.003 \times PaO_2$$

From this equation, it becomes clear that increasing the hemoglobin concentration can protect against tissue dysoxia (the point where tissue oxygenation becomes supply dependent) in the face of hemorrhage.

B. Intraoperatively

There are several intraoperative techniques to minimize blood loss:

1. Positioning
   If positioning is possible, it may be prudent to place the patient in Trendelenburg or reverse Trendelenburg (depending on the operative site) to decrease venous pressure leading to back bleeding.
2. Use of a tourniquet
   If the operative site is an extremity, a tourniquet may be suggested.
3. Deliberate hypotension
   Controlling blood pressure can also limit blood loss by reducing the pressure head for arterial bleeding at the operative site.

4. Hypothermia
   Reducing body temperature will decrease oxygen demand in various organ systems. For example, cerebral metabolic oxygen consumption ($CMRO_2$) drops by 7 % with each drop in degree Celsius. This technique should be employed with caution since coagulopathy may develop and would obviously be counterproductive in situations where there is uncontrolled hemorrhage and potential for DIC such as in this case.
5. Hemostatic agents
   Desmopressin administration can be considered to improve platelet function. Antifibrinolytics may also be given to improve coagulation.
6. Acute normovolemic hemodilution (ANH)
   This procedure involves removing blood from the patient at the beginning of the case in a bag containing anticoagulant (usually CPDA) and replacing the blood with crystalloid and or colloid solution to maintain isovolemia. At a later point, the removed blood is given back to the patient as it contains clotting factors and hemoglobin from the patient. Care must be taken in Jehovah's Witnesses to ensure that the circuit is closed and that the blood does not leave this closed circuit as this violates their religious laws.

Using this technique, the following equation is useful in calculating the desired amount of blood to be removed:

*A*mount of blood to be removed = estimated blood volume (L) × (starting hematocrit – desired [end] hematocrit)/average hematocrit of starting and end [6].

For example:

If the patient is a 70 kg man, the estimated blood volume obtained from a nomogram is 75 mL/kg. This is equal to 75 mL/kg × 70 kg = 5,250 mL of blood volume.

If starting hematocrit is 40 % and the desired is 30 % (see Fig. 23.1), the average will be 35 % (40 + 30/2). Plugging these values into the formula will give the following:

Blood to remove = 5,250mL × (40–30%/35%) = 1,499mL of blood to remove.

This will need to be replaced with an equal amount of colloid or three to four times the amount of crystalloid to maintain isovolemia.

Contraindications to this technique include preexisting anemia (hemoglobin less than 9 g/dL), cardiac disease in which increased cardiac output may be detrimental, and presence of active bleeding or coagulopathy [3, 4]. A hematocrit of approximately 30 % is a reasonable goal (see Fig. 23.1). This results in optimal oxygen transport secondary to the best relationship of Q_t (Cardiac Output) × C_aO_2 (arterial oxygen content) (see Fig. 23.1). When the hematocrit drops to lower levels, however, the supply–demand relationship of oxygenation becomes unfavorable. An increase in cardiac output that results from acute anemia when hematocrit falls below 30 % may result in increased myocardial oxygen consumption from tachycardia, less myocardial oxygen supply from a decreased diastolic time, and diminished oxygen-carrying capacity from the loss of hemoglobin to the heart and all tissues (see CaO_2 equation).

Table 23.1 Glossary of terms used for oxygen being brought to the tissues and used by the tissues

Oxygen being brought to the tissues	Oxygen being used by the tissues
Delivery	Utilization
Transport	Extraction
Supply	Uptake
	Metabolism

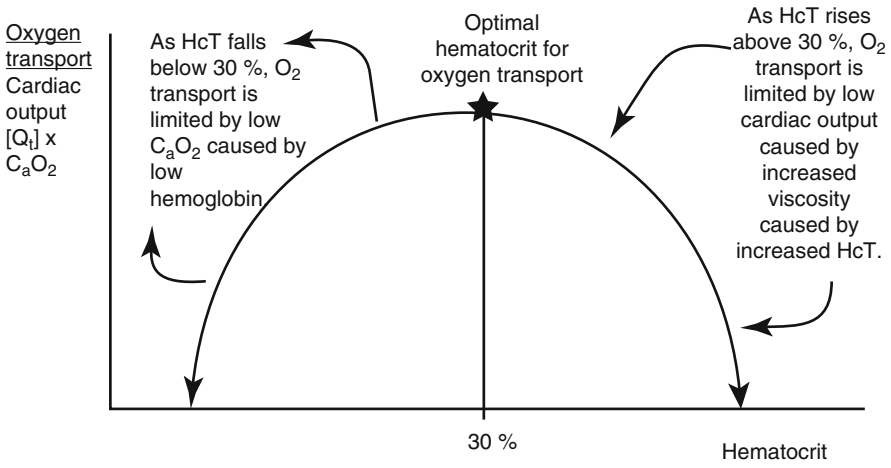

Fig. 23.2 Oxygen transport to the tissues is maximal at a hematocrit of 30 %. *Q* cardiac output, *HcT* hematocrit

L-5. The physiology of anemia

See Table 23.1 for a glossary of terms used in L-5.

Patients who are anemic suffer from imbalance of oxygen supply and demand. As the hematocrit falls below 30 %, O_2 transport/supply ($Q_t \times C_aO_2$) to the tissues decreases because C_aO_2 $[(1.34 \times Hb \times S_aO_2) + (0.003 \times P_aO_2)]$ decreases. O_2 supply/delivery will remain constant for a while because the tissues can increase their extraction of oxygen by decreasing C_vO_2 (Fig. 23.2). When maximal decrease in the C_vO_2 is reached, oxygen utilization becomes supply dependent (dysoxia occurs), and the tissues will rely on anaerobic metabolism to produce ATP (see Fig. 23.3). This utilization of anaerobic metabolism results in a lactic acidosis and shifts the hemoglobin-oxygen dissociation curve to the right, promoting release of oxygen to the peripheral tissues (see Fig. 23.2).

Furthermore, normally 25–35 % of the oxygen bound to hemoglobin is extracted from hemoglobin for tissue metabolism. This explains why normal mixed venous saturation sample from the pulmonary artery is 65–75 % (100 % saturated arterial blood – 25–35 % extraction). In profoundly anemic patients, this extraction ratio may be above 35 %, resulting in lower saturated blood returning to the lungs for oxygen (i.e., mixed venous O_2 saturation less than 65 %). In patients with a fixed shunt, this desaturated venous blood will mix with oxygenated end-pulmonary

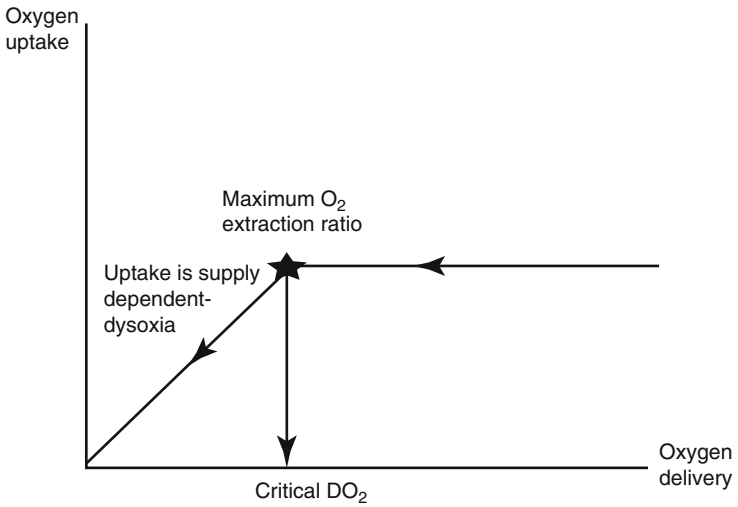

Fig. 23.3 As delivery of oxygen (DO_2) decreases, O_2 extraction or uptake remains constant until a critical point is reached. This implies that the oxygen extraction from C_vO_2 is increasing as delivery falls and the C_vO_2 also decreases. When the oxygen extraction from blood reaches a critical maximal point and the decrease in C_vO_2 is maximal, oxygen utilization becomes O_2 delivery dependent. If O_2 delivery continues to fall, the tissues must use anaerobic metabolism to generate energy needs, which will result in lactic acidosis and, in the long run, death. Note DO_2 delivery of oxygen and can be used interchangeably with oxygen supply or oxygen transport for the purpose of this writing. Also, oxygen uptake on the *Y*-axis can represent oxygen consumption, oxygen metabolism, oxygen extraction, or oxygen utilization for the purposes of this writing (Adapted from [5])

capillary blood leaving the lungs and cause a reduction in the arterial saturation (see Fig. 23.4). This will lead to further oxygen debt in the tissues (see Fig. 23.2). After this point tissue dysoxia (see Fig. 23.2) will occur and cellular injury and death will take place, ultimately leading to demise unless the supply–demand imbalance is corrected.

There are several more important aspects to the physiology of anemia. First, when the hematocrit falls below 30 %, there is a cardiovascular response of tachycardia and increased cardiac output to meet metabolic demands. Tachycardia will increase myocardial oxygen consumption as well as decrease myocardial oxygen supply since diastolic time is decreased. Second, minute ventilation will increase in patients who are spontaneously ventilating. Third, dilutional coagulopathy can become a threat, resulting in further bleeding.

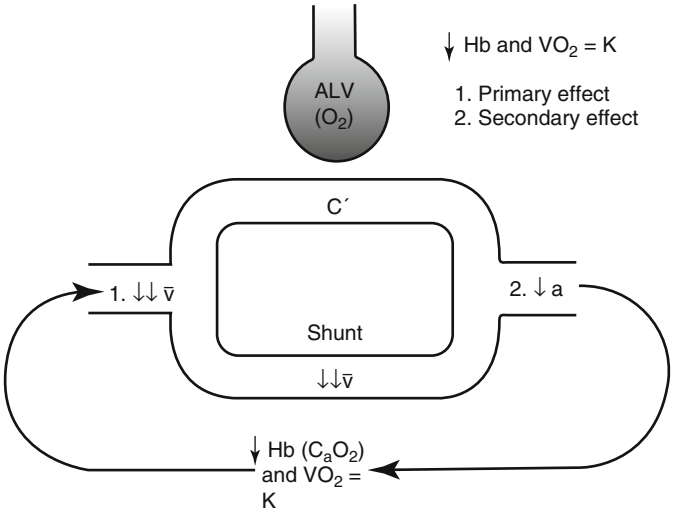

Fig. 23.4 Effect of a decrease in Hb with a constant (*K*) oxygen consumption of (VO_2) on mixed venous and arterial oxygen content. Mixed venous blood (*V*) either perfuses ventilated alveolar (*ALV* O_2) capillaries and becomes oxygenated end-pulmonary capillary blood (*c'*) or perfuses whatever true shunt pathways exist and remains the same in composition (desaturated). These two pathways must ultimately join together to form mixed arterial (*a*) blood. If Hb decreases and oxygen consumption (VO_2) remains constant (*K*), the tissues must extract more oxygen per unit volume of blood than under normal conditions. Thus, the primary effect of a decrease in Hb and a constant VO_2 is a decrease in a mixed venous oxygen content. The mixed venous blood with a decreased oxygen content must flow through the shunt pathway as before (which may remain constant in size) and lower the arterial content of oxygen. Thus, the secondary effect of a decrease in Q_t or an increase in VO_2 is a decrease in arterial oxygen content (Adapted from Hagberg [8])

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Chapter 24
Cardiac and Pulmonary Contusions

Bahareh Khatibi

The patient is a 17-year-old male (60 kg, 69 in) who was the unrestrained passenger in a 60-mph car-vs-bulldozer motor vehicle accident. He presented with a Glasgow Coma Score of 14 and had loss of consciousness at the scene. CT of the chest showed a right pneumothorax and hemothorax, bilateral lung contusions (**L-1**), and fluid surrounding the heart. CT of the spine showed L1–L4 burst fractures as well as a L4 spinous process fracture. Head CT was normal. The patient was intubated and had a right-sided chest tube placed. He was placed on a rotating bed (**L-2**). Over the next several days in the ICU, the patient developed syndrome of inappropriate antidiuretic hormone secretion (SIADH) (**L-3**) as well as ST changes and elevated cardiac enzymes. Echocardiography revealed probable cardiac contusion (**L-4**).

The patient was extubated on post-injury day 4. On post-injury day 8, he was scheduled for lumbar spine surgery. Preoperatively, the patient continued to have SIADH with a Na of 129 and required oxygen by nasal cannula. Induction of anesthesia with lidocaine, etomidate, sufentanil, and rocuronium was uneventful. The patient was placed in the prone position and surgery proceeded uneventfully. Upon completion of the surgery, the patient was allowed to breathe spontaneously and was positioned supine in the ICU bed. Immediately upon extubation, the patient became tachypneic and agitated. He indicated that he had pain, but 10 mcg of sufentanil did not calm him. His breathing became rapid and he complained of inability to breathe. His right chest did not appear to move as well as the left. His oxygen saturation began to drop. A chest x-ray (CXR) was obtained which revealed a right pneumothorax and hemothorax. A new chest tube (**L-5**) was placed that drained serosanguinous fluid.

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Lessons Learned

L-1. What is the natural history of a lung contusion? How does fluid resuscitation affect respiratory function in a patient with lung contusions?

(a) The clinical manifestations of lung injury after thoracic trauma may be insidious, with nonspecific respiratory difficulty and little on CXR in the first few hours; associated changes may not be visible on plain CXR until 4–6 h after injury, presumably because IV fluids may preferentially increase the development of the lesion (bruise) that is already there by causing edema within the lesion. Hemorrhage into the lung leads to pathophysiologic changes that worsen for 24–48 h and then generally resolve by 7 days after injury. In general, the respiratory derangements resolve within 3–5 days, but delayed deterioration may occur. The long-term consequences of pulmonary contusion have not been clearly defined, but can include acute respiratory distress syndrome (ARDS), pneumonia, and empyema [1].
(b) Fluid management in patients with pulmonary contusions is a controversial issue. It has been suggested that overzealous fluid administration in these patients may lead to "wet lung" [2], but relatively few human clinical trials have addressed the impact of resuscitation on pulmonary contusions [1]. Judicious fluid administration is recommended.

L-2. Why are rotating beds utilized? What is the theory behind their use? What is the evidence that they work?

(a) Rotating patients with the uninjured hemithorax in the dependent position can improve oxygenation. This may be due to increasing perfusion to the uninjured lung and decreasing perfusion (and edema) to the injured lung, thus optimizing the ventilation/perfusion relationship [1]. The use of rotating beds has been shown to significantly reduce the incidence of pneumonia and the number of ventilator days after major thoracic trauma [3].

L-3. What is SIADH? How is it diagnosed? How is it treated?

(a) SIADH is hypotonic hyponatremia that is caused by various factors including central nervous system pathology, antidiuretic hormone (ADH)-secreting cancers, and multiple medications.
(b) SIADH is diagnosed by hyponatremia, inappropriate natriuresis, and persistent hypervolemia.
(c) Treatment includes fluid restriction to 1,200–1,800 mL/day and hypertonic saline for symptomatic patients. Medications such as demeclocycline can also be used to treat the chronic hyponatremia of SIADH.

L-4. How are cardiac contusions diagnosed? How are they managed?

(a) There is no gold standard test for the diagnosis of cardiac contusion, and its manifestation varies widely, making the diagnosis a challenge. Three main examinations that should be performed are EKG, troponin I measurement, and TEE [4].
(b) See Fig. 24.1.

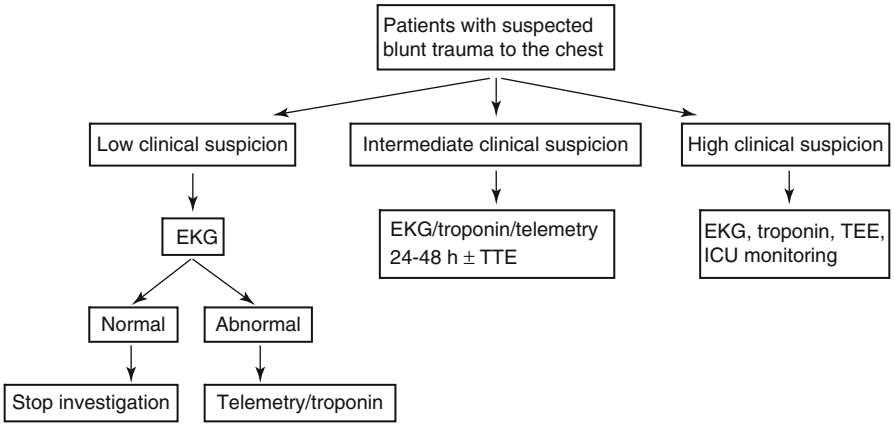

Fig. 24.1 Algorithm in the management of patients with suspected blunt trauma to the chest

L-5. Explain how chest tubes work. What observations could have been made to suggest that the chest tube in this case was not functioning properly?

(a) See Fig. 24.2. Chest tubes are made up of three compartments connected to a drain that is located in the pleural space. The first compartment is a collection chamber, the second compartment serves as a one-way valve, and the third compartment controls the amount of negative pressure that the suction can generate. The collection compartment should be below the level of the patient at all times to prevent the entry of fluid from the collection compartment and chest tube back into the chest. Air is expelled through the drain during exhalation, during spontaneous ventilation, and during inhalation with positive pressure ventilation. The drain will bubble continuously if there is a large leak. If there is no air leak, the fluid will move gently in the drainage tube during quiet, spontaneous breathing.

(b) Observation of synchronous respiratory motion and water seal bubbles suggests the tube is still functioning in the pleural space and all connections are tight. If a leak in the chest tube is suspected, sequential clamping with distal suction before and after the spot in question should be performed. Bubbling air through the water seal when the drainage system is clamped just distal to the point in question, which stops when the clamp is placed proximal to that point, identifies the site of leakage.

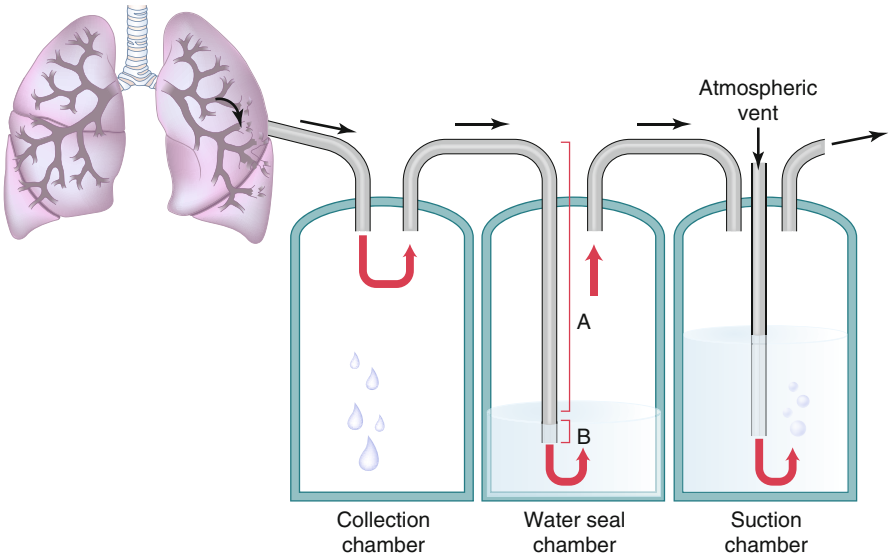

Fig. 24.2 Schematic diagram of functional anatomy of chest tube drainage system. Chest tubes are made up of three compartments connected to a drain in the pleural space. The first compartment is a collection chamber where the amount of draining fluid can be measured. The second compartment is an underwater seal that serves as a reliable, one-way valve that allows air from the pleural space to enter the drainage system, but does not allow air from the drainage system to reenter the pleural space on the next inhalation. *A* determines the resistance to backward flow; *B* determines resistance to forward flow. The third compartment controls the amount of negative pressure that the suction can generate. Excessive negative pressure in the third compartment is neutralized via an atmospheric vent

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Chapter 25
Intraoperative Coagulopathy

Ankur P. Patel

The patient is a 21-year-old female with past medical history significant for hypertension, idiopathic renal failure, epilepsy, and spina bifida who presents as a recipient of renal transplant with neobladder formation. Her past surgical history involved a number of spine surgeries without any complications. On exam, she weighs 39 kg and is 142 cm in height. Her preoperative labs reveal a potassium of 5 meq/dL, blood urea nitrogen (BUN) of 58 (units), HCO_3 of 23, a hematocrit of 33 %, and platelet count of 147,000. Her coagulation labs were prothrombin time (PT) 10.6 with International Normalized Ratio (INR) 1.1 and partial thromboplastin time (PTT) 27.6 (**L-1**).

Due to difficulty obtaining blood draws as well as placing an IV, the decision was made to utilize one of the ports (red, with 1.3-cc priming volume from port to tip) of her indwelling venous two-lumen hemodialysis vascular catheter (Vas-Cath) for access. Prior to utilizing this port, 5 mL of blood was drawn back, and 10 mL of additional blood was drawn for a repeat set of labs in the preoperative, holding area (**L-2**). After this draw, 3 mL of nonconcentrated 1,000 units/mL of heparin was used to flush and lock the port (**L-6**). After appropriate labs confirmed a match with the kidney, the patient was taken to the operating room some hours after this draw and induced utilizing the red port after the same procedure to flush out the heparin was repeated. An 18-gauge peripheral IV was placed in the left hand after induction. The patient was maintained on one minimum alveolar concentration (MAC) of isoflurane in oxygen and air throughout the case.

Immediately upon skin incision, the surgeon noted excessive oozing of blood in the field and requested that the anesthesiologist prepare to have approximately three units of packed red blood cells brought to the room. This oozing continued persistently throughout the case, and two units of packed red blood cells were

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administered after an estimated blood loss of 400 mL and a hematocrit of 25 %. The patient remained hemodynamically stable throughout the 6-h surgery until closure. Immediately after closure of the abdomen, the patient became hypotensive and an additional unit of packed red blood cells was administered with 1 L of albumin. A discussion between the anesthesiologist and the surgeons regarding re-exploration of the abdomen took place, and the decision to observe the drainage output and resuscitate the patient was made.

The patient was taken to the ICU intubated. In the immediate hour following surgery, the drainage output was 250 mL of frank blood and two additional units of packed red cells were given over the next several hours. After failed attempts at a radial arterial line, a femoral arterial line was placed under ultrasound guidance, and a full lab panel was obtained as well as an activated clotting time (ACT). The ACT was 305 s (normal 120–130). Coagulation panel revealed a PT 13.6 s, INR 1.4, and PTT >300 s. Two units of fresh frozen plasma and 50 mg of protamine were administered and labs were repeated: PT 11.8 s, INR 1.2, PTT 28 s, fibrinogen 171, and D-dimer 2,397 ng/mL (normal <250 ng/mL) (**L-4**). The drain output decreased to minimal over the next 4 h. The patient was extubated later in the day without any other complications (**L-5**).

Lessons Learned

L-1. What is the volume capacity of the ports on the two-lumen hemodialysis catheter (Vas-Cath)?

Figure 25.1 shows that there are three ports in this catheter, two identical large ports and a smaller proximal port in the middle. In Fig. 25.2, the three lumens are all separated from one another. In Fig. 25.3, the distal lumen has a priming volume of 1.4 mL and the proximal lumen has a priming volume of 1.3 mL. Both of these lumens are self-contained and isolated from each other so that there is no mixing of contents from each lumen.

Figure 25.4 illustrates the heparin dose–response curve. The following procedure is done to construct a dose–response curve for a patient. This dose–response curve is then used to predict the amount of additional heparin to give and to calculate protamine doses for reversal of anticoagulation.

1. Plot the initial ACT on the *x*-axis (A).
2. Plot the ACT after heparinization (B).
3. Draw the line defined by these two points.
4. If additional anticoagulation is needed, find the desired ACT on that line. The amount of additional heparin needed is the difference on the *y*-axis between the present ACT and the desired ACT.
5. If the third point does not lie on the original line, a new line is drawn originating from the baseline ACT and passing midway between the other two points.
6. For reversal of anticoagulation, the protamine dose is based on the remaining heparin activity, estimated to be the heparin dose corresponding to the latest ACT on the dose–response line.

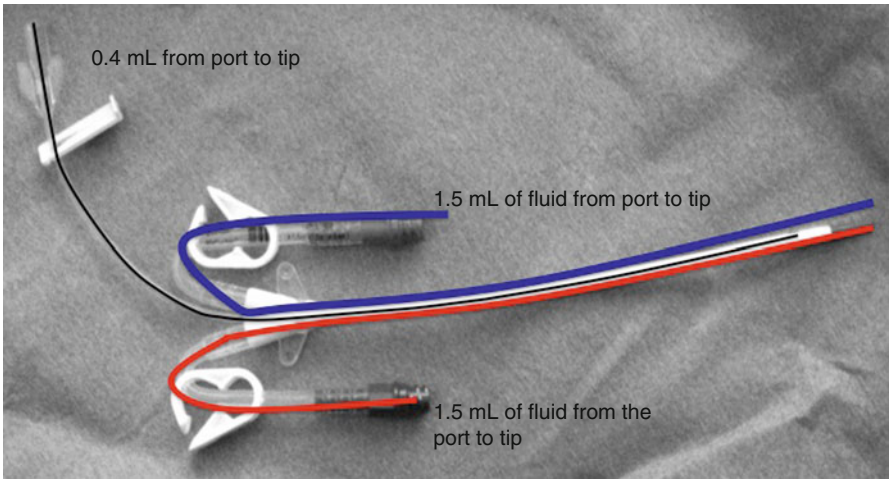

Fig. 25.1 Covidien™ acute triple-lumen dialysis catheter. The volume of dead space (priming volume) from the injection ports to the tip of the catheter is demarcated by the *red and blue lines*. It represents 1.5 mL of fluid through each port which does not mix with any other port's contents along the entire length of the catheter (see Fig. 25.2). The *black line* demarcates the amount of dead space (priming volume) from the port to the tip of the catheter. The priming volume in all three ports is self-contained and isolated from one another (see Fig. 25.2)

L-2. How much fluid should be drawn back when attempting to clear a line of dead-space fluid?

This depends on how much dead space (priming volume) you are trying to clear. For example, in this case, the amount of priming volume in the dialysis catheter to clear starting from the port of the catheter to the tip of the catheter is equal to 1.5 mL of fluid (See Fig. 25.1) for the Covidien catheter. It is 1.3 mL from the red port and 1.4 mL from the blue port of an Arrow Two-Lumen Hemodialysis Catheter. When removing one kind of fluid (heparin) from one small space (one of the lumens of the Vas-Cath) by flushing into it fluid (blood) from a second large space (the vascular system), there is a wash-in (of blood) and washout (of heparin) process that occurs exponentially in the small space (see Table 25.1).

Table 25.2 depicts the exponential decrease in concentration of heparin in the port-to-tip dead space of the catheter as a function of the number of dead-space volumes of blood is aspirated in this dead space by a syringe attached to the dead space. As fluid is aspirated from the catheter, the first 1.5 mL (which represents the total dead-space or priming volume of the catheter) corresponds to removal of 63 % of the dead-space fluid (priming volume), leaving 37 % of the fluid in the catheter. As additional fluid is removed, one can see that the amount of dead-space fluid remaining in the catheter exponentially decreases.

L-3. What coagulopathy should be expected of a patient with renal failure presenting for surgery?

Patients with renal failure, who are receiving regular dialysis, should be expected to have an increase in bleeding time due to chronic uremia and defective platelets.

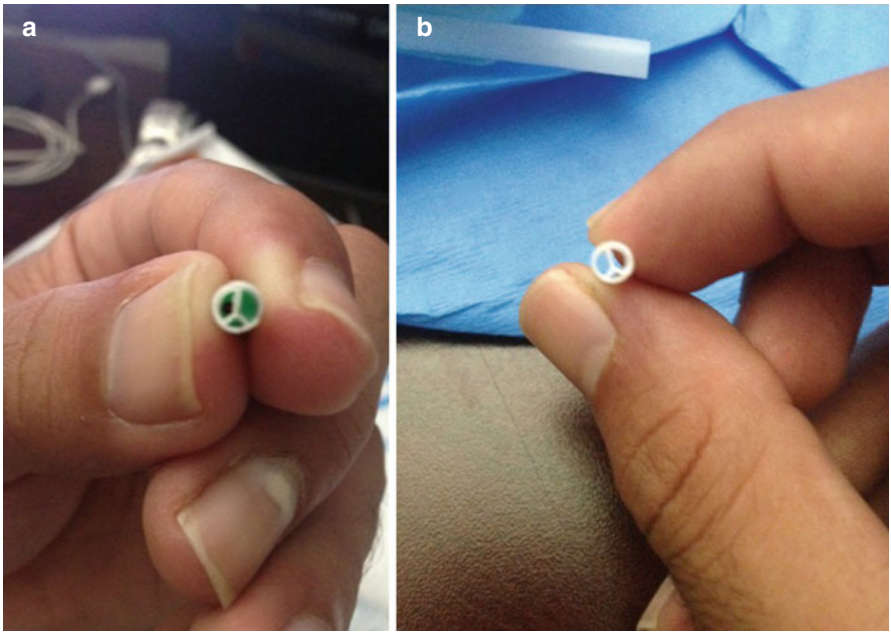

Fig. 25.2 Cross-section view of the Covidien™ acute triple-lumen hemodialysis catheter. (**a**) This view is a cross section near the distal tip of the catheter. There is a green obturator (plastic block) near the tip which blocks off the flow in two of the three lumens. Therefore, fluid exits on the side of the catheter through orifices that are just proximal to the obturator block in these two lumens. The fluid in the lumen on the left, the remaining third lumen at the 9 o'clock position, flows through the distal tip. (**b**) This is the cross section of the middle of the catheter showing the segregation of the priming volume in the three lumens along the length of the catheter

Also, they may have low levels of von Willebrand factor (VWF), which reduces the half-life of factor VIII. Moreover, since they are receiving dialysis through catheters that may be indwelling for long periods of time, the patients are predisposed to iatrogenic heparinization from the "locking" of their access catheters with heparin; in other words, a caregiver may make a mistake at any time and inject something through a heparin-filled ("locked") Vas-Cath lumen [1].

L-4. What is the significance of the D-dimer level?

A D-dimer level is a measure of thrombus formation and breakdown (fibrin split products). A value above normal limits means that the patient is forming clot and lysing the clot faster than any normal process would allow for. It is often drawn to confirm a diagnosis of thrombosis or DIC. If the probability of thrombosis (pulmonary embolus or deep venous thrombosis) is low to intermediate, then a D-dimer of zero rules the diagnosis of thrombosis out. In this regard, it is a sensitive test. If the probability is high and the D-dimer is greater than normal limits (250 ng/mL), another confirmatory test will need to be done (such as pulmonary angiography or lower-extremity ultrasound) since it is not a very specific test. It only tells the

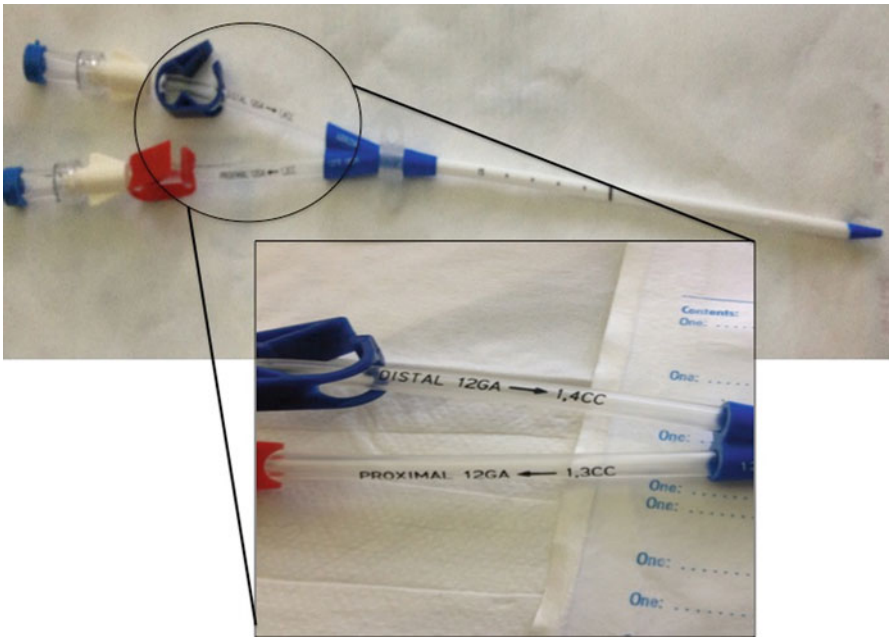

Fig. 25.3 Arrow® two-lumen hemodialysis catheter showing the priming volume in the two ports

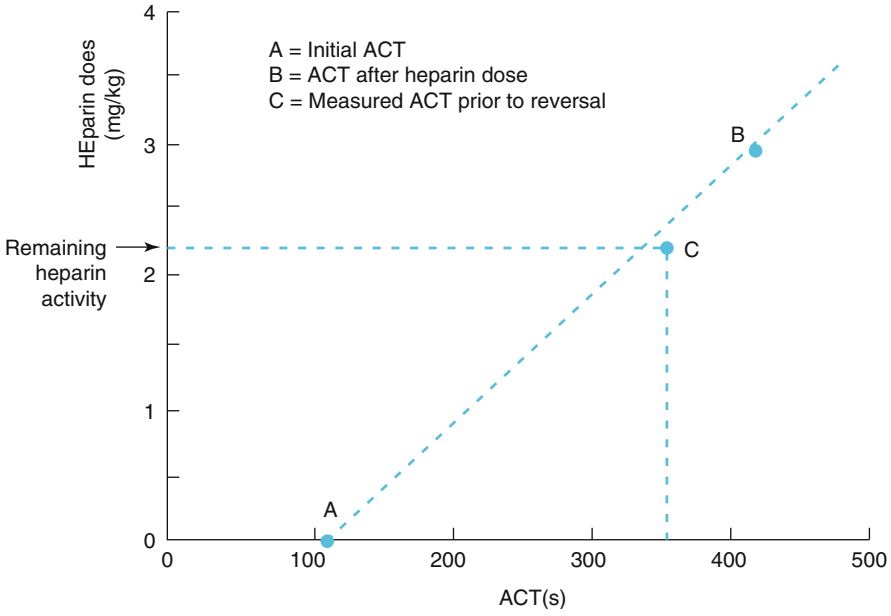

Fig. 25.4 Heparin dose–response curve. Activated clotting time (*ACT*) in seconds versus total heparin dose in milligrams per kilogram (Adapted from [3])

Table 25.1 Wash-in of blood and washout of heparin process

Flush of blood from the vascular system into a lumen of the Vas-Cath in increments of 1 port-to-tip dead space = 1.5 mL	Concentration (% of original) of heparin in a port-to-tip lumen of the Vas-Cath
0	100
1.5	37
3	13.5
4.5	5
6	1.8
7.5	0.7

Table 25.2 The different heparin preparations at UCSD

Heparin concentration (units/mL)	Total volume available (mL)	Uses/locations found
10,000	0.5	Thromboprophylaxis
10,000	10	Infusion center
1,000	10	Operating room cat
100	5	IV line flush
50	500	Therapeutic anticoagulation
2	500	Arterial line bags

physician that there is abnormal thrombus formation and breakdown. Its utility in this case was as a measure of fibrin split products to assess for a disseminated intravascular coagulation (DIC) or a consumptive coagulopathy. D-dimer level should be interpreted with caution since recent surgery, trauma, infection, pregnancy, and liver disease can all raise D-dimer levels. An appropriate use for it would be to monitor the effectiveness of therapy after suspected DIC. At the University of Southern California San Diego (UCSD) Medical Center, the upper limit for normal is 250 ng/mL. In this case, only one value was drawn, and since it was elevated nearly ten times the upper limit of normal, it is plausible to entertain the diagnosis of DIC in this patient [2].

L-5. What conclusions can be drawn from this case?

The data available in the narrative of this case do not permit a clear explanation of what caused the very high ACT and bleeding at surgery. Of course, it is important to clearly aspirate any preexisting residual heparin (or any drug) out of a catheter that is going to be used for the induction of anesthesia or for bolusing medications. It is clear that the problem was not caused by accidental entrainment of heparin from one port by a forceful injection into the other port during induction via the Venturi effect, which was the original clinical hypothesis. After careful study of the design of the hemodialysis catheters used in this institution, this is not likely since the lumens are physically separated. In this case, the total amount of heparin that could have been injected into the patient is 1,500 units of heparin because 3 mL of heparin (1,000 units/mL) was used to flush a 1.3-mL space. This would mean that a 3-mL injection into either port of the lumen would clear the catheter completely and deliver 1.7 mL (1,700 units) of heparin into the patient and leaving the other 1.3 mL in the lumen (assuming the Arrow double-lumen catheter). A similar heparin dose

would have been delivered to the patient if the Covidien™ catheter was used. Since several hours seemed to have elapsed between this flush and the start of surgery, the ensuing coagulopathy cannot be explained by this alone, unless there was a higher concentration of heparin used to flush the ports (drug swap error) based on the typical dose–response curves for heparin (see Fig. 25.2) [3]. There are various different concentrations of heparin available. The available preparations at UCSD are summarized in Table 25.2. Since heparin dose–response curves are individualized for each patient, Fig. 25.4 should be interpreted as a typical example of heparin's effect on a patient. It is clear from this curve that 1,500 units of heparin should not result in an ACT of 300 s as was seen in this case.

In Table 25.2, the unit per mL value is in the left-hand column, the total amount available in mL is in the middle column, and the typical uses or locations that these preparations are found in the hospital are listed in the right-hand column. Of the different heparin preparations at UCSD, only the 10,000-unit/mL preparation available from the infusion center, which is the site where chemotherapy is performed at UCSD (second row), would have been enough to accidentally heparinize the patient enough to bring the ACT to 300.

This case has an interesting differential for the unexpected coagulopathy that was encountered. It is not clear if the patient underwent dialysis prior to the case, but this could be a source for unintentional heparinization that occurred after the initial labs were drawn. More importantly, the anesthesiologist should be aware that a uremic patient with thrombocytopenia is a setup for bleeding and the coagulation parameter that may be abnormal is the bleeding time. This is not a routinely measured lab preoperatively, but it may be over 30 min in patients with uremic platelets [1]. It is the author's opinion that this patient was bleeding from uremic platelets and low VWF and factor VIII levels which led to the initial oozing of blood after incision. This prompted blood transfusion, which led to a coagulopathy resembling DIC from an adverse reaction to the transfusion. DIC has a very broad differential diagnosis and the mainstay of therapy is to treat the underlying cause.

Another possibility is a dilutional coagulopathy that may take place after packed red blood cells are administered without platelets or fresh frozen plasma (FFP). In this case four units of packed red cells were given before any FFP or platelets were given. Factors V and VIII can become diluted when plasma-poor blood is transfused. A dilutional thrombocytopenia may develop; however, it is more common after at least ten units of packed red blood cells has been given.

Finally, the simplest and least complex explanation is that a high concentration of heparin was wrongly used to lock one of the lumens of the Vas-Cath and then flushed into the patient during the induction of anesthesia.

L-6. If the port-to-tip volume = 1.3 and 3 mL of heparin was put in this port, then 1.7 mL (1,700 units) of heparin entered the patient at this point in time. Is this significant?

Since the half-life of heparin is 1 h, there should be very little heparin active in the patient's body several hours later.

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Chapter 26
Hypotension in Chronic Methamphetamine User

Zakir Rangwala

The patient is a 57-year-old male presenting for transurethral resection of bladder tumor (TURBT). Past medical history is significant for chronic methamphetamine use (**L-1**, **L-2**), 35 pack-year smoking history, moderate obesity (body mass index [BMI] 29), and regular alcohol use (approximately 3–4 drinks/day). Patient's vital signs and physical exam were unremarkable (noninvasive blood pressure [NIBP] 136/83 mmHg, heart rate [HR] 80 bpm, respiratory rate (RR) 18/min, O_2 98 %).

After premedication with 2 mg midazolam, the patient was preoxygenated for 5 min with 100 % oxygen and induction was carried out with 200 mg propofol. A laryngeal mask airway (LMA ProSeal) was placed without difficulty, but the patient was noted to have inspiratory stridor despite addition of volatile anesthetics. After attempting to improve ventilation with deepening anesthetic and a downsized LMA, the decision was made to establish a secure airway. The patient was paralyzed with 8 mg vecuronium and intubated easily with an 8.0 endotracheal tube (ETT).

As the case progressed, the patient developed hypotension (**L-2**) with systolic pressures as low as 70 mmHg. The hypotension was unresponsive (**L-2**) to lightening of anesthesia, fluid boluses, and both phenylephrine and ephedrine. The hypotension transiently responded to 10 mcg boluses of epinephrine.

On examination of the anesthesia machine, it was noted that ventilator was delivering 10–15 cm H_2O of positive end-expiratory pressure (PEEP) (**L-3**) despite no preset PEEP setting. Upon further examination, the expiratory valve on the ventilator appeared to be partially stuck. The patient was switched to hand ventilation via oxygen tank and Mapleson circuit and maintained on a propofol drip. The hypotension had partially resolved, and the case progressed uneventfully.

Following, extubation, the patient remained hemodynamically stable. A follow-up chest x-ray was ordered which did not show any signs of aspiration. After further questioning, the patient admitted to methamphetamine use prior to the day of surgery.

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Lessons Learned

L-1: What is the pharmacology of methamphetamine?

A. Methamphetamine is an indirect agonist at norepinephrine, dopamine, and serotonin receptors. It acts by stimulating the release of norepinephrine, dopamine, and serotonin (in a 60:2:1 ratio) from vesicles in CNS nerves into the synapses (Fig. 26.1) [1]. These monoamines, in turn, act directly on their respective peripheral receptors. The acute hemodynamic response to exposure is the result of peripheral stimulation of adrenergic receptors: ↑HR and ↑BP.

B. Methamphetamine also inhibits the reuptake of these neurotransmitters back into the synaptic vesicles in the presynaptic neurons. This contributes to the relative depletion of endogenous catecholamine (Fig. 26.1).

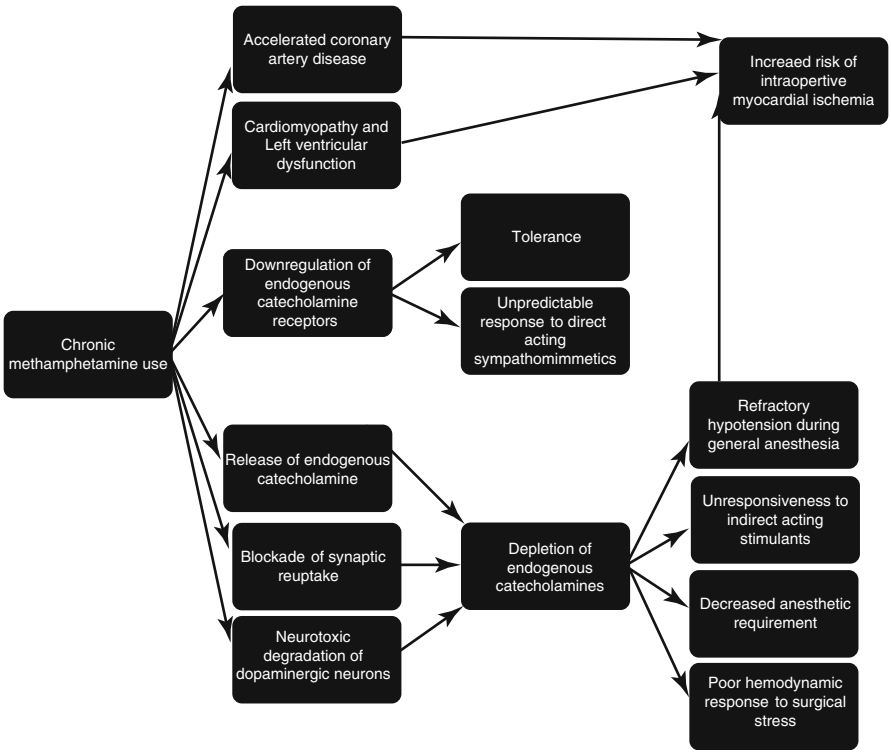

Fig. 26.1 Flowchart detailing the effects and anesthetic implications of chronic methamphetamine use. *See* text of **L-1**A, B and **L-2**A–E

L-2: **How does chronic methamphetamine use contribute to intraoperative hypotension**?

A. Patients with chronic exposure to amphetamines will exhibit tolerance by way of downregulation of endogenous catecholamine receptors, causing an unpredictable response to direct-acting stimulants.
B. Chronic use will lead to depletion of endogenous catecholamine stores via vesicular release into the circulation, neurotoxicity of dopaminergic neurons, and blockade of synaptic reuptake of catecholamine. The depletion of endogenous stores leads to a blunted physiologic and sympathetic response to hypotension while under general anesthesia [2].
C. Refractory hypotension will be unresponsive to indirect-acting agents like ephedrine. Direct-acting vasopressor agents should be readily available to treat low blood pressures. It is important to note that vasopressor agents can have unpredictable effects in chronic methamphetamine users.
D. Endogenous catecholamine stores may not return to normal until days or even weeks following cessation of use.
E. Chronic methamphetamine users are at increased risk of intraoperative myocardial ischemia:

    1. Refractory hypotension can result in poor coronary perfusion pressure.
    2. Excessive sympathetic stimulation can cause coronary artery disease and left ventricular dysfunction as well.

L-3: **What is the mechanism through which excessive PEEP can lead to decreased cardiac output and blood pressure**?

Excessive PEEP has been shown to cause a decrease in mean arterial pressure (MAP) by way of reducing cardiac output. There are six proposed mechanisms that are well supported by experimental data (Fig. 26.2) [3]:

A. Decreased venous return (Fig. 26.2A). Increased intrathoracic pressure from PEEP decreases the venous return gradient, thereby reducing cardiac output (Fig. 26.2A).
B. Increased pulmonary vascular resistance (PVR) and right ventricular dysfunction (Fig. 26.2B):

    1. PVR is lowest at functional residual capacity (FRC) and increases when lung volume moves away from FRC:

        (a) PVR increases at volumes lower than FRC primarily due to hypoxic pulmonary vasoconstriction of large vessels in these low-volume (and hypoxic) lung units.
        (b) PVR increases at volumes higher than FRC due to compression of small intra-alveolar vessels from high-volume lung units.

    2. The increase in PVR increases the work that the right heart has to work against (right heart afterload increases). Because the right heart cannot

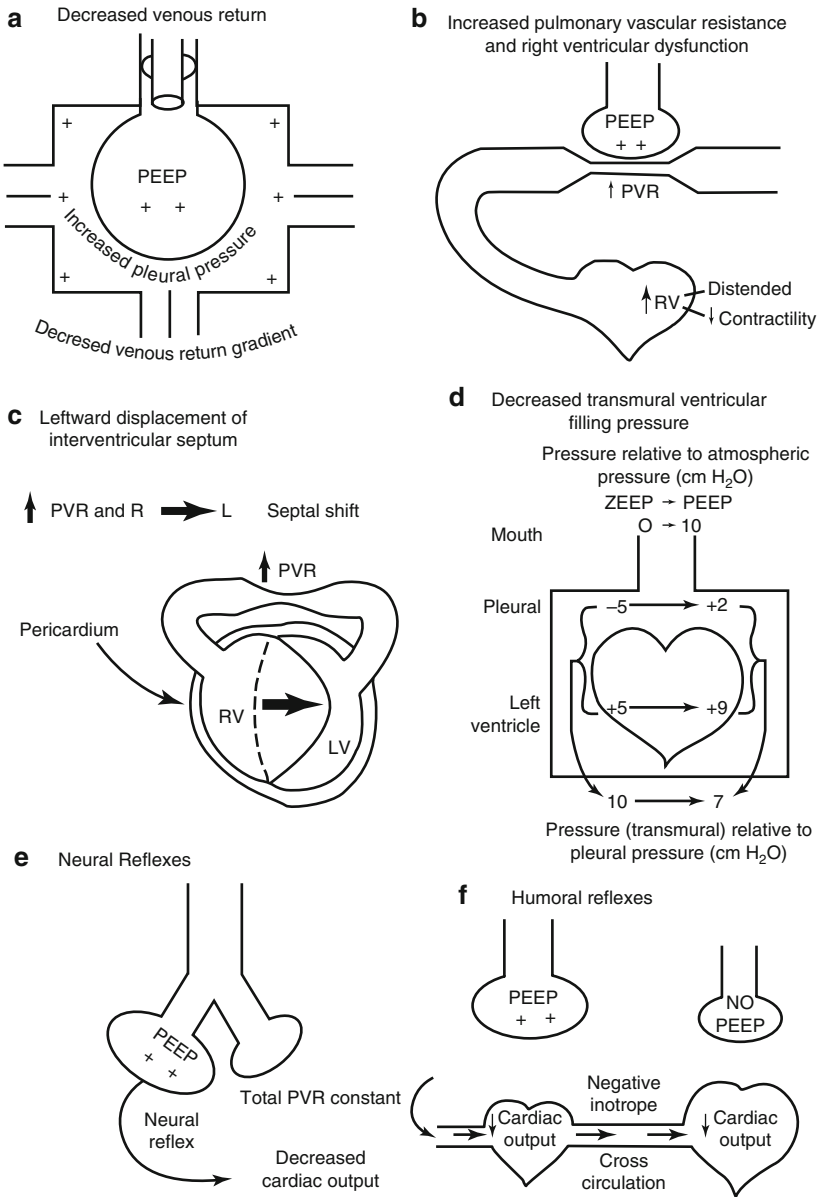

Fig. 26.2 Mechanisms of PEEP-induced decreased cardiac output. *See* text of **L-3**A–F (Adapted from [3])

compensate for excessive afterload pressures, it dysfunctions and the right heart output, and thus left heart output, will decrease.

C. Leftward displacement of interventricular septum (Fig. 26.2C). As stated above, excessive PEEP can increase PVR and right heart afterload. Additionally, it can

also cause the right heart to distend and completely fill the pericardial sac. While within the confines of the stiff and filled pericardium, the only way the right ventricle volume can increase is by leftward displacement of the interventricular septum. The leftward shift will decrease left ventricular filling and output (Fig. 26.2C).

D. PEEP-induced decrease in transmural filling pressures (Fig. 26.2D). Increased PEEP will decrease transmural filling pressures by way of differential transmission of airway pressures to intrathoracic pressures and pericardial pressures. Since transmural filling pressures = intracavitary pressure – pericardial/pleural pressure, an increased sustained PEEP will cause a decrease in transmural filling pressures (Fig. 26.2D).

E. Neural reflexes (Fig. 26.2E). Experimental data has shown that acute pulmonary hyperinflation can cause a decrease in heart rate and cardiac output. Controlling for changes in preload and afterload, it is postulated that there are three afferent receptors in the lung that are stimulated in response to lung distention, and these contribute to a marked fall in blood pressure and stroke volume (Fig. 26.2E).

F. Humoral mediation (Fig. 26.2F). There may also be a PEEP-induced decrease in cardiac output secondary to the release of humoral factors. Experimental data have shown that hyperinflation of lungs can cause release of biologically active materials that can cause depression of cardiac function. Additionally, cross circulation experiments in animals showed that PEEP in one animal in a cross-sectional pair caused a decrease in cardiac output of the other member (Fig. 26.2F).

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Chapter 27
Venous Air Embolism During Arteriovenous Malformation Repair

Zakir Rangwala

The patient is a 36-year-old male who was scheduled to undergo a right occipital-parietal craniotomy for the excision of a small occipital AVM (**L-2**). He had previously suffered an intraventricular hemorrhage 1 week prior to the scheduled surgery. The patient was otherwise healthy and had nothing remarkable in his past medical history.

An upper extremity 18-G peripheral IV and 20-G left radial arterial line were placed prior to taking the patient back to the OR. Following an uneventful, hemodynamically stable induction with fentanyl, vecuronium, and Pentothal, a 9-French left subclavian central venous catheter and a 14-G lower extremity peripheral IV were placed for additional access. A precordial Doppler (**L-5**) was also used for monitoring. After the surgeons placed the patient in a reclining, head flexed forward position (**L-2**), surgery commenced.

Approximately 1.5 h after the surgeons made their initial incision, the rhythmic sound of the precordial Doppler changed slightly (**L-4**, **L-5**). Very soon after, the capnograph monitor displayed a sudden drop of $etCO_2$ from 35 to 24 mmHg (**L-3**, **L-4**, **L-5**). The surgical team was immediately notified (**L-6**, **L-7**) of the possibility of venous air embolism (**L-1**). The surgeons immediately packed the area with saline-soaked gauze (**L-6**, **L-7**), while the anesthesia team used the central line to draw back bright red frothy blood (**L-7**). The anesthesia team continued to draw back from the central line until normal venous-appearing blood drew back consistently and easily.

During this time, the patient was also placed in a Trendelenburg position and nitrous oxide was discontinued, and the patient was ventilated with 100 % oxygen (**L-8**). The patient's MAP remained relatively stable throughout the episode. Within 5–10 min, the $etCO_2$ returned to baseline. The remainder of the surgery proceeded uneventfully, and the patient was extubated and later discharged without sequelae.

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Lessons Learned

L-1: **What is a venous air embolism** (**VAE**)? **What is the mechanism through which a VAE can occur**?

A. VAE is the entrainment of air (either atmospheric or pressurized) from a surgical site into the venous system causing potential hemodynamic and systemic disturbances via the obstruction of pulmonary blood flow.
B. The risk of VAE is present anytime there exists a pressure gradient between the pressure immediately outside an open venous site and the central venous pressure (CVP) or right atrial pressures (RAP). The pressure gradient between the outside and the inside of the vein can occur via one of two ways (Fig. 27.1a, b):

1. Gravitational effects – When the site of the open vein is higher than the right atrium, there exists a relative negative pressure with respect to the RAP, and the intravascular venous pressure becomes subatmospheric. This negative pressure will cause atmospheric air to be entrained into the open vein.
2. Pressurized air – In laparoscopic or endoscopic procedures, insufflated air or carbon dioxide can enter the venous circulation at open venous sites due to supra-atmospheric pressures regardless of height differences.
3. As little as 5 cm H_2O pressure difference between RAP and the outside of an open vessel can cause a hemodynamically significant VAE.

L-2: **What neurosurgical procedures and patient positions are associated with an increased risk of VAE**?

A. Historically, neurosurgical procedures, and specifically surgeries of the posterior fossa in any position and those performed in the sitting position, have been known to have the highest incidence of intraoperative VAE. The large dural sinuses within the cranial vault are incompressible and are thus common sites that air can enter the venous system.
B. Other procedures with reported VAE include spine surgeries, neck surgeries, total hip arthroplasty, cesarean delivery, and liver surgeries.
C. Nonsurgical procedures such as placement of central venous catheters have the potential for VAE to occur.

L-3: **What is the pathophysiology of a VAE**?

A. The factors that determine the severity of morbidity and mortality have to do with the volume and rate of air accumulation as well as the location of the entrainment:

1. Reported lethal volumes of air have been reported from 200 to 500 cc, or 3–5 cc/kg, and it has been postulated that smaller volumes of air are more lethal when entrained in venous openings that are closer to the heart.
2. For reference, a 5-cm H_2O pressure difference across a 14-G catheter can transmit 100-cc air/s.

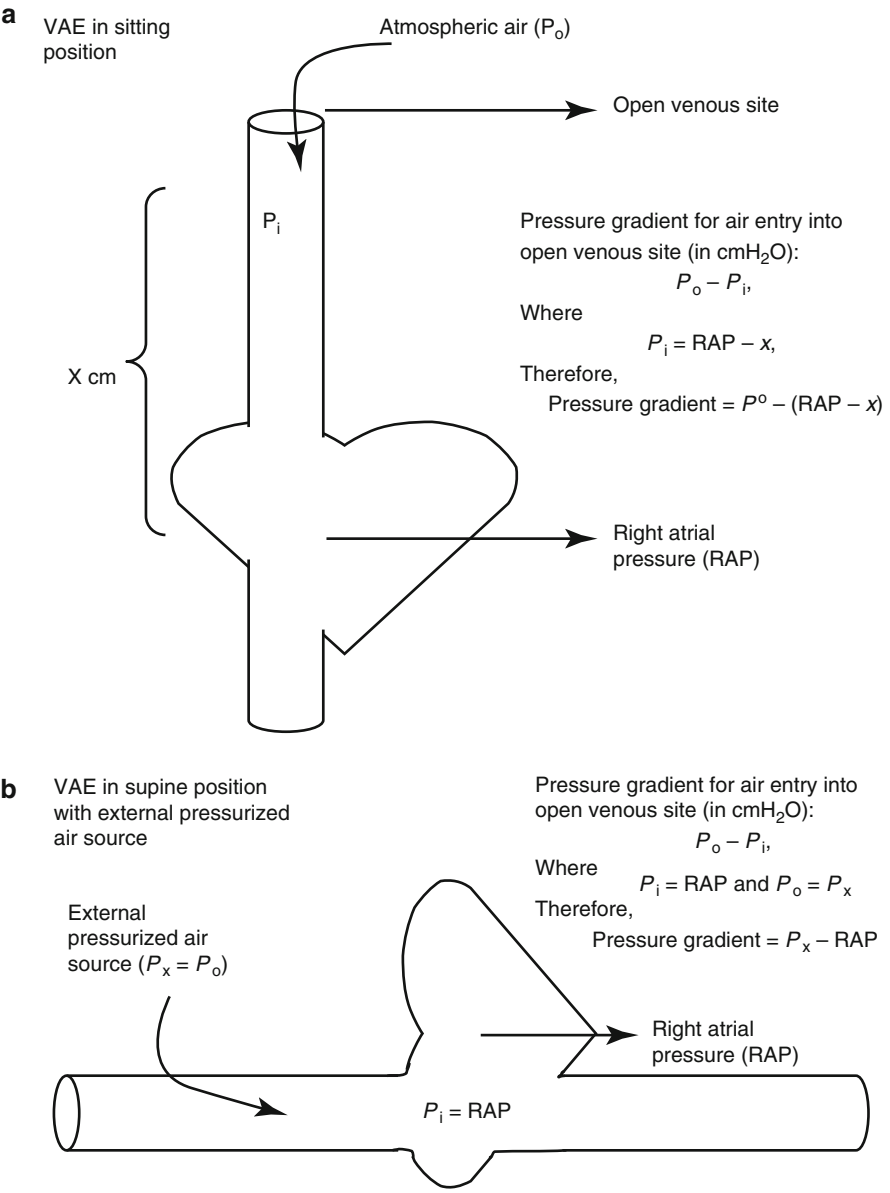

Fig. 27.1 Venous air embolism can occur in (**a**) the sitting position due to gravity's effects on the venous pressure at the open venous site. (**b**) The supine position where the external pressurized air source provides the pressure gradient for air entry into an open venous site. P_i pressure inside vein, P_o pressure outside vein

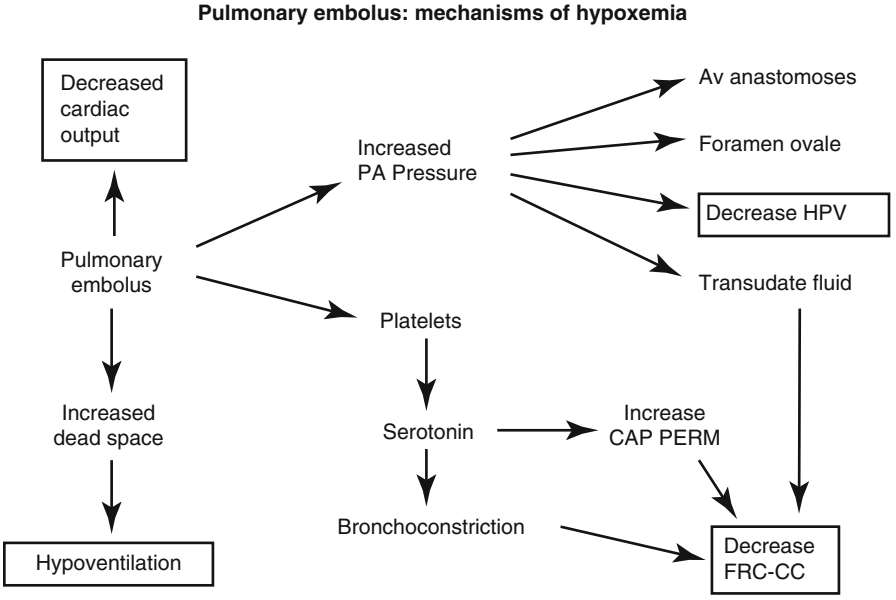

Fig. 27.2 Mechanisms of hypoxemia during embolism. *CAP PERM* capillary permeability, *AV* arteriovenous, *HPV* hypoxic pulmonary vasoconstriction, *PA* pulmonary artery, *FRC* functional residual capacity, *CC* closing capacity (Adapted from Benumof JL. Anesthesia for thoracic surgery. 2nd ed. Philadelphia: Saunders; 1995)

B. Hemodynamics – As stated above, the obstruction of pulmonary blood flow from a VAE can have significant hemodynamic effects:

1. With smaller volumes, entrained air can deposit in the pulmonary circulation, causing obstruction in the pulmonary arterial bed. Neutrophil accumulation and release of vasoactive substances occur, resulting in pulmonary vasoconstriction which can affect right ventricular function and lead to hypotension.
2. Larger volumes of air can produce an "air lock," which can cause immediate hemodynamic collapse secondary to the inability of the right heart to compress against the large air bubble trapped in the right heart. This can present as pulseless electrical activity as a result of total obstruction of right ventricular ejection in the face of preserved electrical cardiac activity.

C. Pulmonary – Hypoxia/hypoxemia is a hallmark of VAE and occurs through a variety of mechanisms (Fig. 27.2):

1. Decreased cardiac output – Occurs as a result of obstruction to pulmonary flow.
2. Hypoventilation – Air bubbles trapped in the alveolar-capillary interface serve as a barrier preventing gas exchange. The physiologic dead space increases and leads to a relative hypoventilation and an increase in $PaCO_2$.

3. Decreased hypoxic pulmonary vasoconstriction (HPV) – A VAE that has travelled into the pulmonary circulation will cause an increase in PA pressures and will lead to decreased HPV and increase intrapulmonary shunting.
4. Decreased functional residual capacity:

   (a) Bronchoconstriction is also known to occur as a result of platelet activation and the release of vasoactive mediators in the setting of VAE.
   (b) Increased pulmonary interstitial edema leads to decreased FRC and will occur via two ways: (1) Serotonin released from activated platelets trapped behind the obstructing air in the pulmonary circulation can increase pulmonary capillary permeability. Increased pulmonary capillary permeability will lead to increased pulmonary interstitial edema. (2) Pulmonary hypertension induced by the obstructing air will also cause increased pulmonary interstitial edema.
   (c) The decreases in lung volume will predispose the patient to hypoxemia as a result of atelectatic lung units.

D. Neurological – Whereas it has been established that up to 35 % of the population has a patent foramen ovale (PFO), paradoxical air emboli are a major concern in the setting of VAE. As stated above, VAE will cause an obstruction to right ventricular outflow, thereby increasing right heart pressures. This relative increase in right-sided pressures will create a right-to-left shunting across the PFO, and air emboli can bypass the pulmonary circulation and enter the systemic circulation. Focal neurological defects and myocardial ischemia can result from even small amounts of air.

L-4: **What are the presenting signs of a VAE**?

A. Cardiovascular – Increased right heart strain may lead to EKG changes. Tachyarrhythmias are also common. Pulmonary artery pressures are increased and cardiac output is decreased. Depending on the amount and rate of air entrained, hypotension can range from mild to complete hemodynamic collapse.
B. Pulmonary – Awake patients may complain of coughing, breathlessness, chest pain, and a sense of "impending doom." Pulmonary rales and wheezing may also be present. With respiratory monitoring, decreases in oxygen saturation and PaO_2 are common.

An acute drop in $etCO_2$ is a sensitive sign of VAE. $PaCO_2$ may increase subacutely as a result of relative hypoventilation. The decrease in PaO_2 and $P_{ET}CO_2$ and increase in $PaCO_2$ is the result of blockage of the pulmonary circulation and concomitant increase in dead space ventilation seen with VAE.

L-5: **What are the most sensitive methods to detect a VAE**? **What are the limitations in each of these methods**?

See Table 27.1 for further information.

Table 27.1 Comparison of methods of detection of vascular air embolus

Detection method	Sensitivity, mL/lg	Availability	Invasiveness	Limitations
Transesophageal echo	High, 0.02	Low	high	Expertise required Expensive Invasive
Precordial Doppler	High, 0.05	Moderate	None	Obese patients
Pulmonary artery catheter	High, 0.25	Moderate	None	Fixed distance Small orifice
Transcranial Doppler	High	Moderate	None	Expertise required
etN_2	Moderate, 0.5	Low	None	Nitrous oxide Hypertension
$etCO_2$	Moderate, 0.5	Moderate	None	Pulmonary disease
Oxygen saturation	Low	High	None	Late changes
Direct visualization	Low	High	None	No physiologic data
Esophageal stethoscope	Low, 1.5	High	Low	Late changes
Electrocardiogram	Low, 1.25	High	Low	Late changes

Adapted from Mirski MA, Lele AV, Fitzsimmons L, Toung TJ. Diagnosis and treatment of vascular air embolism. Anesthesiology. 2007;106:164–77

L-6: What precautionary measures should be taken in procedures where VAE is a significant risk?

A. The anesthesiologist must make an assessment of VAE risk as a function of the specific procedure and positioning for the procedure.
B. Monitoring should be adjusted accordingly. While all anesthetic procedures necessitate the use of pulse oximetry and capnograph monitoring, the anesthesiologist must determine if the placement of a central line for aspiration of air (see below), precordial Doppler, or TEE is appropriate based on VAE risk.
C. Patients should be well hydrated so as to increase CVP to its maximal safest value. An increased CVP will reduce the VAE risk by creating less of a pressure gradient between the pressure outside and inside any open bleeding site (Fig. 27.1).
D. Positioning – While the head-up position carries a higher risk for VAE, it may be prudent to adjust the patient in a flexed leg-up position so as to increase central venous pressure.
E. Regarding the use of PEEP, previously it was thought that using PEEP may help in preventing the incidence of VAE, but studies show that in the seated position, central venous pressure is not augmented enough by PEEP to offset the negative pressure gradient. Additionally, excessive PEEP may compromise hemodynamics (*see* Fig. 54.3) and the increase in right heart pressures may increase the risk of paradoxical emboli across a PFO. Currently, the data for the use of PEEP in procedures where there is a risk of VAE is mixed.

L-7: What is the treatment of VAE?

A. The first step in treatment is recognizing that VAE has occurred. Depending on monitors being utilized, TEE, precordial Doppler, and capnograph values can all be used to aid in diagnosis of VAE.

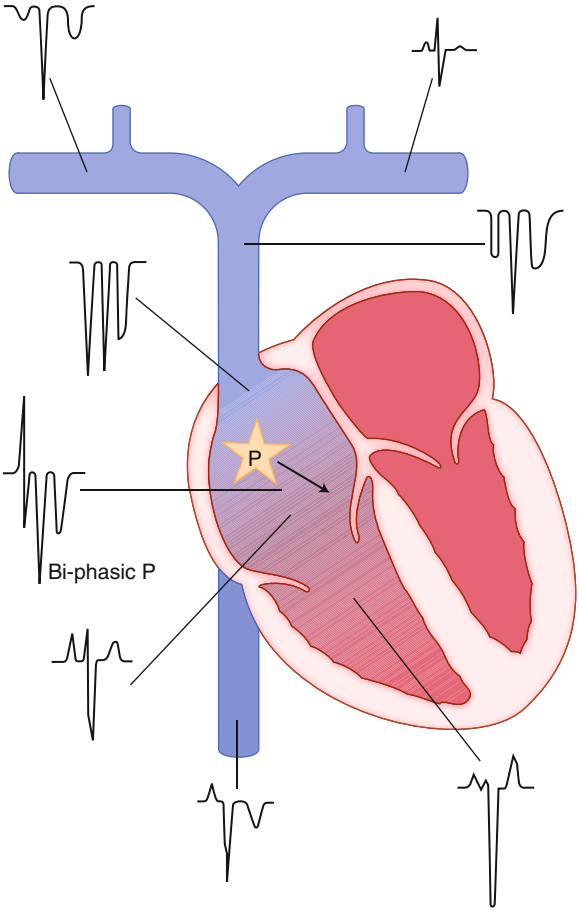

Fig. 27.3 ECG configurations observed at various locations when a central venous catheter is used as an intravascular ECG electrode. The configurations are observed when lead II is monitored and the positive electrode is connected to the catheter. "P" indicates the sinoatrial (SA) node. The heavy *black arrow* indicates the P wave vector. Note the biphasic P wave when the catheter tip is in the mid-atrial position (Adapted from Miller RD, Eriksson LI, Fleisher L, Wiener-Kronish JP, editors. Miller's anesthesia: expert consult. 7th ed. Philadelphia: Churchill Livingstone; 2009)

B. Once VAE is diagnosed, goals of management include:

1. Prevention of further air entrainment – Surgeons should be notified when there is suspicion of VAE and immediately cover the surgical site with saline and saline-soaked gauze to prevent further air entrainment. If possible, the positioning of the table should be altered so that the bleeding site is below the level of the heart, thereby eliminating the negative pressure gradient. Placing the patient in the left lateral decubitus position, also known as the Durant maneuver, may reduce the effects of an air lock and relieve right ventricular obstruction and allow hemodynamic flow. In cranial procedures, bilateral external jugular compression can reduce the negative pressure gradient by increasing the venous pressure at the entrainment site and also help the surgeon to identify the site of bleeding from open dural sinuses:

   (a) Caution should be taken when performing external jugular compression as the prevention of venous drainage may lead to increase intracranial pressure – which may be even more detrimental in certain clinical settings.

(b) Additionally, accidental compression of carotid arteries may lead to ischemia from lack of cerebral perfusion.

2. Aspiration of air – In cases where there is high risk for VAE, placement of a multi-orifice central venous catheter may be warranted, not only for hemodynamic purposes but also to aspirate air from the right atrium in the setting of VAE. Appropriate placement of a multi-orifice catheter may be technically difficult and is commonly done through the use of X-ray, or TEE, or an ECG lead that is attached to the catheter tip. The ideal position is 2 cm distal to the superior vena cava-right atrial junction, where one would find a biphasic P wave on ECG (Fig. 27.3). Although the data are mixed, aspiration of 15–20 cc of air has been shown to improve outcomes in VAE.
3. Hemodynamic support – Because of the hemodynamic disturbances that can occur in VAE, it is important that the anesthesiologist is prepared to support the patient hemodynamically. Inotropic support for the right ventricle should be readily available and resuscitation measures should be taken if hemodynamic collapse occurs. Even in a non-code setting, hemodynamic support can improve outcomes. Closed chest massage can help air locks migrate distally into smaller pulmonary vessels, allowing forward pulmonary blood flow in the larger vessels. 100 % oxygen should be instituted to improve oxygen delivery. There are also studies showing that hyperbaric oxygen may improve outcomes.

L-8: **Why is nitrous oxide detrimental in the setting of VAE**?

A. Although nitrous oxide does not increase the incidence of VAE, it increases the size of the airspace and should be discontinued once VAE is recognized, and the patient should be placed on 100 % oxygen.
B. Nitrous oxide is 34 times more soluble in blood than nitrogen and therefore nitrogen will leave the airspace to go into the blood relatively slowly. Thus, the efflux of nitrogen gas out of an air space is much slower than the influx of nitrous oxide into an airspace. Therefore, the airspace will increase in size.

Chapter 28
Cardiac Tamponade

Zakir Rangwala

The patient is a 75-year-old female, post-op day five from aortic valve replacement and three-vessel coronary artery bypass graft (CABG). Patient had an unremarkable recovery until approximately 1 h following removal of pacing wires at which point chest tube output increased markedly. Transthoracic echocardiogram revealed a large pericardial effusion (**L-1**, **L-2**, **L-3**, **L-4**) with severe right ventricle compression. The cardiothoracic surgery team called for an immediate sternotomy for evacuation of effusion.

Upon arrival to patient's bedside for anesthesia transport directly to the OR, patient's vital signs were as follows: noninvasive blood pressure (NIBP) 80–135/50–78 mmHg, heart rate (HR) 60 bpm, and respiratory rate (RR) 20/min (spontaneous ventilation). O_2 sats >95 % on 4 L face mask. The patient appeared alert and oriented, verbalizing her discomfort as chest pain. While the surgical team requested that the patient be intubated prior to transport, the anesthesia team insisted that the patient should remain spontaneously ventilating (**L-5**, **L-6**) and anesthetic induction should be performed only in the operating room.

Transport was uneventful, and patient was moved to the operating room (OR) table and routine monitors were placed. Additionally, a radial arterial line and defibrillation pads were placed prior to induction. Adequate vascular access was confirmed, and fluids were running as the patient's chest was prepped and draped in case of necessary emergent sternotomy. Induction was then carried out using 100 mg ketamine (**L-8**). Patient was intubated successfully via direct laryngoscopy while spontaneously ventilating (**L-6**, **L-7**). Vital signs remained stable throughout induction.

Anesthetic maintenance was achieved with ketamine and sevoflurane at 0.20–0.25 %. The patient was kept spontaneously ventilating until sternotomy was performed after which the patient was paralyzed and placed on positive pressure

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ventilation (**L-6**, **L-7**). Pericardial effusion and clot were seen on transesophageal echocardiography (TEE) and subsequently evacuated by the surgical team.

Initial labs showed a hematocrit of 22 % and INR of 1.5. Following transfusion of six units packed red blood cells (PRBCs), four units fresh frozen plasma (FFP), and one unit of platelets, oozing at pericardial pacing wire sites ceased. Following chest closure, vital signs remained stable, and no pericardial effusion was noted on TEE. Patient was transported to intensive care unit (ICU) intubated and extubated postoperative day (POD) 1 with an uneventful recovery.

Lessons Learned

L-1: **Describe the anatomy and function of the pericardium**.

A. The pericardial sac is divided into two layers. The outer parietal pericardium attaches to the sternum, diaphragm, and costal cartilage. The inner visceral pericardium, or epicardium, is directly adherent to the heart. The space in between the visceral and parietal pericardium is referred to as the pericardial space.
B. Under normal conditions, the pericardial space contains 10–30 cc of serous fluid.
C. Function is threefold:

    1. The pericardium anatomically fixes the beating heart in place.
    2. The pericardial fluid reduces external friction and acts as a barrier to protect against spread of infection and malignancy.
    3. Mechanical function is to prevent overdistention of heart when blood volume increases and provide a closed chamber of subatmospheric pressure to aid in filling by lowering of extracavitary pressure and increasing filling or transmural cardiac pressures.

        (a) Transmural pressure = intracavitary pressure – pericardial pressure (Fig. 28.1).
        (b) Under non-pathological conditions, the pericardial pressure and pleural pressure are essentially the same, and transmural pressures can also be approximated by the difference between intracavitary and pleural pressures (Fig. 28.1).

L-2: **What is the compliance of the pericardium**?

A. The pericardial sac can accommodate some extra fluid, but depending on the chronicity of fluid accumulation, small increases in effusion can result in very high pericardial pressures (Fig. 28.2) [1].
B. On the flip side, drainage of even small amounts of fluid during critical tamponade will dramatically reduce the pericardial pressure surrounding the heart.

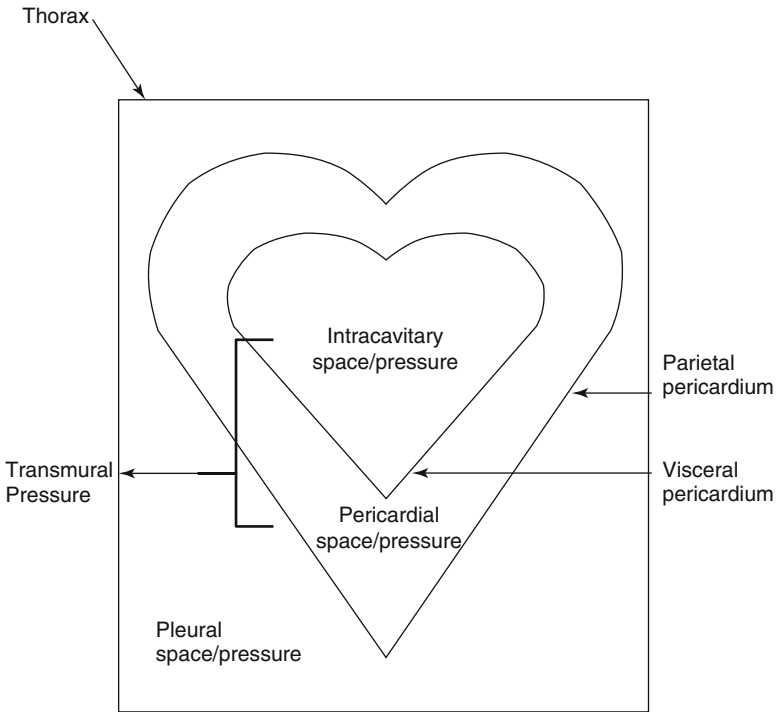

Fig. 28.1 The three spaces that contribute to transmural filling pressures. Under non-pathological conditions, the pericardial pressure and pleural pressure are effectively the same. Thus, the transmural filling pressures is the difference between the intracavitary pressure and the pericardial/pleural pressure. Under conditions where pericardial pressures rise, this assumption cannot be made, and transmural pressures are heavily determined by the pericardial pressure

L-3: **Describe tamponade physiology and the main causes of cardiac tamponade**.

A. When pericardial effusion volume exceeds the distensibility of the pericardial sac, pericardial pressures rapidly increase, thereby causing a relative decrease in transmural filling pressures, while pleural pressures remain constant; therefore, increased pericardial fluid past the limit of pericardial stretch dissociates the pleural and pericardial pressures.

1. When the pericardial pressures exceed the limit of pericardial stretch, transmural pressures can no longer be approximated by pleural pressures and are now heavily influenced by pericardial pressures.
2. Intracavitary pressures will be elevated because of the body's compensatory response of peripheral and pulmonary vasoconstriction to initially prevent collapse by way of increasing central blood volume.

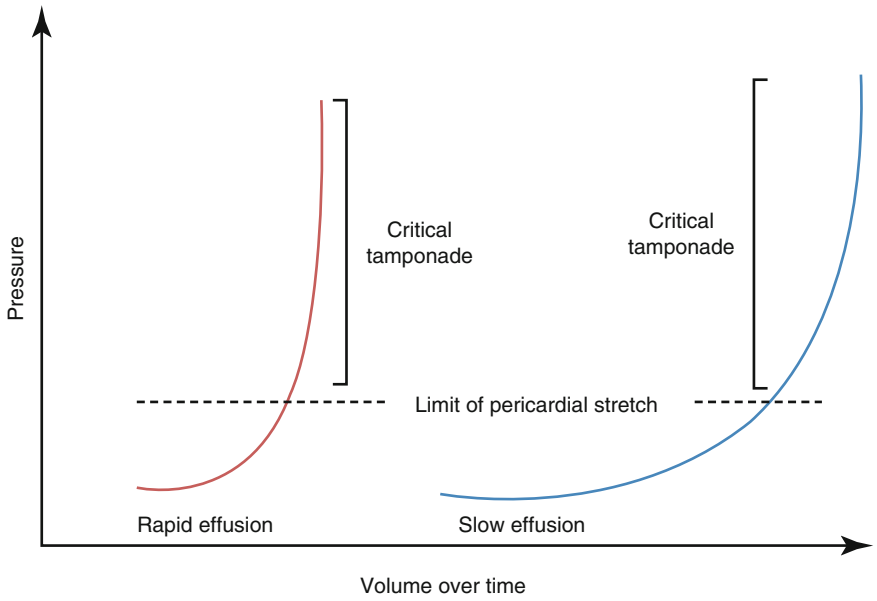

Fig. 28.2 Pericardial pressure-volume curves for rapid (*left*) and chronic (*right*) effusions. In the left panel, the limit of pericardial reserve volume is reached quickly and exceeds the limit of the pericardial stretch. This is followed by a rapid rise in pericardial pressures. In the right panel, the slower effusion allows for more pericardial compliance. The limit of pericardial stretch is reached later, and the rate of rise in pressure is slower (Adapted from [1])

B. As pericardial pressures increase, cardiac chamber collapse occurs during diastole, first in the low-pressure right heart but eventually to all four chambers.
C. As pericardial pressures increase, transmural pressure approaches 0 in all four chambers, preventing venous return and compromising cardiac output.

L-4: **What is pulsus paradoxus and ventricular impedance**?

A. Ventricular impedance refers to the decrease in cardiac output due to ventricular interdependence and septal shifting.

1. During spontaneous ventilation, inspiration generates a negative intrathoracic pressure and augments venous return to the right heart.
2. Because the pericardium restrains filling of all chambers of the heart in tamponade, an increase in right heart filling pressures during diastole will cause a septal bulging to the left and diminish left ventricular filling (ventricular interdependence) contributing to a decreased cardiac output (ventricular impedance).
3. This is readily apparent on TEE.

B. Pulsus paradoxus

1. The clinical manifestation of ventricular impedance
2. Defined as a decrease of systolic blood pressure (SBP) >10 mmHg on inspiration

L-5: **What are the anesthetic goals for management of tamponade**?

A. If possible, pericardiocentesis under local anesthesia should be performed prior to an anesthetic induction.
B. If surgical pericardial drainage is necessary, hemodynamic goals should be to:

1. Maintain intravascular volume and filling pressures to offset pericardial pressure.
2. Maintain increased heart rate—because stroke volume is essentially fixed and cardiac output is heavily rate dependent.
3. Maintain contractility—avoid vagotonic agents.

L-6: **Why is maintenance of spontaneous ventilation important in this case**?

A. Maintaining spontaneous ventilation will preserve the transmural pressure gradient created by the difference in intracavitary pressure and pericardial pressure. That is, the negative pleural pressure created during spontaneous ventilation will offset, to some extent, the detrimental effects of the high pericardial pressure and allow for higher transmural filling pressures (Fig. 28.3).
B. When mechanical ventilation is instituted, the positive intrathoracic pressure is transmitted to the pericardium at approximately 70 %. This pressure is in turn transmitted to the intracavitary pressure at approximately 70 %. This creates an overall decrease in transmural pressure [2].
C. The decrease in transmural filling pressure will contribute to overall hemodynamic collapse.

1. Note: external cardiac compressions are clinically ineffective as intracavitary pressures are already high.

L-7: **When is it safe to paralyze this patient**?

A. When the sternum and the pericardium is open, the high pericardial pressure gradient is eliminated and transmural pressure = intracavitary pressure – atmospheric pressure (0).
B. At this point, it will be safe to paralyze the patient and begin positive pressure ventilation.

L-8: **Why is ketamine a good choice for induction of anesthesia**?

A. Ketamine is an ideal choice because of its sympathomimetic effects and meeting all of our hemodynamic goals: ↑HR, ↑SVR, ↑BP, ↑CO.

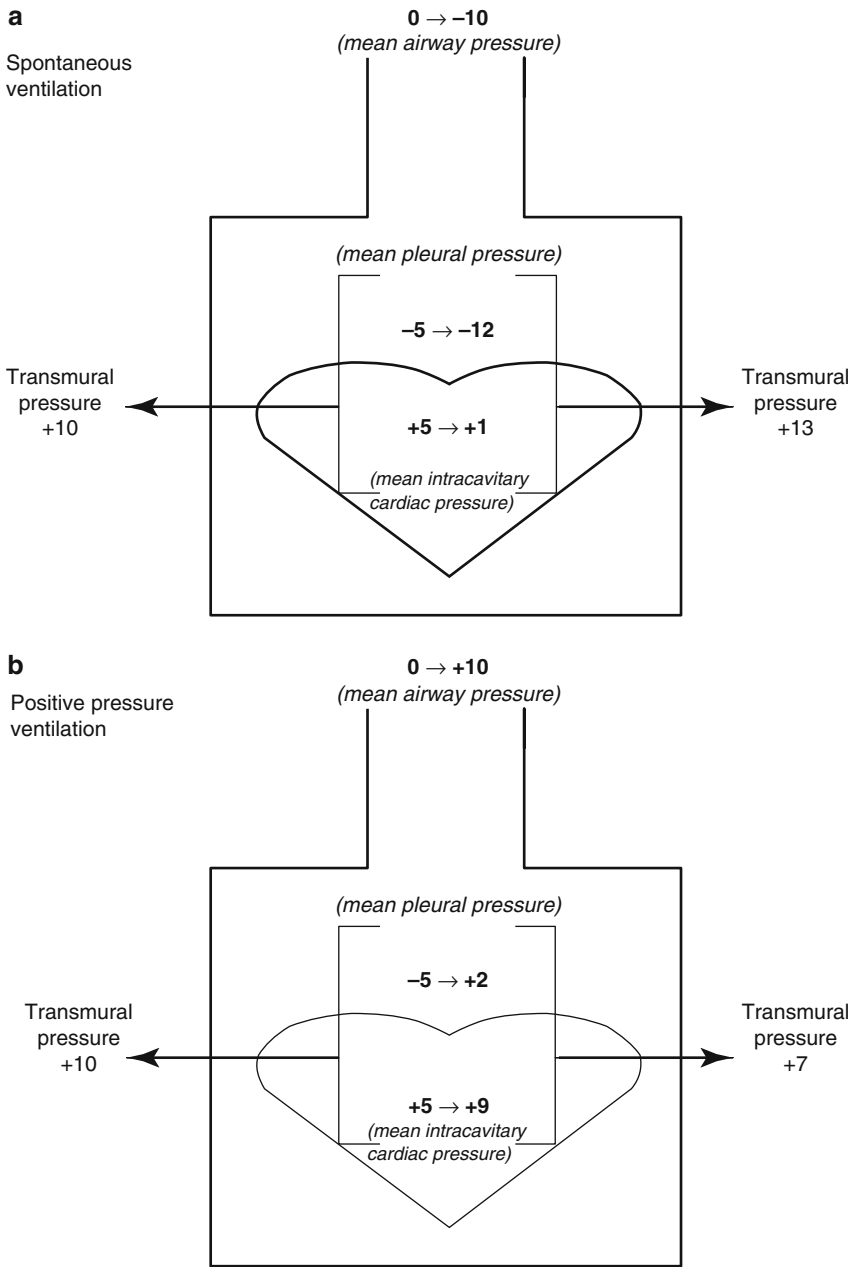

Fig. 28.3 The effects of spontaneous and positive pressure ventilation on transmural filling pressures. (**a**) Spontaneous ventilation will cause a relative increase in filling pressures. (**b**) Positive pressure ventilation will decrease filling pressures. If high enough, positive pressure ventilation can lower transmural pressures enough to cause hemodynamic collapse

B. Additionally, a ketamine induction will allow the patient to continue spontaneous ventilation.
C. It is important to note that ketamine can have the opposite effect on hemodynamics in patients who have already exhausted their sympathetic reserve. Caution must be taken when administering, and inotropic agents like epinephrine or dopamine should be readily available.

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Chapter 29
Case of Intraoperative New-Onset Atrial Fibrillation

Lawrence Weinstein

The patient is a 73-year-old, 80 kg male with a past medical history significant for hypertension, benign prostatic hypertrophy, and remote acute lymphocytic leukemia (now in remission). He presented to the preoperative area for scheduled laparoscopic repair of a right inguinal hernia. Preoperative assessment consisted of a visit to a cardiologist, who determined that the patient had excellent functional capacity, routinely attaining more than four METS (**L-1**) (Tables 29.1 and 29.2) of activity without chest pain, shortness of breath, palpitations, dizziness, nor syncope. Baseline electrocardiogram (EKG) demonstrated normal sinus rhythm at 63 beats per minute (bpm), left axis deviation, and no acute ST segment changes. The patient also went to the anesthesia preoperative clinic, where it was learned that he had had an uncomplicated general anesthetic as a child for tonsil removal.

The patient's medications at the time of surgery included atenolol, lisinopril-hydrochlorothiazide, oral potassium supplements, finasteride, niacin, aspirin, and a multivitamin. He had an allergy to levofloxacin, which caused a rash. Of note, the patient had last taken his atenolol the prior evening and had withheld all other medications on the morning of surgery.

Blood work showed hematocrit of 45.1 %, platelets of 129,000/mcL, sodium of 143 mEq/L, potassium of 3.6 mEq/L, blood urea nitrogen (BUN) of 15 mg/dL, creatinine of 0.9 mg/dL, and blood glucose of 102 mg/dL.

After reviewing medical history on the day of surgery, the patient was brought to the operating room and standard American Society of Anesthesiologists

(ASA) monitors were placed. Baseline vital signs showed a sinus bradycardia in the 50s bpm, blood pressure (BP) of 157/75 mmHg, and oxygen saturation 98 % on room air. He was preoxygenated for 3 min, and anesthesia was induced with midazolam 1 mg, fentanyl 150 μg, lidocaine 80 mg, propofol 200 mg, and vecuronium 5 mg. Ventilation via mask was performed until adequate muscle relaxation was

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Table 29.1 MET values for various leisure activities

Activity	METS
Walking slowly (1–2 mph)	2
Playing musical instrument	2
Walking at average pace (2–2.5 mph)	2.5–3
Slow dancing	2.5–3
Golfing, using cart	2.5–3
Bowling	2.5–3
Fishing	2.5–3
Walking briskly (3 mph)	3.5
Weight lifting	3.5
Leisurely canoeing	3.5
Walking quickly (1 mile in 17 min)	4
Climbing stairs	4
Dancing moderately fast	4
Bicycling slowly (<10 mph)	4
Golf, walking with clubs	4.5
Slow swimming	4.5
Walking very quickly (4 mph)	5
Doubles tennis	5–6
Slow jogging	6
Roller or ice skating	6
Vigorous dancing	6–8
Bicycling (10–16 mph)	6–10
Swimming quickly	6–10
Aerobic calisthenics	6–10
Singles tennis, squash, or racquetball	7–12
Jogging (12 min miles)	8
Skiing	8
Running (6 mph)	10
Running (8 mph)	13.5
Running (10 mph)	16

Adapted from [1]

confirmed with a twitch monitor, and the trachea was easily intubated on the first attempt. After confirmation of endotracheal tube placement via lung auscultation and end-tidal CO_2 ($etCO_2$), anesthesia was maintained with 2 % sevoflurane.

Shortly after surgical incision and abdominal insufflation, the patient's blood pressure increased to the 170s/90s mmHg, with heart rate in the 40–50s bpm (sinus rhythm). This was treated with hydralazine 6 mg, and the BP came down to the 130s/70s mmHg over the next 15 min, still with a heart rate in the 40s bpm. The case proceeded uneventfully over the next 20 min until the surgeon began to sew in the mesh for the hernia repair. At this point, the patient's EKG demonstrated a sudden onset of a narrow complex, irregular tachycardia with ventricular rates between 90 and 120 bpm. Blood pressure remained stable at 150s/80s–90s mmHg, and expired CO_2 was unchanged at 40 mmHg. The surgeons were asked to deinsufflate the abdomen, and the patient was treated with fentanyl 50 μg, esmolol 50 mg (in divided

Table 29.2 MET values for common daily activities

Activity	METS
Standing	1.3
Reading, talking on telephone	1.5
Sitting in class, note taking	1.5–2
Walking indoors, in office	2
Light gardening	2
Light office work, standing light work	2
Walking down stairs	2.5
Cooking, light housework	2.5
Yard work	2.5
Pushing stroller with child, walking dog	2.5
Standing light-moderate work	3
Washing car, mopping, vacuuming	3
Walking on job, moderate pace	3.5
Raking lawn, planting, weeding	4
Painting, paper hanging, moderately heavy lifting	4
Walking with objects 25–49 lb	5
Vigorous gardening, mowing lawn	5
Carpentry, laying carpet	5
Chopping wood	5
Using heavy tools (shovel, pick, spade)	6
Walking with object 50–74 lb	6.5
Loading truck, moving heavy objects	6.5
Walking with object 75–99 lb	7.5
Heavy farming labor	8

Adapted from [1]

doses), and metoprolol 5 mg, which brought the HR down to the 80s bpm. The EKG still showed an irregular narrow complex rhythm, and P waves were not noticed. Consultation with the surgeon revealed that they could complete the right hernia repair in 5 min but that they had discovered a left inguinal hernia that they also intended to fix as long as the patient was anesthetized. Given that the patient was hemodynamically stable, it was agreed that the right-sided hernia repair would be finished and that the patient's status would be reassessed prior to proceeding to repair the left inguinal hernia. The abdomen was reinsufflated, and the hernia repair was completed on the right. During this time, the pulse again increased to the 110s–120s bpm, and the decision was made to wake the patient up. Heart rate was brought down to the 90s bpm with an additional 30 mg esmolol, and neuromuscular blockade was reversed with neostigmine 4 mg and glycopyrrolate 0.6 mg. Upon emergence, with the patient breathing spontaneously, the HR again increased, this time to 140 bpm, with blood pressures in the 170s/90s mmHg. Sevoflurane was turned back on to keep the patient asleep and defibrillator pads were applied to his chest. A 12-lead EKG was performed, which demonstrated atrial fibrillation (**L-2**, **L-3**, **L-4**, **L-5**, **L-6**, **L-7**) with rapid ventricular response (Figs. 29.1, 29.2 and

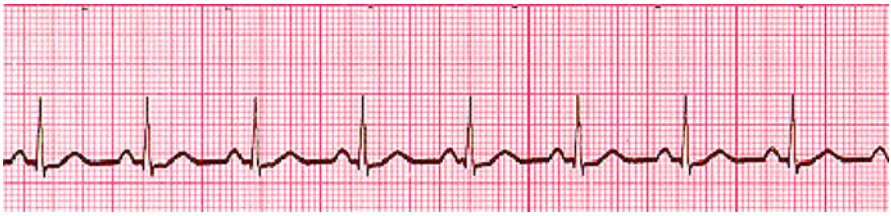

Fig. 29.1 Normal sinus rhythm. Note the normal *P wave* preceding each QRS complex. The rhythm is regular (Adapted from the Ambulance Technicians Study [2])

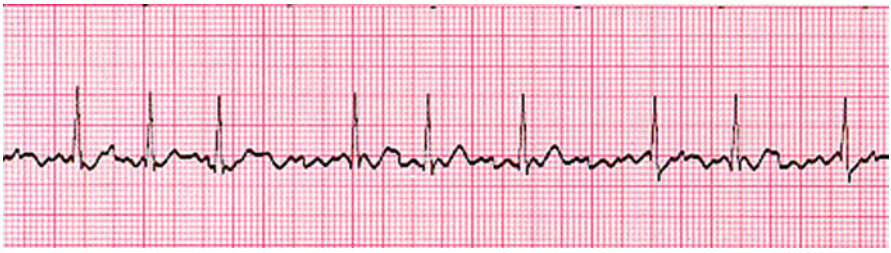

Fig. 29.2 Atrial fibrillation. Note the *irregularly irregular* temporal pattern. There are not discernable *P waves*, and the baseline consists of small, irregular *f waves* (Adapted from the Ambulance Technicians Study [2])

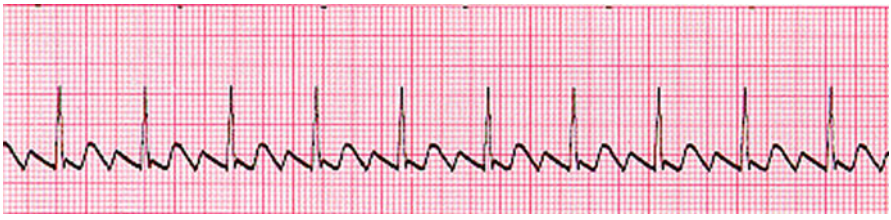

Fig. 29.3 *Atrial flutter* – note the regular, sawtooth appearance of the *atrial flutter waves*. This figure represents atrial flutter with 2:1 ventricular conduction; every 2nd atrial contraction results in a ventricular beat (Adapted from the Ambulance Technicians Study [2])

29.3). The heart rate was controlled with additional esmolol 50 mg, metoprolol 5 mg, and verapamil 10 mg. Once the pulse was in the 80s–90s bpm, the patient's anesthesia was eliminated, and he was extubated uneventfully and brought to the postanesthesia care unit (PACU). Vital signs in the PACU were O_2 saturation 100 % on 8-L face mask, pulse 102 bpm (atrial fibrillation on EKG), BP 153/74 mmHg, respiratory rate (RR) 14 bpm, and temperature 97.4 °F. Postoperative labs were significant for serum potassium of 2.9 mmol/dL, and supplemental potassium was given in the PACU.

The patient remained stable in the PACU and eventually spontaneously reverted to sinus rhythm in the 70s bpm. He denied any chest pain, shortness of breath, or recall of intraoperative events. He was discharged home on the second postoperative

day, with an increased dose of atenolol and instructions to follow up with his cardiologist.

Lessons Learned

L-1. What is a MET? Why do we care if a patient can achieve four METS without symptoms of chest pain or shortness of breath?

MET is short for a *metabolic equivalent*. Briefly, it is an expression of basal metabolic rate or, more specifically, basal oxygen consumption where one MET equals 3.5 mL O_2/kg/min [3, 4]. When assessing functional capacity preoperatively, anesthesiologists ask patients about various life task or exercise capabilities. Different tasks require varying amounts of work/oxygen consumption. By obtaining information on a patient's activity, the anesthesiologist can gain knowledge about their body's tolerance to work. This is important because patients who cannot achieve 4 METS of work are considered to have poor functional capacity and have a relatively higher risk of perioperative cardiovascular morbidity. Conversely, patients who can achieve 10 METS of activity have excellent functional capacity and are at very low risk for perioperative cardiac events, even in the presence of preexisting stable cardiac disease or risk factors [5].

Table 29.1 lists various leisure activities and their MET values. Because many patients do not regularly participate in leisure sports, Table 29.2 provides MET values for common household and workplace activities.

L-2. Describe the pathophysiology, physical exam signs, and EKG characteristics of atrial fibrillation.

With atrial fibrillation, regular sinus node impulses are overwhelmed by rapid, random, electrical discharges produced by areas of irritable atrial tissue. It is thought that atrial fibrillation is a result of atrial fibrosis with loss of atrial muscle tissue. Fibrotic change can be a result of aging, chamber dilatation, or inflammatory processes.

The predominant physical finding of atrial fibrillation is an "irregularly irregular" pulse, with or without tachycardia. Cardiac auscultation may reveal variable intensity of the first heart sound. The jugular venous pulse will notably lack an *a* wave, as there is no organized atrial contraction with atrial fibrillation. Atrial fibrillation with rapid ventricular response may also present with hypotension or hypertension, depending on the individual patient's comorbidities.

The 12-lead EKG is the diagnostic test of choice for atrial fibrillation. The chaotic atrial activity will be evidenced by rapid, small, irregular deflections from the electrical baseline, without visible *P* waves (Figs. 29.1 and 29.2). These small deflections, known as *f* waves, are best seen in lead V_1, II, III, and aVF and may resemble a "ragged baseline." Sometimes, the *f* waves may be difficult to discern, and the baseline may appear flattened. Ventricular QRS complexes are irregularly spaced and can be narrow or widened, depending on the state of the ventricular conducting system and the bundle of His [6].

It is easy to confuse atrial fibrillation with atrial flutter, another supraventricular tachyarrhythmia. Atrial flutter will appear as a more regular rhythm with a "saw-tooth" atrial baseline (Fig. 29.3).

L-3. What are the symptoms of atrial fibrillation in an awake patient?
The awake patient developing atrial fibrillation will likely present with a history of palpitations or irregular fluttering sensation in the chest. Often these symptoms are accompanied by a sense of anxiety.

If the atrial fibrillation causes a drop in blood pressure, patients may report a history of light-headedness, dizziness, or syncope [7].

Sometimes symptoms will occur only with exertion, including shortness of breath, weakness, and chest pain.

In some cases, a patient's comorbidities can make atrial fibrillation a devastating condition, resulting in congestive heart failure with profound shortness of breath, severe hypotension with syncope, or angina. Examples of such comorbid conditions include coronary stenosis (rapid ventricular rate can cause demand ischemia) and conditions associated with impaired left ventricular diastolic filling (such as severe left ventricular hypertrophy with diastolic heart failure, aortic stenosis, or hypertrophic obstructive cardiomyopathy); with such conditions, the left ventricle does not effectively passively fill during diastole. Consequently, atrial contraction plays a more important role in achieving adequate left ventricular end-diastolic volume and, subsequently, stroke volume. Loss of atrial contraction with new-onset atrial fibrillation in such circumstances can result in a sudden drop in cardiac output and profound hypotension.

L-4. What are the risk factors for the development, or triggering, of atrial fibrillation?
There are numerous risk factors and causes of atrial fibrillation. It is useful to break down predisposing factors into categories, which helps to separate chronic conditions from reversible ones and may make differential diagnosis more manageable. Table 29.3 lists causes of atrial fibrillation [7].

Of the categories listed in Table 29.3, the patient in this case had a history of *hypertension*, one of the most common conditions associated with predisposition to atrial fibrillation. His EKG showed *left axis deviation*, consistent with probable *left ventricular hypertrophy*, which is a risk factor for diastolic heart failure and elevated pressure in the pulmonary circulation. Finally, the patient was on diuretics and potassium supplementation, so that he may have been at risk to be hypokalemic, despite a "low-normal" potassium value preoperatively.

L-5. How is acute atrial fibrillation evaluated and managed?
The first step an anesthetist should take upon recognition of *any* rhythm change is to assess all vital signs to see if the new rhythm is hemodynamically stable or unstable (*see* Fig. 29.4). Unstable atrial fibrillation is associated with hypotension and perhaps a drop in end-tidal carbon dioxide (reflecting decreased cardiac output). If congestive heart failure occurs as a result of unstable atrial fibrillation, jugular venous distention may be present, and there may be oxygen desaturation secondary to diminished cardiac output and pulmonary edema. In a conscious patient,

Table 29.3 Causes of atrial fibrillation

Category of cause	Specific conditions
Atrial dilation – any process that chronically increases atrial pressure or volume can lead to dilated atrial walls with consequent fibrotic change	Coronary artery disease Hypertension with left ventricle hypertrophy Valve disease (mitral stenosis/regurgitation) Hypertrophic cardiomyopathy Pulmonary hypertension Intracardiac thrombi or tumor
Inflammatory and infiltrative – disease states that cause structural change to atrial wall tissue with resultant fibrosis of obliteration of muscle mass	Pericarditis Myocarditis Amyloidosis Sarcoidosis Age-induced atrial fibrosis Infections, pneumonia
Endocrine and electrolyte	Hyperthyroidism Pheochromocytoma Hypokalemia Hypomagnesemia
Drugs	Alcohol Caffeine
Neurogenic causes	Stroke Subarachnoid hemorrhage
Idiopathic atrial fibrillation *Familial*	

congestive heart failure would present as acute shortness of breath. If there is cardiac ischemia associated with the arrhythmia, chest pain may be present and EKG ST segment changes may be seen, though these can be difficult to rely on if there is a preexisting bundle branch block or if the ventricular rate is too rapid to easily interpret the ST segments.

If new-onset atrial fibrillation is deemed *unstable*, then immediate *direct current electrical cardioversion* is indicated [5]. Situations that fall into this category include:

- Ongoing myocardial ischemia
- Severe hypotension unresponsive to pharmacologic intervention
- Acute congestive heart failure
- Angina or severe shortness of breath in an awake patient [8]

For patients with new-onset atrial fibrillation who do not meet the above criteria for immediate electrical cardioversion, management should focus first on *controlling the HR* (*see* Table 29.4). Excessive ventricular tachycardia can impair left ventricular filling by decreasing time spent in diastole, which can lead to a drop in cardiac output and, consequently, blood pressure. Additionally, tachycardia creates

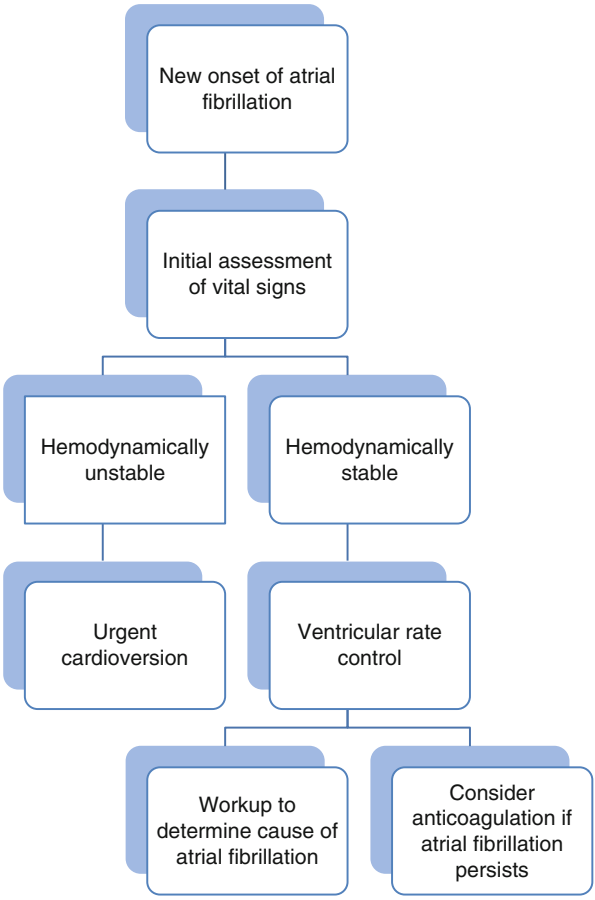

Fig. 29.4 Management algorithm for new-onset atrial fibrillation (Adapted from [8])

increased oxygen demand by the myocardium, increasing the likelihood of ischemia. In the OR setting, treatment is generally initiated with fast-acting intravenous agents to quickly lower the ventricular response rate to atrial fibrillation. Drugs commonly used include:

- *Beta antagonists*: esmolol, metoprolol, and propranolol
- *Calcium channel blockers*: diltiazem or verapamil

The above classes of drugs decrease heart rate and cardiac output. They should be used with caution in the setting of known heart failure or severe left ventricular dysfunction, as further decreases in contractility might hurt such patients, worsening the heart failure. Another type of patient for whom beta-blockers and calcium channel blockers may be harmful is the patient with a known accessory pathway such as Wolff-Parkinson-White syndrome. Blocking AV nodal conduction in such patients can lead to favoring of the accessory pathway and worsening reentrant tachycardia.

Table 29.4 Intravenous agents for acute heart rate control of atrial fibrillation

Drug	Loading dose	Onset	Maintenance dose	Major side effects
Drugs effective for acute heart rate control of atrial fibrillation in patients who do NOT have heart failure or an accessory pathway				
Esmolol	0.5 mg/kg over 1 min	3–5 min	0.06–0.2 mg/kg/min	Hypotension, heart block, bradycardia, bronchospasm, heart failure
Metoprolol	2.5–5 mg bolus over 2 min; up to three doses	5 min	Not applicable	Hypotension, heart block, bradycardia, bronchospasm, heart failure
Propranolol	0.15 mg/kg	5 min	Not applicable	Hypotension, heart block, bradycardia, bronchospasm, heart failure
Diltiazem	0.25–0.75 mg/kg	2–7 min	5–15 mg/h infusion	Hypotension, heart block, bradycardia, heart failure
Verapamil	0.075–0.15 mg/kg over 2 min	3–5 min	Not applicable	Hypotension, heart block, bradycardia, heart failure
Evidence favors the efficacy of the following drug for acute rate control of atrial fibrillation in patients with a known accessory pathway				
Amiodarone	150 mg over 10 min	Days	0.5–1.0 mg/min	Pulmonary toxicity, worsened arrhythmia, liver damage
Drugs useful for acute rate control of atrial fibrillation in patients with heart failure but NO accessory pathway				
Digoxin	0.25 mg every 2 h, up to 1.5 mg	2 h	0.125–0.25 mg daily	Digitalis toxicity, heart block, bradycardia
Amiodarone	150 mg over 10 min	Days	0.5–1.0 mg/min	Pulmonary toxicity, worsened arrhythmia, liver damage

Data from [8]

For patients with known heart failure, *amiodarone* or *digoxin* may be better choices for rate control in that they do not depress myocardial contractility as much as beta antagonists and calcium channel blockers. Amiodarone is also safe in the setting of a known accessory pathway [8].

If tachycardia is initially accompanied by mild to moderate hypotension, then *phenylephrine*, an alpha-1 agonist, may be an appropriate drug for initial treatment.

This drug increases blood pressure by increasing vascular tone. The increased pressure can cause an increase in vagal tone via autonomic reflexes, with resultant decrease of heart rate.

Once new-onset atrial fibrillation is determined to be hemodynamically stable and heart rate control is successfully achieved, it is then appropriate to order tests and labs to find potential underlying causes of the arrhythmia [9]. Workup includes:

- 12-lead EKG – presumably this has already been done to establish the diagnosis of atrial fibrillation. But a formal interpretation should be made looking for evidence of potential causes, such as left ventricular hypertrophy or myocardial ischemia.
- Serum electrolytes to check for hypokalemia or hypomagnesemia.
- Complete blood count.
- Thyroid function tests.
- Chest x-ray to screen for cardiomegaly or chronic obstructive pulmonary disease
- Echocardiogram to evaluate for atrial or ventricular dilation, valvular lesions, ventricular function, pericarditis, and pulmonary hypertension.
- Drug and alcohol screen in a young patient without other medical history.
- Other blood tests may be indicated based on the rest of the clinical history and physical exam.

Once initial tests have been ordered, it is important to communicate with the patient's primary caregiver to inform them of the new atrial fibrillation. At this point, the decision may be made to involve a cardiology consultant to assist in guiding further management of the arrhythmia.

If atrial fibrillation does not spontaneously resolve, patients may need to be started on anticoagulation therapy with heparin or warfarin [9], to prevent clot formation in the stagnant atria. This decision is made after the acute intraoperative management of new-onset atrial fibrillation and will not be discussed further.

L-6. What types of patients might become most unstable with sudden onset atrial fibrillation? How would they be treated?

When one's heart goes from sinus rhythm to atrial fibrillation, there is no longer a coordinated atrial contraction prior to ventricular contraction and closure of the tricuspid and mitral valves. As a result, any ventricular filling is dependent on passive filling during diastole. Therefore, patients with impaired diastolic filling will have a greater drop in left ventricular end-diastolic volume and, subsequently, stroke volume, and cardiac output. Acute drops in cardiac output can result in hypotension, which may predispose to coronary ischemia. The following are conditions associated with decreased passive diastolic filling, and therefore risk factors for rapid hemodynamic instability should atrial fibrillation occur:

- Aortic stenosis
- Mitral stenosis
- Severe left ventricular hypertrophy with diastolic heart failure
- Severe right ventricular hypertrophy secondary to pulmonary hypertension or chronic obstructive pulmonary disease (COPD)
- Hypertrophic obstructive cardiomyopathy (HOCM)

In addition to the above list, patients with known coronary artery stenosis are at risk for demand ischemia in the setting of atrial fibrillation with tachycardia, as increased heart rate will increase myocardial oxygen demand.

Patients who become hemodynamically unstable with new-onset atrial fibrillation should be treated via electrical cardioversion to restore a normal sinus rhythm.

L-7. How would one emergently convert atrial fibrillation back to sinus rhythm?

In the event of new-onset, *unstable* atrial fibrillation, the treatment is to restore sinus rhythm as quickly as possible. This is accomplished via *direct*, synchronized, *electrical cardioversion* at 150 J when using a biphasic defibrillator.

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Chapter 30
Valvular Disease

Timothy M. Maus

A 60-year-old, 81 kg, and 66-in. tall male presented for redo sternotomy (**L-1**) for aortic valve and mitral valve replacement secondary to rheumatic disease with critical aortic stenosis (AS) (**L-2**) and severe mitral stenosis (MS) (**L-3**). His past medical history was significant for coronary artery disease (CAD), congestive heart failure (CHF) with pulmonary hypertension (pHTN), systemic hypertension, dialysis dependent end-stage renal disease, systemic hypertension, type II diabetes mellitus, and stroke. Detailed cardiac history included previous CABG with patent left internal mammary artery (LIMA) to left anterior descending (LAD) artery and occluded saphenous vein graft (SVG) to posterior descending artery (PDA), with severe pHTN of 90/41, right atrial pressure (RAP) of 30, pulmonary capillary wedge pressure (PCWP) of 35, and a cardiac index of 2.16 (**L-4**). Aortic valve area was calculated at 0.89 cm^2 and mitral valve was 1.1 cm^2. Current medications included metoprolol, nifedipine, aspirin, sevelamer, cinacalcet, and sertraline.

After routine monitors and arterial line were placed, the patient was induced intravenously with 5 mg midazolam, 750 mcg fentanyl, 20 mg etomidate, and 100 mg rocuronium. The patient was hyperventilated by bag-mask ventilation. BP and HR were maintained 150s/70s mmHg and 70s bpm, respectively, throughout induction. DL with Macintosh size three blade revealed a grade III view. Decision was made to switch to a Macintosh size four blade. The patient's heart rate after the initial DL increased to 110 bpm and was treated with 30 mg esmolol. While continuing mask ventilation, the HR decreased to 35 bpm (**L-5**) with a decrease in blood pressure to 60/50 mmHg. Twenty mcg of epinephrine was administered, and cardiac surgeons were notified. The circulator prepped the patient's groins, and perfusionist readied the bypass machine. The HR remained 35 bpm with BP of 60/50 mmHg. A second 20 mcg epinephrine bolus was administered with initiation

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of chest compressions (to aid circulation of resuscitation drugs). Without significant response to the small doses of epinephrine, 1 mg epinephrine was administered with an immediate increase of HR to 110 bpm and blood pressure to 205/85. The patient was simultaneously intubated and a combination of inhaled sevoflurane and 10 mg incremental doses of esmolol were administered, returning the blood pressure to baseline. The remainder of the procedure and postoperative course were uneventful. The patient was successfully extubated and discharged on POD 7.

Lessons Learned

L-1: What are perioperative concerns for redo sternotomy?

- A previous sternotomy with significant scar tissue and sternal wires precludes rapid access to the pericardium and heart, thereby increasing the risk of complications with reentry. This lends itself to several concerns for the consultant anesthesiologist. With delayed access to the pericardial space, the ability to rapidly internally defibrillate or cardiovert is removed. Therefore alternatives for arrhythmia treatment must be in place, typically via external defibrillation pads placed prior to induction (Table 30.1).
- In addition to arrhythmia management, delayed access to the pericardium precludes rapid commencement of cardiopulmonary bypass in the event of hemodynamic compromise. Therefore discussion with the cardiothoracic surgeon prior to induction should include the potential for femoral access (wires or cannulae) to allow for femoral-to-femoral bypass should hemodynamic instability on induction occur.
- Beyond delayed access to the pericardium, the approach may be complicated by the scar tissue and its adherence to surrounding structures and repairs. These may include right atrial free wall, prior coronary artery bypass grafts, and specifically right internal mammary artery grafts that cross the midline. Violation of the right atrial free wall or prior surgical repair may result in acute decompensation secondary to hemorrhage or acute ischemia. Therefore knowledge of the prior surgery details will be imperative to the preoperative and preinduction decision making.
- The notable slow access and potential for complications yielding instability prompt the surgeon to utilize an oscillating saw which minimizes although does not eliminate the risk of injury to surrounding structures. Large-bore intravenous access as well as the immediate availability of typed and crossed and checked-in blood in the operating suite is necessary in anticipation of potential complications.

Table 30.1 Perioperative concerns for redo sternotomy

1. Place external defibrillation pads
2. Know if femoral-to-femoral access can be utilized if persistent hemodynamic instability occurs
3. Risk of major bleeding:
(a) Have checked packed red blood cells available in operating room
(b) Obtain large-bore IV access

Table 30.2 Hemodynamic goals in aortic stenosis

Parameter	Desired end point	Problem with greater value	Problem with lesser value
Heart rate	Low end of normal range	↓ Systolic time ↓ Stroke volume ↓ Cardiac output	↓ Cardiac output (fixed stroke volume)
Heart rhythm	Normal sinus rhythm	Atrial fibrillation not tolerated	Bradyarrhythmias not tolerated
Preload	Euvolemia	Pulmonary edema	Hypotension
Afterload	High end of normal range	↓ End organ perfusion with excessive SVR elevation	↓Coronary perfusion pressure
Contractility	Maintained	↑ Myocardial oxygen consumption	Hypotension

L-2: What are hemodynamic goals for aortic stenosis?

- The hemodynamic goals for aortic stenosis relate to the fixed stroke volume due to decreased aortic valve area, as well as the decreased diastolic function secondary to compensatory left ventricular hypertrophy. The goals may be broadly divided into achieving optimal heart rate, heart rhythm, preload, afterload, and contractility (Table 30.2).
- Rate – The HR is often kept at the low end of normal range in patients with aortic stenosis, with particular attention to preventing tachycardia. Tachycardia reduces the amount of time in systole, thereby reducing the amount of blood passing through the stenotic lesion. In addition, tachycardia decreases diastolic filling in a patient with likely decreased diastolic function.
- Rhythm – Normal sinus rhythm is highly desirable, as patients with AS may rely on 40–50 % of their cardiac output from their atrial contraction. This relates to their poor diastolic filling and compliance, where the atrial kick provides a boost in LV volume prior to contraction. Prompt cardioversion may be necessary in the setting of deviation from normal sinus rhythm.
- Preload – Euvolemia is typically the target volume status. Preload reduction is not recommended as the patient is dependent on a fixed stroke volume. Volume loading in a patient with significant diastolic dysfunction may result in pulmonary edema.
- Afterload – The patient with AS often possesses significant left ventricular hypertrophy, and therefore perfusion of the hypertrophied left ventricle is systemic pressure and SVR dependent, and in that respect, the patient is at risk for myocardial ischemia. In addition, a decrease in afterload does not result in an increase in cardiac output (as would normally occur) because the stroke volume is fixed. Thus there is a positive benefit to maintain SVR and no benefit to reducing SVR.
- Contractility – As previously stated, the stroke volume is fixed. Augmenting contractility is unlikely to increase cardiac output; however, augmenting contractility will increase myocardial oxygen demand and lead to potential ischemia.

L-3: What are hemodynamic goals for mitral stenosis?

- The hemodynamic goals for MS relate to the fixed left ventricular inflow as well as elevated left atrial pressure and size. The goals may be broadly divided into

Table 30.3 Hemodynamic goals in mitral stenosis

Parameter	Desired end point	Problem with greater value	Problem with lesser value
Heart rate	Low end of normal range	↓ Diastolic time ↓ Cardiac output	↓ Cardiac output (fixed stroke volume)
Heart rhythm	Normal sinus rhythm	Chronic atrial fibrillation often tolerated	Bradyarrhythmias not tolerated
Preload	Euvolemia	Pulmonary edema	Difficult to reduce left atrial preload
Afterload	Normal range	Does not affect mitral flow	Does not affect mitral flow
Contractility	Maintained	Unrelated to mitral flow	Unrelated to mitral flow

achieving optimal HR, heart rhythm, preload, afterload, and contractility (Table 30.3).

- Rate – The HR is often kept at the low end of normal range in patients with MS, with particular attention to preventing tachycardia. Tachycardia reduces the amount of time in diastole, thereby reducing left ventricular filling in a patient with already decreased LV filling.
- Rhythm – While normal sinus rhythm is desirable, left atrial distension often results in chronic atrial dysrhythmias. Cardioversion is often unsuccessful because severe left atrial distension causes atrial dysrhythmia reoccurrence. In addition, chronic MS leads to left atrial enlargement and stasis of blood, significantly increasing risk of thrombus generation. Cardioversion therefore must be used with caution to prevent thrombus dislodgement and systemic embolization.
- Preload – Mitral stenosis leads to a chronic state of left atrial volume overload and a propensity for pulmonary edema despite an underfilled left ventricle. Augmenting preload is not beneficial as the left atrium is already distended and only leads to further pulmonary edema.
- Afterload – Normal afterload is maintained as the lesion is prior to the left ventricle, and therefore altering afterload will not change the mitral valve inflow.
- Contractility – Similar to afterload, the lesion is prior to the left ventricle, and therefore augmentation does not particularly increase cardiac output in mitral stenosis.

L-4: What is the clinical significance and anesthetic implications of pulmonary hypertension?

- The right heart is typically a low-pressure system, functioning as a conduit for venous blood returning from the head and body to the lungs for gas exchange. The right ventricle as a thin small chamber is well suited to accommodating and transferring volume to the low-pressure circuit and is quite sensitive to afterload increases. Pulmonary hypertension, which causes significant right ventricular afterload and RV strain, can lead to chronic right ventricular dysfunction and failure. Decreased right ventricular cardiac output yields an underfilled left ventricle and systemic hypoperfusion.

Table 30.4 Pharmacologic treatment of intraoperative bradycardia

Glycopyrrolate	0.2–1 mg IV bolus
Atropine	0.4–1 mg IV bolus
Dopamine	5–10 μg/kg/min IV infusion
Epinephrine	10 μg–1 mg IV bolus

- Reversible causes of increased PVR should be avoided. These include the avoidance of hypoxia, hypercarbia, and hypothermia. In severe pHTN, patients with significant RV dysfunction, the small increase in PVR caused by nitrous oxide administration may lead to further RV decompensation. Therefore, nitrous oxide may be best to avoid in such patients. Maintenance of systemic BP prevents decreased coronary perfusion pressure to the already compromised right ventricle and further decompensation.

L-5: What are the causes and treatment of intraoperative bradycardia?

- Intraoperative bradycardia may result from several systemic disturbances including hypoxia, hypothermia, excessive vagal tone (laryngoscopy, surgical stimulation, abdominal insufflation), drug toxicity (beta-blocker, calcium channel blocker, neostigmine), iatrogenesis (oculocardiac reflex, carotid massage), and myocardial ischemia. Underlying cardiac disease including permanent conduction abnormalities must be considered (Table 30.4).
- Beyond identifying the cause of bradycardia, acute treatment for symptomatic bradycardia typically is divided into chemical and electrical modalities. Chemical means include glycopyrrolate, atropine, dopamine, and epinephrine. Electrical means include both transcutaneous as well as transvenous pacing.

Recommended Reading

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Part III
Obstetrics-Related Cases

Chapter 31
Labor Epidural with Unrecognized Dural Puncture, Causing High Sensory Block, Hypotension, Fetal Bradycardia, and Post-dural Puncture Headache

Thomas L. Archer

A 30-year-old woman, gravida 2 para 1, was admitted in labor at 40 weeks 4 days estimated gestational age with spontaneous rupture of membranes. She had had an effective and uncomplicated epidural for her first delivery. Additional past medical history, vital signs, and physical exam were unremarkable. Oxytocin augmentation of labor was begun; the pain of contractions increased and an anesthesiologist was called at 0140. After evaluation and informed consent, he attempted to place an epidural catheter at the L3–4 interspace. Loss of resistance to saline was obtained, but the epidural catheter could not be passed beyond the tip of the Tuohy needle, despite additional injections of saline, rotation of the needle, and the use of a stiffer catheter. The catheter was finally placed at L2–3 but only after additional repositioning of the needle, injection of extra saline, and after asking the patient to increase her spinal flexion while the needle was in the epidural space. Despite these difficulties, no abundant fluid return was noted at any time during placement of the Tuohy needle (**L-1**, **L-2**, **L-3**).

At 0208 a test dose of 3 mL lidocaine 1.5 % with epinephrine 1:200,00 was given through the catheter and was described as negative for signs of intravenous or intrathecal injection. Blood pressure (BP) at that time was 111/68 mmHg, and heart rate was 66 beats per minute (bpm). Another 3 mL of the same solution was given at 0212. At 0215 the patient then positioned herself in the supine position with a tilt to the left. She reported that her legs felt "very heavy," and she denied feeling anything during a uterine contraction. She reported that the epidural was working "faster" than it had with her first delivery, although she was still able to move her legs (**L-4**, **L-5**). The patient's blood pressure at that time was 81/51 mmHg, and heart rate was 53 bpm. At 0219 a continuous patient-controlled epidural analgesia (PCEA) infusion of bupivacaine 0.1 % and fentanyl 2 μg/mL was started at 10 mL/h, with a patient-controlled bolus of 5 mL, a "lockout interval" of 15 min, and a maximum hourly

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dose of 25 mL (**L-6**). Repeat BP was 84/56 mmHg while HR was 86 bpm. The anesthesiologist again confirmed that the patient could move her legs before he left the room at approximately 0225 (**L-7**).

From 0221 until 0400, the recorded BPs ranged from 86 to 107 mmHg systolic and from 48 to 67 mmHg diastolic.

At 0430 the fetal HR decreased to 60 bpm and the patient was placed in the full left lateral decubitus position (**L-8**). Oxytocin was discontinued and oxygen was applied. The obstetrician and anesthesiologist were called. The patient stated that her hands were numb. The BP was 80/41 mmHg, and the heart rate was 78 bpm. The anesthesiologist gave phenylephrine 100 μg intravenously, and the BP increased to 124/70 mmHg at 0439. At 0440 the anesthesiologist noted that the patient was breathing well and that she had full strength in her hands. The epidural infusion was stopped, and the anesthesiologist explained to the patient, nurse, and father of the baby that it was possible that some of the epidural infusion had been going into the spinal fluid, producing an effect that was greater than normal. At 0455 a fetal scalp electrode was applied. At that time the patient's hands were no longer numb. She could feel cold sensation to ice down to the T2 dermatome, and her vital signs were described as "stable."

From 0500 until 0630, the patient was pain free. Her BP ranged between 88–136 mmHg systolic and 52–79 mmHg diastolic, and there were no more episodes of fetal bradycardia. At 0635 she was described as "shaking from the epidural" and feeling "pressure but no pain." At 0654 the BP was 134/84 and the HR was 71 bpm. At 0700 (change of shift), the outgoing anesthesiologist reported to the anesthesiologist who was relieving him that it appeared to him that the epidural was functioning "too well" and that there might be a hole in the dura which was producing an exaggerated effect by allowing some of the epidural dose to directly enter the cerebrospinal fluid through the hole.

At that same time, the new nurse noted that the patient was having pain with uterine contractions. The newly arrived anesthesiologist was called, and she was requested to "turn the epidural back on." At 0735, before any injection through the "epidural" catheter, fetal bradycardia recurred, and the patient was placed in various positions, ending up with her on her hands and knees. Terbutaline 0.25 mg was given subcutaneously for possible uterine hypertonus (**L-9**). At 0739 3 mL of bupivacaine 0.25 % plus fentanyl 10 μg was injected through the "epidural" catheter. After this the patient became comfortable within a few minutes and her hands became numb (**L-10**). The patient was then checked; her cervix was fully dilated and she began to push with each uterine contraction. At 0845 the patient delivered a male infant with Apgars of 6 and 9 after an episode of shoulder dystocia.

At 0930 the patient sat up after delivery to breast feed her infant and complained of a frontal and occipital headache (**L-11**). The headache persisted throughout the day, diminishing in intensity when she lay flat and increasing when she sat upright. By the next morning (24 h after delivery), the headache had disappeared and the patient went home with her baby. The following morning (48 h after delivery), the headache had returned and the patient spoke by phone with the anesthesiologist on

call. The patient came to labor and delivery (L&D) for evaluation (**L-12**) and received an epidural blood patch (EBP) approximately 54 h after delivery (**L-13**, **L-14**). The EBP greatly diminished the intensity of the headache, and the patient went home with instructions to continue to take oral analgesics and to call the obstetric (OB) anesthesia team if the headache recurred. The headache did recur the following morning (roughly 72 h after delivery) and was "worse than ever." She said she felt fine except for her postural headache and was eating well, showering, taking care of the baby, and was able to perform all of her normal activities. The patient denied fever, chills, anorexia, stiff neck, photophobia, or back pain or tenderness, although she did say her back felt slightly stiff. The patient was managed by phone, although she was offered the option of coming back to the hospital for further evaluation and a possible second EBP or neurological imaging (**L-15**, **L-16**). At home the patient took ibuprofen and hydrocodone plus acetaminophen and received adequate pain relief. The anesthesiologist called her daily, and on the morning of the sixth day after delivery, she reported that her headache had disappeared. The patient expressed appreciation for the careful follow-up.

Lessons Learned

L-1: Unrecognized dural puncture
The absence of a "gush" of cerebrospinal fluid (CSF) through the Tuohy needle does not exclude the possibility of unrecognized dural puncture. Likewise, a negative test dose or aspiration does not eliminate this possibility. *Vigilance and eternal skepticism about one's "epidural" are essential to safe OB anesthesia practice*.

L-2: Reasons for unrecognized dural punctures
Dural punctures are sometimes created and not recognized for the following reasons:

(a) A plug of tissue or a clot at the tip of the Tuohy needle prevents the return of CSF through the needle, despite the creation of a hole in the dura.
(b) The Tuohy needle punctures the dura but then the tip is moved out of the CSF.
(c) The Tuohy needle creates an incomplete tear of dura which later becomes complete due to maternal straining or pushing.
(d) Rotation of the Tuohy needle in the epidural space may damage the dura.

L-3: Proper use of ultrasound might reduce the rate of unintentional dural puncture
Ultrasound *may* be helpful in decreasing the incidence of unintended dural puncture by showing the approximate depth of the ligamentum flavum, but this assertion is unproven and controversial. More studies are required in this area. In the future, new technology, such as ultrasound or optical imaging directly from the tip of a needle, may be able to reduce inadvertent dural puncture.

L-4: Complications of unrecognized dural puncture
Accidental (and often unrecognized) dural puncture is a common complication of epidural placement and can lead to the following complications:

(a) High sympathetic block causing maternal hypotension, nausea, vomiting, and loss of consciousness
(b) High motor block causing maternal weakness, with possible aspiration, respiratory paralysis and death
(c) Acutely decreased placental perfusion and fetal hypoxia ("fetal distress," as manifested by fetal heart rate decelerations)
(d) Post-dural puncture headache

There are many different acceptable regimens for the initial dosing of a labor epidural, but it is abnormal for the patient to get complete and rapid pain relief and weak legs from a total epidural dose of 6 mL of lidocaine 1.5 % with epinephrine 1:200,000.

L-5: An "epidural" that works "too well": a red flag
An "epidural" block which develops more rapidly than normal should be a "red flag," alerting the anesthesiologist to a potentially fatal situation, particularly in the middle of the night when everyone's guard is down. The anesthesiologist needs to stay with the patient until she is sure that a high block or hypotension is not developing. This time can be used for doing paperwork, setting up the infusion pump, and establishing additional rapport with the patient and family members.

L-6: These are typical labor epidural infusion settings

L-7: Epidurals that work too well are a great danger
Despite prompt hypotension and rapid block onset, the resident was apparently reassured by the fact that the patient could still move her legs. He started PCEA with routine settings, left the room 17 min after the test dose, and did not come back to check on the patient. These were errors in judgment that could have resulted in maternal or fetal death.

L-8: Aortocaval compression and other threats to fetal oxygenation
The effects of sympathectomy on maternal BP and cardiac output (venous return) depend greatly upon maternal position and attendant aortocaval compression. Maternal hypotension or fetal HR decelerations require us to immediately place the laboring woman onto one side (preferably left side down). In theory, the laboring patient should never be supine, especially once an epidural block is in place. In practice, this rule is frequently ignored, for ease in fetal monitoring ("finding the baby"), for patient comfort and convenience, and for Foley placement, vaginal exams, intrauterine pressure catheter (IUPC) placements, and so forth. Some patients and their fetuses will tolerate the mother's lying supine after an epidural is in place; others will not. As anesthesiologists we must resist a tendency toward complacency about maternal positioning once an epidural is in place.

As shown in Figs. 31.1, 31.2, 31.3, 31.4, and 31.5, a labor epidural, by lowering aortic BP, can be the "last straw that breaks the camel's back" in terms of fetal

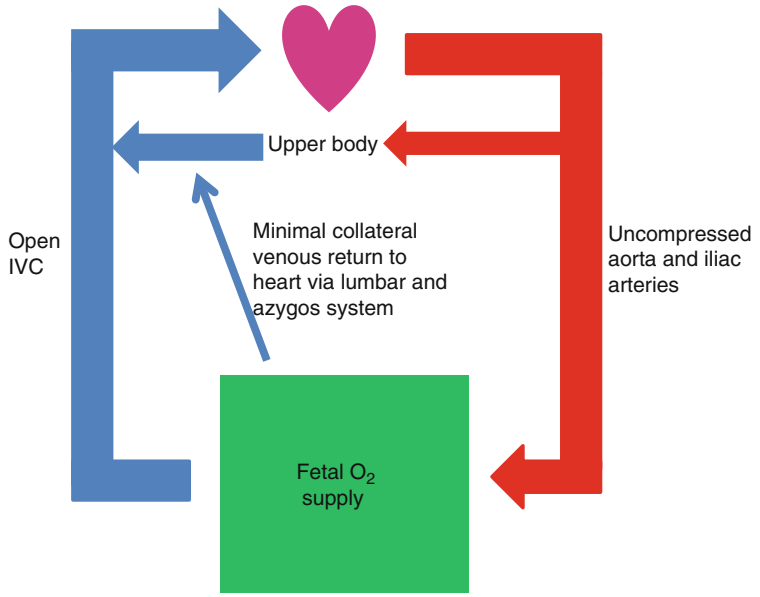

Fig. 31.1 With no uterine contractions, no aortocaval compression, no placental vascular disease, and with normal aortic BP, the fetus receives plenty of oxygen. Since the inferior vena cava (*IVC*) is wide open, minimal amounts of uterine venous blood flow back to the heart via the collateral channels (lumbar veins, vertebral plexus, and azygos vein)

oxygenation when placental perfusion and fetal oxygenation are already precarious due to some or all of the factors shown in Figs. 31.2, 31.3, and 31.4 (uterine contractions, aortocaval compression, and placental vascular disease). Hence, anesthesiologists can easily precipitate "fetal distress" in patients. We must be vigilant to prevent or correct uteroplacental perfusion problems over which we have direct control, such as aortic hypotension and aortocaval compression. Also, the oxytocin infusion rate is not under our direct control, but if the patient is receiving oxytocin and there is "fetal distress," the oxytocin should be stopped.

L-9: Uterine Hyperstimulation is a sometimes overlooked cause of fetal hypoxemia and "distress"

Anesthesiologists often assume that fetal bradycardia is due to maternal hypotension and vena caval compression, and this is often the case. However, we must not overlook the possibility that fetal hypoxia can be caused by uterine hyperstimulation and tetany, particularly when labor is being augmented by oxytocin infusion. Maternal hypotension is treated with left uterine displacement, vasopressors, fluid, and oxygen. Uterine tetany is treated with the uterine smooth muscle relaxants terbutaline or nitroglycerin.

L-10: Intrathecal "epidural" catheter

The anesthesiologist coming on duty had been told that the "epidural" catheter was acting as though it were intrathecal or near a tear in the dural, resulting in

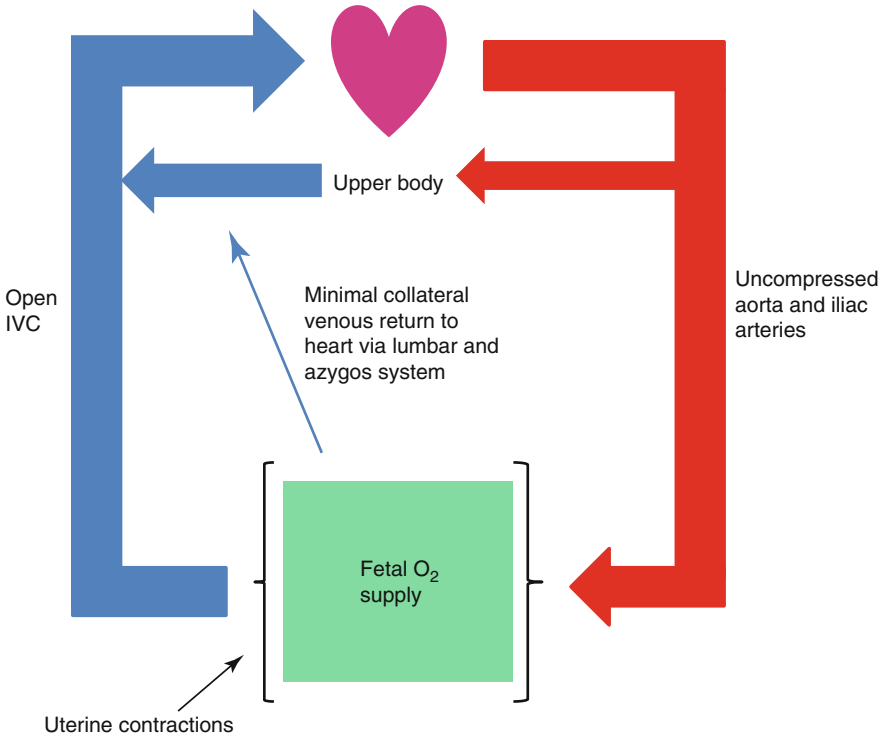

Fig. 31.2 Uterine contractions periodically cut off maternal blood flow to the placenta. Excessive frequency or duration of uterine contractions ("hyperstimulation" or "uterine tetany," often caused by oxytocin augmentation of labor), can cause the fetus to become hypoxic, but under normal circumstances, the fetus has sufficient reserve to tolerate the periodic oxygen supply interruptions inherent in uterine contractions

abnormally low-dose requirements for analgesia but otherwise working acceptably. She chose to use the catheter for analgesia but reduced the dose drastically and was vigilant for a high block. A common bolus dose for severe pain near delivery—assuming that the epidural catheter was functioning normally—might be 10 mL of either lidocaine 1 % plain or bupivacaine 0.25 %.

L-11: Post-dural puncture headache after an "epidural"
Development of a classic PDPH within a few hours of epidural block placement is a sign of unrecognized dural puncture with a large needle. PDPH due to a small needle (e.g., 25 gauge) generally takes 24 h to develop.

L-12: Post-dural puncture headache: logistics and management
The new mother has an infant to care for and may have other health problems or pain related to the delivery. Your institution may want to facilitate the readmission of patients with suspected post-dural puncture headache (PDPH) so that these patients do not have to wait long periods of time to be seen in the emergency

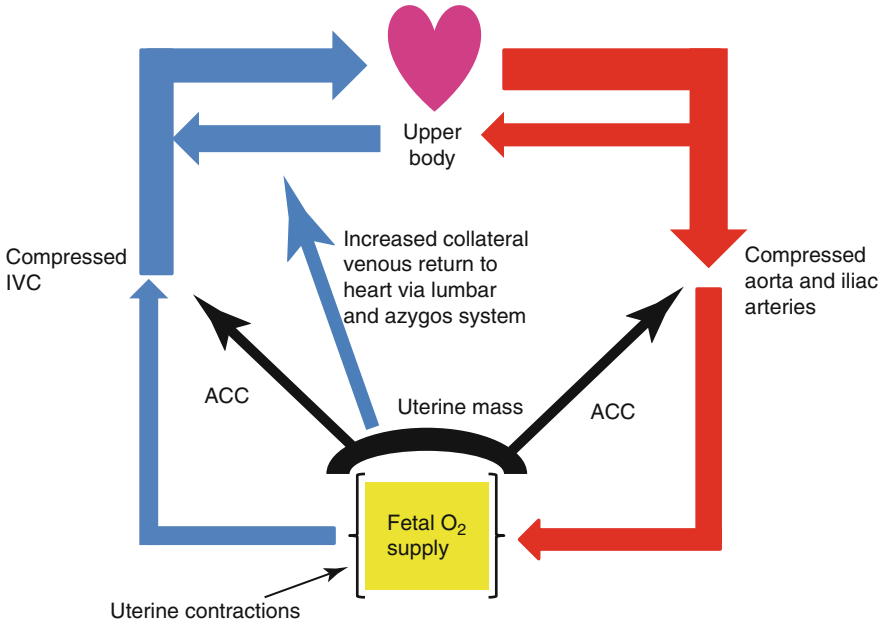

Fig. 31.3 Aortocaval compression (*ACC*) is an additional threat to the fetus' oxygen supply. Compression of the aorta can reduce uterine artery pressure below aortic pressure, and compression of the inferior vena cava (*IVC*) increases uterine venous pressure, thereby reducing uterine perfusion pressure. Additionally, global venous return is reduced, which reduces cardiac output and activates the sympathetic nervous system in an attempt to maintain aortic pressure. During normal gestation, collaterals develop to carry uterine venous blood back to the heart in the presence of IVC compression or obstruction. This collateral blood flow normally occurs via the ovarian veins, ascending lumbar veins, vertebral venous plexus, and azygos vein, but these pathways are quite variable

department. If a PDPH is diagnosed and an EBP is decided upon, consideration should be given to having an experienced anesthesiologist perform the placement of the epidural needle for the blood patch.

L-13: Epidural blood patches involve risks and inconvenience
Blood patches are not always successful and present their own risks and inconvenience. Patients are often hesitant to undergo a second intervention in their back with a needle.

L-14: Post-dural puncture headache: rule out other causes of the headache!
PDPH causes significant disability and inconvenience. In speaking with the patient, a PDPH should not be minimized or disregarded. Careful, sympathetic, and objective follow-up is important. There are many possible causes of peripartum headache, only one of which is dural puncture, and we must rule out other causes of headache (e.g., tension, caffeine withdrawal, sinusitis, migraine, hypertension, subdural hematoma, dural sinus or cortical vein thrombosis, arteriovenous malformation [AVM], brain tumor, meningitis).

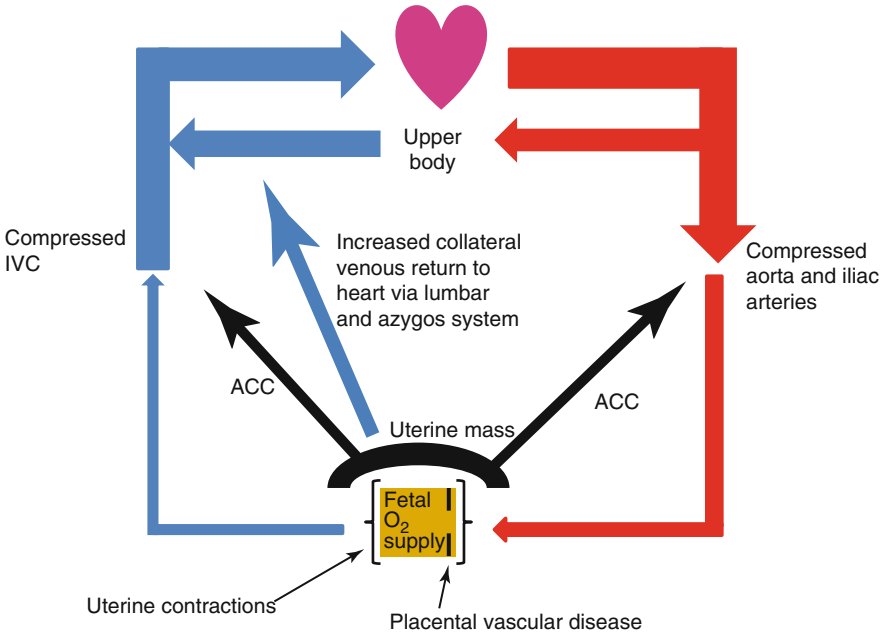

Fig. 31.4 If the patient has placental vascular disease (commonly caused by preeclampsia, abruption, or diabetes), placental perfusion and fetal oxygenation become even more precarious

L-15: Post-dural puncture headache usually goes away on its own
Most episodes of PDPH will disappear on their own within a few days to a few weeks, although longer durations have been reported.

L-16: EBP: wait at least 24 h after the onset of the headache. If it fails, review your diagnosis
Most authorities recommend waiting at least 24 h after the onset of PDPH before performing an EBP. If the first EBP is unsuccessful, a second may be performed, but the optimum interval between the two EBPs is unknown. Two EBPs can be performed before neuroimaging studies are performed but not three. Atypical features or clinical deterioration should prompt neuroimaging of the head.

Figures 31.1, 31.2, 31.3, 31.4, and 31.5 illustrate the concept that the fetus is at the end of a long chain of processes which transport oxygen from the maternal to the fetal circulation. A break in any of the links of this chain will cause the fetus to experience hypoxic stress, manifest as loss of fetal HR variability, fetal bradycardia, and, eventually, death. As shown in the following schematic diagrams, partial interruptions of various oxygen transport processes can summate to cause a hypoxic crisis for the fetus. A given single stress (such as hypotension due to an epidural) may merely be the "straw that breaks the camel's back," if other links of the chain are already weakened.

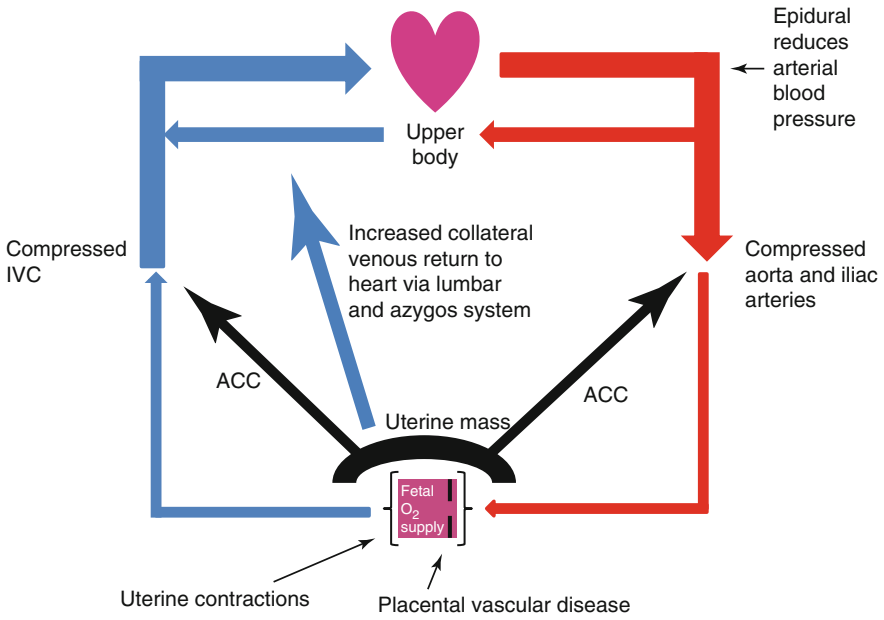

Fig. 31.5 When an epidural is administered in the setting of one or more preexisting threats to fetal oxygenation, it can serve as the "straw that breaks the camel's back" and lead to rapid deterioration of the fetus—manifest as fetal bradycardia or loss of fetal HR variability. The anesthesiologist must be particularly vigilant of avoiding hypotension and ACC in patients who have preexisting threats to fetal oxygenation, such as frequent uterine contractions or placental vascular disease

Chapter 32
Acute Pulmonary Dysfunction Immediately After Cesarean Delivery Under General Anesthesia

Thomas L. Archer

A 28-year-old woman, 65 in. tall and weighing 220 lb (BMI 37), gravida 3 para 1, presented at 38 2/7 weeks estimated gestational age with a small amount of vaginal bleeding. She had a primary cesarean section 4 years earlier for macrosomia, received 14 units of blood for postpartum hemorrhage, and was diagnosed with von Willebrand disease type 2b (**L-1**). Ten weeks prior to admission, prothrombin time (PT), factor VIII, and von Willebrand factor (vWF) *antigen* were normal, but vWF *activity* was low at 18 % of normal. Activated partial thromboplastin time (aPTT) and Ivy bleeding time were prolonged at 43.1 s and greater than 20 min, respectively (**L-2**). Automated platelet count was reported as "clumping." The peripheral smear revealed abundant platelet clumping in the feathered edge, with decreased platelets in the regular part of the smear (**L-3**).

The morning before surgery, the patient received vWF/factor VIII concentrate (Humate-P, 6,000 U), after which the vWF activity normalized. The decision was made to proceed with cesarean section and to have blood and platelets available for transfusion. General anesthesia was planned because of the decreased platelets seen on peripheral smear and the history of hemorrhage. Physical examination was unremarkable.

A second IV was placed and during 3 h prior to surgery, the patient inadvertently received 3 L of lactated ringers solution through the two IVs. She urinated three times before going to the operating room. Despite this excessive fluid administration, the patient's lungs were clear to auscultation immediately prior to induction, she did not complain of any respiratory distress, and her oxygen saturation on room air was 97 %. A Foley catheter was placed before induction of anesthesia and drained 300 mL of pale urine. Cefazolin 2 g was administered and no rash or respiratory symptoms were noted at that time. In addition to routine monitors, the Aesculon Electrical Cardiometry system (Cardiotronic, Inc., La Jolla, California) was applied.

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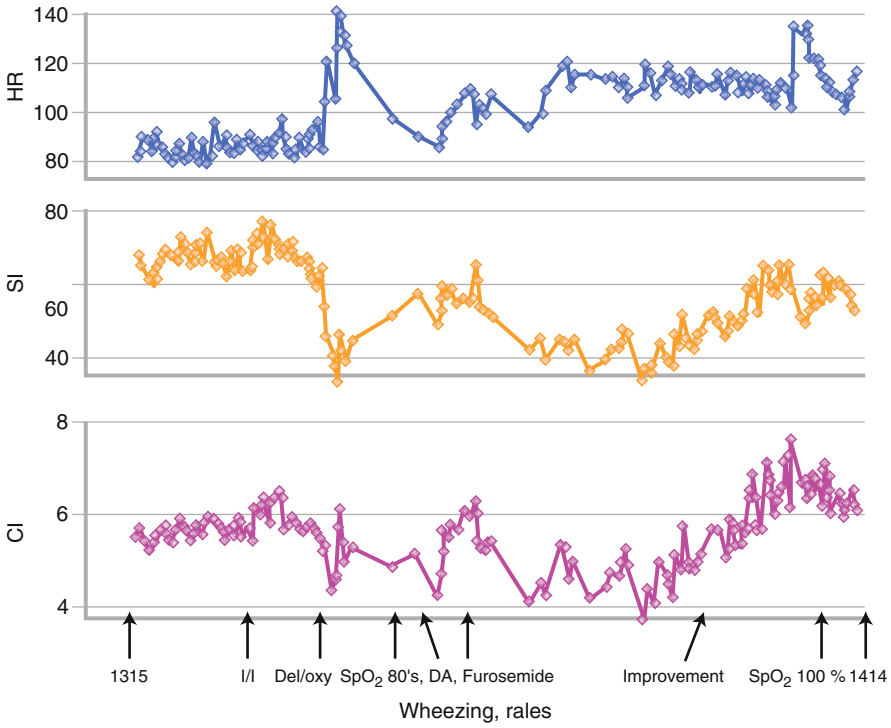

Fig. 32.1 Hemodynamics of cesarean delivery under general anesthesia, complicated by sudden hypoxemia and pulmonary edema. Intubation and incision (*I/I*) at 1325 are followed by delivery and oxytocin administration (*Del/oxy*) at 1330. At 1336, hypoxemia, wheezing, and rales develop, associated with tachycardia and decreased stroke and cardiac indices, suggesting left ventricular failure. At 1338, anesthesia is deepened (*DA*) with fentanyl, propofol, and sevoflurane and muscle relaxation is provided with vecuronium. Furosemide administered at 1342 is followed by diuresis, clinical improvement, and increased stroke and cardiac indices. *SI* stroke index, *CI* cardiac index, *HR* heart rate

General anesthesia and muscle relaxation were induced with propofol 200 mg IV and succinylcholine 140 mg IV. The trachea was intubated at 1325, with the tube positioned at a depth of 18 cm at the teeth. Auscultation revealed normal breath sounds bilaterally and the SpO_2 was 100 %. Anesthesia was maintained with N_2O 50 % in oxygen and sevoflurane 1 % inspired until a female infant was born with Apgars of 6 and 9 at 1330. After delivery a bolus of oxytocin 2 U was given followed by a "wide open" infusion of oxytocin 40 U/L of lactated ringers solution (**L-4**). N_2O was increased to 70 %, the sevoflurane was reduced to 0.2 % inspired, and fentanyl 100 mcg and midazolam 2 mg were administered.

Six minutes after delivery (1336), the patient began to "buck" on the endotracheal tube and the surgeon requested more relaxation. The chest was uncovered to better observe chest movement and a blanching rash was noted. Also, the SpO_2 had decreased from 100 to 85 %. Blood pressure was 156/98 (**L-5**). Vecuronium 3 mg, propofol 80 mg, and fentanyl 100 mcg were administered to deepen anesthesia and facilitate both surgery and ventilation. Auscultation of the lungs revealed diffuse, bilateral rales

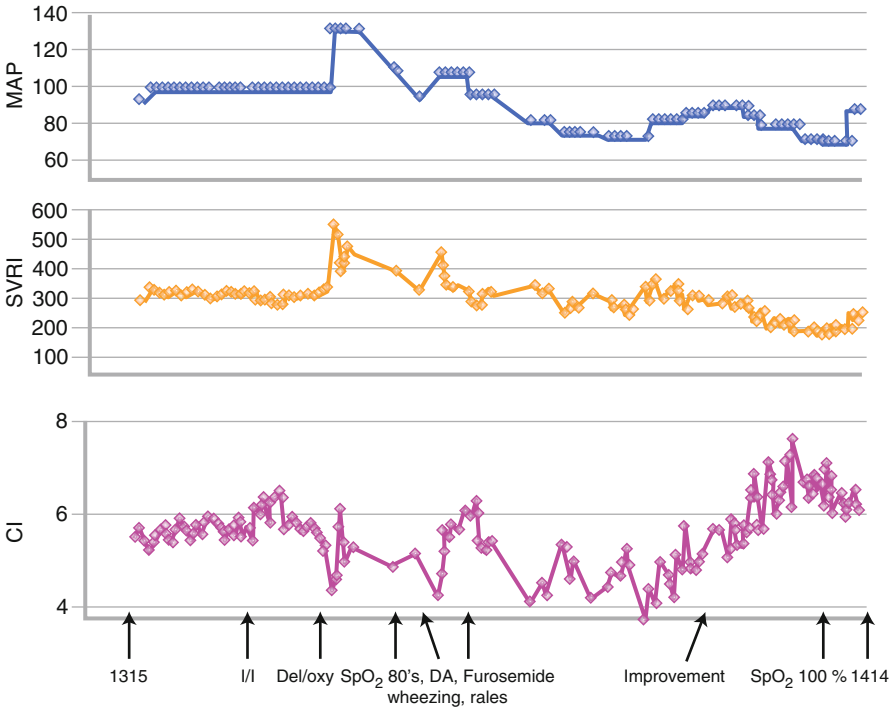

Fig. 32.2 Same case and time course as in Fig. 32.1, but here the upper two panels display mean arterial pressure (*MAP*, by cuff) and systemic vascular resistance index (*SVRI*), as calculated from CI and MAP. Pulmonary deterioration was associated with hypertension and high SVRI, consistent with fluid overload and light anesthesia. Deepening of anesthesia (*DA*) was followed by an increase in CI and furosemide was followed by an initial decrease in CI and a later, prolonged increase, compatible with the patient's clinical recovery. Other abbreviations as in Fig. 32.1

and wheezing. One-hundred percent oxygen was administered and the patient was ventilated by hand with PEEP. Albuterol was administered into the endotracheal tube because of a provisional diagnosis of bronchospasm of unknown etiology (**L-6**).

To further deepen anesthesia and provide bronchodilation, the sevoflurane was briefly increased to 3 % inspired and then decreased to 1 %. The trachea was suctioned for modest amounts of clear secretions.

At 1340 it was noted that the cardiac index (CI) and stroke index (SI) had decreased somewhat compared to predelivery levels, as shown in Fig. 32.1 (**L-7**). Mean arterial pressure and systemic vascular resistance index (SVRI) were high, as shown in Fig. 32.2 (**L-8**), and coagulation in the surgical field appeared normal (**L-8**). These facts, together with the timing of the hypoxemia and our knowledge of the previous administration of a large amount of IV fluid, led to the tentative diagnosis of acute volume overload heart failure, precipitated by autotransfusion and relief of vena caval compression at delivery (**L-9** and Figs. 32.3, 32.4, and 32.5). The patient received furosemide 40 mg IV at approximately 1342 and a brisk diuresis ensued. The fiber-optic

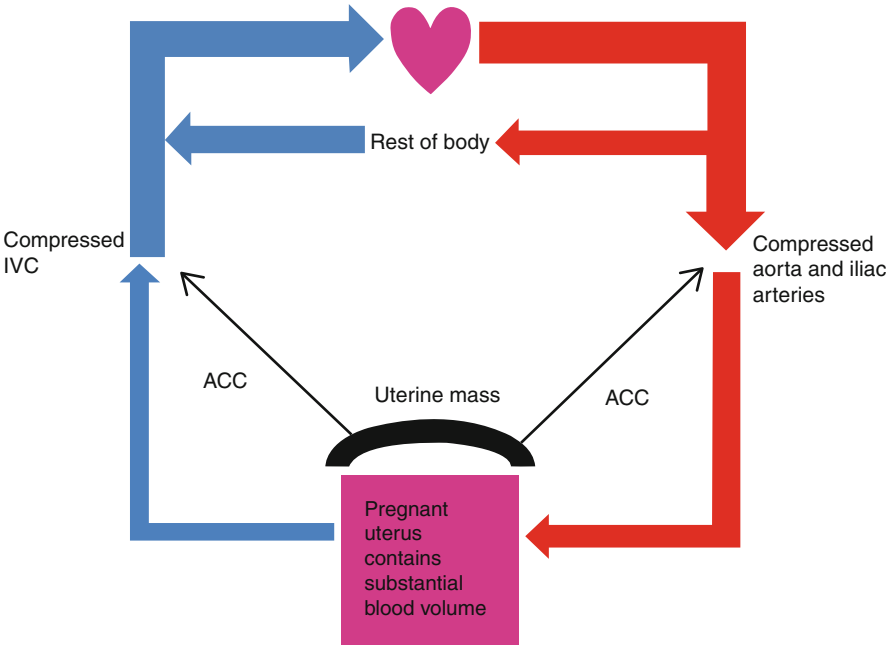

Fig. 32.3 Prior to delivery, venous return is limited by inferior vena cava (*IVC*) compression and blood volume "stored" within uterus. *ACC* aortocaval compression

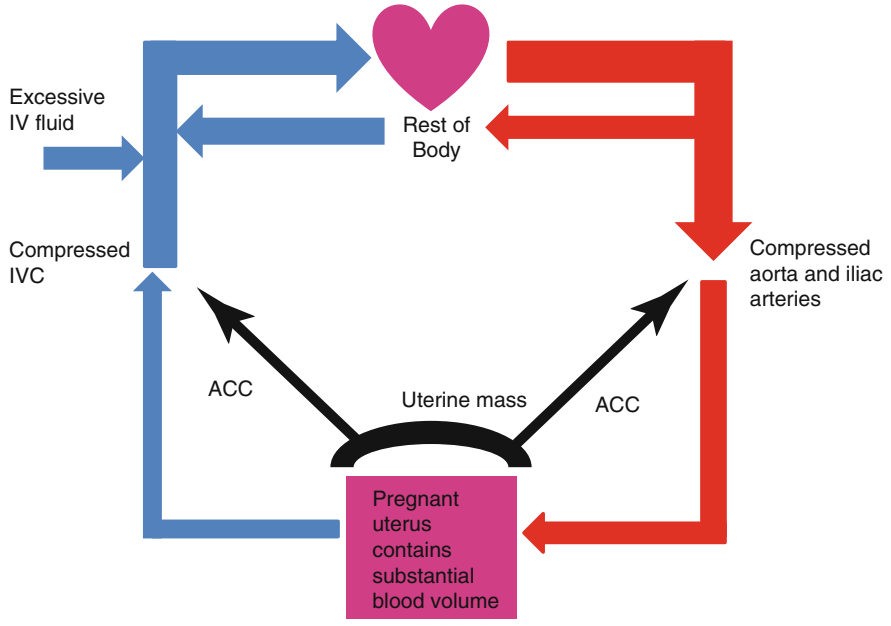

Fig. 32.4 Iatrogenic fluid overload increases circulating blood volume, but patient is asymptomatic. *ACC* aortocaval compression

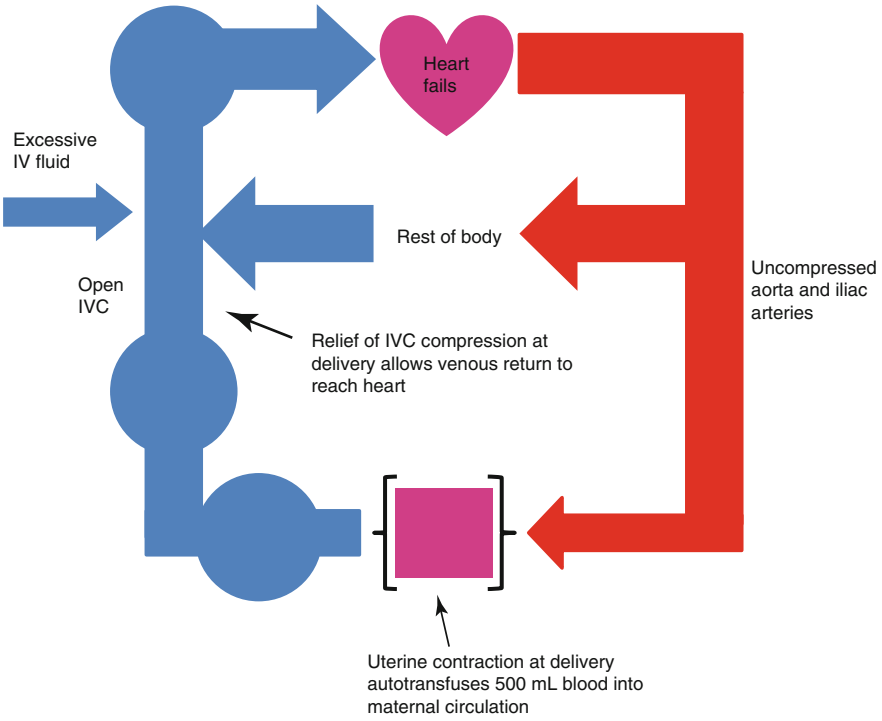

Fig. 32.5 Autotransfusion and relief of aortocaval compression overwhelm the heart with venous return

bronchoscope revealed that the tip of the endotracheal tube was 4 cm above the carina and that, despite copious watery secretions, there were no mucus plugs or foreign bodies in the airway (**L-10**). Thereafter, during diuresis, CI increased as the SpO_2 increased and the lung auscultatory findings improved, as shown in Fig. 32.1 (**L-11**).

By the end of surgery at 1455, the lungs were clear and the SpO_2 was 100 %. The patient was extubated and recovered uneventfully. During surgery hemostasis was normal. Estimated blood loss was 850 mL. Urine output from Foley placement until the end of surgery was 2.5 L. By the end of the procedure, the rash on the chest had disappeared.

Lessons Learned

L-1: von Willebrand disease
von Willebrand disease (vWD) is the most common inherited bleeding disorder and affects approximately 1 % of the population. Patients with vWD may give a history of easy bruising, nosebleeds, menorrhagia, and excessive bleeding after surgery. von Willebrand factor (vWF) is a large multimeric glycoprotein which is made by megakaryocytes and vascular endothelium and which has an affinity both for

disrupted vascular endothelium and for platelet receptors. Hence, vWF helps bind platelets to disrupted vascular endothelium. vWF also carries factor VIII within it and, thereby, physically maintains the coagulation activity of factor VIII close to the disrupted vascular endothelium where it is needed.

vWD types 1 and 3 are due to a quantitative deficit of structurally and functionally normal vWF. Type 1 accounts for 70–80 % of patients with vWD, and in these patients, the administration of desmopressin (DDAVP) causes normal vWF to be released from vascular endothelium, thereby ameliorating the coagulation disorder. DDAVP is not effective for treating vWD Type 2b, however, since this condition is caused by a structurally and functionally abnormal vWF which binds with excessive avidity to platelet receptors, causing platelet clumping and the destruction of both platelets and the attached abnormal vWF. In vWD type 2b patients, it has been thought that administration of DDAVP would be counterproductive, since it would cause the release of additional abnormal vWF which would exacerbate thrombocytopenia and coagulopathy. Hence, vWD type 2b should be treated with exogenous, functionally normal vWF/factor VIII complex, which is available as "Humate-P," a human plasma-derived preparation which has undergone viral inactivation treatment. Cryoprecipitate also contains vWF/factor VII complex, but this product has not undergone viral inactivation treatment.

The laboratory evaluation of vWD involves determination of both vWF "antigen" and "activity" (also called ristocetin cofactor activity). vWD types 1 and 3 are characterized by reductions in both vWF antigen and activity, whereas vWD type 2b involves normal vWF antigen but abnormally low activity. Humate-P or cryoprecipitate can correct low activity.

Clinically important points to remember are:

(a) vWD type 1 is the commonest form and can usually be successfully treated with DDAVP.
(b) DDAVP should probably not be used to treat vWD type 2b, which should be treated with exogenous, normal vWF, either as vWF/factor VIII complex (Humate) or as cryoprecipitate.
(c) vWD type 2b is often associated with thrombocytopenia.

L-2: PTT, PT, and bleeding time in von Willebrand disease

The aPTT is abnormal in about 25 % of patients with vWD type 2b. PT is normal, since factor VIII is not involved in the "extrinsic pathway" of coagulation. The bleeding time is seldom obtained in current perioperative practice since it is not very sensitive or specific for any condition.

L-3: Platelet clumping

"Platelet clumping" can occur in vitro in many patients due to EDTA anticoagulant, cold agglutinins, and for other reasons. Platelet clumping and destruction also occur in vivo in vWD type 2b so that in this case the significance of platelet clumping on the peripheral smear was not clear. We were prepared, however, to transfuse the patient with red cells and platelets.

L-4: Intravenous oxytocin after delivery
We administered an oxytocin bolus in this case because we were concerned about coagulopathy and we wanted the uterus to contract rapidly. Until recently, a 5-unit bolus of oxytocin after delivery was fairly common, but many practitioners are currently using boluses much smaller than 5 units, if they use a bolus at all. Boluses of oxytocin can cause arteriolar and venous dilation, hypotension, and decreased systemic vascular resistance. Despite the venodilation due to oxytocin, cardiac output usually increases at delivery due to relief of aortocaval compression and autotransfusion. Oxytocin 40 U/L in lactated ringers is twice the usual infusion concentration after delivery. The obstetricians had asked us to "double the oxytocin" because of the history of bleeding.

L-5: When hypertension is associated with pulmonary dysfunction
The fact that the patient was hypertensive made us think that an anaphylactic reaction or amniotic fluid embolus were less likely causes for the pulmonary dysfunction.

L-6: "All that wheezes is not asthma"
"All that wheezes is not asthma." Our differential diagnosis at this point for acute hypoxemia and wheezing during cesarean delivery under general anesthesia included:

(a) Incipient mainstem intubation with mechanical airway obstruction
(b) Acute fluid overload pulmonary edema due to excessive preoperative fluid infusion and relief of aortocaval compression and autotransfusion
(c) Anaphylactic reaction with bronchospasm and increased alveolar capillary permeability
(d) Aspiration of gastric contents, with mechanical airway obstruction and alveolar capillary damage
(e) "Amniotic fluid embolism" ("anaphylactoid syndrome of pregnancy")

L-7: Cardiac output increases after a normal delivery, due to relief of aortocaval compression, autotransfusion, and oxytocin administration
A decrease in cardiac output and stroke volume after delivery is highly abnormal. Cardiac output normally increases 25–75 % immediately after delivery due to relief of aortocaval compression, autotransfusion, and oxytocin administration.

L-8: Systemic vascular resistance
These facts made anaphylaxis or amniotic fluid embolus (AFE) less likely diagnoses, since both those conditions would have been associated with a low SVRI and a low MAP. Also, an AFE is frequently associated with disseminated intravascular coagulation (DIC).

Bolused oxytocin is a potent arteriolar dilator and usually decreases SVRI under both neuraxial and general anesthesia (Fig. 32.6). Hence, the increase in SVRI after delivery and oxytocin administration seen in this case was unusual and unexpected and probably due to high sympathetic tone in the setting of light anesthesia. It may well be that light general anesthesia postdelivery in this already volume-overloaded patient was the event that precipitated the heart failure.

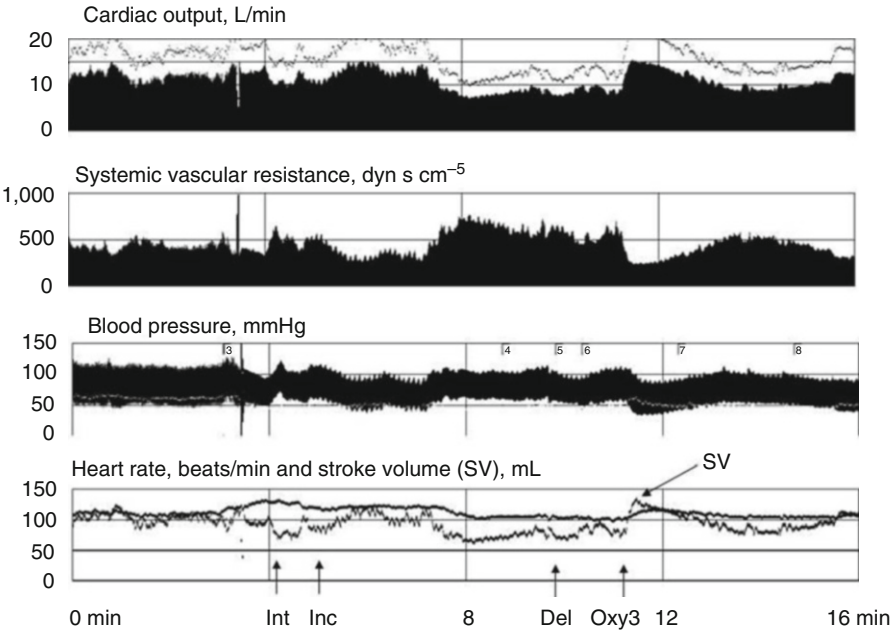

Fig. 32.6 Normal hemodynamic response to delivery (*Del*) and oxytocin 3 unit bolus (*Oxy3*) under general anesthesia, measured with LiDCO pulse contour analysis. Oxytocin bolus after delivery increases stroke volume and cardiac output and decreases systemic vascular resistance and blood pressure. *Int* intubation, *Inc* incision, *SV* stroke volume. *Please note*: *this is not the case described in the rest of the text*

L-9: Cardiac output increases after delivery. Oxytocin dilates resistance arterioles, thereby decreasing systemic vascular resistance

Delivery causes relief of aortocaval compression and uterine contraction causes autotransfusion, both of which cause a sudden increase in venous return to the heart. Oxytocin, besides contracting the uterus, decreases systemic vascular resistance (SVR) and afterload by dilating resistance arterioles (0.1 mm in diameter and smaller). If the heart is unable to "handle" this suddenly increased venous return by increasing cardiac output, the heart can fail suddenly. Figures 32.3, 32.4, and 32.5 illustrate this pathophysiology. Examples of conditions in which the heart has trouble increasing cardiac output would be mitral or aortic stenosis, cardiomyopathy, or the high afterload accompanying light general anesthesia.

L-10: Misdiagnosis of "bronchospasm"

Partial or incipient mainstem intubation is often misdiagnosed as "bronchospasm" under general anesthesia. This is particularly common when the patient is obese and is placed into Trendelenburg position and when the anesthesiologist has not meticulously avoided over-insertion of the endotracheal tube (ETT) during intubation. Use of the fiber-optic bronchoscope (FOB) enables the anesthesiologist to definitively exclude incipient mainstem intubation as a cause of "bronchospasm" and/or high

peak inspiratory pressures. Empirical withdrawal of an ETT which is probably well placed runs the risk of accidental extubation. “Taking a look” with the FOB also allows the rapid identification of aspirated stomach contents, mucus plugs, or foreign bodies in the airway.

L-11: Diuresis in heart failure due to fluid overload

Diuresis was accompanied by increased SpO_2, improvement in breath sounds, and by increasing CI, all of which was consistent with successful treatment of heart failure due to fluid overload, although an allergic reaction was still a possible diagnosis in this case.

Chapter 33
Jehovah's Witness with Placenta Previa and Increta for Cesarean Hysterectomy

Thomas L. Archer

A 34-year-old, 97 kg woman, gravida 3 para 2, who had undergone two previous classical cesarean sections, was referred at 32 weeks and 6 days estimated gestational age for management of placenta previa. The patient had no vaginal bleeding prior to admission. On admission to the University of California San Diego (UCSD), placenta accreta or percreta was suspected (L-**1**, and Figs. 33.1 and 33.2).

The patient was a Jehovah's Witness who refused allogeneic red blood cell transfusion, but would accept crystalloid and colloid solutions, fresh frozen plasma, cryoprecipitate, factor VIIa, dialysis, and autologous transfusion via a continuous circuit which did not lose its connection with her body, including both acute normovolemic hemodilution (ANH) and intraoperative cell salvage (**L-2**).

The patient's hemoglobin on admission was 10.9 g/dL. Mean red cell volume (MCV), mean cell hemoglobin concentration (MCHC), and serum iron were within normal limits (**L-3**).

Erythropoietin 40,000 units was given subcutaneously once 12 days prior to surgery, followed by 10,000 units daily for 8 days prior to surgery. Ferrous sulfate 325 mg was given orally three times per day for 12 days prior to surgery (**L-4**).

At 34 weeks and 4 days estimated gestation, when her hemoglobin was 10.9 g/dL (unchanged from admission), the patient was taken to the operating room for cesarean hysterectomy. Sequential compression devices were applied to the patient's calves (**L-5**).

A 16-gauge intravenous cannula was placed in each forearm. The patient was given sodium citrate 30 mL orally and famotidine 20 mg and metoclopramide 10 mg intravenously (IV). The patient was given midazolam 3 mg IV to facilitate line placement. A 20-gauge right radial arterial line was inserted, and the PulseCO system (LiDCO Ltd, Cambridge, UK) was connected for the trending of cardiac

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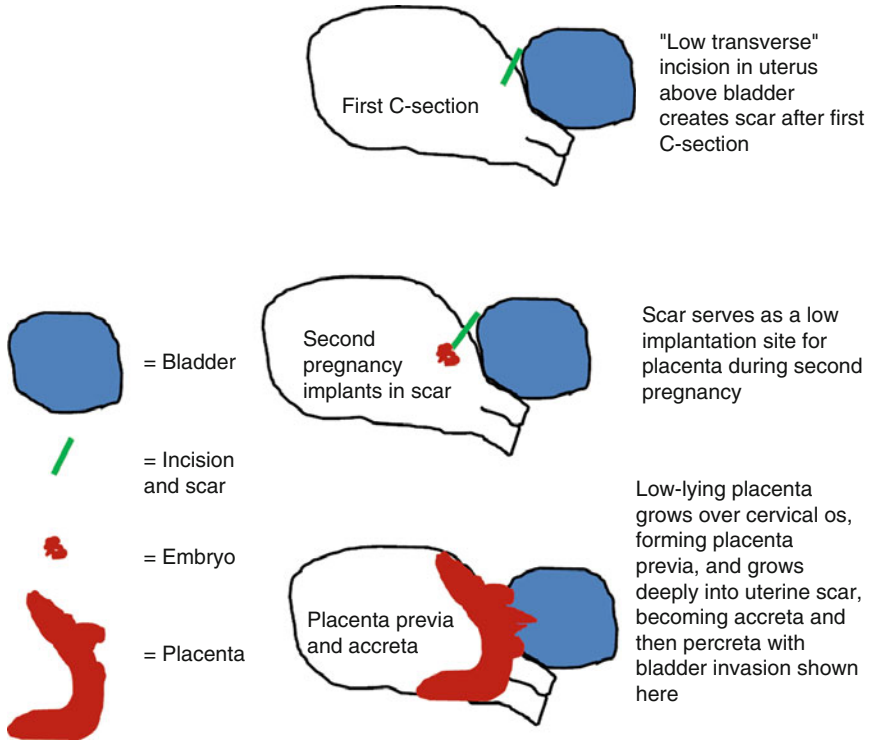

Fig. 33.1 Placenta previa in a patient with a previous cesarean section should raise suspicion of placenta accreta, which will necessitate cesarean hysterectomy

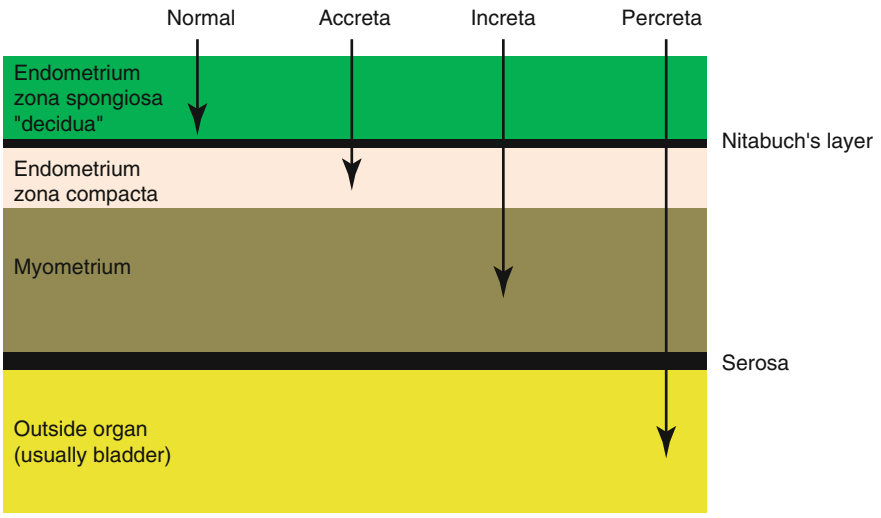

Fig. 33.2 Placental invasion of uterus

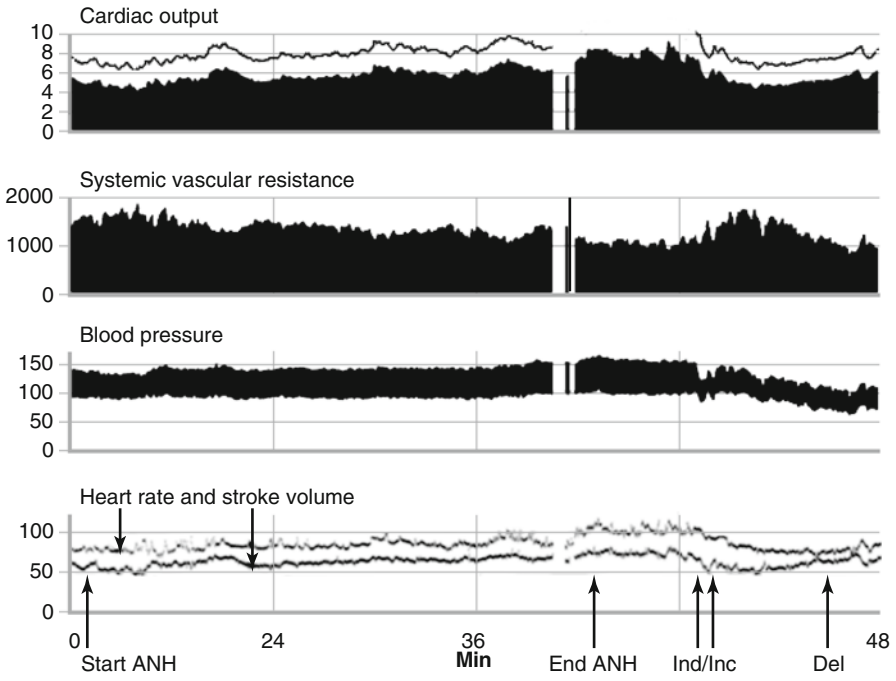

Fig. 33.3 Acute normovolemic hemodilution (*ANH*) from a hemoglobin of 10.9–7.8 g/dL causes increasing heart rate (beats/min), stroke volume (mL), and cardiac output (CO, L/min) and decreasing systemic vascular resistance (*SVR*, dyne.cm.sec $^{-5}$). Blood pressure (mmHg) is well maintained due to infusion of hetastarch and crystalloid to maintain normovolemia. Induction of general anesthesia (*Ind*) is associated with a decrease in blood pressure and incision (*Inc*) is followed by increased SVR and decreased CO. *Del* delivery, without oxytocin administration

output (CO) and systemic vascular resistance (SVR) by means of pulse contour analysis (**L-6**).

A baseline arterial blood sample in the operating room showed a hemoglobin of 10.9 g/dL and normal blood gases. An 8.5-French right internal jugular venous sheath was placed. Over the course of 38 min, 2,100 mL of blood was removed into standard citrate phosphate dextrose (CPD) storage bags, which remained in a continuous circuit with the patient throughout the operation. During blood removal, hetastarch 1,000 mL, normal saline 900 mL, and Normosol-R 2,600 mL were infused to maintain normovolemia (**L-7**, **L-8**).

All fluids were warmed prior to administration (**L-9**). Left uterine displacement and oxygen 6 L/min by facemask and circle system ($FiO_2 = 1.0$) were maintained during hemodilution until induction. Fetal heart rate stayed in the range of 140–150 beats per minute, with preserved variability, throughout hemodilution. After ANH was complete, an arterial blood sample revealed a hemoglobin of 7.8 g/dL. As seen in Fig. 33.3, SVR gradually decreased and CO increased during the course of ANH (**L-7**, **L-8**).

Following preoxygenation and IV administration of fentanyl 150 mcg and lidocaine 100 mg, anesthesia was induced with propofol 140 mg IV. Tracheal intubation was facilitated with succinylcholine 140 mg IV. Anesthesia was maintained with 50 % nitrous oxide and 1 % sevoflurane in oxygen until delivery, at which time the nitrous oxide was increased to 66 % and the sevoflurane was decreased to 0.5 % (**L-10**).

The surgical team used a separate suction system to remove amniotic fluid (**L-11**). Blood which was not contaminated with amniotic fluid was suctioned into the collection bottle of a Medtronic AT-1000 cell saver machine.

A healthy male infant, with Apgar scores of 7 and 8 at one and 5 min, respectively, and weighing 2,765 g, was delivered through a vertical uterine incision cephalad from the placenta. No attempt was made to deliver the placenta and no oxytocin was administered (**L-12**).

Hysterectomy was performed with limited blood loss. The subsequent pathology report described findings of placenta increta and previa with near-total penetration of the lower anterior uterine wall at the previous cesarean section site.

After hemostasis had been achieved, the anesthesia team returned the 2,100 mL of ANH blood to the patient over a period of 81 min. The cell saver machine had not received enough blood to allow processing (**L-13**). Hemoglobin determined 24 min after initiation of blood replacement was 8.4 g/dL. Furosemide 10 mg IV was given twice during blood infusion in order to facilitate elimination of the intravenous fluid administered during hemodilution. Urine output for the case was 1,600 mL. After the intravenous injection of indigo carmine, the patency of both ureters was verified at cystoscopy (**L-14**).

At the end of surgery, neuromuscular blockade was reversed. The patient's lungs were clear to auscultation, hemoglobin saturation was 100 %, and the trachea was extubated. The patient was taken to the surgical intensive care unit. Blood loss was estimated at 1,000 mL. In addition to the 2,100 mL of ANH blood, the patient received normal saline 1,200 mL, Normosol-R 3,600 mL, and hetastarch 1,000 mL. The patient's hemoglobin on the first postoperative day was 9.0 g/dL and she made an uneventful recovery.

Lessons Learned

L-1: Current placenta previa in patient with prior cesarean section: a "red flag" for placenta accreta, increta, or percreta

Placenta accreta, increta, and percreta (see Figs. 33.1 and 33.2) are becoming more common, primarily due to increased cesarean section rates. The number of Jehovah's Witnesses is also increasing, having doubled since 1980 to a current estimated 6.3 million.

The combination of *placenta previa* and *previous cesarean section* should immediately trigger a suspicion of *placenta accreta*. The mechanism of this association is illustrated in Fig. 33.1. The previous low transverse uterine scar creates a nidus for implantation of the embryo in a subsequent pregnancy. Since implantation is low in

the uterus, the placenta forms near the cervical os and grows over it, forming placenta previa. The preexisting uterine scar provides a pathway for the invasion of the placenta more deeply than usual into the uterine wall, thereby forming placenta accreta, increta, or percreta, depending on the depth of penetration (Fig. 33.2). The incidence of types of abnormally adherent placentation is accreta 78 %, increta 17 %, and percreta 5 %. The gold standard for diagnosis of placenta accreta is MRI, but ultrasound can often detect this condition as well.

Any trauma to the uterus (cesarean section, myomectomy, or even a dilation and curettage [D&C]) can cause a nidus for implantation and a pathway for excessive invasion of the uterine lining. A placenta which is implanted to an excessive depth will not separate from the uterine wall after delivery, and this condition necessitates a hysterectomy after the baby is delivered (cesarean hysterectomy).

L-2: Jehovah's witness
Different Jehovah's Witnesses will accept different fluids, blood derivatives, and blood salvage techniques, depending on their specific beliefs. Hence, it is important for the anesthesiologist to clarify in advance of surgery exactly what the patient will accept. Many hospitals have forms for organizing this information. It is also essential that the patient and the surgical, nursing, and anesthesia teams be in full agreement before surgery as to the plan for compensating blood loss in order for the compensation plan to be both timely and effective.

L-3: Iron deficiency anemia
These results exclude iron deficiency in this patient. Iron deficiency anemia is by far the most common cause of anemia in women of childbearing age and is characterized by a low mean cell volume (MCV), mean cell hemoglobin concentration (MCHC), serum iron, and serum ferritin.

L-4: Erythropoietin
Erythropoietin (EPO) is most often used to treat anemia in patients with chronic kidney disease, but it is also indicated to decrease the need for allogeneic blood transfusion prior to elective, noncardiac, and nonvascular surgery. In the studies which justify this practice, ferrous sulfate was administered to patients together with the EPO, and ferrous sulfate was given to this patient as well, despite the absence of iron deficiency. Possible complications of EPO administration include stroke, myocardial infarction, hypertension, venous thrombosis, and stimulation of cancer growth [1].

L-5: Deep vein thrombosis
Pregnancy is a hypercoagulable state and sequential compression devices are standard of care for cesarean section patients. Additionally, administration of erythropoietin increases the incidence of deep vein thrombosis.

L-6: Pulse contour hemodynamic monitoring
There are several systems currently on the market for the estimation and trending of CO by analyzing an arterial waveform. Despite the fact that the technology is controversial and in a state of flux, I believe it can be useful for detecting hemodynamic trends.

L-7: Acute normovolemic hemodilution

Acute normovolemic hemodilution has gone in and out of favor due, respectively, to its potential for minimizing allogeneic transfusion and to questions about the procedure's cost and practicality. However, and at the same time, the risks of allogeneic blood transfusion due to infection and immunomodulation are becoming increasingly recognized.

Blood volume peaks at 34 weeks of gestation at 7.6–9.4 % of body weight. Assuming the lower value, this patient's blood volume was approximately 7,400 mL. We removed 28 % of that volume (2,100 mL) into CPD bags, with continuous replacement of warmed crystalloid and colloid, and her hemoglobin decreased from 10.9 to 7.8 g/dL, a 28 % reduction. This degree of hemodilution was considerable because acute anemia might have caused fetal hypoxia. However, we felt such a degree of hemodilution was justified for the following reasons: the patient faced an operation involving possible torrential blood loss and we were absolutely prohibited from giving allogeneic blood; we would be continuously monitoring the fetal heart rate during the hemodilution (which could be stopped at any time); and the fetus would be exposed to maternal anemia only briefly.

We calculated normovolemic replacement for the 2,100 mL of blood removed as follows: 1,000 mL of blood was replaced by 1,000 mL of 6 % hetastarch. 1,100 mL of additional blood removal required three times that volume of crystalloid (3,300 mL) and we administered 3,500 mL.

The preoperative insertion of internal iliac or uterine artery balloons, to be inflated after delivery, and postdelivery embolization of uterine arteries via previously inserted catheters (neither of which was performed in this case) are additional options for minimizing blood loss during cesarean hysterectomy.

L-8: Hemodynamics of acute normovolemic hemodilution and anemia

Hemodilution causes blood viscosity to decrease and blood viscosity is one determinant of systemic vascular resistance. For laminar blood flow through a uniform tube (which does not describe blood flow in the human body), the resistance of the tube is inversely proportional to the fourth power of the radius and proportional to the viscosity of the blood (Poiseuille's Law). Blood viscosity, in turn, depends on the hematocrit. Hence, as ANH occurs, SVR decreases simply because the hematocrit is decreasing. If any tissue hypoxia were to occur, this would also cause arteriolar dilation, further decreasing SVR. CO, in turn, is inversely proportional to SVR. Figure 33.3 demonstrates that progressive hemodilution is associated with a decreasing SVR and an increasing CO, while blood pressure is well maintained.

Moderate chronic anemia is relatively well tolerated in pregnancy, by both mother and fetus, and during acute normovolemic hemodilution in pregnant sheep, fetal oxygen consumption is maintained until the maternal hematocrit is reduced by more than 50 % [2].

ANH has been demonstrated to be safe and effective for limiting allogeneic blood transfusion in other types of surgery. Severe anemia is well tolerated as long as normovolemia and normothermia are maintained. Tissue perfusion and oxygen consumption are well maintained during normovolemic anemia due to increases in both cardiac output and oxygen extraction [3].

The key to safe ANH is the maintenance of blood volume, cardiac output, and normothermia during hemodilution and surgery.

L-9: Normothermia
Maintenance of normothermia is important during surgery, since hypothermia can cause coagulopathy, wound infections, and increased postoperative cardiac events. For this reason, all fluid and blood products should be warmed during operations with the potential for heavy blood loss.

L-10: Neuraxial block vs. general anesthesia for cesarean hysterectomy
Neuraxial block is the "standard" anesthetic for cesarean section, other things being equal, but general anesthesia (GA) is often used for cesarean hysterectomy for the following reasons:

(a) GA eliminates the need to worry about the patient's airway, emotional state, anxiety, pain, nausea, shivering, and movement of arms and upper body.
(b) GA allows maintenance of an intact and compensating sympathetic nervous system in the face of heavy blood loss.
(c) Allows the team to focus on hemodynamics and volume replacement.
(d) The desire of the patient to be awake for the birth of her baby must be balanced against the advantages of GA, and the choice of anesthetic technique should be made after a thorough discussion with the patient and the OB team.
(e) Neuraxial morphine without local anesthetic, either as a single shot spinal or given through an epidural catheter, is an option for postoperative pain relief since neuraxial narcotics do not cause sympathectomy. However, if internal iliac balloons are placed preoperatively for possible hemorrhage control, any neuraxial block must be performed before the placement of the balloons, since the sheaths in the femoral arteries necessary for balloon placement do not allow the patient to flex at the hip for subsequent neuraxial block placement.
(f) GA is often associated with a lower CO and a higher SVR than neuraxial anesthesia. Figure 33.3 demonstrates that during relatively light GA, surgical stimulation increases the SVR, and decreases the CO due to activation of the sympathetic nervous system and associated increased arteriolar tone. This decrease in CO and well-maintained SVR can be desirable in hypovolemic patients, i.e., a reactive sympathetic nervous system helps maintain organ perfusion.

L-11: Amniotic fluid embolus and cell salvage: an unwarranted fear
Concern is often expressed about causing "amniotic fluid embolus" if cell salvage is used in obstetric cases, but cell salvage is now often used in complicated obstetric cases for the following reasons:

(a) A separate suction setup is used to remove amniotic fluid. Blood is suctioned to the cell saver only after all of the amniotic fluid has been removed.
(b) The normal saline wash process performed during cell salvage is highly effective in removing all types of contaminants from salvaged blood, including fetal squamous cells, lamellar bodies, and amniotic fluid. No cases of amniotic fluid embolus have been reported in patients to whom properly washed cell saver blood has been administered.

(c) Despite points (a) and (b), many authorities recommend the use of "leukocyte reduction filters" in addition to routine blood filters when administering the cell salvage product to the patient. These filters provide an additional level of safety to the process and can be obtained from the blood bank.
(d) Cell salvage is particularly appropriate for patients who may have torrential bleeding, as in placenta percreta, and for whom allogeneic transfusion is not an option, but it is often used as an adjunct in cesarean hysterectomy even for patients who are able to receive allogeneic blood.
(e) The syndrome called "amniotic fluid embolus" is of unknown etiology and is probably not caused by amniotic fluid. An alternative term for the syndrome is "anaphylactoid syndrome of pregnancy."

Furthermore, cell salvage blood has many advantages over banked, allogeneic blood:

(f) Salvaged RBCs have normal 2,3-DPG (2,3-diphosphoglycerate) levels and red cell deformability, unlike banked red cells.
(g) Risk of infection and clerical error is eliminated.

Cell salvage has the following disadvantages:

(h) At the very most, only 50 % of shed RBCs can be recovered and readministered.
(i) No clotting factors or platelets are present in the cell salvage product.

L-12: Oxytocin and cesarean hysterectomy
Oxytocin is often not administered after delivery of the baby during cesarean hysterectomy, since contraction of the uterus might lead to the shearing off of the normal areas of the placenta which are not abnormally adherent to the uterus. Such unwanted placental separation could increase hemorrhage during hysterectomy. The anesthesiologist should discuss this issue with the obstetrician prior to surgery.

L-13: Cell salvage
Due to the design of the cell saver, the "bowl" in which red cells are washed with saline must be filled with packed red cells before any of those cells can be given back to the patient. Partially filled "bowls" cannot be given back. The minimum amount of salvaged blood in the collection bottle in order to result in a full bowl is generally considered to be approximately 500 mL.

L-14: Ureteral damage during hysterectomy
Accidental ligation of a ureter near the bladder is a possible complication of hysterectomy, particularly when the uterus is large, hemorrhage is abundant and surgery is performed rapidly, as can be the case with cesarean hysterectomy. Methylene blue or indigo carmine can be injected intravenously in order to color the urine. At cystoscopy the egress of blue dye from both ureteral orifices confirms that the ureters are intact. Both dyes scavenge nitric oxide and can cause hypertension if they are given rapidly. Both dyes also transiently lower SPO_2 readings.

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Chapter 34
A Pregnant Patient with Mitral Stenosis

Seth T. Herway and Thomas L. Archer

A 36-year-old woman, gravida 3 para 1011 (**L-1**) at 30 weeks gestation, presented to labor and delivery (L&D) for obstetric evaluation. Anesthesia was consulted because the patient reported a heart condition secondary to childhood rheumatic fever (**L-2**). With the current pregnancy, the patient reported progressive shortness of breath with normal activities such as walking to the bus stop (**L-3**).

The patient had been diagnosed with rheumatic heart disease in the Philippines. Echo obtained at that time was unavailable. She had a cesarean section (CS) in the Philippines in 2009 under general anesthesia with no complications. She had been told that the CS needed to be performed under general anesthesia due to her heart condition. She moved from the Philippines to the United States 3 months prior to her presentation to be with her parents after finding out she was pregnant again.

Echocardiogram at University of California San Diego (UCSD) revealed severe mitral stenosis and severe mitral regurgitation. She had a severely enlarged left atrium with moderate pulmonary hypertension. Mitral valve area was 1.2 cm^2, mitral mean gradient 16 mmHg, pulmonary artery pressure 49 mmHg systolic, aortic valve area 1.9 cm^2, LVEF 60 %, and LA volume index was 112 mL/m^2 (normal = 16–28 mL/m^2). See Figs. 34.1 and 34.2 for patient and normal example, respectively.

Due to the patient's cardiac condition, the cardiology, anesthesia, and obstetrics teams met to develop plans for care (**L-4**, **L-5**, **L-6**).

The decision was made to schedule the patient for repeat CS when she would be at 37 weeks and 1 day gestation. The plan was to perform the operation in the main operating rooms (rather than L&D) under general anesthesia (**L-5**). The patient was to be admitted to the cardiology service 2 days prior to scheduled CS and to be admitted to the surgical intensive care unit (SICU) postoperatively. The immediate plan was for cardiology to titrate beta-blockade with metoprolol to a resting heart

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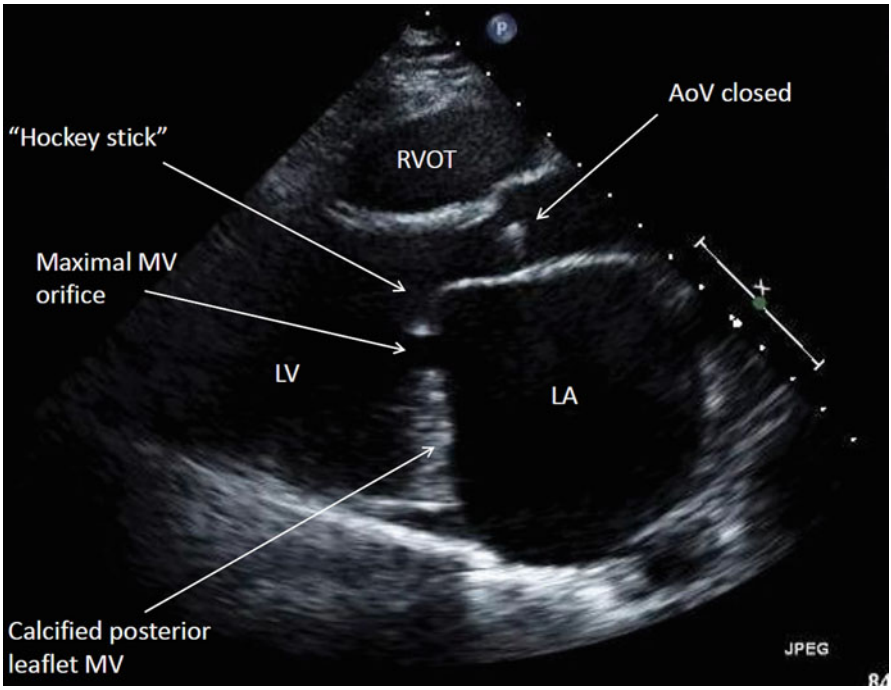

Fig. 34.1 Left parasternal long axis echocardiography view in diastole, with maximum mitral valve orifice shown. Left atrium is greatly enlarged. Anterior leaflet of mitral valve has "hockey stick" appearance and posterior leaflet is heavily calcified. *AoV* aortic valve, *LA* left atrium, *LV* left ventricle, *MV* mitral valve, *RVOT* right ventricular outflow tract

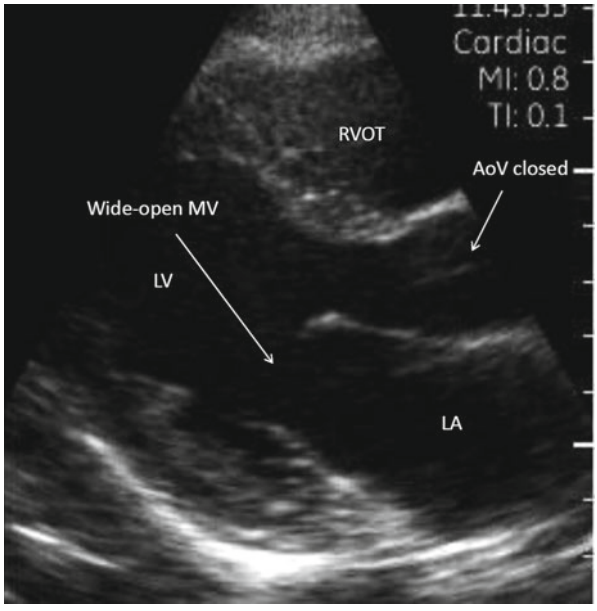

Fig. 34.2 Normal heart, left parasternal long axis echocardiography view in diastole. Mitral valve (*MV*) leaflets are wide open. *AoV* aortic valve, *LA* left atrium, *LV* left ventricle, *RVOT* right ventricular outflow tract

rate of 70–85 beats per minute (bpm). She was also to be anticoagulated with Lovenox which would be changed to heparin at 36 weeks of gestation (**L-6**).

The final anesthetic plan developed was to perform the CS under general anesthesia (without any neuraxial anesthesia), with cardiac anesthesia staff present for intraoperative transesophageal echocardiographic (TEE) evaluation if necessary.

When the patient was at 36 weeks and 1 day of gestation (1 week prior to her planned admission) she presented to L&D with severely painful contractions and with her cervix dilated to 7 cm. The OB anesthesia service received an urgent page at 2305 and saw the patient immediately. She was in extreme distress, actively contracting with 10/10 pain, tachycardic with a heart rate in the 130s, uncooperative, and without IV access. The main OR was notified to set up for a stat CS. Cardiac anesthesia was also paged. The main OR reported that the ORs were currently running at capacity and there was no nursing staff available to open an additional room. The obstetric service wanted to proceed immediately in the obstetric operating suite but because the obstetric operating rooms are too small to accommodate the TEE machine, the decision was made to utilize obstetric nursing staff in one of the main operating rooms. Coordinating the OR location and staffing resulted in a delay such that the patient was brought to a main operating room suite at 2335; approximately 30 min after the anesthesia service had first been paged (**L-7**). While this was being coordinated, IV access was obtained and the patient was transported to a main operating suite.

Standard monitors, an arterial line, and electrocautery pads were quickly applied in the OR. The patient's heart rate (HR) was in the 130s. Esmolol 60 mg and fentanyl 300 mcg were given. The patient's HR slowed minimally, remaining in the 120–130 range. As the surgical site was being prepped and drapes were being hung at 2338, the patient became unresponsive (**L-8**). It was impossible to determine the patient's cardiac rhythm because of noise in the electrocardiogram (EKG) due to the surgical site being sterilely prepared at this moment. A rapid sequence induction was immediately performed with 14-mg etomidate and 100-mg succinylcholine, and the airway was secured with a 7.0-mm endotracheal tube. Skin incision, uterine incision, and delivery were all accomplished at 2339. During this time the EKG tracing could again be seen as sinus tachycardia. Labetalol 10 mg was given and the HR decreased to 80. Anesthesia was maintained with sevoflurane. The patient's HR remained in the 80s for the remainder of the case. Cardiac anesthesia arrived to perform TEE, which showed severe left atrial enlargement and moderate mitral stenosis with mild to moderate mitral regurgitation (MR). Moderate aortic insufficiency with no aortic stenosis and normal left and right ventricular function were also noted. Surgery ended at 0042. The decision was made to keep the patient intubated and to transfer her to the SICU, but due to lack of bed availability in the SICU, care was maintained by the anesthesia team in the postanesthesia care unit for 3 h until a SICU bed became available.

The patient was not able to be extubated until 2 days postoperatively due to hemodynamic instability that was managed with esmolol and phenylephrine drips (**L-9**). Her postoperative course was complicated by endometritis which was treated with antibiotics and by wound separation with a superficial hematoma that required evacuation. She also experienced a thrombocytopenia that could not be attributed to

a disseminated intravascular coagulopathy (DIC) or a heparin-induced thrombocytopenia (HIT) and eventually resolved. She was ultimately discharged 11 days postoperatively with appropriate obstetric and cardiology follow-up.

Lessons Learned

L-1: "**Gravida**," "**para**," **and other obstetric terminology**
G means "gravida," the number of times a woman has been pregnant. P means "para," the number of times a woman has given birth to a fetus (living or dead) older than 20 weeks gestational age. In the "TPAL" system, the "P" or "para" is sometimes expressed more fully as four values, representing "term deliveries, preterm deliveries after 20 weeks gestation, abortion or miscarriage before 20 weeks gestation, and living children." These parameters can be remembered by the mnemonic "Texas Power and Light." This patient who was G3P1011 had one term delivery by CS, no preterm deliveries of an infant greater than 20 weeks gestation, one miscarriage prior to 20 weeks of gestation, and she had one living child.

L-2: **Mitral stenosis due to rheumatic heart disease is the most prevalent clinically significant valvular lesion in pregnant women** [1]
Rheumatic fever is an inflammatory condition resulting in fever, rash, joint pain, shortness of breath, and other signs and symptoms which can develop 2–3 weeks following an infection with *Streptococcus pyogenes* (group A Strep by the Lancefield system). It has been estimated that rheumatic fever develops in about 3 % of individuals who experience an untreated strep infection. By means of antigenic mimicry, this infection can result in inflammatory sequelae in the heart, joints, skin, and brain. About half of individuals affected with acute rheumatic fever go on to develop some form of rheumatic heart disease. This is most typically cardiac inflammation that involves primarily valvular endocardium. Rheumatic fever and rheumatic heart disease have become relatively rare in developed countries, probably due to the widespread use of antibiotics to treat streptococcal infections.

Although recognition and treatment is improving, as of 2012, rheumatic heart disease still causes an estimated 200,000–250,000 premature deaths each year and is the major cause of cardiovascular death in children and young adults in developing countries [2]. Current estimates are that 15–20 million people worldwide are affected with rheumatic heart disease.

Those affected with rheumatic heart disease often do not recall previous acute rheumatic fever symptoms or episodes. They typically present due to the onset of shortness of breath many years after an acute rheumatic fever attack. Mitral valve incompetence is the most common valvular lesion in patients with rheumatic heart disease. Mitral stenosis typically takes longer to develop and is a result of persistent valvulitis that results in bicommissural fusion.

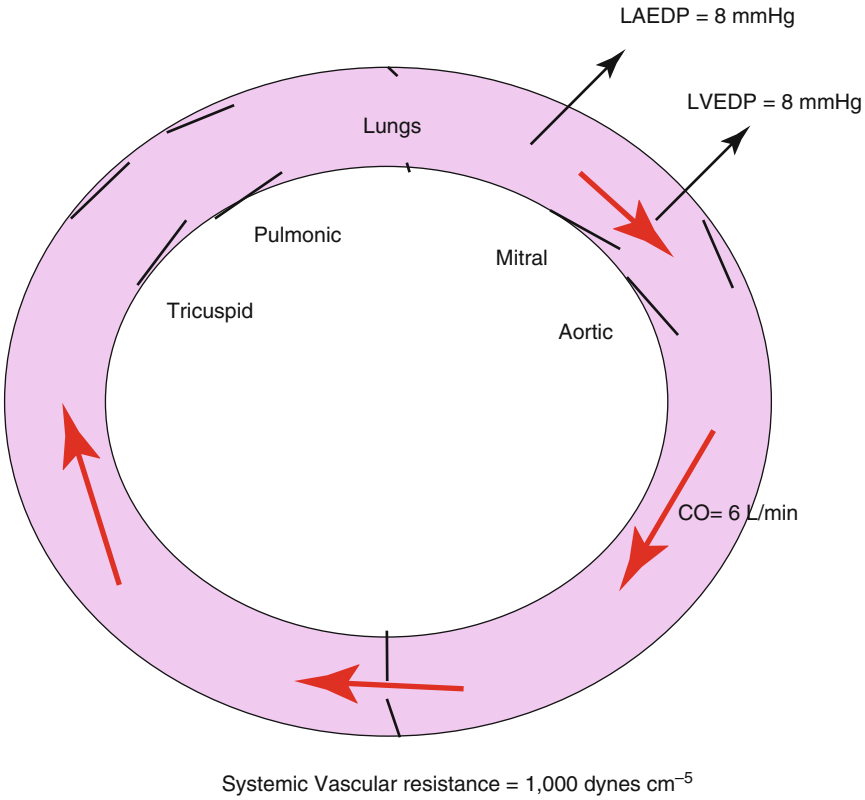

Fig. 34.3 Normal heart valves offer little resistance to flow, no matter what the cardiac output. For this reason, left atrial end diastolic pressure (*LAEDP*) equals left ventricular end diastolic pressure (*LVEDP*) and the lungs are not subjected to high capillary pressures, regardless of cardiac output. Pulmonary artery pressure is normal

L-3: **The normal hemodynamic changes of pregnancy tend to make mitral stenosis more symptomatic**

Systemic vascular resistance decreases during pregnancy and cardiac output increases to 50 % above baseline by approximately the 30th week of gestation. Heart rate and stroke volume increase 20 and 30 %, respectively. There is an increase in total blood volume of 40–50 % and an increase in red cell mass of 20 %, leading to a reduction in hematocrit and blood viscosity. All of these normal cardiovascular changes of pregnancy tend to make mitral stenosis more symptomatic and many patients experience their first symptoms during pregnancy. Figures 34.3, 34.4, and 34.5 illustrate this physiology in graphic form and it is important to note that both normal pregnancy and neuraxial anesthesia tend to decrease systemic vascular resistance.

Of course, there is nothing we can or should do about the normal hemodynamic changes of pregnancy. But with respect to neuraxial anesthesia, decreases in

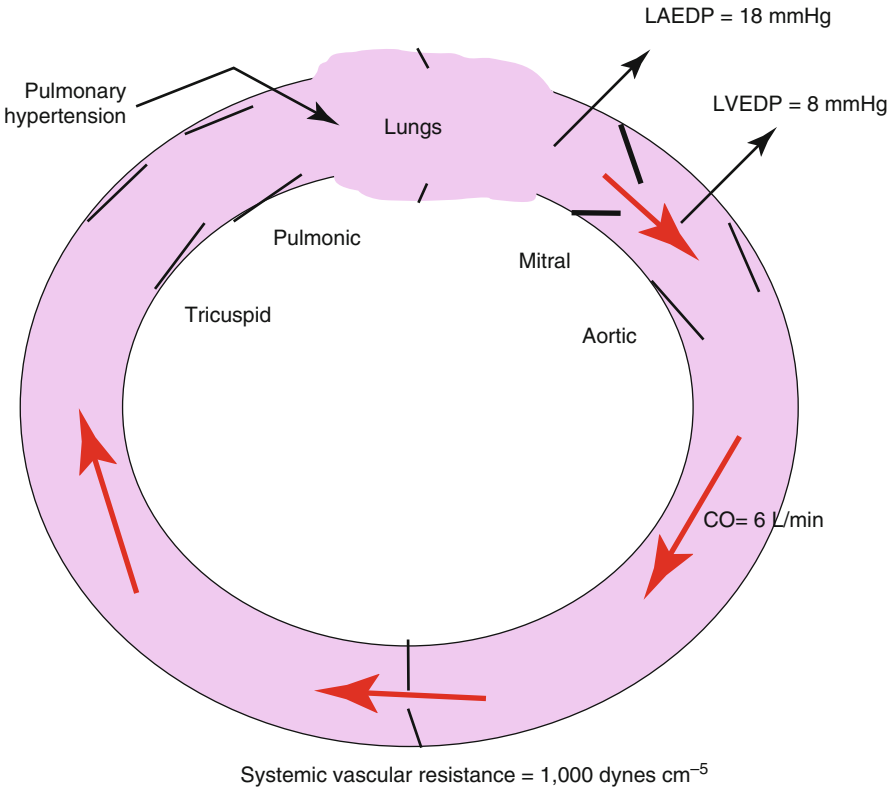

Fig. 34.4 "Compensated" mitral stenosis: a stenotic mitral valve offers appreciable resistance to flow and the pressure gradient across the valve is proportional to the cardiac output (Ohm's law). In this example, left atrial end diastolic pressure (*LAEDP*) is increased, but the patient may be asymptomatic because the LAEDP is not high enough to cause symptoms. Pulmonary artery pressure is mildly elevated. Specific numerical values are for illustrative purposes only. *LVEDP* left ventricular end diastolic pressure, *CO* cardiac output

systemic vascular resistance should be prevented by the use of phenylephrine. For the prevention or treatment of hypotension after neuraxial anesthesia in a mitral stenosis patient, phenylephrine is superior to ephedrine and the administration of abundant fluids, since the latter treatment would tend to increase the cardiac output which could precipitate pulmonary edema as shown in Figs. 34.4 and 34.5. Maintenance of SVR with phenylephrine allows the cardiac output to remain "normal" without a decrease in blood pressure.

Cardiac output peaks at about 60–80 % above nonpregnant baseline just after delivery due to autotransfusion of approximately 500 mL of blood from the contracting uterus and the relief of aortocaval compression. The autotransfusion of blood from the uterus increases central blood volume and pressure. This postpartum increase in central blood volume and pressure can be exacerbated by the regression of epidural anesthesia and subsequent return of venous tone. For these reasons, the patient with mitral stenosis is at the highest risk of decompensation immediately

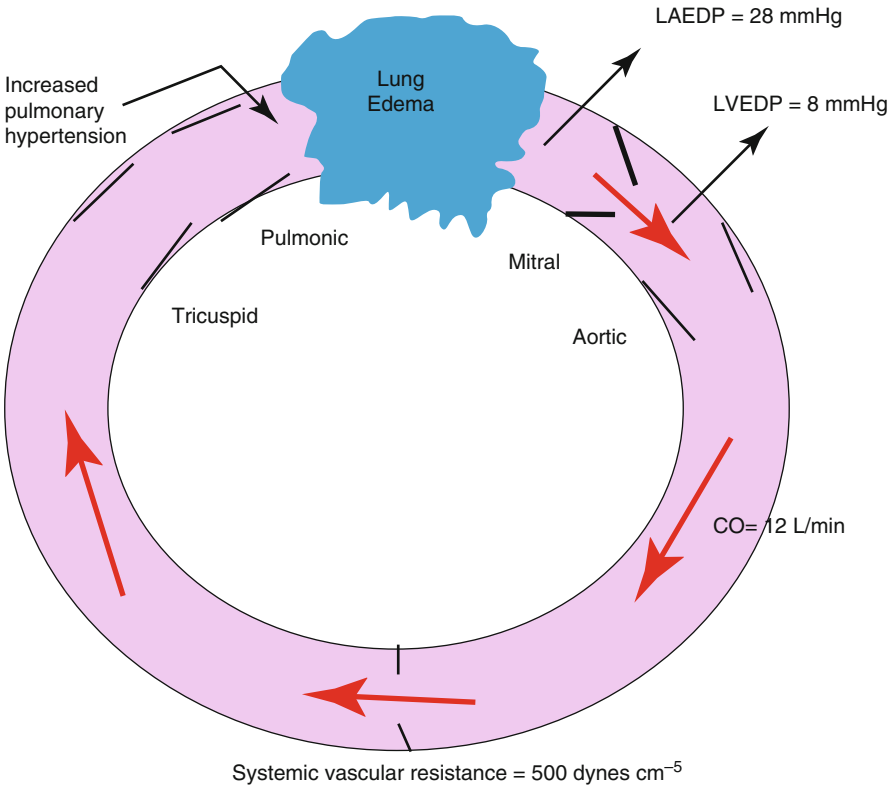

Fig. 34.5 "Decompensated" mitral stenosis: when systemic vascular resistance decreases and cardiac output increases due to pregnancy or neuraxial anesthesia, the pressure gradient across the mitral valve increases, elevating left atrial end diastolic pressure (*LAEDP*) and setting the stage for pulmonary edema and increasing pulmonary hypertension. Specific numerical values are for illustrative purposes only. *LVEDP* left ventricular end diastolic pressure, *CO* cardiac output

after delivery. In the days following delivery, edema fluid is "mobilized" and enters the circulation and cardiac output remains elevated for 2–3 weeks after delivery.

L-4: **The key to hemodynamic management of mitral stenosis in pregnancy is**: "**Don't rock the boat**!" **maintain normal heart rate**, **cardiac output**, **heart rhythm**, **venous return**, **systemic vascular resistance**, **and cardiac contractility**
For patients with mitral stenosis, pregnancy makes a bad situation worse.

Mitral stenosis retards filling of the left ventricle, tending to decrease stroke volume and cardiac output. Despite this reduction in cardiac output, pregnancy demands an increased cardiac output to serve the needs of the developing fetus and to maintain normal blood pressure in the face of decreasing systemic vascular resistance.

Mitral stenosis also slows the emptying of the left atrium, which results in elevation of both left atrial and pulmonary artery circulatory volumes and pressures. This can lead to dilation of the left atrium and/or pulmonary edema.

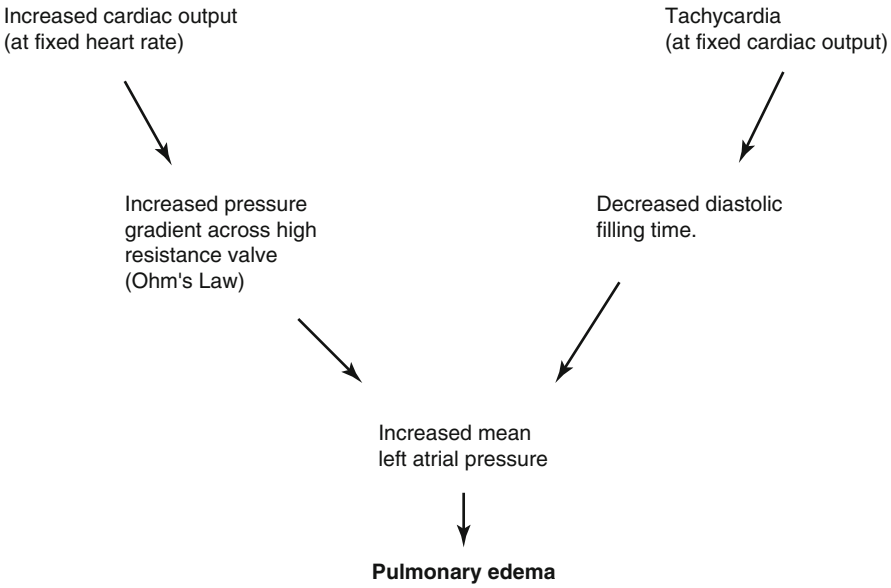

Fig. 34.6 In mitral stenosis, avoid increases in cardiac output and tachycardia. Both conditions independently increase left atrial pressure and can precipitate pulmonary edema

Furthermore, pregnancy itself and painful labor can cause tachycardia, which will decrease diastolic filling time in a patient already experiencing decreased left ventricular (LV) filling.

Figure 34.6 shows how increased cardiac output and tachycardia independently increase left atrial pressure in mitral stenosis, causing symptomatic "decompensation."

Patients with mitral stenosis compensate poorly to changes in venous return. Excessive venous return due to pregnancy itself, pain, or autotransfusion can precipitate pulmonary edema, while inadequate venous return caused by aortocaval compression, sympathectomy, or the Valsalva maneuver can lead to inadequate LV filling and hypotension. Hence, pregnant patients with mitral stenosis need a constant, adequate venous return, neither too high nor too low.

As if the preceding factors were not bad enough, pain, anxiety, and alveolar hypoxia (as well as hypercarbia and acidosis) can all increase pulmonary vascular resistance and pulmonary artery pressures. Further increases in right ventricular pressure can then cause the interventricular septum to encroach into the left ventricular cavity, even further reducing stroke volume. Hence, pain relief, anxiolysis, and alveolar normoxia are all important for preventing further deterioration in laboring patients with mitral stenosis.

The hemodynamic picture of the pregnant patient with mitral stenosis is complex, but the hemodynamic management of pregnant patients with mitral stenosis is easily understandable in light of the known pathophysiology of the disease and is summarized in Table 34.1.

Table 34.1 Hemodynamic management of mitral stenosis in pregnancy and during anesthesia

"**Don't rock the boat**!"		
Parameter	**Goal**	**Achieve goal with**
Heart rate	Low end of normal	Values *below* normal: Rate augmentation needed if hemodynamically unstable Values *above* normal: Tight pain control with neuraxial anesthesia (NA) or general anesthesia, phenylephrine, beta blocker
Heart rhythm	Normal sinus rhythm	Exercise caution with cardioversion due to likelihood of thrombus formation in dilated left atrium. Cardioversion often unsuccessful because dilated left atrium prone to resume arrhythmia
Venous return (VR)	Normal—avoid extremes	Avoid *decreased* VR: Proper positioning with LUD and avoiding pushing with NA or general Avoid *increased* VR: Prevent pain and consider continuing epidural after delivery
Systemic vascular resistance	Normal	The lesion is prior to the left ventricle and thus changes in afterload do not affect mitral valve inflow
Contractility	Maintain	The lesion is prior to the left ventricle and thus changes in contractility do not affect mitral valve inflow

L-5: **In the pregnant patient with mitral stenosis, attention to hemodynamics is probably more important than the specific anesthesia technique**

There are no controlled studies that have determined the best type of anesthetic for pregnant patients with cardiovascular disease. The decision to pursue regional versus general anesthesia is left to the practitioner's clinical judgment and particular skills. Regardless of the technique chosen for the pregnant patient with mitral stenosis, the anesthesiologist should focus on maintaining appropriate hemodynamics, as outlined in (**L-4**) and Table 34.1.

Due to familiarity with general anesthesia (GA) for the management of patients with cardiac pathology, many anesthesiologists choose GA for cesarean section in patients with cardiac conditions. Some feel that GA allows tighter control of hemodynamics. GA is often associated with a lower cardiac output than neuraxial anesthesia, and this is an advantage, other things being equal. GA with endotracheal intubation allows greater control over oxygenation and ventilation and also offers the ability to utilize transesophageal echocardiography.

Concerns with GA include the potential for tachycardia and increases in systemic and pulmonary artery pressures during laryngoscopy and intubation. Positive pressure ventilation may compromise venous return and inhalational anesthetic agents may depress the myocardium, promote uterine atony, and anesthetize the fetus.

Neuraxial techniques diminish the cardiovascular response to pain and minimize the Valsalva maneuver by minimizing the pushing reflex. A carefully administered

Table 34.2 New York Heart Association (NYHA) class definitions for all types of heart disease

NYHA class	Symptoms
I	No symptoms and no limitation in physical activity. Ordinary activity does not cause undue fatigue, palpitation, shortness of breath
II	Slight limitation of physical activity. Comfortable at rest but ordinary physical activity results in fatigue, palpitation, shortness of breath
III	Marked limitation of activity due to symptoms even during light physical activity (such as walking one block). Comfortable only at rest
IV	Severe limitations. Experiences symptoms even while at rest

neuraxial anesthetic can be titrated to the stage of labor and level of pain. The additional benefit of maternal awareness for delivery should not be overlooked. There are many case reports of epidural techniques being utilized for cesarean section in mild, moderate, and severe mitral stenosis [1, 3–5]. These reports all stress the importance of slow induction of epidural anesthesia with judicious fluid administration. These reports also note that the use of intrathecal or epidural opioids can limit the use of local anesthetics and thus minimize hemodynamic changes. Kee et al. reported management of three parturients with mitral stenosis without significant hemodynamic changes and without any local anesthetic boluses by initiating anesthesia with intrathecal fentanyl with subsequent maintenance using diluted epidural bupivacaine and fentanyl infusion [6]. Continuous spinal anesthesia is less commonly reported, but has been used with success. Dresner et al. published a case series of 33 women with cardiac disease who underwent cesarean section successfully managed with continuous spinal anesthesia [7].

Neuraxial anesthesia causes sympathectomy which reduces systemic vascular resistance. The normal hemodynamic response to decreased systemic vascular resistance (SVR) is an increase in cardiac output, and this is a danger in patients with mitral stenosis. After neuraxial block, hypotension commonly occurs. This is most often due to vasodilation and loss of SVR. It is appropriate to treat this loss of SVR with phenylephrine to avoid a reflex increase in the cardiac output.

With either general or neuraxial anesthesia, the authors recommend tight hemodynamic monitoring and control, with gradual titration of the neuraxial anesthetic and the use of phenylephrine to maintain adequate systemic vascular resistance [8, 9]. During labor, adequate pain control with neuraxial analgesia is of key importance to avoid tachycardia and increased cardiac output.

Another possible disadvantage of continuous epidural anesthesia is the risk of epidural hematoma in a patient who may need anticoagulation for urgent heart surgery.

L-6: **Mitral stenosis early in pregnancy**: **medical interventions and anticoagulation**

For women with mild to moderate heart disease (New York Heart Association class I or II; see Table 34.2), medical management is appropriate. Beta-blockers may be utilized to prolong the diastolic filling time. Prompt treatment of atrial fibrillation is indicated. Volume overload can be treated with diuretics. Reduction of salt intake and limitation of physical activity are also appropriate. Cardiac output reaches its

maximum by 28 top 32 weeks of gestation and the patient who has been able to reach this gestational age may not experience further deterioration.

As a rule of thumb, pregnancy increases the New York Heart Association (NYHA) functional class by one. Women with severe disease (NYHA class III/IV or mitral valve area <1.0 cm^2) appear to have fewer complications during pregnancy if they undergo balloon mitral valvuloplasty or have valve surgery before conceiving [10]. Percutaneous balloon mitral valvuloplasty can be considered after conception but risks to the fetus need to be adequately considered [11].

Although there are no controlled studies to guide anticoagulation in pregnant patients with mitral stenosis, most authors agree that anticoagulation is indicated even in the absence of atrial fibrillation [8]. Opinions on anticoagulation in pregnant patients with cardiac conditions are largely extrapolated from American Heart Association guidelines for nonpregnant patients [9]. Several authors recommend the use of Coumadin throughout pregnancy other than during the period of organogenesis (6–12 weeks gestation) and close to delivery (>36 weeks gestation), when heparin should be used.

L-7: **Preconception and early prenatal care and counseling are of great importance for patients with mitral stenosis**

Both preconception and predelivery education for the patient are important in patients with severe mitral stenosis. Ideally, women with mitral stenosis should be evaluated and counseled by a cardiologist prior to becoming pregnant in order to better understand their disease and the risks of labor and delivery. Balloon valvuloplasty or valve surgery can be discussed at this time.

Once pregnant, patients need to be aware of the deleterious effects of pain, anxiety, and associated tachycardia on their cardiovascular physiology and they need to be encouraged to seek medical care immediately when any stress arises.

Although it may not be economically or socially feasible, consideration should be given to admitting patients with severe disease well before their anticipated delivery in order to be able to intervene promptly when delivery is indicated. In a patient with severe mitral stenosis, delay in care can be catastrophic.

The following parameters predict morbidity and mortality in pregnant patients with cardiac disease [12, 13]:

A. Aortic valve area < 1.5 cm^2
B. Mitral valve area <2.0 cm^2
C. NYHA class 3 or 4
D. Left ventricular ejection fraction (LVEF) <40 %
E. Prior primary cardiac events (defined as pulmonary edema, symptomatic arrhythmia, stroke, cardiac arrest, or death)

In patients with none of these predictors, incidence of a primary cardiac event (same definition as above) is 5 %. With one predictor it is 27 % and with two or more it is 75 %.

In mitral stenosis, specifically, the overall mortality is reported to be less than 1 % with mild disease and between 5 and 15 % in those with severe disease or with atrial fibrillation.

L-8: **The patient probably lost consciousness due to tachycardia and decreased blood pressure**

We can only speculate about the cause of this patient's loss of consciousness on the operating room (OR) table prior to anesthesia induction. At the time, the surgical site was being prepped and there was noise in the EKG that obscured the underlying rhythm. Once the noise in the EKG ceased, the patient was found to be in sinus tachycardia, so there was probably no true cardiac arrest to explain the syncope. With a heart rate in the 130s and the probable increase in right ventricular and pulmonary pressures that were likely present in this anxious patient in pain, it seems most likely that there was inadequate filling of the left ventricle which resulted in hypotension and cerebral ischemia. Aortocaval compression may have contributed to the hypotension as well. Intubating the patient, reducing the heart rate, and providing left lateral positioning were the correct things to do, and the patient survived what was probably a near-fatal episode of tachycardia and systemic hypoperfusion.

L-9: **The mitral stenosis patient is still in danger after delivery**

Even after delivery, the care of these patients is far from over. Most deaths occur between the 2nd and 9th days postpartum [14]. Postpartum patients with mitral stenosis should be admitted to an intensive care unit following delivery. The most common complications are arrhythmia and pulmonary edema. The significant increase in cardiac output postpartum as well as the possibility of decreased surveillance may be responsible for the high incidence of postpartum deterioration. Many clinicians advocate continuing epidural anesthesia for 24 h postoperatively to help minimize the postpartum increase in cardiac output. Diuretics can be employed to help manage the fluid balance. Hemodynamic goals postpartum are the same as those sought throughout pregnancy and labor.

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Chapter 35
Unrecognized Uterine Hyperstimulation Due to Oxytocin and Combined Spinal-Epidural Analgesia

Thomas L. Archer

The anesthesia resident was called to evaluate a 32-year-old gravida 1 para 0 in labor at term for epidural placement because of severe labor pain at 6–7 cm of cervical dilation. The patient had previously asked not to be visited by the anesthesiologist because she wanted to have her baby without pharmacological interventions for pain (**L-1**).

The patient's labor was being augmented with an oxytocin infusion, and she was having strong contractions every 2–3 min (**L-2**). Other than the severe pain, she had no medical problems and her pregnancy had been problem-free. When the anesthesia resident first saw her, she was screaming and writhing in bed with each contraction. The patient's blood pressure was 130/80 mmHg and her heart rate was 122 beats per minute (bpm). After an expeditious history, physical exam and the obtaining of informed consent, the resident gave the patient 100 mcg of fentanyl intravenously in divided doses over 2 min and the anesthesia attending gave the patient another 50 mcg a few minutes later while the resident was setting up for the epidural (**L-3**). The attending anesthesiologist suggested a combined spinal-epidural (CSE) because of the severe degree of pain and he drew up 1 mL of 0.25 % bupivacaine and 0.5 mL of fentanyl (25 mcg), for a total intrathecal injection volume of 1.5 mL (**L-4**).

With the help of the intravenous (IV) fentanyl, the patient was able to position herself and hold still for the epidural and the resident easily located the epidural space on her first attempt. She passed the spinal needle into the CSF using the needle-through-needle technique and injected the intrathecal dose. The patient received rapid and complete pain relief within a couple of minutes and after 5 min was asking whether the contractions had stopped.

The patient's cell phone rang, she answered it and—with a smile on her face—told the person calling her that the anesthesiologist had done a wonderful job and that she was now completely pain-free, whereas she had been in agony 30 min before.

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The anesthesia resident, feeling pleased with her care, finished taping the epidural catheter in place and was setting up the infusion pump for the continuous epidural infusion when the nurse said, "OK, dear, we're going to give you a little oxygen and turn you onto your left side since baby isn't very happy right now." The anesthesia resident glanced at the clock and noticed that approximately 15 min had passed since the intrathecal injection of the CSE (**L-5**). Her attending had left the room by then and she turned to the nurse and said, "Yes, let's get her all the way over onto her left side, open up her fluids, give her the oxygen, and then let's check her blood pressure." The blood pressure was 105/64 and the pulse was 78. The resident then gave the patient 100 mcg of phenylephrine, having diagnosed the problem as maternal hypotension, aortocaval compression, and acute uteroplacental insufficiency causing fetal hypoxemia and bradycardia (**L-6**).

"What's the fetal heart rate?" she asked the nurse.

"It's about 60–80, but I may be getting Mom's pulse rate. I've been having trouble getting the heart tones for a while now and when we changed position I lost the baby. I'm going to call the doctor (**L-7**)."

At that moment an obstetric resident rushed through the door, saying, "How're we doing here? How long have we been down?"

"I'm not sure," the nurse replied. "I've been having trouble getting the baby on the monitor and I may be getting the Mom's heart rate. I had the baby pretty well before we turned, but now I can't get it back."

"OK, so let's put on a fetal scalp electrode right now," the OB resident said (**L-8**).

By that time other obstetrical residents and nurses had arrived. One nurse continued to search for the fetal heart tones using the external Doppler while another nurse helped the OB resident place a fetal scalp electrode. The placement was prompt and the fetal heart rate was confirmed to be 64.

At this point the anesthesia resident said, "She did get a little hypotensive after the CSE, but we turned her onto her side and gave her oxygen, fluids, and phenylephrine and now her BP is 130/68, which is back to normal. So I think we're OK there" (**L-9**).

The fetal heart rate remained in the 60s and the OB resident then said, "OK, well then, tell them to get ready in the back. I think we'd better take her to the OR just in case the heart rate doesn't recover. Let's get moving. Please call my attending STAT."

The anesthesia resident confirmed that the patient was alert and able to breathe deeply and move her legs, although they were slightly weak. Her grip strength was good. She told the nurses to call her attending as well and she started to unplug the bed from the wall. "Ma'am, you and your baby are going to be OK but we'd better take you to the OR just in case you need a CS. We'll get you completely numb with medication through the epidural," she said.

In the OR the patient was able to help move herself onto the OR table (**L-10**) and the anesthesia resident administered 5 mL of 2 % lidocaine with bicarbonate and epinephrine in order to augment the epidural block in preparation for cesarean section. The fetal heart rate from the fetal scalp electrode was 58. She tested the block and the patient had a T6 level to ice, although she could still move her legs. "We'll get the level up real quick, in case you have to do a C-section," she told the OB

resident. The patient was lying in the full left lateral position and the anesthesia resident was giving her 100 % oxygen by face mask from the circle system of the anesthesia machine (**L-11**).

By that time the OB and anesthesia attendings had arrived and were informed of the sequence of events.

The OB attending felt the patient's abdomen and said, "She's right in the middle of a contraction now. How often has she been having them?"

The nurse said, "I'm not sure. She was in a good pattern prior to the epidural, but we've been focused on getting the heart tones. That's why we put on the fetal scalp electrode."

"She may be hyperstimulated," said the OB attending. "Let's get some terbutaline in the room" (**L-12**).

A nurse rapidly returned with an ampule of terbutaline.

"Yes, her uterus is still hard," said the OB attending. "It hasn't relaxed at all. Go ahead and give her the terbutaline."

The nurse gave the ampule of terbutaline subcutaneously in the patient's thigh.

The anesthesia resident—finally realizing what was going on—then said, "I've got some nitroglycerin spray on my cart. Do you want me to give some under her tongue?"

"Yes, that would be great," replied the OB attending.

The anesthesia resident got a small red spray bottle from the anesthesia cart, had the patient open her mouth and raise her tongue, and directed two sprays at the base of her tongue. "There," she said. "Maybe that will help too."

The attending obstetrician then asked, "How long has the oxytocin been turned off?" and the OB resident and the nurse exchanged a rapid glance. "Oh wow," the nurse said, staring at the infusion pump. "It's still on. I'm turning it off right now."

"That would be a good idea," said the OB attending.

The nurse turned off the oxytocin infusion, which had been running during the entire episode of fetal bradycardia.

Two minutes later the obstetrician said she could feel the uterus relaxing. Soon afterwards the fetal heart tones recovered to 160.

The patient was observed in the OR for another 15 min and was then returned to her labor room. The oxytocin was very gradually reintroduced, a good contraction pattern developed once again and 1 h later her cervix was completely dilated. The epidural portion of the CSE worked well. The patient was pain-free but was still able to move her legs and to push effectively and she delivered a vigorous baby girl vaginally after a 90-min, pain-free second stage of labor (**L-13**).

Lessons Learned

L-1: **See laboring patients early**: **if they let you**

In our labor and delivery unit, we try to see patients for an anesthesia consultation early in labor, before labor pain becomes intense. This allows time for a relaxed and thorough evaluation of the patient and an unhurried discussion of the analgesic

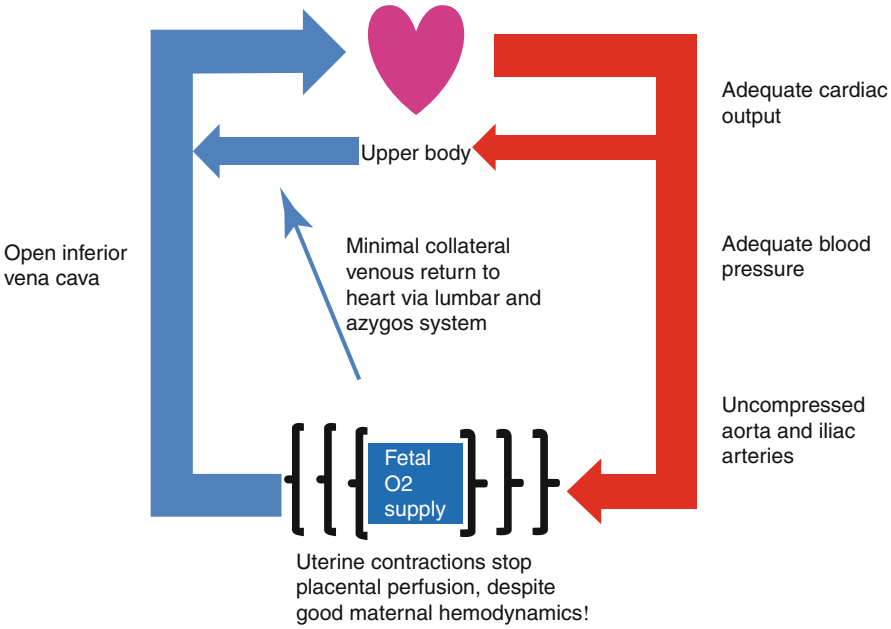


Fig. 35.1 Despite adequate cardiac output, uterine artery pressure, and the lack of obstruction of the inferior vena cava, excessive uterine contractions may asphyxiate the fetus, since the placenta is not perfused by the mother during uterine contractions. With an epidural in place, contractions may be painless and hence the obstetric care team may be unaware that they are occurring unless they look (or rather feel) for them. If an intrauterine pressure catheter (IUPC) is in place, it will allow the increase in intrauterine pressure to be appreciated, but otherwise, a tetanic uterine contraction must be detected by *palpation of the uterus*, which should be within the skill set of the obstetric anesthesiologist

options. It also allows time for the anesthesia team to become aware of any possible problems in the patient's care, such as poor IV access, a poor airway, obesity or a history of poor blood coagulation, previous back problems, or difficulties with anesthesia. When patients specifically ask not to be visited by "anesthesia" during labor, it may make their care more problematic and increase their risk of complications, since it is suboptimal to meet a patient for the first time when a crisis has already developed.

L-2: **Placental perfusion can be precarious and uterine contractions stop it**

Maternal blood flow to the placenta stops during uterine contractions, as shown in Fig. 35.1 of this case, and excessive uterine contractions can cause fetal hypoxemia. Other threats to fetal oxygenation are shown in Figs. 31.1, 31.2, 31.3, 31.4, and 31.5.

In this lesson we offer a closer view of placental perfusion—from the point of view of the intervillous space, which is the location of the interface for gas exchange between the maternal and fetal circulations.

Figure 35.2 shows normal placental perfusion. Fetal blood in the capillaries of the chorionic villi receive oxygen by diffusion from the "lakes" of continually

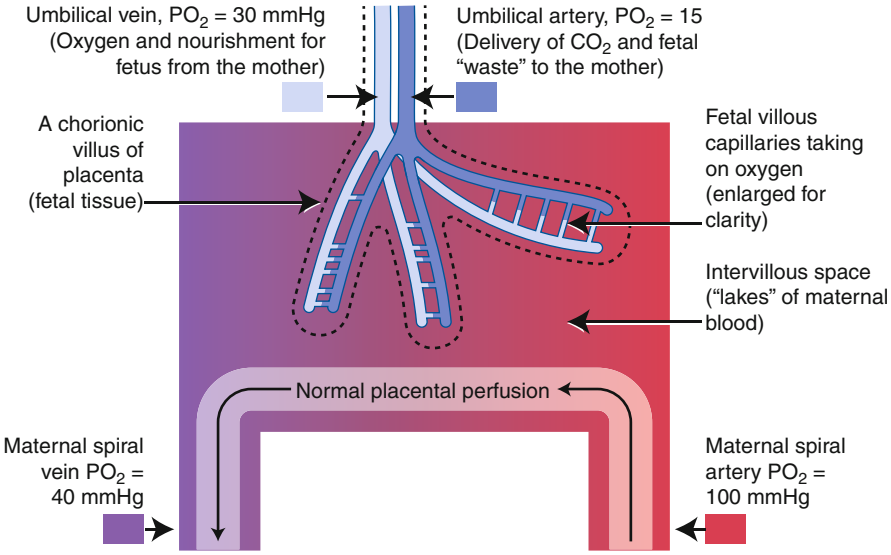


Fig. 35.2 Even under the best of circumstances, maternal blood in the intervillous spaces has what we normally think of as a low pO_2—probably around 40 mmHg—and the pO_2 of umbilical venous blood never even reaches this figure. Fetal arterial blood has an even lower pO_2, because varying amounts of deoxygenated blood are mixed with umbilical venous blood before the mixture is pumped to the fetal tissues via the fetal aorta. The blood perfusing the fetal heart and brain has a higher pO_2 than that which is pumped back to the placenta and the lower body due to the functions of the patent foramen ovale and ductus arteriosus. A P50 of approximately 20 mmHg and a high hemoglobin concentration are two mechanisms by which the fetus is able to live aerobically, despite what would be very low oxygen tensions for an adult human being

refreshed maternal blood within the intervillous spaces. The pO_2 of that maternal blood represents the absolute upper limit of what umbilical venous blood could possibly achieve. In fact, the pO_2 of umbilical venous blood is well below that of the maternal blood in the intervillous spaces due to limitations on the diffusion of oxygen and also because the placenta itself consumes a lot of oxygen.

As shown in Fig. 35.3, any acute or chronic reduction in maternal placental perfusion will even further reduce the pO_2 of the intervillous spaces, making fetal oxygenation even more precarious.

Figure 35.4 shows this process of reduction of intervillous perfusion carried to an extreme: tetanic uterine contractions cause a cessation of intervillous blood flow, and despite the fact that the fetus continues to perfuse the placenta from its side (for a while, until fetal cardiac arrest occurs), there is no oxygen for the fetal blood to pick up!

Acute reversible causes of poor placental perfusion are:

1. Maternal hypotension
2. Aortocaval compression
3. Excessive uterine contraction (hyperstimulation, as in this case)

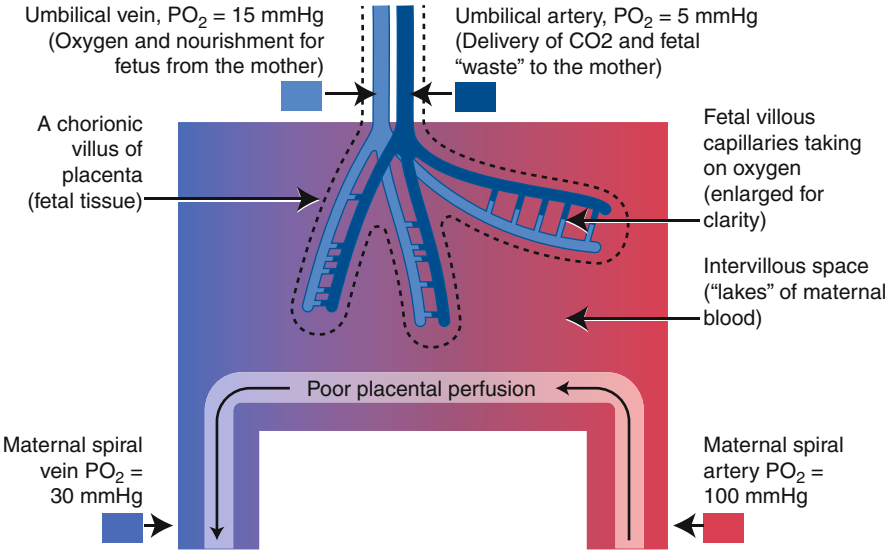


Fig. 35.3 Since fetal oxygen consumption is relatively fixed (or at least cannot decrease below a certain level without fetal damage or death), any decrease in placental perfusion must be accompanied by increased oxygen extraction from intervillous blood, leading to an even further reduction in intervillous blood pO_2. There are many causes of reduced placental perfusion—both acute and chronic and reversible and irreversible—and the patient needs to be managed in labor in such a way as to prevent further decreases in placental perfusion. Chronic and nonreversible reductions in placental perfusion such as those due to chronic hypertension and preeclampsia cannot be helped, but other acute and reversible factors—such as aortocaval compression and maternal hypotension—can and must be recognized and corrected, or better, prevented. *Reversible causes of reduced placental perfusion*: *hypotension*, *aortocaval compression*, *and excessive uterine contractions*. *Nonreversible causes*: *placental disease*, *preeclampsia*, *hypertension*, *diabetes*, *renal disease*, *and placental abruption*

Acute nonreversible causes of reduced placental perfusion are:

1. Placental abruption
2. Prolapsed umbilical cord

Chronic and nonreversible causes of reduced placental perfusion are:

Placental vascular disease, as in preeclampsia, chronic hypertension, diabetes, chronic partial placental abruption, renal disease, and lupus.

You should understand the concept of "fetal intrauterine resuscitation." If "fetal distress" is due to one of the three reversible causes of reduced placental perfusion mentioned above, we can perhaps avoid an emergency CS by recognizing and then correcting the reversible cause.

Furthermore, *you should understand that the chronic nonreversible causes of poor placental perfusion mentioned above make the patient more sensitive to the*

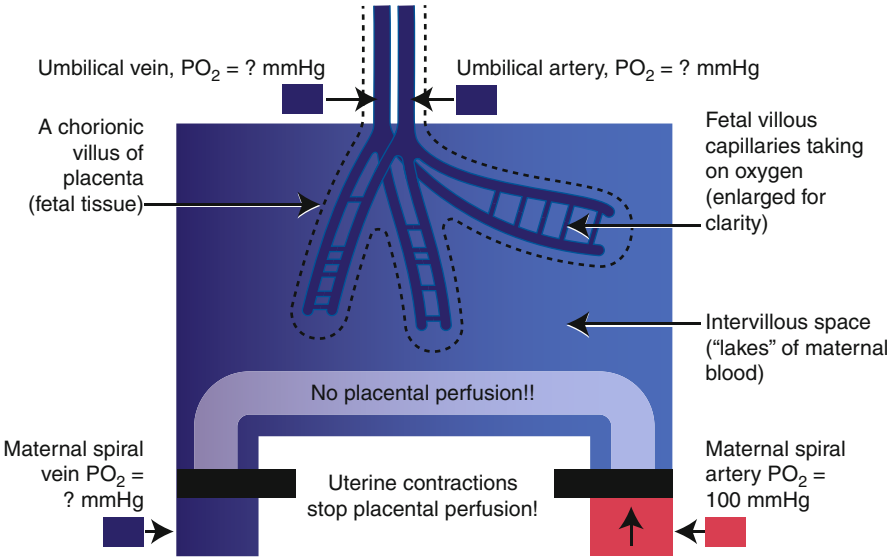


Fig. 35.4 This is a "close-up" picture of the situation in the placenta during a tetanic uterine contraction. No maternal blood enters the intervillous spaces. The fetal circulation continues (until fetal cardiac arrest ensues) but there is no oxygen in the intervillous spaces to pick up! Unless this condition is corrected promptly, the fetus will die. The point of this case is that *fetal intrauterine resuscitation*—in this case by relaxing the uterus—is preferable to an emergency C-section

acute reversible causes. Hence, when we perform a labor epidural, we should be aware of coexisting placental conditions (such as chronic hypertension) which may make placental perfusion more precarious and increase the risk that our epidural will precipitate an episode of fetal hypoxemia.

L-3: **Intravenous fentanyl is usually helpful prior to epidural block placement**
My practice is to administer IV fentanyl in adequate doses when a patient needs an epidural and has significant labor pain. The *advantages* are:

(a) The patient receives meaningful pain relief rapidly.
(b) The patient can position herself better and hold still for the epidural placement, increasing the chances of successful epidural placement and reducing the risk of accidental dural puncture.
(c) The discomfort associated with epidural placement itself is diminished.
(d) Adequate IV fentanyl is especially important in patients for whom epidural placement is anticipated to be difficult (e.g., morbidly obese parturients).

The *disadvantages* adduced by practitioners who do not frequently administer fentanyl in this situation are as follows:

(a) Possible maternal and neonatal respiratory depression
(b) Maternal dysphoria and sleepiness
(c) Systemic analgesia that can mask a poorly functioning epidural

In my opinion, the advantages of IV fentanyl prior to epidural placement outweigh the disadvantages for most patients; however, *if you are going to administer fentanyl to the patient prior to epidural placement, it is essential to monitor her level of consciousness and respiration and make sure that the nurse is physically supporting the patient while she is sitting upright*.

L-4: Combined spinal-epidural (CSE) analgesia has advantages and disadvantages

Common intrathecal doses for a CSE are as follows: bupivacaine 0–2.5 mg and fentanyl 0–25 μg, in a total volume of 1–1.5 mL.

Performing a CSE is always a "double-edged sword." One the one hand, it provides a rapid onset of analgesia or anesthesia. On the other, the epidural catheter remains untested and may fail later on during labor or during C-section. Furthermore, as occurred in this case, the rapid pain relief associated with a CSE may promote excessive uterine contractility ("hyperstimulation"), which in turn causes fetal hypoxia and bradycardia. (See **L-12** for possible mechanism.)

L-5: When facing "fetal distress," don't forget to assess the patient for uterine hyperstimulation

Hyperstimulation due to rapid pain relief usually occurs within 30 min after pain relief. This is also the period of time during which maternal hypotension and aortocaval compression usually declare themselves and it is of key importance to look for *both* phenomena whenever "fetal distress" follows an epidural block. In my experience, I have found that often neither the nurse nor the anesthesia resident thinks of this possibility at this point. The nurse is focused on "finding the baby" and the anesthesia resident is focused on setting up the epidural infusion pump and detecting and correcting hypotension and aortocaval compression if they should occur.

L-6: The most common cause of "fetal distress" after epidural is maternal hypotension: but don't forget to assess for hyperstimulation!

Most episodes of fetal bradycardia after epidural or CSE placement are due to maternal hypotension, with or without aortocaval compression. The resident saw a mild reduction in blood pressure after her block and assumed that this was the cause of the fetal bradycardia, as it usually is. However, 5–10 % of the time, the pain relief due to the neuraxial block may contribute to uterine hypertonicity, especially if oxytocin is being used for labor augmentation and *excessive uterine contractions can precipitate acute fetal hypoxemia*.

Hence, it is essential that at least one experienced person (nurse, midwife, or MD) palpate the uterus frequently during episodes of "fetal distress" to evaluate uterine tone. In the rush to "find the baby" (the fetal heart tones), this step was forgotten.

L-7: "Fixation errors" or "cognitive tunnel vision": forgetting the big picture

Sometimes obstetric care providers focus excessively and exclusively on "finding the baby," that is, detecting the fetal heart rate with the external Doppler. While important, a single-minded "fixation" (see below for a discussion of "fixation errors") on this parameter—particularly when it is difficult to detect—may prevent the detection of other problems, such as (in this case) a hypertonic uterus. Detecting

fetal bradycardia—while of key importance—is *not* a therapeutic intervention, and in this case, the team forgot to check for uterine hyperactivity and to turn off the oxytocin infusion—perhaps because everyone was focused on "finding the baby."

The obstetric care team was falling into what cognitive psychologists call a "fixation error" or "cognitive tunnel vision." The nurse was focused on finding the baby. Her sole emphasis was on detecting the fetal heart rate. When she had difficulty finding the fetal heart tones, her impulse was to fix the problem of "detecting the fetal heart rate" rather than focusing on the more important problem of fixing possible fetal hypoxia.

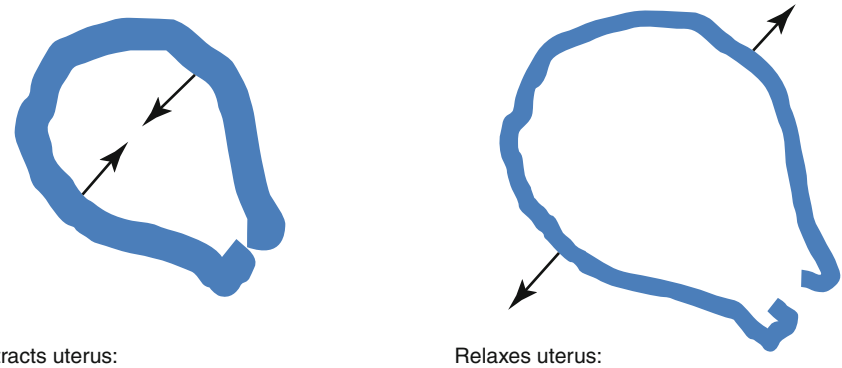
The anesthesia resident was perhaps thinking more physiologically. She had learned to look for hypotension and aortocaval compression as the most common causes of "fetal distress" following a neuraxial block and her thinking was supported by the fact that the BP was somewhat lower.

L-8: **Situation awareness**: **avoiding** "**fixation errors**."
The obstetrics resident follows the nurse's lead and—quite sensibly—decides to measure fetal heart rate with a fetal scalp electrode (often abbreviated as "FSE"). The cervix at this point was well dilated and the membranes were ruptured, so placement of a FSE would be anticipated to be easy. Placement of the FSE was not an absolute mistake at this point. The big relative mistake was forgetting to look at the entire situation: painful labor, oxytocin infusion, rapid pain relief by CSE, etc. Looking at the *entire situation* and constantly *reviewing one's initial assessment* is called "situation awareness" and is a key mental technique for managing a crisis well.

L-9: **The** "**blood pressure problem**" **may not be the real problem**.
Within the limitations of her knowledge and experience, the anesthesia resident is performing well. She has recognized and successfully treated hypotension and possible aortocaval compression. The problem is that the problem she has fixed is not the real problem! The real problem is a tetanic uterus. The obstetric care team has "cognitive tunnel vision" at this point.

L-10: **Simple observations can be of key importance**. **How well can she move her legs**?
The anesthesia provider should carefully observe whether the patient can move herself onto the OR table or not since that observation gives important information about the quality of the epidural and spinal block already in place.

L-11: **Left uterine displacement and 100 % oxygen are not always enough**
The patient is lying on her left side to prevent aortocaval compression and is receiving 100 % oxygen by face mask and the circle system. Both maneuvers would ensure maximum oxygen delivery to the placenta and fetus, *if* maternal arterial blood could traverse the uterine wall and reach the intervillous spaces. However, as shown in Fig. 35.4, a strong uterine contraction has cut off all blood flow to the intervillous space of the placenta, and if the contraction is tetanic, the fetus will be asphyxiated, despite the measures mentioned above. Giving 100 % oxygen to the patient at this point is also a good idea in case general anesthesia will be required, but 100 % oxygen is not helping the fetus, since the tetanic uterine muscle is preventing maternal blood from reaching the placenta.



Contracts uterus:

Normal labor
oxytocin

Methylergonovine ("Methergine")
Prostaglandin F2-alpha ("Hemabate")
Misoprostol

Relaxes uterus:

Pain (epinephrine effect?)
Beta agonists (terbutaline, epinephrine)
Nitroglycerin (IV or SL)
Volatile anesthetics
Magnesium sulfate

Fig. 35.5 This figure summarizes the effects of various pharmacologic agents on uterine tone

L-12: **Rapid pain relief in labor can promote hyperstimulation**, **possibly due to decreasing epinephrine in maternal blood**

The causes of uterine "hyperstimulation" following rapid pain relief in labor are not fully understood, but most authorities believe it has to do with the decrease in circulating epinephrine which occurs when severe labor pain is suddenly relieved. Beta-agonists (such as epinephrine and terbutaline) cause uterine relaxation and once the uterine relaxant effect of epinephrine is removed, influences promoting uterine contraction (such as concurrent oxytocin administration) become dominant and contractions increase or even become tetanic.

Figure 35.5 shows common pharmacologic agents which have effects on uterine tone.

L-13: **Successful management of a crisis requires both detailed knowledge and situation awareness**. **The correct path is often uncertain and you may have to perform many interventions at once**

It took a fresh look on the part of more experienced eyes (the OB attending) to recognize that something else was going which caused the "fetal distress": unrecognized uterine hyperstimulation. An ongoing oxytocin infusion and rapid pain relief were contributing factors. The failure to turn off the oxytocin infusion could have been prevented, perhaps, by an ability to stand back from the current situation and "scan the environment" so as to understand the entire context of the case, rather than focusing solely on obtaining the fetal heart rate and preparing for an emergency C-section to save the baby.

When fetal bradycardia occurs, it is not always obvious what the cause is and sometimes obstetric caregivers adopt a bundle of measures simultaneously: stopping the oxytocin, maternal position change, oxygen, fluids, and the administration

of a vasopressor such as ephedrine (which conveniently has beta-agonist effects which may help relax the uterus)—all in an attempt to address whatever is the cause of the fetal hypoxia—whether it is hypotension, aortocaval compression, nuchal cord, other cord compression, or (as in this case) hyperstimulation.

Fetal intrauterine resuscitation is a key concept for obstetric and obstetric anesthesia caregivers. *Try to correct the fetal oxygenation problem in utero without having to rush to C-section*. There are several *reversible* causes of failure of fetal oxygenation which can be corrected without resorting to immediate C-section: hypotension, aortocaval compression, hyperstimulation, and—less commonly—a maternal hypoxic episode due to a seizure. It is often in the best interest of both fetus and mother to allow the fetus to be resuscitated in utero rather than subjecting the mother to an emergency C-section with all of the risks attendant on that scenario.

In this case the team actually performed very well in many respects: nursing, anesthesia, and obstetrics communicated well together in an explicit, open, and constructive manner, and despite the crisis the team performed with apparent calm and worked well together. The team members called for help promptly—and the attendings who came did, in fact, solve the problem!

Before the arrival of the OB attending, the team simply did not have sufficient knowledge of the clinical possibilities to be able to think of hyperstimulation as a possible cause of the syndrome they were encountering.

But more knowledge is not always sufficient in a crisis. One must retain the capacity to keep the "big picture" in view and to change one's thinking as to what is going on. The key lesson of this case is to continually be willing to revise your assessment of what is happening to the patient and to try not to "fixate" on one diagnosis or course of action which may be incorrect. This problem of "fixation error" or "cognitive tunnel vision" is why many airplanes get flown into mountains and why some patients die despite having remediable conditions.

Chapter 36
Super-Morbidly Obese Patient for Elective Repeat Cesarean Section

Thomas L. Archer

A 32-year-old woman, gravida 2 para 1, was referred to the UCSD Obstetric Anesthesia consultation service (**L-1**) at an estimated gestational age of 36 weeks and 4 days because of super-morbid obesity (**L-2**) and a prior bad experience with anesthesia. Her current weight was 356 lb, height was 70 in., and her body mass index (BMI) was 51. She had recently been diagnosed with obstructive sleep apnea, but had not yet been fitted with a continuous positive airway pressure (CPAP) device

History Obtained During Consultation

She gave a history with her first pregnancy of failed epidural anesthesia for labor and a subsequent cesarean section (C-section) performed under general anesthesia (GA), after a very unpleasant awake fiberoptic intubation. Her labor had been long and painful and the anesthesia team had been unable to place a labor epidural despite many attempts. She suffered greatly in labor despite intravenous (IV) narcotics and ultimately had to have a C-section due to the failure of her cervix to dilate past 6 cm.

For the C-section, the anesthesia team had again tried to place an epidural in the operating room, thinking that improved light and positioning and the help of a second anesthesiology attending justified another attempt at epidural placement. After an hour, the anesthesia team was able to place an epidural catheter but the resulting block was dense in her right leg and abdomen but only partial on the left, and the block could not be made bilateral by pulling back the catheter.

The anesthesia team had then desisted from further attempts and induced GA after an awake fiberoptic intubation, which the patient found to be very traumatic.

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The rest of the C-section proceeded uneventfully, although the baby was born somewhat depressed due to the GA and prolonged surgery time. The baby required bag and mask ventilation briefly but then became vigorous and had no sequelae.

Physical and Ultrasound Examination Obtained During Consultation

On physical examination during the current consultation, the patient was massively obese and had a Mallampati class 3–4 airway with a large tongue and an obese neck. Her larynx was posterior (the thyromental distance being at least 6 cm), prognath was good, submandibular compliance was good, and the anesthesia attending and resident felt that she could probably be ventilated via mask and/or intubated with a standard laryngoscope despite her obesity. The rest of her physical exam was unremarkable.

A standard obstetrical ultrasound machine from labor and delivery was used to examine her lumbar spine in the sitting position with maximum flexion. Facet joints, transverse processes, and weak spinous process "shadows" were seen at two different lumbar levels. There was no spinal rotation evidenced by the fact that the facet joints and transverse processes on both sides were equally distant from the skin surface. The spinous processes that could be seen in the lumbar region were approximately 2.5 cm to the left of a line drawn directly cephalad from the gluteal cleft. The approximate depth to the ligamentum flavum was 10 cm when the ultrasound probe was only minimally compressing the tissue. A very weak posterior longitudinal ligament echo was seen at two different levels. The echo was maximally bright with an ultrasound probe angle of about 5° cephalad from perpendicular to the skin, and hence that was judged to be the proper insertion angle (*Note*: a description of how to use ultrasound to obtain all this information is given in the Appendix figures (Figs. 36.A1, 36.A2, 36.A3, 36.A4, 36.A5, 36.A6, 36.A7, 36.A8, 36.A9, 36.A10, 36.A11, 36.A12, 36.A13, 36.A14, 36.A15, and 36.A16). The author strongly suggests reading these at this point in order to fully understand the rest of the case).

The patient was counseled that we believed we could see appropriate landmarks in her spine using ultrasound and that we felt we had a reasonable chance of success in placing a continuous lumbar epidural when she returned to the hospital for her C-section. We emphasized, however, that we could not guarantee success with epidural placement and that an awake fiberoptic intubation and general anesthesia might still be required.

Patient Returns for Her Elective C-Section

The following description includes steps taken before, during, and after the ultrasound-assisted placement of the epidural catheter.

When the patient returned for her elective C-section, we followed our UCSD Obstetric Obesity Bundle, as follows (*Steps 1–10*):

1. Routine measures were taken, including reduction in gastric acidity and volume (oral sodium citrate, IV famotidine, and metoclopramide) and pulmonary embolism prophylaxis with sequential compression devices.
2. The OR technician put wideners on both sides of the OR table and then covered it with an "Air-Pal" inflatable mattress for use in sliding the patient from the OR table to her hospital bed after surgery was completed.
3. The anesthesia resident "ramped" the head of the OR table to facilitate spontaneous ventilation and intubation if it were to be required.
4. Laryngeal mask airways were checked and a Glidescope and fiberoptic tower were brought into the OR.
5. A bed was obtained in the surgical intensive care unit for postoperative monitoring because of her known obstructive sleep apnea, and Respiratory Therapy had been notified that she would need a CPAP device while in the hospital. This point was thought to be especially relevant since the patient might be receiving neuraxial, IV, and oral opioids for postoperative pain.
6. Before taking the patient back to the OR, we talked with her again at length and achieved "psychological buy-in" with the patient for the upcoming attempt at a possibly difficult neuraxial block (**L-3**).
7. Also before going back to the OR with the patient, we tried to "manage her expectations," meaning that we tried to make her realize and accept the fact that the neuraxial anesthesia might not be perfect (she might feel pressure and some discomfort, etc.) and that we hoped she would try to accept some discomfort in order to avoid the risks of GA (**L-4**).
8. Once in the OR, we tried to help the patient to relax using a warm, cheerful, and sympathetic interpersonal manner and by warming the room and applying warm blankets to her shoulders and anterior chest (**L-5**).
9. We applied oxygen by face mask to the patient in the sitting position since we planned to use IV analgesia prior to our epidural placement.
10. We used adequate IV analgesia (fentanyl 100 μg) before starting the block in order to *prevent and avoid* distress (rather than reacting to it once the patient was in pain and emotionally upset or angry).

For the ultrasound-assisted placement of the epidural catheter and surgery we proceeded as follows (*Steps 11–20*):

11. We used a standard obstetric ultrasound machine to acquire the following key pieces of information: two Tuohy needle insertion points (a "best" level and a "backup" level), the best needle insertion angle and the approximate depth from the uncompressed skin surface to the ligamentum flavum (See Figs. 36.A1, 36.A2, 36.A3, 36.A4, 36.A5, 36.A6, 36.A7, 36.A8, 36.A9, 36.A10, 36.A11, 36.A12, 36.A13, 36.A14, 36.A15, 36.A16 and **L-6**).
12. In this patient we found the "best" and "backup" lumbar insertion points; each one was about 2.5 cm to the left of the line running directly cephalad from the gluteal cleft (**L-7** and Figs. 36.A4, 36.A8, and 36.A11).

13. We estimated the depth to the ligamentum flavum at both insertion points to be about 10 cm and we reconfirmed that our insertion angle should be 5° cephalad from perpendicular to the skin. See Fig. 36.A12.
14. The resident used abundant local anesthesia to create an "insertion line" for the Tuohy needle (**L-8** and Fig. 36.A16).
15. The resident tried to insert the Tuohy needle in one smooth, continuous pass to a depth of 8.5 cm, but the needle contacted bone at a depth of 7 cm, as shown in Fig. 36.A15. Believing this structure to be spinous process, she removed the needle and reinserted it 1.0 cm below the first insertion site but with the same insertion angle, as shown in Fig. 36.A16. After passing through many centimeters of fat, the resident could feel slightly gritty tissue starting at about 7 cm and she continued to advance the needle until the tip was at a depth of 8.5 cm, as shown in Fig. 36.A14.
16. The resident then applied the glass loss of resistance syringe and advanced the needle in the usual fashion, encountering first a very gritty ligamentum flavum and then achieving a clear cut loss of resistance at 10.7 cm. See Figs. 36.A14 and 36.A16.
17. The resident expanded the epidural space with normal saline, passed the catheter into the space, and withdrew the Tuohy needle in the usual fashion. The catheter was left at 15 cm at the skin, an estimated 4.3 cm into the epidural space.
18. The test dose through the epidural catheter was negative, and the patient was then dosed with 100 μg fentanyl epidurally, followed by two 5 mL doses of lidocaine 2 % with bicarbonate and epinephrine. The patient remained sitting upright until both of her thighs started to turn numb (**L-9**). We then asked her to lie on her side and we affixed the epidural catheter to the skin only *after* she had laid down. This delay in affixing the catheter to the skin until after the patient lies down is known as the Cohen Maneuver and is performed in order to reduce the possibility that adoption of the recumbent posture will pull the epidural catheter out of the epidural space (**L-10**).
19. A solid bilateral block developed within 15 min after a total of 15 mL of lidocaine 2 % with bicarbonate and epinephrine had been injected epidurally in divided doses. The patient experienced some tingling in the hands but had no hand weakness and no trouble breathing.
20. The patient was comfortable during uneventful though prolonged surgery and a vigorous baby girl was delivered.

Lessons Learned

L-1: **Obstetric anesthesia consultation service**
Even though it is extra work for the OB Anesthesia Service in the short run, at UCSD we feel that having a "consultation service" within the labor and delivery unit has several advantages and that—in the long run—it leads to better and less stressful care for both the patient and the staff.

The goals of the anesthesia consultation for the morbidly obese pregnant patient should be as follows:

(a) Explaining to the patient the challenges involved in her care in order to obtain her "psychological buy-in" (Jon Benumof) for facing the difficult road ahead.
(b) Discussion of anesthesia options, with emphasis on the possible difficulty of neuraxial block placement and the possible role of ultrasound in facilitating the performance of the block.
(c) Possible need for awake fiberoptic intubation with explanation of the rationale for this and the specific technique.
(d) Examination of the lumbar spine with ultrasound in an attempt to see whether landmarks are visible and the approximate depth of structures in order to know what length of needle will be required and at what depth the loss of resistance should be expected.
(e) Evaluation of peripheral veins to estimate difficulty of obtaining IV access.
(f) A formal anesthesia consultation gives time for in-depth resident education about the obese parturient.

L-2: **Managing maternal obesity requires commitment and coordination of care**

The UCSD Reproductive Medicine Department has a Maternal Weight and Wellness Program to attempt to address the medical and psychological needs of the morbidly obese parturient in an organized fashion. An anesthesia consultation is usually a part of this comprehensive planning process.

Taking care of these patients is very difficult for all concerned, but one member of the Reproductive Medicine faculty had a strong interest and commitment to this area. She was able to obtain institutional support for this endeavor, arguing that the University should attempt to excel in the care of these patients, since obesity in pregnancy is a common and important problem.

The Anesthesia Department realized as well that these patients are difficult to care for, but rather than continue to take care of these patients on an ad hoc basis, we decided to be more proactive and to attempt to develop an "obesity bundle" of techniques for taking care of these special patients (see section "Patient returns for her elective C-section" in the narrative of the case for the specifics of the obesity bundle). Both Departments realized that if we did a good job taking care of these patients, many such patients might be referred to us.

L-3: **Psychological buy-in**

"Psychological buy-in" is a term I learned from Jonathan Benumof, MD, in the context of awake fiberoptic intubation. The concept is of key importance in medicine and means that the patient realizes the importance and potential difficulty of what we are trying to do together for her care. Most obese patients realize that their weight makes their care more difficult and it is often helpful to mention this in a measured and respectful way in order to establish a shared and realistic "mental model" for the upcoming attempt at neuraxial block. Our goal was to make the patient an ally in our common quest for a good outcome.

L-4: **Management of expectations**
"Management of expectations" is another key concept for medical practice. For example, if a patient expects absolutely no discomfort after major abdominal surgery, she will be disappointed and perhaps angry when confronted with reality, even though her care and outcomes were the best achievable with current methods.

L-5: **Patient relaxation**
Patient relaxation is not only good for her mental state but will also help the patient flex her spine and open the interlaminar foramina, thereby increasing the size of our "target" in the cephalad-caudad dimension.

L-6: **Ultrasound to facilitate neuraxial block placement**
It is beyond the scope of this case report to fully explain how to use ultrasound to assist with neuraxial block placement and the reader is referred to excellent references [1, 2]. The illustrations contain an explanation of the rationale and technique for transverse plane, ultrasound-aided neuraxial block which borrows heavily from the work of Jose Carvalho, MD, and KJ Chin, MD.

L-7: **Know where spinous processes are**
Many patients have some degree of scoliosis, and the spinous processes are not always directly cephalad from the gluteal cleft. It is possible that the previous right-sided block was due to epidural catheter placement within the right side of the epidural space, since the true midline was shifted to the left in this patient. See Figs. 36.A1, 36.A2, 36.A3, 36.A4, and 36.A5 for the technique of finding the spinous processes with ultrasound.

L-8: **Infiltrate an "insertion line" for the epidural needle**.
We assumed that we might have to move our insertion point up or down somewhat in the line of the spinous processes, and hence we deeply infiltrated an "insertion line" in order to be able to adjust our insertion point up or down without stopping to reanesthetize the tissues.

L-9: **Leave the patient sitting until you are sure the block will work**.
Leave the patient in the sitting position until you are sure that the block is developing bilaterally. It is very awkward both physically and emotionally for the patient to lay her down and then have to sit her up for a second attempt at epidural placement. On the other hand, do not wait *too* long to lay the patient down because her legs may become weak and she may not be able to help position herself! It is particularly helpful to have the patient position herself properly on the "ramp" before you get too far along. A very good maxim of airway management when you are planning regional anesthesia is to assume that the regional anesthesia will fail and hence to position the patient optimally at the very outset of the case for a possible intubation or other airway maintenance maneuver.

L-10: **The Cohen Maneuver**
The Cohen Maneuver is named after Sheila Cohen, MD. Insert and leave the catheter no more than 4–5 cm within the epidural space when the patient is seated. You will

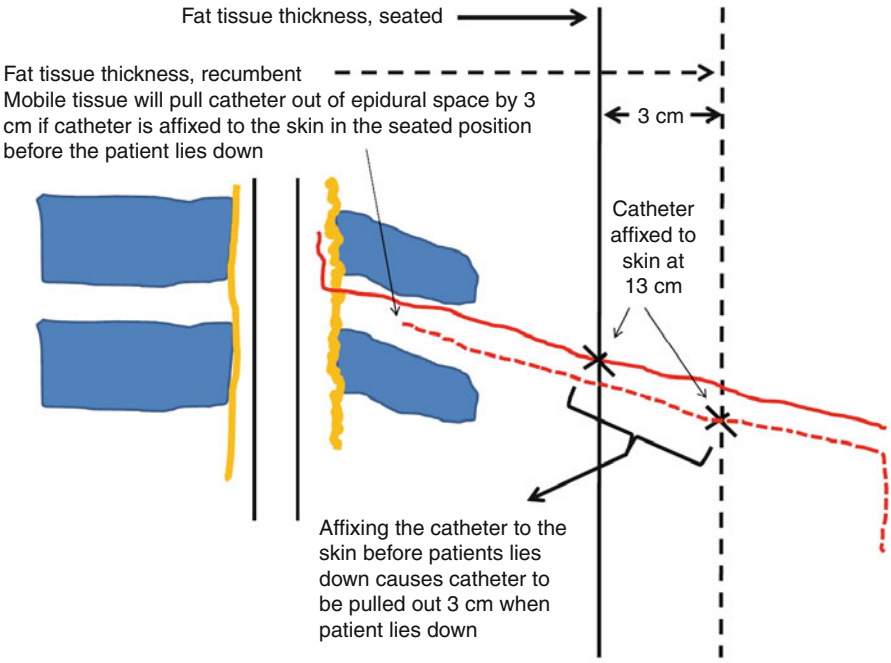

Fig. 36.1 When a seated obese person lies down, lumbar adipose tissue moves outward (gets thicker). If the epidural catheter is affixed to the skin *before* the patient lies down, the outwardly migrating adipose tissue drags the epidural catheter with it and may pull the tip of the catheter out of the epidural space. The solution to this is the "Cohen Maneuver" shown in Fig. 36.2

be performing the Cohen Maneuver to prevent the catheter from being pulled out when the patient lies down, and hence it is unnecessary to leave it in farther. Leaving the catheter farther in the space may lead to a one-sided block. See Figs. 36.1 and 36.2 for how the Cohen Maneuver works. The key idea is that when an obese patient lies down, her lumbar adipose tissue moves outward. If the epidural catheter is affixed to the skin while she is seated, the mobile adipose tissue affixed to the catheter will pull it out of the epidural space as she lies down. This is avoided by allowing the adipose tissue to slide outward along the epidural catheter and then affixing the catheter to the skin only *after* it has moved outward from the spine.

Appendix: Ultrasound-Guided Neuraxial Block

If every patient had palpable spinous processes underlying a line running directly cephalad from the gluteal cleft, neuraxial block would be easier than it is and ultrasound would never be needed

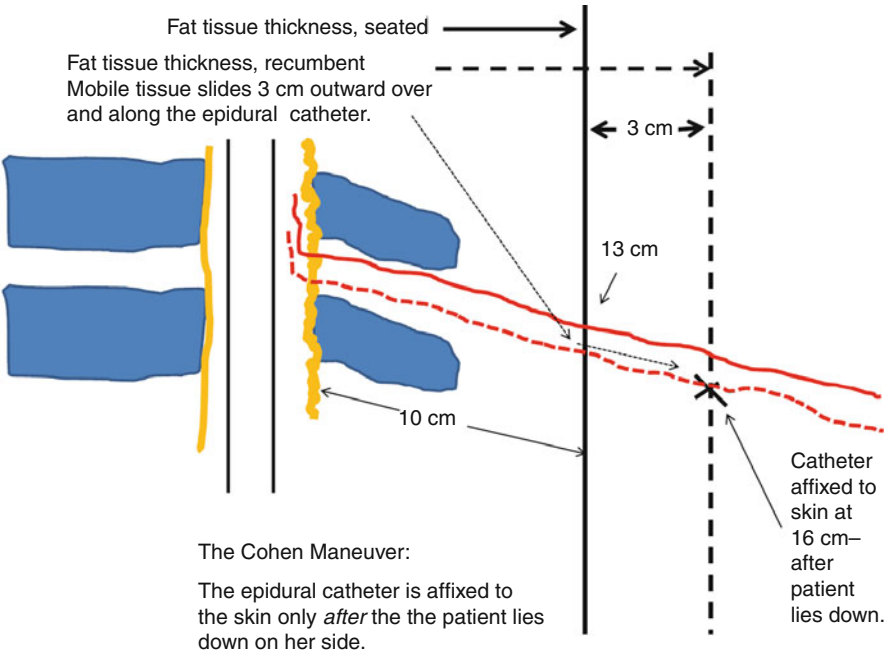

Fig. 36.2 The Cohen Maneuver consists in laying the patient on her side before affixing the epidural catheter to the skin. This allows the outwardly mobile adipose tissue to slide outward over and along the epidural catheter, rather than pulling it out of the epidural space. The catheter is affixed to the skin only *after* the adipose tissue has moved outward as far as it is going to go

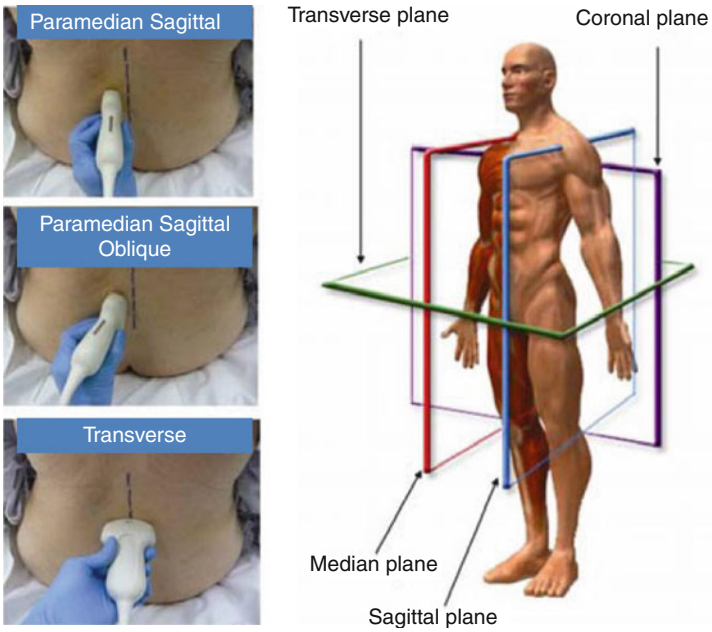

Fig. 36.A1 The left panel shows the three commonly used orientations of the curved obstetric ultrasound probe for examination of the lumbar spine. We only used the transverse view in this case. The labels on the mid-back (paramedian sagittal, paramedian sagittal oblique, and transverse) refer to the plane in which the ultrasound probe is held. Standard anatomical planes are illustrated and overlaid on the human figure to the right (Adapted from Chin et al. [1])

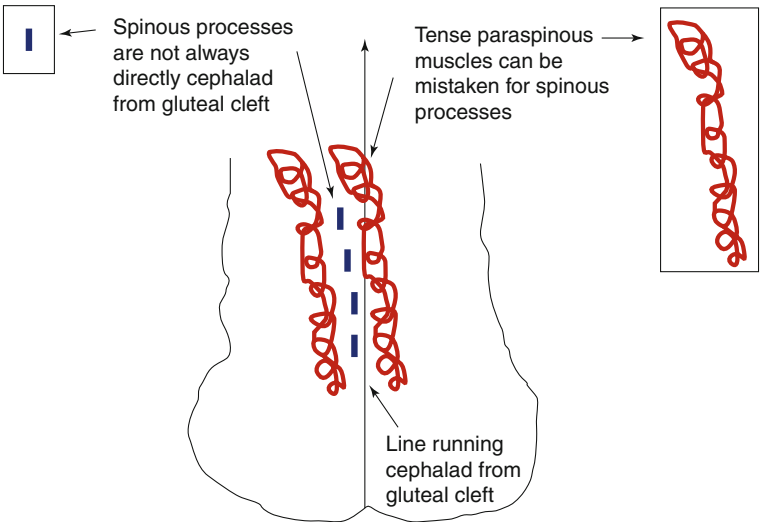

Fig. 36.A2 Scoliosis (spinous processes off the midline) and tense paraspinous muscles (which can be mistaken for spinous processes) are two common pitfalls in performing neuraxial block without ultrasound. Obesity or edema can make spinous processes difficult or impossible to palpate and tense or well-developed paraspinous muscles can be mistaken for bony spinous processes. In cases with very well-developed paraspinous muscles, the paraspinous muscle is what the examining finger feels and the spinous processes themselves are hidden in the "valley" between the hard paraspinous muscles

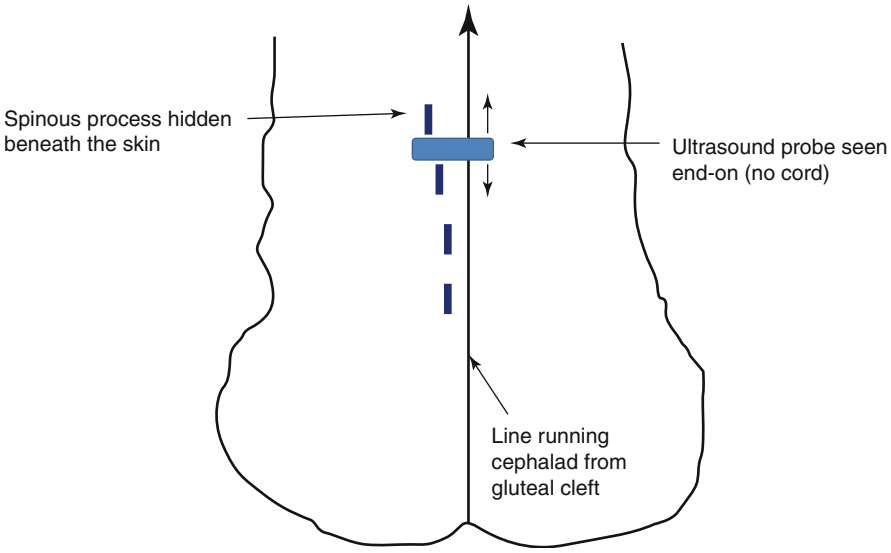

Fig. 36.A3 My ultrasound examination of the lumbar spine begins with a *search for a spinous process*. We know that there are several of them more or less in the middle of the lower back, but we need to *precisely* locate at least one. We have seen already how many people are fooled by scoliosis and by tense paraspinous muscles. We scan the lumbar region where we think the midline is—but we have to keep an open mind, since the spinous processes are not always straight cephalad from the gluteal cleft. Once we find a spinous process by seeing its "shadow," as seen in Fig. 36.A6, our next job is to center the probe over it, as shown in Figs. 36.A4 and 36.A6

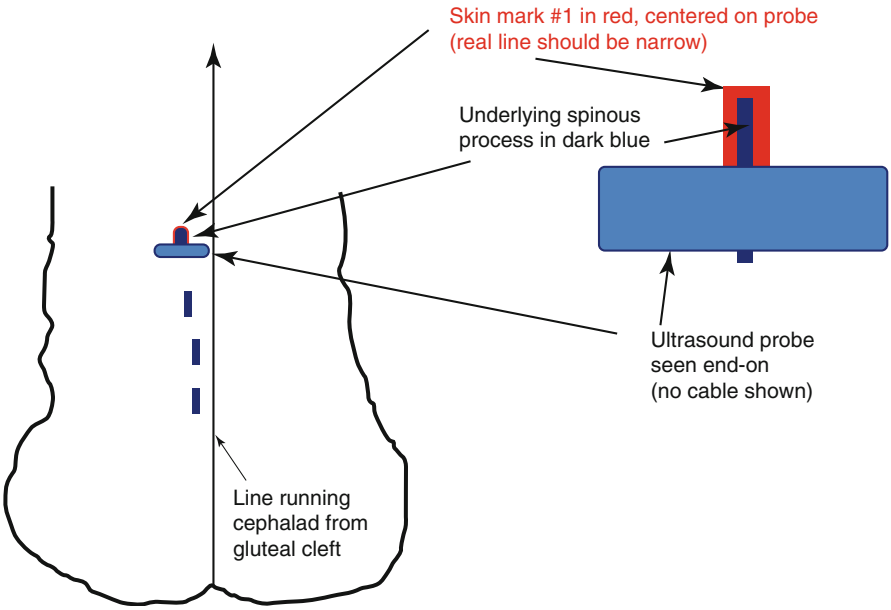

Fig. 36.A4 This figure shows how the curved ultrasound probe (the same used by obstetricians) can "see" through fat, edema, or muscle to *display the true location of the spinous processes*. Once we have found a spinous process by moving the probe up and down the back, *the skin is marked* at the center of the ultrasound probe *immediately or just above the cephalad margin of the probe when the spinous process is precisely centered below the center of the probe, as seen on the ultrasound screen*. An actual ultrasound image corresponding to this situation is shown in Fig. 36.A6. When the center point of the ultrasound probe is over a spinous process, the image in Fig. 36.A6 will be seen. This is the true midline, *but only at that interspace*, since the amount of deviation of the spinous processes off the "midline" (as erroneously judged by the gluteal cleft) may vary from level to level

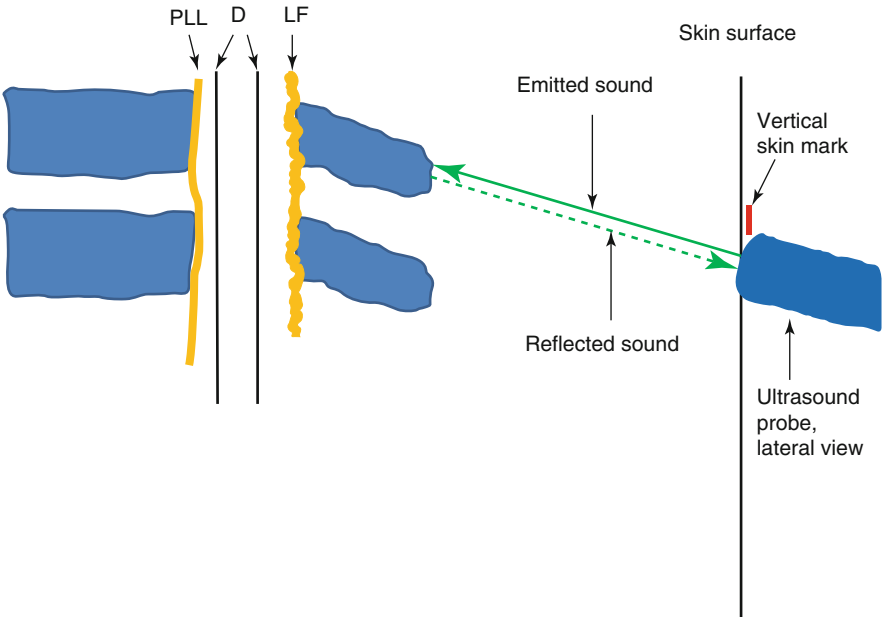

Fig. 36.A5 In this figure we see a *side view of the process* of defining the exact right-left position of the spinous process. The spinous process itself has already been *found* by moving the transverse ultrasound probe up and down the middle of the back until the spinous process "shadow" is seen. The ultrasound bounces off the spinous process, giving the image seen in Fig. 36.A6. What is shown in here is the marking of the skin immediately above the center of the ultrasound probe *once the spinous process shadow underlies the middle of the probe on the ultrasound screen*, as shown in Fig. 36.A6. *Since you will be relying on these skin marks for needle placement, they need to be made with meticulous care and should be checked again after they are made. Millimeters count for success.* Abbreviations: *PLL* posterior longitudinal ligament, *D* dura, *LF* ligamentum flavum

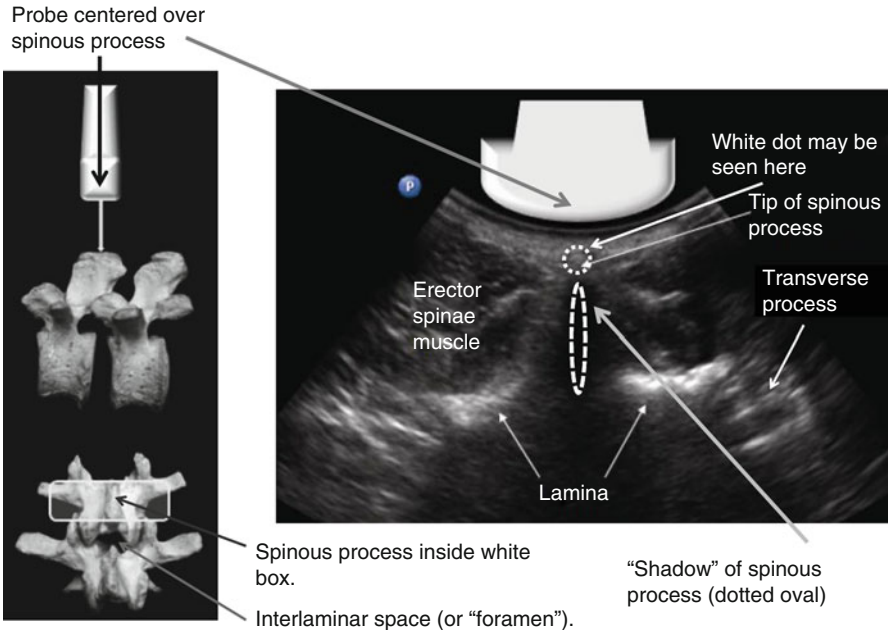

Fig. 36.A6 *Transverse ultrasound view of lumbar spine, with center of probe positioned over the spinous process*, as shown in left panel. The white rectangular box in the lower half of the left panel contains the tip of the spinous process as its center. With probe in this position, nothing is "seen" deep to that structure—only a spinous process "shadow" (as shown in *right panel*). Sometimes, a white dot is seen at the tip of the spinous process, casting the black "shadow" deep to it. This white dot landmark can further allow exact identification of the midline (defined as the spinous process itself at that level). True midline is identified with the probe in this position, and the skin immediately or just cephalad to the center of the probe and overlying the spinous process is marked as shown in Fig. 36.A4 at the center of the ultrasound probe. If performed meticulously, the skin mark should *directly overlie the spinous process at that level* (Adapted from Chin et al. [1])

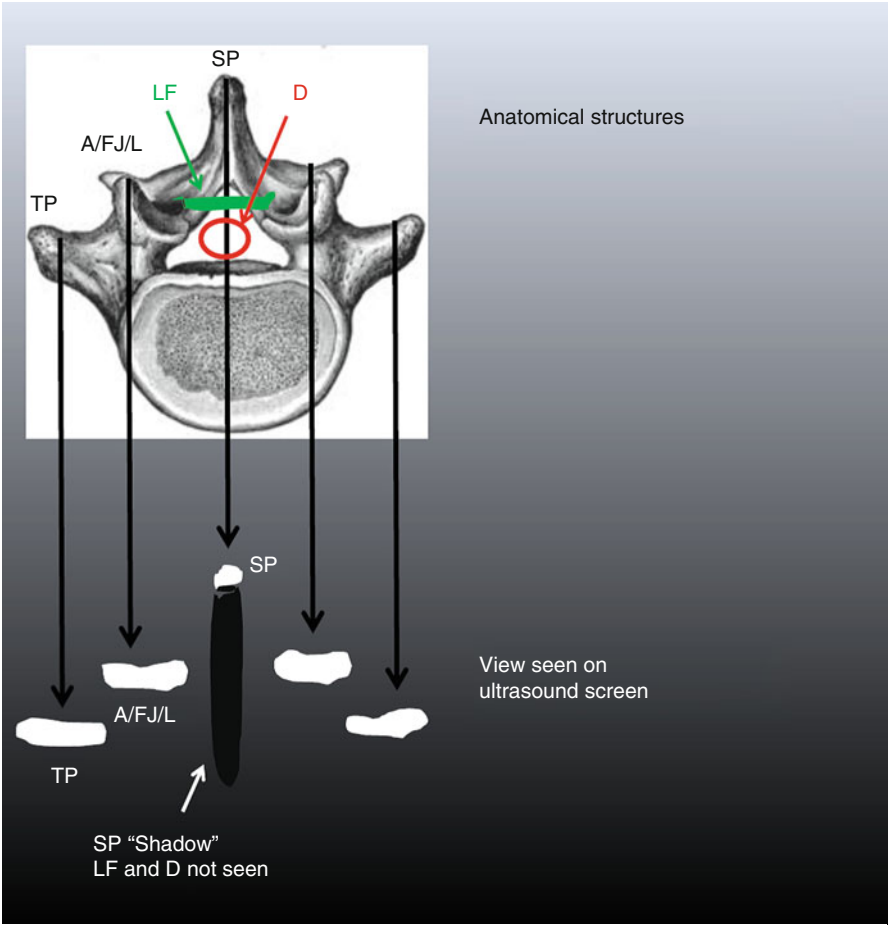

Figure 36.A7 *Schematic diagram explaining anatomic origin of the image seen in the transverse ultrasound view over a spinous process* (see Fig. 36.A6). At this level in the cephalad-caudad dimension, the ligamentum flavum (*LF*) and dura (*D*) cannot be "seen" by the ultrasound probe, since the spinous process is in the way. Abbreviations: *SP* spinous process, *TP* transverse process, *A* articular process, *FJ* facet joint, *L* lamina, *SP* spinous process (Adapted from Chin et al. [1])

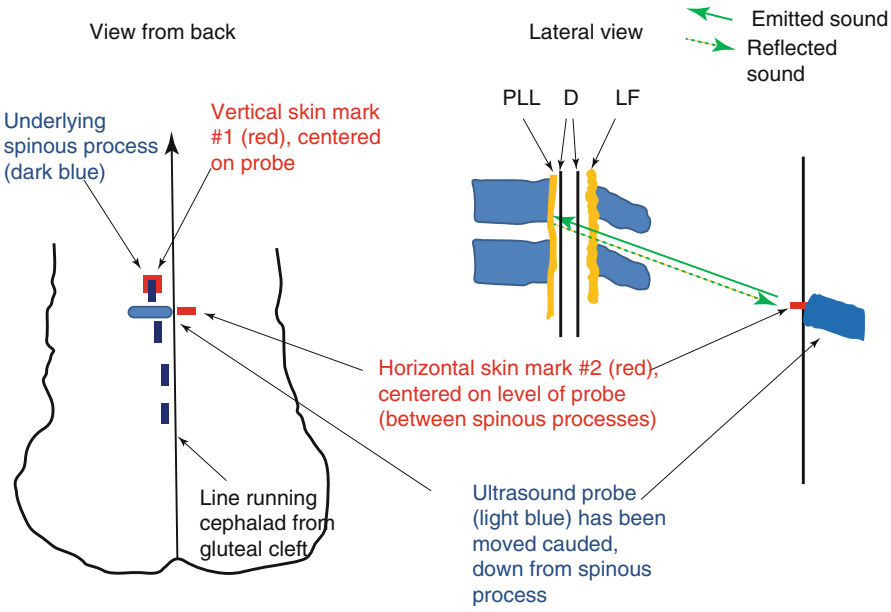

Fig. 36.A8 *To define the insertion point* in the cephalad-caudad axis, the ultrasound probe is moved slightly caudad until the spinous process "shadow" disappears and the underlying ligamentum flavum (*LF*) and posterior longitudinal ligament (*PLL*) are seen, as in Fig. 36.A9. Mark the middle of the *side* of the probe to define this level on the cephalad-caudad axis. *D* dura

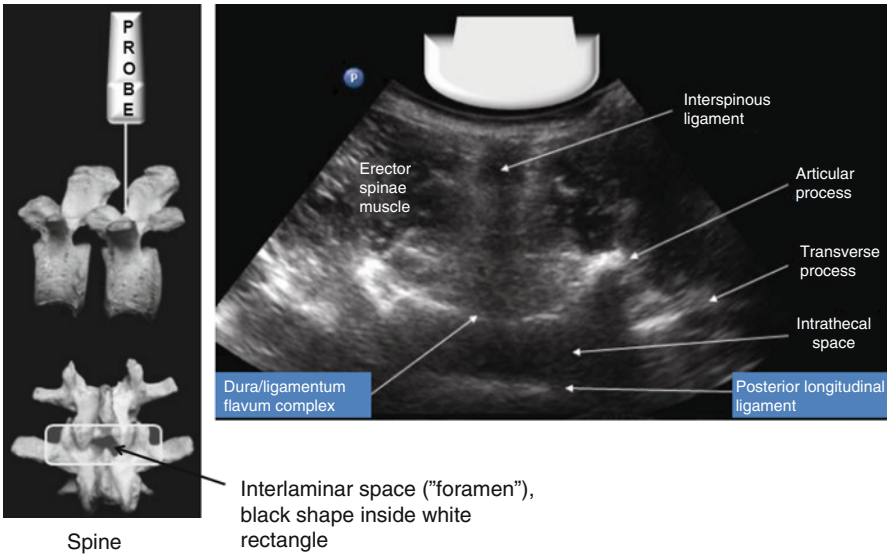

Fig. 36.A9 Transverse ultrasound view of lumbar spine *between* spinous processes (as shown in the left panel of Fig. 36.A9). In comparison to Fig. 36.A6, the ultrasound probe has been moved caudad a few centimeters until it overlies the supraspinous and interspinous ligaments (as shown in the upper portion of the left panel), at which point the image in the right panel is seen. The supraspinous and interspinous ligaments do not reflect much sound (compared to bone) and they are only seen as an attenuated, stippled mass. We can now appreciate structures *deep* to the interspinous ligaments (ligamentum flavum/dura complex, LF/D, and posterior longitudinal ligament, PLL) which are seen through the interlaminar foramen, as shown in the lower image in the left panel of Fig. 36.A6. *Note that in the ultrasound view, articular processes flow seamlessly into the facet joint and lamina and it is of no significance to identify exactly what structure is seen in this area (hence, I am calling this structure the A/FJ/L in* Figs. 36.A7 and 36.A10). The middle of the *side* of the US probe is marked on the skin to define the insertion point on the cephalad-caudad axis, as shown in Fig. 36.A8. The intersection of horizontal and vertical lines drawn through the two skin marks defines one insertion point, as shown in Fig. 36.A11 (Adapted from Chin et al. [1])

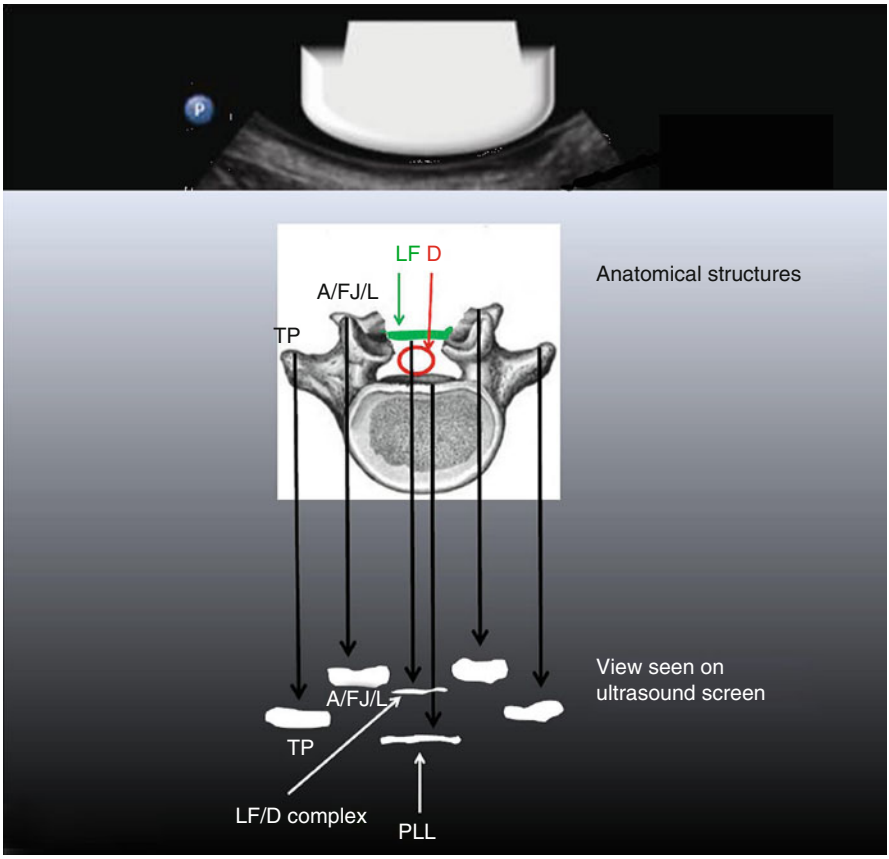

Fig. 36.A10 When the *transverse plane of the ultrasound probe passes between the spinous processes*, the underlying *ligamentum flavum/dura complex and the posterior longitudinal ligaments are "seen"* through the interlaminar foramen, that is, they reflect sound back to the probe and their reflections become visible on the screen, as shown in this schematic (Fig. 36.A10) and in Fig. 36.A9. Abbreviations: *TP* transverse process, *A* articular process, *FJ* facet joint, *L* lamina, *LF* ligamentum flavum, *D* dura, *PLL* posterior longitudinal ligament (Adapted from Chin et al. [1])

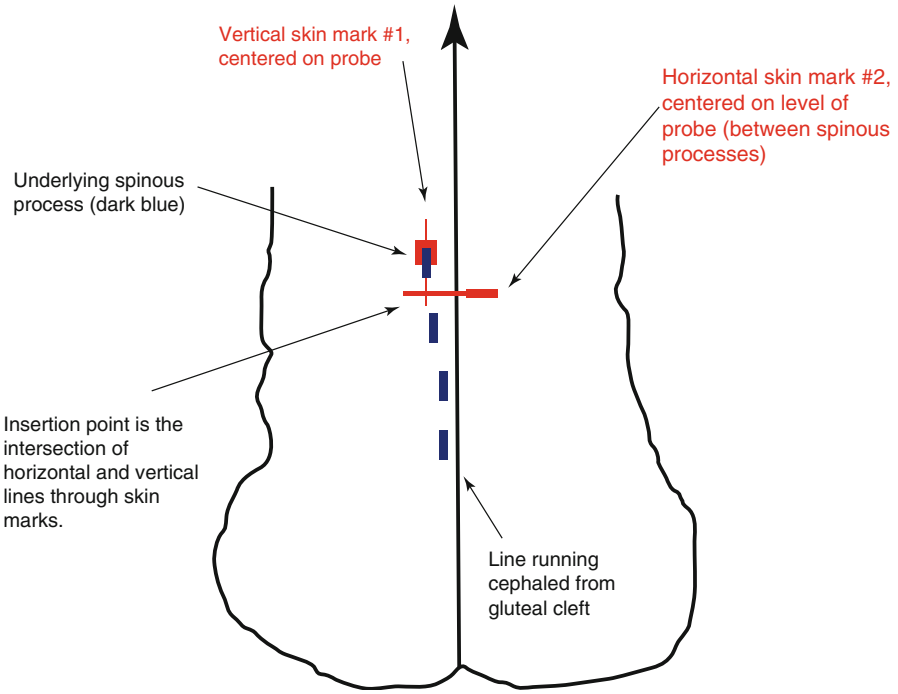

Fig. 36.A11 *The insertion point for the epidural needle is defined by the intersection of horizontal and vertical lines through the skin marks, as shown here*. See Fig. 36.A8 for the making of the horizontal mark and Fig. 36.A4 for the making of the vertical mark. The marks need to be made with meticulous care, checking the exact position more than once and being sure not to inadvertently pull the skin to one side with the probe, only to have the skin move relative to the underlying spinous process once the probe is removed from the skin. The probe should be vertically applied to the skin of the obese patient's back, and any position changes should be performed by lifting the probe off the skin and reapplying it vertically in a new position, rather than by sliding the probe over the potentially mobile skin. This technique avoids making a skin mark which does not faithfully overly the underlying structure whose position is being marked. Once the insertion point is defined, all that remains (in theory) is to define the insertion angle and the insertion depth, as shown in Figs. 36.A12 and 36.A13. The insertion angle is almost always 5–15° cephalad from a line perpendicular to the skin

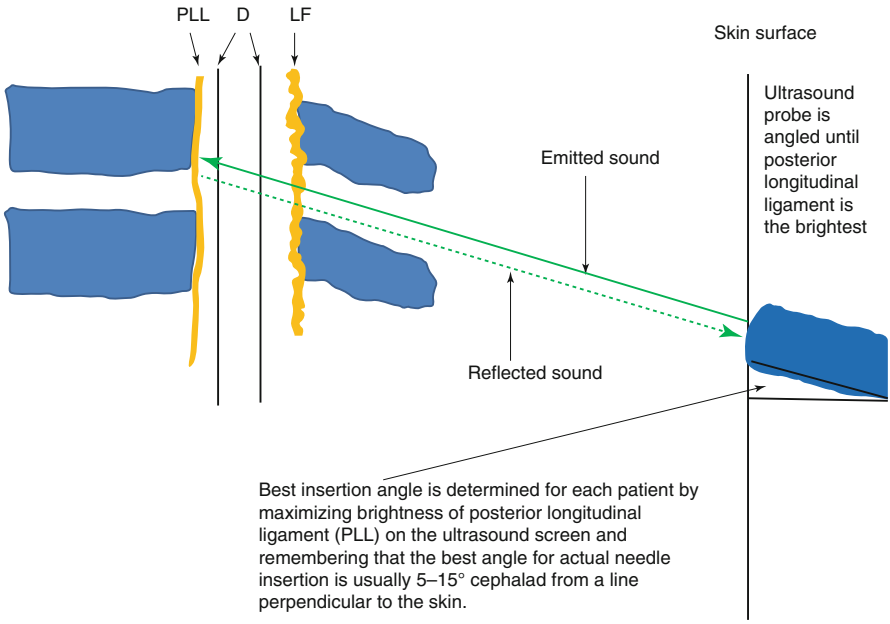

Fig. 36.A12 Because of the slight downward slant of the spinous processes in the lumbar region, the *angle of insertion* (for the midline approach) should always be 5–15° cephalad from a line perpendicular to the skin surface. In any given case, the best angle will vary, but it should be estimated for each patient by noting the ultrasound probe angle at which the reflection of the posterior longitudinal ligament (as shown in Figs. 36.A9 and 36.A10) is the brightest. *The insertion angle should not be changed if subsequently the insertion point is changed.* If you use the midline approach, it is usually futile to greatly change the insertion angle, particularly in obese patients. In more slender patients, the insertion angle may be changed slightly, but only slightly. What needs to be changed—if success is not achieved promptly—is the *insertion point, not the insertion angle*. Abbreviations: *PLL* posterior longitudinal ligament, *D* dura, *LF* ligamentum flavum

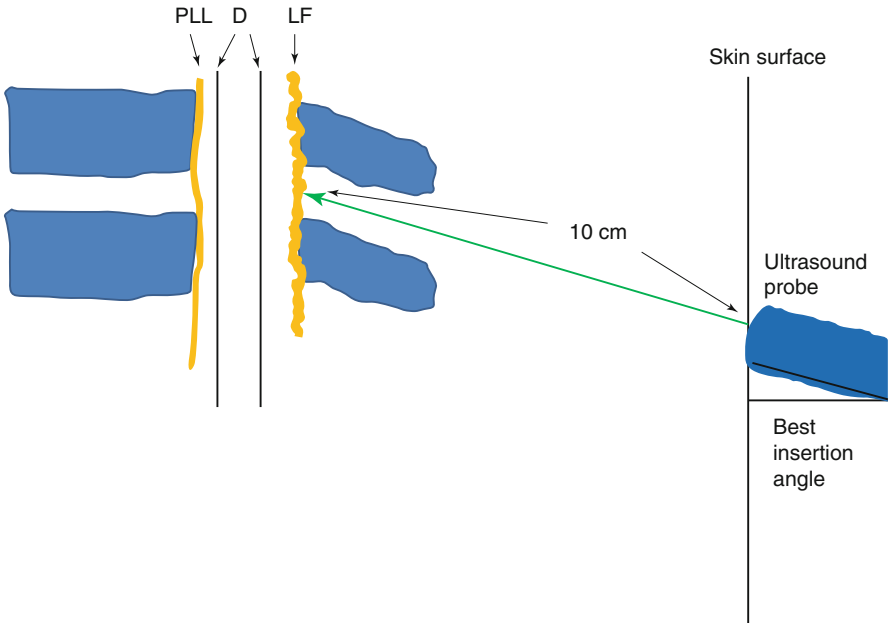

Fig. 36.A13 With ultrasound in this patient, depth to ligamentum flavum is estimated at 10 cm. Hence, we know three very important facts: (1) A longer needle is required than the standard 8-cm Tuohy needle. (2) Our initial needle pass can safely be made to 8.5 cm. We can insert the Tuohy needle with a high degree of confidence initially to within 1.5 cm of the ligamentum flavum (Jose Carvalho, MD), thereby saving time, discomfort. and frustration for both the patient and ourselves. This distance (1.5 cm less than the estimated depth) has been found empirically to provide a reasonable margin of safety to prevent accidental dural puncture. The initial pass can and should be made relatively quickly, *while all the time paying careful attention to the texture of the tissues encountered by the tip of the needle*. This tactile information can give you additional information about the course of your needle and the probability of success, as shown in Figs. 36.A14 and 36.A15. (3) We can expect loss of resistance at about 10 cm, but the actual depth of the ligamentum flavum is usually greater than estimated by about 0.5–1.0 cm because there is usually some degree of tissue compression by the ultrasound probe. Abbreviations: *PLL* posterior longitudinal ligament, *D* dura, *LF* ligamentum flavum

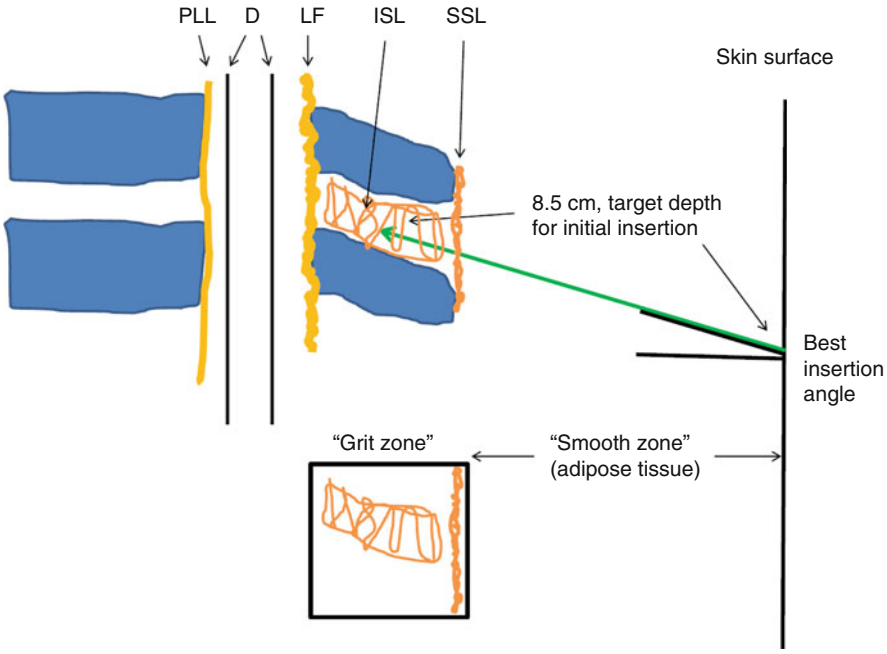

Fig. 36.A14 Knowing the depth to the ligamentum flavum (10 cm in this example) allows us to make an initial smooth pass with the needle to a depth of 8.5 cm, while paying careful attention to the texture of the tissues traversed by the needle tip. The tactile information you acquire with your initial needle pass is important. After the epidural needle pops through the resistance of the skin, the practitioner should feel a "smooth zone" of adipose tissue, which offers minimal resistance to passage of the needle. If, as shown in Fig. 36.A14, you then contact "gritty" or "crunchy" material in the latter part of the initial needle pass (after the "smooth zone"), you have probably contacted (first) supraspinous and then (second) interspinous ligaments. If the needle tip can substantially enter the "grit zone" (comprised of supraspinous and interspinous ligaments), the tip of the needle is probably already beyond the tips of the bony spinous processes and your chances of being able to contact the ligamentum flavum are pretty good. Abbreviations: *PLL* posterior longitudinal ligament, *D* dura, *LF* ligamentum flavum, *ISP* interspinous ligament, *SSL* supraspinous ligament

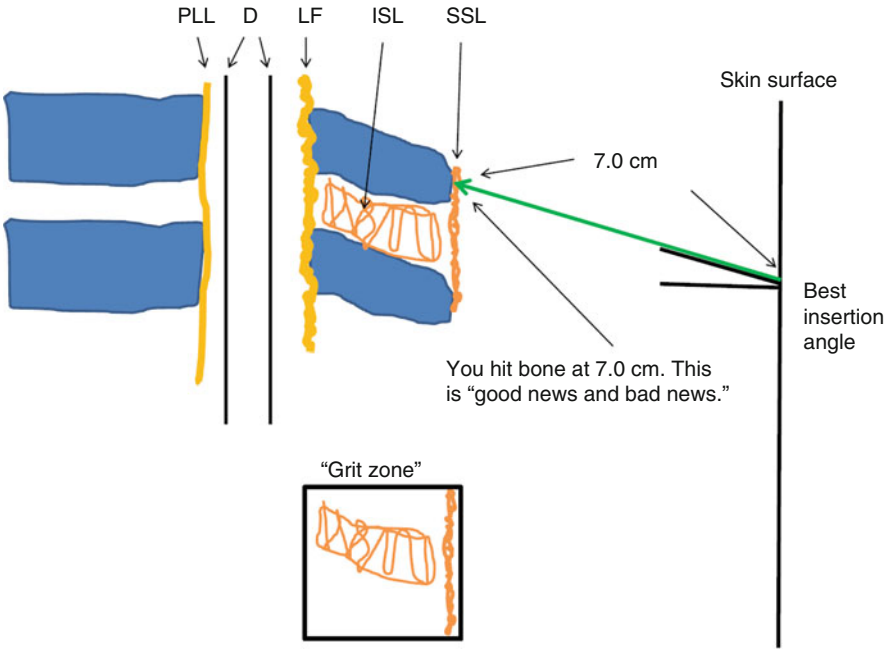

Fig. 36.A15 If, on the other hand, as shown in here, you contact bone before arriving at your initial target depth (8.5 cm in the example shown), it is a "good news/bad news" situation. The good news is that you know you are in the midline, since the only bone you could have contacted that shallow is spinous process. The bad news is that you are hitting spinous process and have to move your insertion point up or down by anywhere from several millimeters to a centimeter or more. *As usual, however, your angle of insertion should remain constant*. Abbreviations: *PLL* posterior longitudinal ligament, *D* dura, *LF* ligamentum flavum, *ISP* interspinous ligament; *SSL* supraspinous ligament

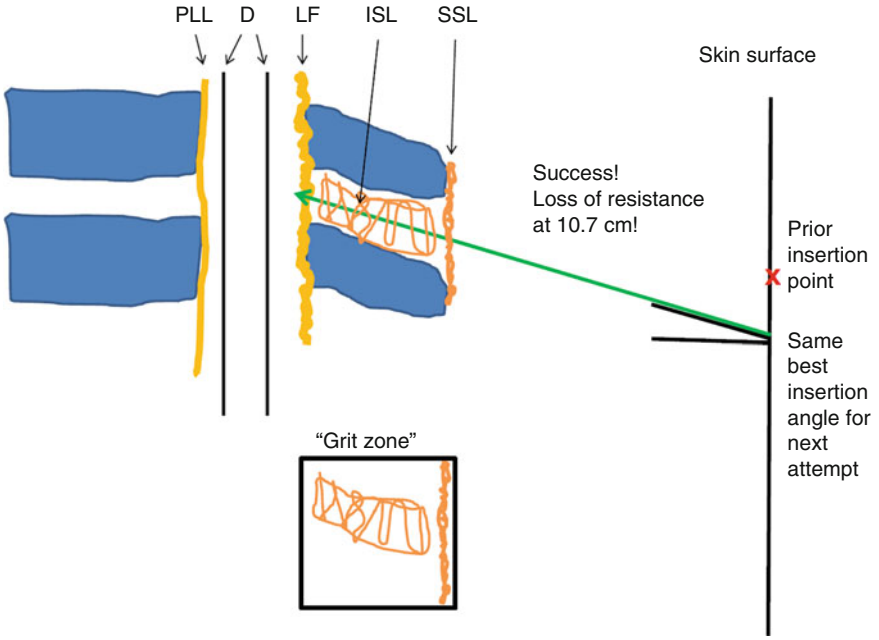

Fig. 36.A16 Success! The insertion point has been shifted caudad by 1.0 cm and the Tuohy needle slips between the spinous processes. Liberal infiltration of local anesthetic along a vertical "insertion line" (as opposed to a single point) overlying the spinous processes ensures that insertion points can be changed up or down easily and quickly, without having to take time to re-inject local anesthetic. In Fig. 36.A16 this "insertion line" had to include at least both the "prior insertion point" and the ultimately successful insertion point. *However, the insertion angle was not changed*. The actual depth to the ligamentum flavum is usually somewhat deeper (0.5–2 cm) than what is estimated in obese patients, because compression of adipose tissue by the ultrasound probe decreases the apparent distance from the probe surface to the underlying structures. Abbreviations: *PLL* posterior longitudinal ligament, *D* dura, *LF* ligamentum flavum, *ISP* interspinous ligament, *SSL* supraspinous ligament

References

1. Chin KJ, Perlas A, Chan V, Brown-Shreves D, Koshkin A, Vaishnav V. Ultrasound imaging facilitates spinal anesthesia in adults with difficult surface anatomic landmarks. Anesthesiology. 2011;115(1):94–101.
2. The Society for Ultrasound in Regional Anesthesia (USRA): http://www.usra.ca/vspine.php. Accessed 1 Feb 2013. Click on the "lumbar spine ultrasound" link and follow the instructions.

Chapter 37
Probable Amniotic Fluid Embolus

Sun Choe Daly

The patient is a 75 kg, 5′4″, 38-year-old gravida 1 para 0 at 40.5 weeks gestational age seen for placement of continuous labor epidural (CLE). The patient's vital signs were: blood pressure (BP) 99/53 mmHg, heart rate (HR) 74 beats per minute, temperature 98.8 °F, and respiratory rate (RR) 16 respirations per minute. The patient denied any significant past medical history, prior surgeries, or drug allergies. The patient was being induced with buccal misoprostol. Her cardiac and pulmonary examinations were normal. Her airway exam was Mallampati class III, 4 cm thyromental distance, full range of motion of her neck, intact dentition, and she was able to prognath. The CLE was placed without difficulty at the lumbar 3–4 vertebral space with loss of resistance to air and saline at 4.5 cm, and the catheter was secured at 9.5 cm at the skin. Her baseline hemoglobin was 12.9 g/dL, hematocrit was 39.9 %, and platelet count was 199,000/μL.

Six hours after CLE placement, the patient had spontaneous rupture of membranes, complained of nausea, was noted to have posturing with loss of consciousness for less than 1 min with a heart rate of 140 bpm. When the anesthesiologists arrived, the patient's eyes were open and she was spontaneously ventilating but not responsive to verbal questioning. Fetal HR was ~60 bpm and maternal HR was ~140 bpm. Amniotic fluid embolus (AFE) was considered to be a possible cause of this patient's distress (**L-1**). The patient was supplied with 100 % oxygen via Mapleson circuit and sealing facemask. The patient's BP was unable to be measured with the noninvasive blood pressure cuff. After the administration of phenylephrine 200 μg and ephedrine 20 mg, her BP was ~90/40 mmHg. The patient's CLE was immediately disconnected.

The patient was transferred to the operating room for emergent cesarean section. She was positioned with left uterine displacement on a ramp of blankets to optimize the sniff position, and rapid sequence induction with cricoid pressure was performed

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with etomidate 20 mg and succinylcholine 100 mg, and the obstetricians made incision. Increased jaw muscle tension (**L-2**) was noted with inability to open the mouth for intubation and rocuronium 100 mg was immediately administered. Intramuscular succinylcholine was prepared in case IV access had failed, but it was not needed. A non-triggering anesthetic was used for maintenance during the case to avoid the possible development of malignant hyperthermia (MH).

A radial arterial line and second peripheral IV line were placed in the right upper extremity. Examinations of arterial blood gases, coagulation studies, complete blood count, and fibrinogen levels were sent off immediately. Obstetric hemorrhage transfusion protocol was also initiated and product replacement began immediately for anticipated massive hemorrhage (**L-3**).

Supportive measures (**L-4**) were carried out for a presumptive diagnosis of AFE. During the case, the patient displayed classic symptoms and signs of AFE with hypoxemia, respiratory acidosis, shock, hemorrhage, and disseminated intravascular coagulation (DIC) (**L-5**). A diagnosis of DIC was made according to international criteria (**L-6**). DIC is commonly seen in AFE and can be seen in several other obstetric conditions (**L-7**).

The initial laboratory examinations revealed a metabolic acidosis which reached its nadir 3 h into the case with pH at 7.15 despite aggressive resuscitation with crystalloids, blood products, and phenylephrine to maintain perfusion. There was a large alveolar-arterial gradient with PaO_2 reaching a nadir of 81 mmHg on 100 % inhaled oxygen 3 h into the case. The hemorrhage was profound with her anemia reaching its nadir hemoglobin of 6.2 g/dL and hematocrit of 18 % 1 h into the case, which was treated with aggressive blood product administration of 13 units of packed red blood cells (PRBCs). The patient also developed thrombocytopenia with a nadir of 51,000/μL 3 h into the case with continued treatment with three platelet aphaeresis packs. The patient's coagulopathy (**L-8**) reached its maximum 1 h into case with prothrombin time (PT) of 22 s, International Normalized Ratio (INR) of 1.9, and activated partial thromboplastin (aPTT) time of 85 s and was treated throughout the case with 11 units of fresh frozen plasma (FFP). The patient was noted to have profound hypofibrinogenemia at the start of the case with less than 40 mg/dL initially which was treated with two units of cryoprecipitate. The patient also developed a respiratory acidosis with peak $PaCO_2$ of 52 mmHg 3 h into the case, which was treated with increasing minute ventilation from 6 to 8 L/min.

After 3 h of aggressive resuscitation guided by serial laboratory examinations, the surgical team closed the abdomen. But due to poor respiratory mechanics, the abdomen was reopened and greater than 1 L of blood was evacuated. The abdomen was then packed and the patient was transported to ICU. Estimated blood loss was 8 L and urine output was 2 L over 4 h.

The epidural catheter was left in situ at the end of the procedure due to continuing coagulopathy and thrombocytopenia. On postoperative day (POD) five, there was concern for infection due to an increasing white blood cell (WBC) count. The patient's coagulation parameters had normalized but she was still thrombocytopenic with a platelet count of 30,000/μL. Due to concern for epidural abscess formation in light of increasing WBC, the decision was made to discontinue the epidural

catheter. Although the patient was still intubated, she was off sedation, following commands and fully able to cooperate with a neurologic exam and able to communicate whether she felt back pain. These were criteria needed in order to monitor for epidural hematoma formation (**L-9**). The patient was transfused platelets and her platelet count rose to 86,000/μL and her catheter was discontinued. Neurologic examinations were conducted every 1 h for the next 48 h. The patient had no complications from her epidural catheter removal.

The patient had a complicated course over the next week with multiorgan failure. The patient required several treatment modalities including continuous renal replacement therapy for acute kidney injury, nitric oxide for adult respiratory distress syndrome (**L-10**), and multiple transfusions to treat her coagulopathy. The patient steadily improved and was extubated on POD 9 and without neurologic sequelae although she continues to require hemodialysis. The baby was transferred immediately to the neonatal intensive care unit, intubated, and cooled. The baby was extubated on POD 4 and was discharged home on POD 14.

Lessons Learned

L-1: What is the differential diagnosis of cardiopulmonary arrest in this patient?
See Table 37.1.

L-2: What is the significance of masseter spasm in this patient?
Masseter spasm is a rare reaction to intravenous administration of succinylcholine and has been linked to MH susceptibility. Although masseter spasm is rare, succinylcholine is known to cause an increase in jaw tension in adults and children [1]. It is possible that an ineffective dose of succinylcholine can also be a cause of difficulty with intubation, but this patient's jaw tension increased after succinylcholine administration. This has been documented in studies measuring jaw tension after administration of succinylcholine even when loss of twitch was noted at the adductor pollicis [2]. In a retrospective chart review study, about 1 % of children anesthetized with halothane and succinylcholine presented with masseter spasm. In 12 of 15 patients who had muscle biopsies in this study, 100 % of them were noted to be susceptible to MH with calcium uptake studies [3, 4]. Although this isolated masseter spasm is unlikely to be associated with MH, it may be prudent to avoid triggering agents once this phenomenon is noted and to increase vigilance (Fig. 37.1).

Succinylcholine's effect on jaw muscle tone is likely distributed normally in a population. When jaw rigidity prevents tracheal intubation after succinylcholine administration, a nondepolarizing muscle relaxant should be given, as was done in this case. As many as 50 % of this latter group may be susceptible to MH if associated with limb rigidity. Somewhere in the area of the declining curve is the boundary for the MH population.

Table 37.1 Differential diagnosis of cardiopulmonary arrest in this patient

	Positive Indicators	Negative Indicators
Amniotic fluid embolus	Spontaneous rupture of membranes, chemical induction (misoprostol), increased maternal age, increased gestational age, fetal distress, seizure, hypotension	Rare event, estimated at 1:8,000–83,000 births, a diagnosis of exclusion
Pulmonary embolus	Pregnancy is a hypercoagulable state, immobility due to CLE	No known source, the patient had intermittent venous compression stockings as a preventative measure
Venous air embolus	Patient had IV access	Difficult to give large enough air bolus through regular IV tubing on a pump to cause CP arrest
Anaphylaxis	Patient was being administered misoprostol	No known drug allergies, no cutaneous reaction, no bronchospasm
Aortocaval compression	Gravid uterus	Patient's hemodynamics were not improved with left uterine displacement
Anesthetic complication such as high spinal	Patient had an epidural; the catheter could have migrated into the subarachnoid space	The patient had a stable working epidural for 6 h prior to the event
Hemorrhage from uterine rupture	Possible to have uterine rupture with chemical induction without pain due to neuraxial block	No fetal parts were palpated on abdomen
Local anesthetic toxicity	The patient had continuous and patient-controlled dosing of her epidural with a mixture of local anesthetic and narcotic solution	The patient was receiving a very low concentration of bupivacaine and had a negative response to test dose on placement of epidural for IV placement

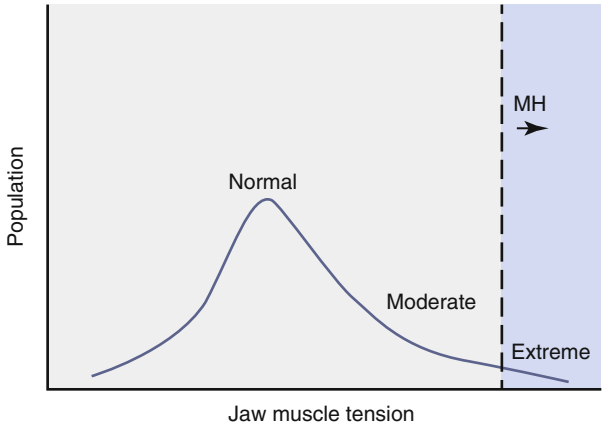

Fig. 37.1 Jaw muscle tension distribution after succinylcholine administration (Adapted from Zhou et al. [4])

L-3: How do you tailor blood product replacement in massive obstetric hemorrhage [5]?

1. Control bleeding using surgical and/or placement of intravascular balloons or emboli under radiologic guidance.
2. Aim to restore circulating blood volume using colloid and blood products.
3. Control exacerbating factors for abnormal coagulation such as hypothermia, hypocalcemia, and acidosis.
4. Administer blood products:

   (a) Red cells

   Use O negative red cells first.
   If no record of red cell antibodies, ABO, and Rh compatibility, crossmatched blood should be available within 30 min (maximum of 45 min).
   Replace red cells as required to maintain circulating blood volume.
   Use fluid warmer to avoid hypothermia.

   (b) FFP

   Transfuse one unit of plasma to every one unit of red cell.
   Aim for PT and aPTT less than 1.5 times normal (normal for PT <15 s and APTT <35 s).

   (c) Platelet transfusion

   One to two adult doses after 1.5–2.0 blood volume replacement (equivalent to eight to ten bags of red cells).
   Aim for a platelet count over 50,000/μL.

   (d) Fibrinogen

   Cryoprecipitate (dose = two donation pools).
   Fibrinogen concentrates (4 g).
   Aim for fibrinogen level over 100 mg/dL.

L-4: What is the treatment for AFE [6]?
General supportive measures in the treatment of amniotic fluid embolism.

1. Treat hypoxia with 100 % oxygen.
2. Treat hypotension by fluid resuscitation with isotonic solutions and vasopressors.
3. Treat diminished left ventricular contractility with fluids and inotropic therapy.
4. Treat DIC and coagulopathy with FFP, cryoprecipitate, fibrinogen, and factor replacement.
5. Treat hemorrhage with RBC transfusions and thrombocytopenia with platelets.

L-5: What are the classic symptoms and signs of AFE?
See Fig. 37.2.

L-6: How do you diagnose DIC?
International society on thrombosis and hemostasis diagnostic scoring system for overt DIC [5].

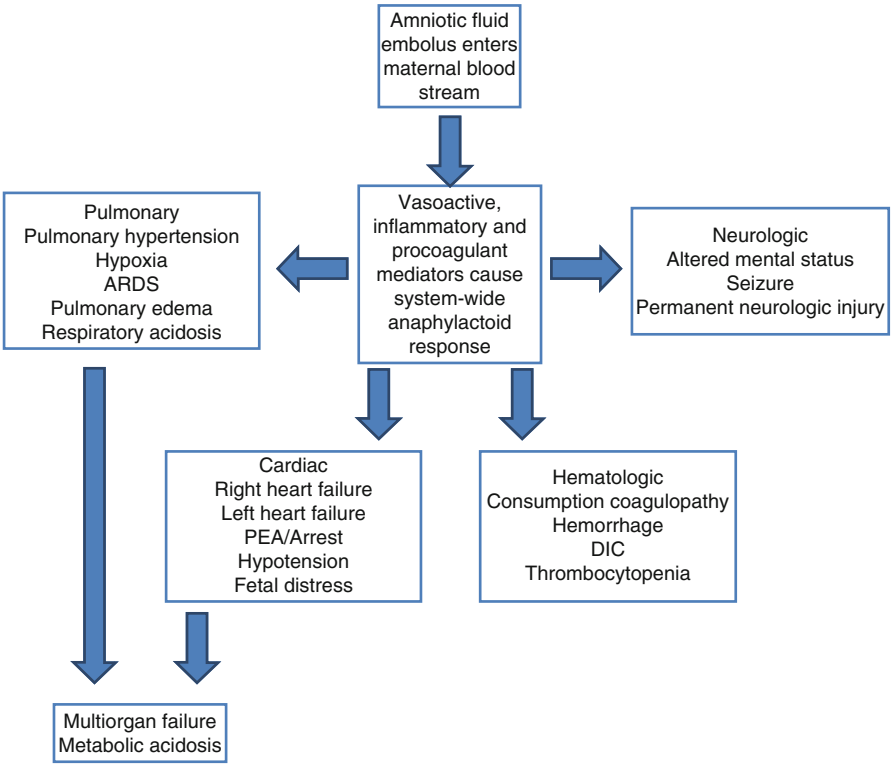

Fig. 37.2 Classic signs and symptoms of AFE. *AFE* amniotic fluid embolus, *DIC* disseminated intravascular coagulation, *PEA* pulseless electrical activity, *ARDS* adult respiratory distress syndrome

1. Risk assessment: Does the patient have an underlying disorder known to be associated with overt DIC?
   - If yes: proceed
   - If no: do not use this algorithm
2. Order global coagulation tests (prothrombin time, platelet count, fibrinogen, fibrin-related markers).
3. Score the test results.
   - Platelet count (>100,000/μL = 0, <100,000/μL = 1, <50,000/μL = 2)
   - Elevated fibrin marker (e.g., D-dimer, fibrin degradation products) (no increase = 0, moderate increase = 2, strong increase = 3)
   - Prolonged prothrombin time (<3 s = 0, >3 s but <6 s = 1, >6 s = 2)
   - Fibrinogen level (>100 mg/dL = 0, <100 mg/dL = 1)
4. Calculate score.
   - >5 compatible with overt DIC: repeat score daily
   - <5 suggestive for non-overt DIC: repeat next 1–2 days

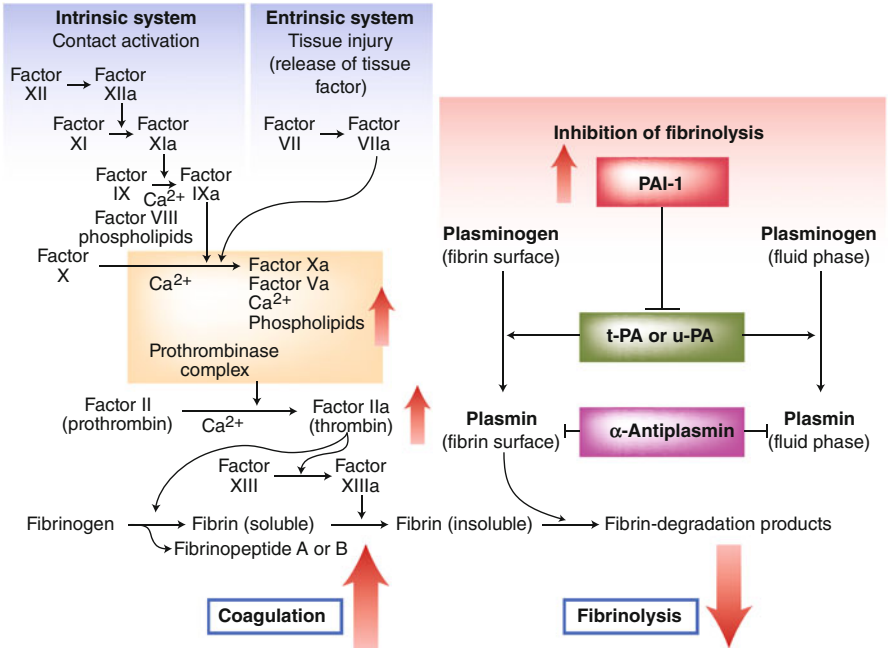

Fig. 37.3 Coagulation cascade. Contact activation: collagen, *PAI-1* plasminogen activator inhibitor, *t-PA* tissue plasminogen activator, *u-PA* urokinase plasminogen activator

L-7: What are some commonly described causes of DIC in obstetrics?

Amniotic fluid embolism
Intrauterine fetal demise
Hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome
Preeclampsia/eclampsia
Placental abruption and placenta previa
Septic abortion and intrauterine infection
Postpartum hemorrhage
Acute fatty liver of pregnancy

L-8: What are the intrinsic and extrinsic coagulation cascades and where does DIC intervene?

The placenta is at the center of the cause of DIC in AFE due to its procoagulant bias. There is a massive generation of thrombin in DIC, which can aggregate on the phospholipids released during cell lysis. The placenta expresses PAI-1, which decreases fibrinolysis (see Fig. 37.3).

L-8a: What part of the coagulation cascade does PT measure?

PT measures the extrinsic pathway mainly. It measures activity of factors I, II, V, VII, and X, excluding factor VIII, and normal values are 10–15 s. Coumadin inactivates the vitamin K-dependent factors II, VII, IX, and X, and protein C and S and prolongs PT and INR.

Table 37.2 Adjunct therapies used in amniotic fluid embolism [6]

Strategy	Therapeutic goal
Intra-aortic counterpulsation	Increase coronary perfusion pressure, decrease afterload
Extracorporeal membrane oxygenation	Oxygenation
Cardiopulmonary bypass	Oxygenation and pump function
Plasma exchange transfusions	For use when DIC is refractory to replacement treatment. To remove inflammatory cytokines, cell debris, etc.
Uterine artery embolization	Stop hemorrhage from uterus
Continuous hemofiltration	Treatment of acute kidney injury
Cell salvage combined with blood filtration	Transfuse red blood cells during massive hemorrhage
Serum protease inhibitors	Treatment for DIC
Inhaled nitric oxide	Improve oxygenation, reduce pulmonary hypertension
Inhaled prostacyclin	Improve oxygenation, reduce pulmonary hypertension
High-dose corticosteroids	Reduce further production of inflammatory mediators

L-8b: What part of the coagulation cascade does the aPTT measure?
PTT measures the intrinsic part of the pathway, measuring factors I, II, V, VIII, IX, X, XI, and XII, excluding factor VII, and normal values are 25–35 s. Heparin activates antithrombin III and inactivates factor Xa, prolonging PTT.

L-9: What is a safe platelet count to discontinue or place an epidural catheter with regard to epidural hematoma formation?
Epidural hematomas are rare and catastrophic complications of neuraxial anesthesia. The risk of hematoma formation has been estimated to be 1:150,000–220,000. Although many obstetric patients present with platelet counts of less than 100,000/μL, there are few recommendations for placement or removal of epidural catheters in light of thrombocytopenia. If one is concerned for possible epidural hematoma formation, serial full neurologic examinations must be performed, and any signs or symptoms of spinal cord ischemia should be investigated without delay. A recent review article has suggested the following guidelines for neuraxial anesthesia or lumbar puncture in a patient with thrombocytopenia [7].

It is safe to place/remove epidural or spinal anesthetic with platelet count greater than or equal to 80,000/μL and lumbar puncture with platelet count >40,000/μL with the following conditions:

1. The platelet count is stable.
2. There is no other acquired or congenital coagulopathy.
3. The platelet function is normal and the patient is not on an antiplatelet drug.
4. The patient is not on an anticoagulant. If the patient is on a low molecular weight heparin, 12 h should have elapsed from the last dose of a prophylactic dose or 24 h after a therapeutic dose before an epidural or spinal anesthetic is placed.

L-10: What are additional strategies can you use in treatment of AFE?
See Table 37.2. [6]

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Chapter 38
Emergent Cesarean Section

Christopher Edwards

The patient is a 26-year-old gravida 1 para 0 at 39 weeks 4 days of gestation brought emergently to the operating room for cesarean section secondary to fetal bradycardia (**L-1**). Past medical history was only significant for a previous cesarean section in Mexico. The fetus was known to have intrauterine growth retardation and oligohydramnios.

In the operating room, the patient was monitored with standard American Society of Anesthesiologists (ASA) monitors, a rapid sequence induction was performed, and endotracheal intubation (**L-2**) followed uneventfully. Once the airway was secured, surgery proceeded.

Lessons Learned

L-1: **What is thought to be the reason for early**, **variable**, **and late decelerations**? In order to understand the changes in fetal heart rate (FHR), we first need to understand how the FHR is monitored. There are various techniques to measure the FHR; both external and internal measurements are utilized in clinical practice. An example of an external monitor is fetal Doppler ultrasonography. The Doppler method records both the FHR and the uterine contraction strength and time interval. An example of an internal monitor is a fetal scalp electrode. Both methods allow for continuous FHR monitoring. The normal FHR is between 120 and 160 beats per minute and has both short-term and long-term variability. Short-term variability is considered to be present when heart rate differs between two to three adjacent beats.

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Table 38.1 Patterns of fetal heart rate tracing [2]

Patterns of fetal heart rate tracing	Timing	Causes	Dangerous
Early decelerations	Occurs at the time of each uterine contraction	*Fetal head compression* leading to increased fetal vagal response. Decrease in heart rate is usually less than 20 bpm from baseline	NO
Variable decelerations	Vary in relation to the uterine contraction	*Umbilical cord occlusion*, either partial or complete	Depends on the timing, length, and severity of fetal bradycardia
Late decelerations	Occurs 10–30 s after the uterine contraction	Secondary to fetal hypoxemia, usually related to *uteroplacental insufficiency*	Yes—ominous sign of fetal distress

Long-term variability is a rolling sine wave that occurs three to six times per minute with variation of at least 6 bpm [4]

Table 38.1 and Fig. 38.1 describe and show, respectively, various types of classic fetal heart rate tracings.

L-2: **What is the obstetric difficult airway algorithm**?

Airway management should always begin with a thorough evaluation of the patient's airway including history and physical exam. The airway exam should include measurement of thyromental distance and a Mallampati classification. If a difficult airway is anticipated and time permits other options including regional anesthesia, local anesthesia or an awake intubation should be considered. In the event that general anesthesia with endotracheal intubation is the only option, the obstetric (OB) anesthesia difficult airway algorithm (Fig. 38.2) differs from airway management in the non-obstetric patient because of the impact of FHR on management. Once the patient is anesthetized and endotracheal intubation is unsuccessful but mask ventilation is adequate, consideration of fetal wellbeing becomes important. If no fetal distress is present, awaking the patient seems prudent. If fetal distress is present, there are only three options: (a) awaken the patient (higher risk of fetal demise), (b) surgical airway, or (c) maintain mask ventilation (higher risk of aspiration) and proceed with surgery. Both awaking the patient and a surgical airway are definitive therapies. If mask ventilation is chosen, then one must consider whether ventilation is spontaneous vs. controlled, whether to maintain cricoid pressure, whether to suction the stomach, and what is the best way to position the patient's head (turned to the side and head down). Regardless of the decision-making process, one should always have a well-developed backup plan with all available equipment prepared and ready to use in the event an emergency develops.

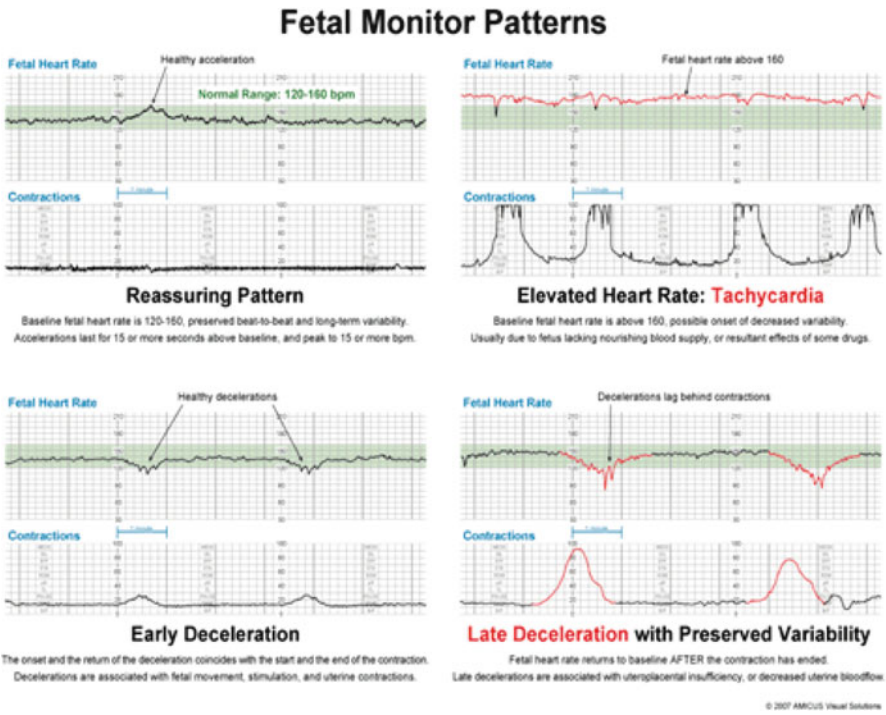

Fig. 38.1 Various fetal heart tracings [1]

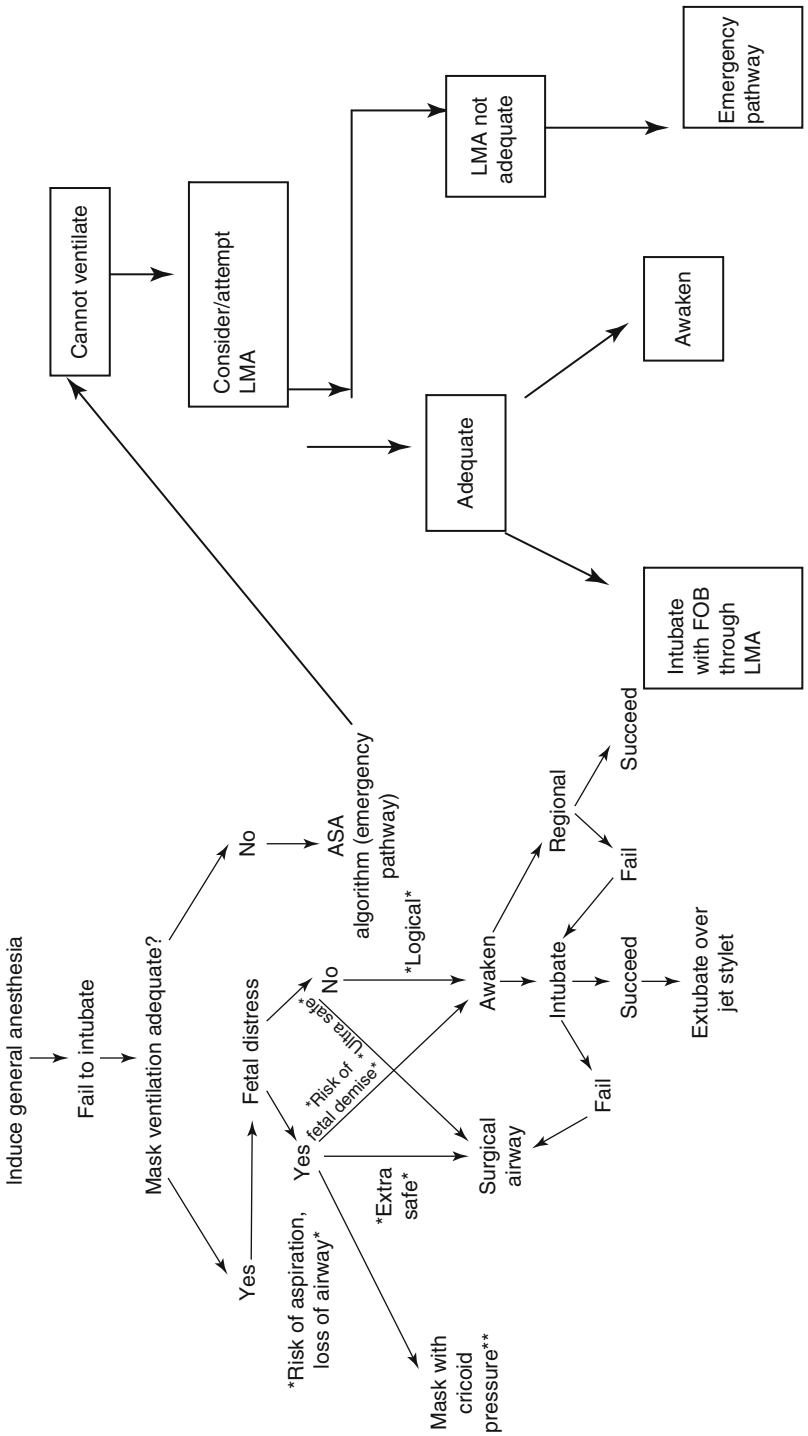

Fig. 38.2 Obstetric difficult airway algorithm. ** indicated that while this is not a an optimal technique it is an optional rescue technique [3]

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Chapter 39
Pregnancy Plus Atrial Septal Defect vs Eisenmenger Syndrome

Christopher Edwards

The patient is a 20-year-old female gravida 1 para 0 at 22 weeks and 3 days (**L-1**) scheduled for a dilation and extraction (D&E) of fetus with multiple congenital anomalies. During a routine physical exam, she was noted to have a holosystolic murmur, and upon further review, she noted a recent decrease in exercise tolerance, specifically new onset shortness of breath. A transthoracic echocardiogram showed a 4-cm atrial septal defect (ASD), depressed right ventricular function, severe right atrial enlargement with central venous pressure (CVP) greater than 50, and pulmonary hypertension (**L-2**); the diagnosis of Eisenmenger syndrome was considered.

Prior to the scheduled D&E, laminaria (**L-3**) were placed to aid in cervical dilation. A lumbar epidural (**L-4**), two large-bore IVs, and an arterial line were placed for this procedure, and with each subsequent laminaria change, the epidural was bolused to aid with pain control (**L-5**). After adequate cervical dilation, she was taken to the operating room for the scheduled D&E under general anesthesia. After completion of the D&E, sometime later she had an ASD repair.

Lessons Learned

L-1: **What are the cardiovascular changes of pregnancy**?

A. *Cardiovascular Parameters*—Table 39.1 shows the important pregnancy-induced cardiovascular changes.

B. *Blood Pressure*—As a result of the 50 % increase in cardiac output and the 20 % decrease in SVR, there is only a mild reduction in the systemic blood pressure. The SVR, cardiac output, and blood pressure changes are thought to occur

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Table 39.1 Pregnancy-induced cardiovascular changes

Parameters	Change
Cardiac output	+50 %
Stroke volume	+25 %
Heart rate	+25 %
LVED volume	Increased
LVES volume	No change
Ejection fraction	Increased
Pulmonary capillary wedge pressure	No change
CVP	No change
Systemic vascular resistance (SVR)	−20 %

Table 39.2 Factors affecting SVR/PVR

↑SVR/↓PVR—increases L→R shunt ↓SVR/↑PVR—decreases L→R shunt	
Decrease SVR	*Decrease PVR*
1. Induction agents	1. Alkalosis
2. Volatile agents	2. Hypocarbia
3. Epidural	3. High Fi02
Increase SVR	4. Volatile agents
1. Pain	*Increase PVR*
2. Sympathetic nervous system activation	1. Acidosis
3. Stress	2. Hypercarbia
	3. Hypoxia
	4. N2O
	5. PEEP

secondary to circulating prostacyclins, progesterone, and estrogens in addition to the low-resistance placenta (Table 39.2).

C. *Aortocaval Compression*—Aortocaval compression is highly dependent on the position and size of the uterus relative to the inferior vena cava (IVC) and aorta. Aortocaval compression is maximal in the supine position, significantly less in the lateral position, and least in the prone position. Aortocaval compression typically starts at 16–18 weeks gestation and continues until delivery.

L-2: **What is Eisenmenger syndrome**?

Eisenmenger syndrome is a reversal of blood flow through a communication between the right and left sides of the heart. In the normal heart, pressures on the left side of the heart are greater than pressures on the right side of the heart (Fig. 39.1 [left side]). With an unrepaired ASD/ventricular septal defect (VSD), left-to-right shunting of blood over time causes the right side of the heart to develop right ventricular hypertrophy (Fig. 39.1 [right side]). It is thought that the chronic increase in right-sided blood flow destroys the pulmonary endothelium resulting in an increase in pulmonary vascular resistance (PVR). When the PVR becomes greater than the SVR, reversal of flow in a now right-to-left manner occurs and Eisenmenger syndrome develops. Clinically this is seen as cyanosis and hypoxemia due to admixture of deoxygenated blood. Other late signs and symptoms include erythrocytosis, hemoptysis, palpitations, edema, and syncope. We hypothesize that during

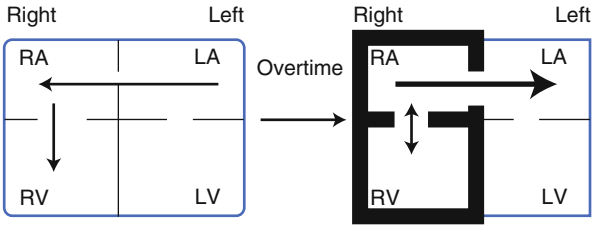

Fig. 39.1 *Left side* of figure shows normal direction of blood flow through an ASD. *Right side* of figure shows reversal of blood flow in Eisenmenger syndrome. *Arrows* indicate direction of blood flow

pregnancy, the decrease in SVR caused an increase in right-to-left shunt which leads to hypoxemia and shortness of breath.

L-3: **What are laminaria**?
Laminaria is a tool used to dilate the cervix during induction of labor or help to dilate the cervix before an abortion. It is derived from the laminaria japonica and laminaria angustata, both kelp species. The laminaria are formed into a rodlike structure and inserted into the cervix. It works by drawing water from the cervical tissue to the laminaria, which can swell three to five times its original diameter, leading to cervical dilation. This concept is similar to Merocel packing in the nose or Tampax in the vagina.

L-4: **What are the anesthetic concerns for a patient with an ASD undergoing a D&E**? **An epidural vs general**?
Having an ASD can affect normal physiology in many ways, thus changing how anesthetic drugs interact with the body. To understand how this works, a better understanding of what physiologic changes take place is needed. With an ASD, there is a left-to-right shunt. This type of shunt slows uptake of anesthetic vapors from the lungs and may dilute intravenous drugs slowing induction. This is usually of little clinical consequence. The main hemodynamic concerns are those associated with changes in the SVR and PVR as these parameters will ultimately affect the quantity and direction of blood flow across the shunt. Below is a list of common anesthetic entities that affect SVR and PVR.

Understanding the effect of changes in hemodynamics is important when delivering an anesthetic to a patient with an ASD. Multiples types of anesthetics are acceptable to use as long as the expected changes are carefully thought about and dealt with (Fig. 39.2).

A. *With a* right-to-left shunt, *it would be reasonable to choose an anesthetic that ↑SVR/↓PVR. The drugs that are best in this regard* (*maintaining SVR*) *are etomidate* (*mean ± SD*; *-10 ± 14 %*) *and ketamine* (*mean ± SD*; *0 ± 33 %*).
B. *With a* left-to-right shunt, *it would be reasonable to choose an anesthetic that ↓SVR/↑PVR. The drugs that are best in this regard are propofol* (*mean ± SD*; *-15–25 %*), *volatile agents*, *and neuraxial techniques* (*note that most IV induction agents have little effect on PVR*.)

Finally, with an ASD or any communicating lesion, extreme care to avoid any paradoxical air embolism needs to be considered. Meticulous care to avoid even small amounts of air in IV tubing to prevent catastrophic outcomes (especially in a right-to-left shunt) including coronary and cerebral air emboli is critical.

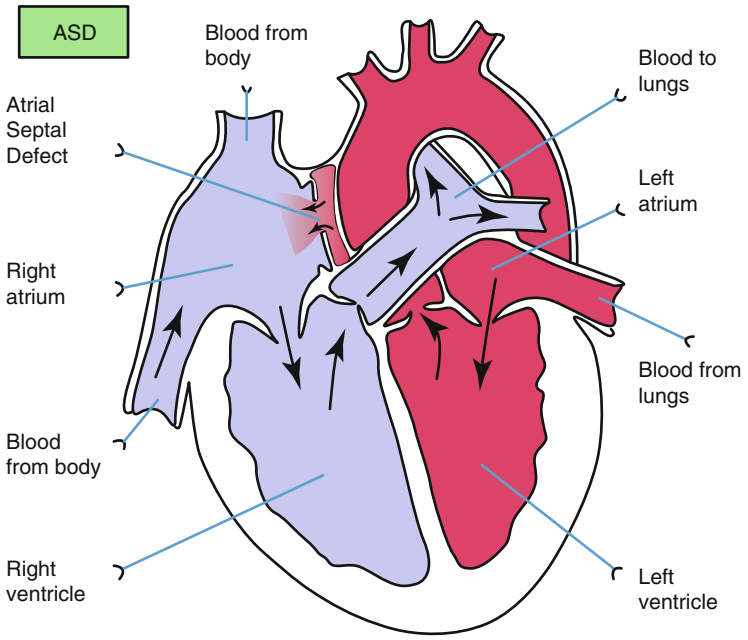

Fig. 39.2 Depiction of an ASD

L-5: **Why is pain control important for a patient with an ASD**?
Pain has both psychological and physical effects on the human body. In a patient with an ASD and in considerable pain, such as a laboring female, there are important physiologic consequences to controlling pain. Direction and quantity of blood flow through the ASD depends on pressures on either side of the lesion, namely, SVR and PVR. If SVR > PVR, then blood flow will be left to right. If PVR > SVR, then blood flow will reverse and a right-to-left shunt develops. SVR tends to increase in patients with pain; as this occurs, the shunt fraction from left to right increases. The increase in left-to-right shunt can lead to pulmonary congestion, pulmonary edema, and shortness of breath. Using adequate pain control techniques may help to maintain normal physiologic parameters in patients with an ASD.

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Chapter 40
Uterine Abruption

Erica K. Stary

A 20-year-old gravida 2 para 1 female (59 kg, 63 in.) at 33 weeks' gestation, with past medical history of asthma and with current preeclampsia, presented for induction of labor. After placement of a successful continuous lumbar epidural (CLE), she underwent spontaneous vaginal delivery at 0100. The anesthesia team was called at 0300 to give the CLE a bolus for attempted manual extraction of retained placenta (**L-2C**). The patient had continued bleeding since delivery that was thought to be from uterine atony or retained placenta (**L-1**, **L-2**). However, her blood pressure at this time was 70/30 mmHg (heart rate [HR] 100–110 beats per minute [bpm]), so an epidural bolus was not given.

An arterial blood gas (ABG) was sent at 0300, and an additional 18 g PIV was placed. ABG results were as follows: pH 7.41, $PaCO_2$ 27 mmHg, PaO_2 96 mmHg, base excess −7.1, bicarb 17 meq/L, and hematocrit 26. Intravenous fluids were given until packed red blood cells (PRBCs) arrived. The patient appeared clinically better, and the anesthesia team was dismissed, with the plan for the obstetricians to follow serial hematocrit levels and to transfuse more PRBCs as needed.

At 0530, the anesthesia team was called for ongoing hemorrhage. Mean arterial pressures were 60–70 mmHg, her heart rate was 130–140 bpm, and the patient was exhibiting decreased mental status and worsened pallor (**L-3**). Blood loss was estimated at 2,000–2,500 cc by the obstetricians at that time. An arterial line was placed by anesthesia. From 0300 to 0600, 5u PRBCs and 1 g of calcium chloride were given, with an ABG at 0640 of pH 7.33, $PaCO_2$ 24 mmHg, PaO_2 218 mmHg, base excess −11.8, bicarb 12 meq/L, and hematocrit 36.

By 0700, the patient clinically appeared coagulopathic (**L-4**, Fig. 40.1 and Table 40.1). Labs were sent, with international normalized ratio (INR) 1.4, fibrinogen 115 mg/dL, and partial thromboplastin time (PTT) 73 s. Cryoprecipitate, fresh frozen plasma (FFP), and more PRBCs were ordered, and the patient was moved

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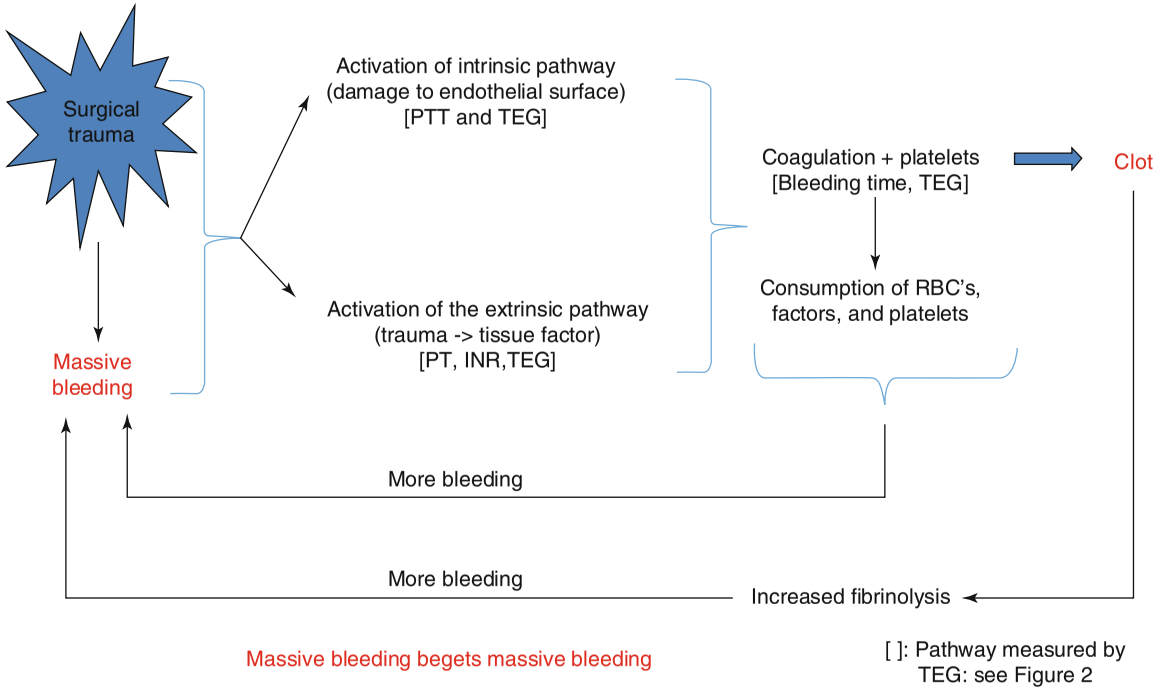
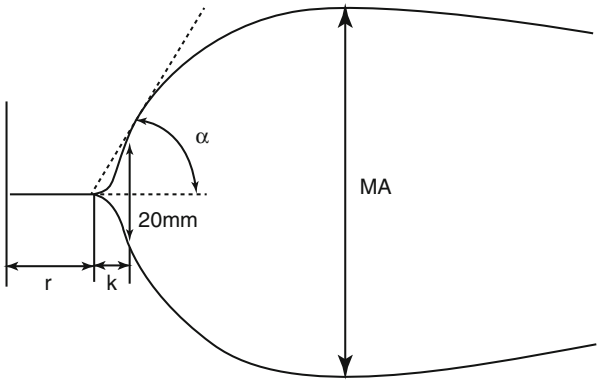


Fig. 40.1 Relationships between surgical trauma, massive bleeding, coagulation, and clot

Table 40.1 Thromboelastography (TEG): typical pattern, variables measured, normal values, and therapy



TEG®

Variable	"Reaction time (**r**)" is the time from initiation of the TEG®, until the amplitude of the trace reaches 2 mm	"Alpha angle (α)" is the angle formed between the middle of the TEG® and a line drawn between the *r*- and *k*-times "Clot formation time" (**k**) is the time from the r-time until the TEG amplitude reaches 20 mm	"Maximum amplitude (**MA**)" is the greatest amplitude reached on the trace
Measures	"r" corresponds to initial fibrin formation, generated via the intrinsic pathway. It is functionally related to plasma clotting and inhibitor factor activity	The α angle and k-time are directly related, as they are both a function of rate of fibrin polymerization "k" is a measure of the clot formation kinetics	MA corresponds to maximum clot strength and elasticity. It is directly related to the quality of fibrin and platelet interaction. Platelets have the greatest influence on the final clot strength
Normal values	*r*: 21–30 mm	40–60° *k*: 3–12 mm	50–60 mm
Therapy for abnormal values	Coagulation factors (fresh frozen plasma (FFP)[a])	Fibrinogen (cryoprecipitate[b])	Platelets

[a]FFP is the plasma from a whole unit of blood; all coagulation factors are in normal concentrations

[b]Cryoprecipitate includes fibrinogen, factor VIII, factor XIII, and von Willebrand factor

urgently to the main operating room for emergency exploratory laparotomy and hysterectomy. The patient was intubated prior to transport, as she was increasingly unstable. Once in the operating room, a central-line cordis catheter was placed. FFP, platelets, and cryoprecipitate were given, and an ABG at 0715 was 7.27/$PaCO_2$ 28 mmHg, PaO_2 116 mmHg, base excess −13.2, bicarbonate 12 meq/L, and hematocrit 24.

Upon opening the abdomen, a 3–4 cm posterior uterine rupture (**L-1C**) (**L-5**) extending through the broad ligament, cervix, and posterior vagina was found. There was also a massive hematoma dissecting from the broad ligament to the vesicouterine peritoneum. Bleeding was eventually controlled surgically, and a supracervical hysterectomy was done. Further evaluation revealed a posterior wall bladder defect and anterior wall vaginal defect, which were repaired (**L-1C**) (**L-4**). Total estimated blood loss for the surgery was about 3 L, with total blood loss about 5–5.5 Ld since delivery. The patient was adequately resuscitated and transferred to the intensive care unit for further monitoring. Labs upon arriving to the intensive care unit were as follows: pH 7.4, $PaCO_2$ 38 mmHg, PaO_2 266 mmHg, base excess −1.6, hematocrit 30, INR 1.2, PTT 32 s, and fibrinogen 301 mg/dL.

Lessons Learned

L-1: Differential diagnosis of antepartum bleeding

A. Placenta Previa
   Placenta previa is defined as abnormal implantation of the placenta, which is immediately adjacent to, partially covering, or completely covering the cervical os. Typically this presents with painless vaginal bleeding in the second or third trimester but can present earlier. Lack of abdominal pain and abnormal uterine tone distinguishes it from, but does not rule out, placental abruption. Incidence is about 3.6 per 1,000 pregnancies [1].
B. Placental Abruption
   Placental abruption is defined as complete or partial separation of the placenta from the vaginal wall (decidua basalis) before the delivery of the fetus. Bleeding may be overt or may be hidden behind the placenta. Symptoms include vaginal bleeding with uterine tenderness and increased uterine activity. Often these women present in preterm labor, with nonreassuring fetal heart rates. Placental abruption is the most common cause of disseminated intravascular coagulation (DIC) in pregnant patients and occurs in 10 % of cases. Incidence is estimated at 0.4–1 % of pregnancies, with a 12 % perinatal mortality rate [1].
C. Uterine Rupture
   Uterine rupture can be devastating but thankfully is a rare occurrence. **L-3** lists risk factors. Maternal presentation with uterine rupture is variable, although fetal heart rate decelerations are most often present. Abdominal pain existing between contractions and with point tenderness was historically thought to be involved in all cases; however, this may not be the case in at least 10 % of cases. There has also been dispute as to whether a timely diagnosis of uterine rupture is possible with epidural analgesia in place. However, several case studies point out that if analgesia as opposed to anesthesia is properly maintained, then pathologic pain will be felt even though analgesia is provided by a continuous lumbar epidural run at normal rate. Referred shoulder-tip pain may serve as an early

indicator of uterine rupture in women with a functioning epidural. The mechanism of referred shoulder-tip pain is likely subdiaphragmatic irritation of the phrenic nerve when perforation of a visceral organ extends into the peritoneum, which can be felt even under neuraxial anesthesia [2]. Other symptoms of uterine rupture include vaginal bleeding, maternal hypotension, cessation of labor, maternal tachycardia, and fetal compromise. A true diagnosis is confirmed with exploration of the uterus during laparotomy. Uterine rupture increases the risk of neonatal mortality by 60-fold [1].

D. Vasa Previa

Vasa previa occurs when fetal vessels cross over or run in close proximity to the inner cervical os. These vessels are unsupported by the umbilical cord or placental tissue and are at risk of rupture when the supporting membranes rupture. If these fetal vessels rupture, the bleeding is from the fetoplacental circulation, and fetal exsanguination will rapidly occur, leading to fetal death. The clinical picture usually includes rupture of membranes and painless bleeding, followed by fetal compromise. Vasa previa has been estimated to occur in 1 in 2,500 deliveries, and it has one of the highest fetal mortality rates (50–75 %) [1].

L-2: **Differential diagnosis of postpartum bleeding**

A. Uterine Atony

Uterine atony is the most common cause of postpartum hemorrhage, accounting for up to 80 % of primary postpartum hemorrhage and occurring in 4–6 % of pregnancies [1]. It is a failure of the uterus to contract adequately at parturition, in response to endogenous uterotonic agents. Conditions associated with uterine atony include multiple gestations, macrosomia, polyhydramnios, high parity, prolonged labor, tocolytic agents, augmented labor, chorioamnionitis, or a high concentration of volatile anesthetic if a general anesthetic is used. Symptoms include postpartum bleeding and a soft uterus on exam. See Table 40.2 for drug therapies for uterine atony.

B. Genital Trauma

Most commonly, genital trauma includes lacerations and hematomas to the perineum, vagina, and cervix. However, this category also includes genital tract lacerations and retroperitoneal hematomas. Retroperitoneal hematomas, since they can be concealed and undiagnosed for prolonged periods of time, are the most dangerous of genital trauma. Injury typically occurs during cesarean sections but also can occur following a rupture of a low-transverse uterine scar during labor. Symptoms vary depending on the size of and the rate of growth of the hematoma. However, any time a postpartum patient exhibits hypotension, tachycardia, and/or an unexplained drop in hematocrit, retroperitoneal hematoma should be in the differential.

C. Retained Placenta

Retained placenta is defined as failure to deliver all or any part of the placenta after vaginal delivery. Manual extraction by the obstetricians is usually the treatment of choice for this condition, which occurs in as many as 3.3 % of deliveries [1].

Table 40.2 Drug therapies for uterine atony

Agent	Dose and route	Contraindications	Side effects	Notes
Oxytocin	20–60 unit per L of IV infusion	None	Decreased SVR and hypotension with bolus	Short duration of effect
Methergine	0.2 mg IM	Hypertension Preeclampsia CAD	Thromboembolic? Severe N/V	Long duration of effect
Hemabate	0.25 mg IM or IU	Reactive airways Pulmonary HTN Hypoxemic Patients	Bronchoconstriction Shivering Diarrhea Temp. elevation	May repeat every 15 min, up to 2 mg
Misoprostol	0.8–1 mg PR	None	Shivering Temp. elevation Diarrhea N/V	Off label use

IV intravenous, *SVR* systemic vascular resistance, *IM* intramuscular, *IU* intrauterine, *PR* per rectum, *CAD* coronary artery disease, *HTN* hypertension, *N/V* nausea and vomiting, *Temp* temperature

D. Placenta Accreta
Placenta accreta is defined broadly as abnormally adherent placenta. There are three types: *placenta accreta vera* is defined as adhering to the myometrium, *placenta increta* includes invasion of the myometrium, and *placenta percreta* includes invasion of the uterine serosa or other pelvic structures. The presence of a placenta previa increases the risk of placenta accreta, as does a history of having prior cesarean sections. In a prospective study by Silver et al. when placenta previa occurred in patients with no prior, one prior, two prior, or four prior cesarean sections, there was an incidence of placenta accreta in 3, 11, 40, and 67 %, respectively [3]. For this reason, there should be a high index of suspicion of placenta accreta for any pregnant patient who has had a prior cesarean section and who has placenta previa. Imaging such as ultrasound or magnetic resonance imaging will help to elucidate the extent of the placental invasion and will serve to guide preparations for the delivery.

E. Uterine Inversion
Uterine inversion is the turning inside out of the uterus. It is exceedingly rare and rarely causes severe morbidity or mortality in areas of the world with adequate healthcare. Although usually obvious because symptoms include hemorrhage and a mass in the vagina, sometimes it is not as readily apparent and diagnosis is delayed. The inverted uterus is associated with uterine atony. Once the uterus has been manually replaced, uterotonics are administered, as a firm uterus is desired.

L-3: **Clinical signs of hemorrhagic shock in obstetrics patients**
Table 40.3 lists the physiologic responses to hemorrhage in antepartum and postpartum patients. Blood volume increases progressively from 6 to 8 weeks gestation and

Table 40.3 Physiologic responses to hemorrhage in obstetrics patients by percent blood loss*

Acute blood loss, mL	Blood loss, %	Physiologic response
900	15	Asymptomatic
1,200–1,500	20–25	Tachycardia and tachypnea
		Narrowed pulse pressure
		Orthostatic hypotension
		Delayed hypothenar refilling
1,800–2,100	30–35	Worsening tachycardia and tachypnea
		Hypotension
		Cool extremities
>2,400	40	Shock
		Oliguria/anuria

Adapted from Francois and Foley [4]
*Total blood volume of 6,000 mL

reaches maximum at about 32–34 weeks, after which time not much change is observed. The increase in plasma volume (40–50 %) is relatively greater than that of red cell mass (20–30 %) resulting in hemodilution and a decrease in hemoglobin concentration. The increased blood volume serves two purposes. First, it facilitates maternal and fetal exchanges of respiratory gases, nutrients, and metabolites. Secondly, it reduces the impact of maternal blood loss at delivery.

L-4. Coagulopathy in a bleeding patient
In cases of massive hemorrhage, patients are not only losing blood through primary traumatic injury, but also the bleeding itself leads to a coagulopathic state, which promotes more bleeding. Figure 40.1 portrays this concept. Depicted within the diagram are the interrelationships between traumatic bleeding, consumption of factors, and the laboratory coagulation tests that become abnormal. Table 40.1 depicts thromboelastrography (TEG), which is a test of whole-blood clotting. Although it was not used for the present case, in cases of massive hemorrhage, it gives valuable information about what parts of the coagulation cascade or platelets are abnormal, to help clinicians with treatment decisions.

Simple Guidelines for Evaluating a TEG

When evaluating a TEG, treatment will be dictated by the current clinical situation, along with clinical judgment. However, there are a few simple rules to help with judgment:

1. R parameter is a period of time from initiation of the test to the initial fibrin formation. If the R parameter is elongated, this means there is delayed clot formation, so suspect:
   (a) Heparin effect
   (b) Factor deficiency

Table 40.4 Conditions associated with uterine rupture

Previous uterine surgery
Trauma
Indirect:
Blunt (e.g., seat belt injury)
Excessive manual fundal pressure
Extension of cervical laceration
Direct:
Penetrating wound
Intrauterine manipulation
Forceps application and rotation
Postpartum curettage
Manual placental extraction
Version and extraction
External version
Inappropriate use of oxytocin
Grand multiparity
Uterine anomaly
Placental percreta
Tumors (trophoblastic disease, cervical carcinoma)
Fetal problems (macrosomia, malposition, anomaly)

2. Alpha angle is the angle between the line in the middle of TEG and the line tangential to the developing "body" of the thromboelastogram. The alpha angle represents the acceleration (kinetics) of fibrin buildup and cross-linking. If this angle is small, then the patient is low on fibrinogen and/or coagulation factors, and cryoprecipitate and/or FFP should be administered.
3. Maximal amplitude is a measure of platelet aggregation and quantity. Small maximum amplitude often suggests thrombocytopenia, and platelets should be considered.

L-5: **Risk factors for uterine rupture**

Uterine rupture is rare in the primigravid patient or those without prior cesarean section or a scarred uterus. Table 40.4 lists the conditions that are associated with uterine rupture.

In this case, a young woman with no prior cesarean sections, it is unknown why she was at risk for uterine abruption. It is possible the laceration started at her cervix during the vaginal delivery and extended upwards. It is also possible that the attempted manual extraction of the placenta led to the abruption, but this is less likely, as she was already exhibiting signs and symptoms of hemorrhage and subsequent hypovolemia before these attempts. Of course, there is always the chance that this patient had a uterine anomaly, which is essentially a diagnosis of exclusion, but may be the only viable option. There are no case reports of uterine rupture being caused by the pressure exerted on the uterus during placing a continuous lumbar epidural in the sitting position.

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Part IV
Pediatric-Related Cases

Chapter 41
Neonatal Resuscitation Following Spontaneous Vaginal Delivery

Michael Bronson

The mother of the patient is a 25-year-old gravida 2 para 1 female at 35 weeks' gestation who presented in labor. The pregnancy had been complicated by gestational diabetes with poorly controlled blood glucose. A continuous lumbar epidural catheter was placed uneventfully for labor analgesia. The neonate was delivered vaginally with a very tight, nonreducible nuchal cord (**L-1**). The cord was then divided and the baby was delivered with some difficulty. After delivery, the neonate was without respirations or a pulse. The neonatal intensive care unit (NICU) team was present for delivery and initiated resuscitation (**L-2**).

The patient was intubated by the NICU team, and two doses of epinephrine were administered with ongoing cardiopulmonary resuscitation (CPR) (**L-3**). Ventilation was noted to be ineffective, and further inspection showed that the endotracheal tube (ETT) was at 15 cm at the gumline (**L-4**). The ETT was pulled back while CPR continued. A cord gas that was sent at the time of delivery showed pH 6.88, CO_2 54 mmHg, O_2 43 mmHg (maternal $FiO_2 = 0.40$), base deficit 16.9 mEq/L, lactic acid 138 mg/dL (**L-5**, **L-6**). With the ETT now at 10 cm at the gumline, ventilation improved and heart rate (HR) increased >100 beats per minute (bpm) following an additional dose of epinephrine. Now relatively stable and with acceptable vital signs, the neonate was transported to the NICU for further post-resuscitation care.

Lessons Learned

L-1. **What is a nuchal cord**? **What are the consequences of a nuchal cord**?
A nuchal cord occurs when the umbilical cord forms a loop around the fetal neck. This is a very common finding at delivery with a reported incidence between 14.7

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Table 41.1 Risk factors for requiring neonatal resuscitation

Maternal factors	Pregnancy factors
1. Diabetes	1. Premature rupture of membranes
2. Hypertension	2. Preterm labor
3. Infection	3. Multiple gestation
4. Substance abuse	4. Chorioamnionitis
5. No prenatal care	5. Placenta previa
6. Age <16 or >35 years	6. Placenta accreta
7. Significant cardiac, pulmonary, renal, or neurologic disease	7. Polyhydramnios/oligohydramnios
	8. Meconium-stained amniotic fluid
	9. Non-reassuring fetal heart rate patterns

and 33.7 % [1–3]. The exact significance of a nuchal cord has been highly disputed. Nuchal cords have been associated with fetal demise, impaired fetal growth, and neurodevelopmental abnormalities in smaller studies(involving 46–180 patients) and case reports [4–6]; however, large retrospective studies (involving >24,000 patients) have not shown an association with nuchal cord and adverse perinatal outcomes [1, 2].

L-2: **What is the algorithm for neonatal resuscitation**?

It is estimated that about 10 % of newborns will need some support at birth, while roughly 1 % will require extensive resuscitation to survive [7, 8]. Certain clues may assist the provider in predicting who may require resuscitation at birth so that appropriate equipment and personnel can be present at the time of delivery (Table 41.1) [7–9]. Much like adult cardiopulmonary resuscitation, the foundation of the neonatal resuscitation algorithm is the ABCs: airway, breathing, and circulation (Fig. 41.1).

Initial steps in the resuscitation of the neonate (Table 41.2) involve determining (1) the gestational age, (2) whether meconium is present in the amniotic fluid, (3) the presence of breathing or crying, and (4) muscle tone [7]. It should be noted that if meconium is present in the amniotic fluid and the newborn is not vigorous (weak respiratory efforts, poor muscle tone, HR <100 bpm), then it is recommended that the trachea be suctioned for meconium prior to proceeding with other steps. If the initial assessment of the neonate is non-reassuring, then preparation for cardiopulmonary resuscitation should be started, all the while administering tactile stimulation (flicking soles of feet and rubbing their back in the hopes that spontaneous ventilation will begin) and providing warmth.

Then an evaluation of the neonate's respirations and HR will indicate whether supportive care (continuous positive airway pressure [CPAP] and/or oxygen saturation (SpO_2) monitoring) or positive-pressure ventilation (PPV) with SpO_2 monitoring is needed (Fig. 41.1):

- Labored breathing and HR >100 bpm → supportive care
- Apneic/gasping or HR <100 bpm → PPV with SpO_2 monitoring

More recent guidelines for neonatal resuscitation recommend that room air be used for the initial resuscitative measures of the term infant as opposed to 100 % oxygen [8]. To support this recommendation, numerous studies were reviewed that

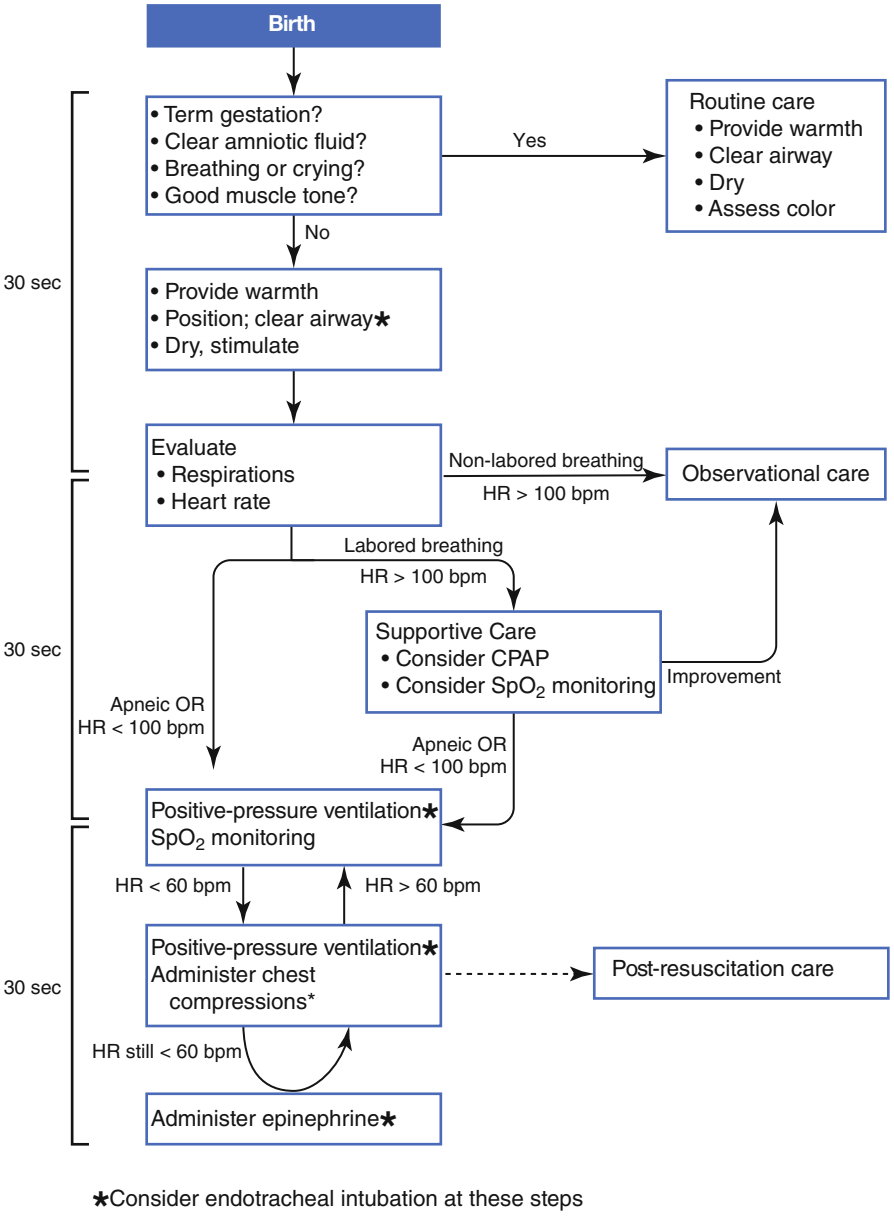

Fig. 41.1 Algorithm for neonatal resuscitation (Adapted from Perlman et al. [8])

showed no benefit to using 100 % oxygen over room air, and in fact, current evidence suggests that exposing the neonate to 100 % oxygen can have untoward effects such as increased mortality, neurologic disability, carcinogenicity, and

Table 41.2 Initial assessment of the neonate

1. Gestational age?	Preterm babies have underdeveloped lungs, muscle weakness, and lesser ability to maintain body temperature. The more the patient is preterm, the greater the likelihood resuscitation is necessary
2. Clear amniotic fluid?	If meconium is present in the amniotic fluid, then meconium may also be in the trachea. If the baby is vigorous, then there is not enough meconium to completely block the airway and vice versa. Therefore, if the baby is not vigorous, the trachea should be suctioned for meconium immediately
3. Breathing/crying?	Breathing or crying with observable chest movement = good respirations. Gasping is a sign of significant respiratory depression and/or severe neurologic dysfunction and should lead to immediate intervention
4. Muscle tone?	A limp newborn will be unable to adequately expand their lungs and sustain respiration so will most likely require positive pressure and/or intubation for ventilatory support

Table 41.3 Targeted preductal SpO_2 after birth

Minutes	SpO_2, %
1	60–65
2	65–70
3	70–75
4	75–80
5	80–85
10	85–95

oxidative stresses leading to organ dysfunction [10–15]. Current guidelines support the use of preductal (from right wrist or hand) SpO_2 monitoring and titrating oxygen supplementation to targeted preductal SpO_2 values during the resuscitative process (Table 41.3). In preterm infants requiring resuscitation, it is recommended to begin with 30 % oxygen and also titrate support to preductal SpO_2 targeted values [8].

During PPV, there should be adequate chest movement and bilateral breath sounds, and if not, steps should be taken to ensure adequate ventilation (i.e., tighter mask seal, clearing airway, proper positioning of head, adjusting level of positive pressure). Ventilation of the lungs has been deemed the single most important and effective step for the successful resuscitation of the neonate, which may require endotracheal intubation at any time in order to be successful [7]. (*See* black asterisks in Fig. 41.1 and last paragraph of **L-2**.)

If, after 30 s of successful PPV, the HR remains less than 60 bpm, chest compressions should be initiated. Compressions with the provider's thumbs is the preferred technique, and compressions should occur at a rate of 90 per minute while breathing is continued at 30 breaths per minute. After 30 s of adequate cardiopulmonary resuscitation, the HR should be reassessed, and if still less than 60 bpm, epinephrine (concentration of 1:10,000, dose of 0.1–0.3 mL/kg IV or 0.3–1.0 mL/kg down ETT) should be administered while CPR is continued.

At any point during resuscitation, it may become necessary to place an endotracheal tube to secure a patent airway. Indications for endotracheal intubation include

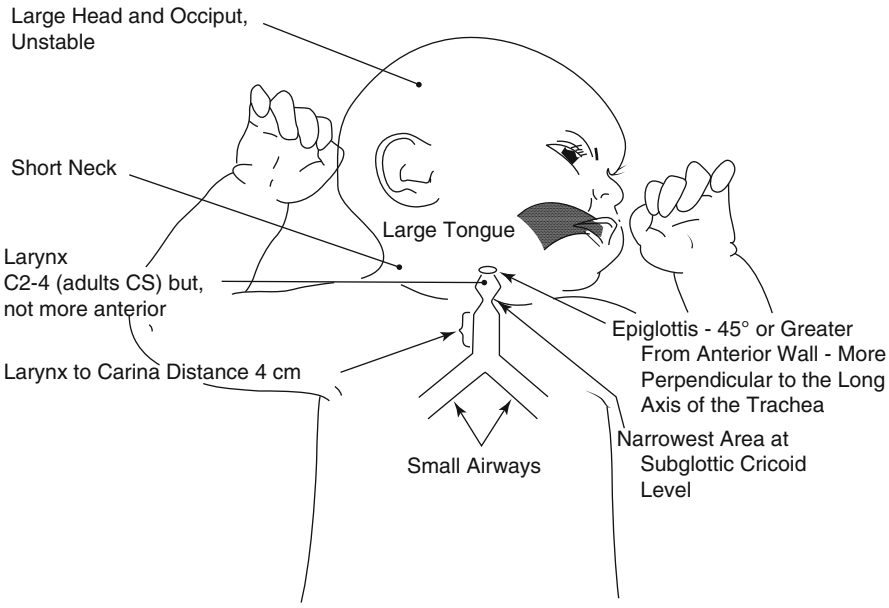

Fig. 41.2 Infant airway anatomy (Adapted from Benumof [18])

the following: (1) to suction meconium from trachea, (2) improve ventilation, (3) facilitate coordination with chest compressions, and (4) administer epinephrine [7, 9]. Important anatomic differences from the adult airway, tips for successful intubation of the neonate, appropriate endotracheal tube size, and depth of insertion are discussed below (**L-3** and **L-4**).

L-3. What are the differences between the neonatal and adult airway?
Fundamental anatomic differences between the neonatal and adult airway have been well described (Fig. 41.2) [16–18]; the neonate has a/an:

1. Large head/prominent occiput
2. Short neck
3. Small mandible
4. Relatively large tongue
5. Epiglottis that is larger, stiffer, and often omega-shaped
6. Larynx that is located at C2–C4 (C5 in adults)
7. Cricoid ring which is the narrowest part of the airway (glottis in adults)
8. Smaller tracheal diameter (approximately 4 mm vs. 16 mm in adults)
9. Shorter distance from larynx to trachea (approximately 4 cm vs. 12 cm in adults)

These features make airway management potentially more challenging in the neonate. The larger head, prominent occiput, and short neck make placement of the infant into a proper "sniff position" more difficult. This may be overcome with the aid of a roll under the shoulders and a doughnut-shaped pillow to stabilize the head. The presence of a relatively large tongue compared to the oropharynx leads to

Table 41.4 Key concepts in neonatal airway management

Differences from adults	Clinical conceptual implications	Practical airway management
1. Large head/prominent occiput	More difficult to place in "sniff position"	Shoulder roll
	More difficult to stabilize head	Doughnut-shaped pillow
2. Short neck	More difficult to place in sniff position	Shoulder roll
3. Small mandible	More difficult to displace tongue during laryngoscopy	Proper tongue sweep right → left
4. Relatively large tongue	Difficult mask ventilation	Insertion of oral airway
	More difficult to displace tongue during laryngoscopy	Proper tongue sweep right → left
5. Relatively larger epiglottis	More difficult to visualize larynx	Miller blade is preferred to lift epiglottis
6. Cephalad larynx	More difficult to visualize larynx	Miller blade is preferred
7. Narrowest part of airway at cricoid ring	ETTs passing through larynx can meet resistance in the subglottis	ETT size carefully selected (Table 41.5)
		Uncuffed ETTs traditionally preferred
8. Smaller tracheal diameter	ETTs passing through larynx can meet resistance in the trachea	ETT size carefully selected (Table 41.5)
		Uncuffed ETTs traditionally preferred
9. Shorter tracheal distance	More potential for inadvertent mainstem intubation and extubation	Meticulous awareness to depth of insertion (Table 41.6)
		Avoid extreme head flexion/extension

upper airway obstruction during mask ventilation, so an oral airway may need to be inserted to facilitate mask ventilation.

The relatively small mandible and larger tongue will make laryngoscopy more difficult, as displacement of the tongue into the submandibular space will be limited. In addition, a larger epiglottis and more cephalad larynx could make visualization of the glottis more challenging, and for this reason, a Miller blade is the preferred laryngoscope when intubating a neonate [18].

Since the tracheal diameter is reduced compared to the adult and because the narrowest part of the airway is subglottic (at the cricoid ring) which is not in the field of view of the intubating practitioner, the size of endotracheal tube must be carefully selected (see below, **L-4**), and uncuffed endotracheal tubes are usually preferred in the neonate [16]. Additionally, the shorter trachea makes the chance of mainstem intubation and unintended extubation much greater compared to the adult, so the depth of insertion should be carefully monitored (see below, **L-4**). Following intubation, bilateral breath sounds should be confirmed before and after

Table 41.5 Endotracheal tube selection for neonates

Weight (g)	ID (mm)
<1,000	2.5
1,000–2,000	3.0
2,000–3,000	3.5
>3,000	3.5–4.0

Table 41.6 Endotracheal tube depth of insertion for neonates

Weight (kg)	Depth (cm)
1	7
2	8
3	9
4	10

securing the endotracheal tube at the gumline of the neonate to ensure proper positioning. Extreme flexion/extension of the head should be avoided to prevent inadvertent mainstem intubation or extubation.

Table 41.4 summarizes key clinical conceptual implications of the neonatal airway compared to adults and the significance of these differences to practical airway management.

L-4: **What size ETT should be used in neonates and what is the appropriate depth of insertion**?

Table 41.5 summarizes the appropriate internal diameter (ID) of endotracheal tubes for neonates based on weight [7, 18].

To be complete, general guidelines for endotracheal tube size selection in pediatric patients is as follows:

- 6 months–2 years: 4.0–4.5 mm
- >2 years: (age + 16)/4 = ID (mm)

Table 41.6 summarizes appropriate depth of insertion for endotracheal tubes in neonates based on weight [7, 18]. Note that weights of 1, 2, 3, and 4 kg correspond to depths of 7, 8, 9, and 10 cm, respectively.

Equations for depth of ETT insertion in pediatric patients are as follows:

- 1–2 years: age + 10 = depth (cm)
- 2 years: (age/2) + 12 = depth (cm)

L-5. **How are cord gases interpreted**?

An analysis of cord gases at the time of delivery gives valuable information regarding the condition of the neonate at birth. Conditions such as fetal hypoxia can lead to acid–base abnormalities that can be detected via cord gas sampling. Fetal hypoxia results from (1) impaired maternal oxygenation, (2) inadequate placental perfusion or (3) if delivery of oxygenated blood from the placenta is impeded [19]. The inability of the fetus to undergo oxidative metabolism in the peripartum period leads to a state of anaerobic metabolism. This results in the production of organic acids, importantly lactic acid and keto acids, creating a metabolic acidosis in the fetus [19, 20].

The analysis of umbilical artery gases gives the best estimate of fetal acid–base status as it is carrying deoxygenated blood from the fetus to the placenta. Normal values for umbilical artery blood gases are pH 7.27, $PaCO_2$ 49 mmHg, PaO_2 18 mmHg, and base excess −4 mEq/L [20, 21]. A commonly cited cutoff for the diagnosis of fetal acidemia is a pH <7.0 [20, 21]. Fetal acidemia has been associated with increased risk of neonatal mortality, need for cardiopulmonary resuscitation and intubation, cerebral palsy, hypoxic–ischemic encephalopathy, intraventricular hemorrhage, and seizures [20–23]. A $PaCO_2$ gradient between the fetal (relatively high) and maternal (relatively low) blood is responsible for diffusion across the placenta and is facilitated by maternal hyperventilation that occurs with pregnancy [19]. The significance of a base deficit is discussed below (**L-6**). Low PaO_2 (<18 mmHg) in umbilical artery blood is difficult to interpret [13].

L-6. What is the significance of the base deficit on cord gas analysis?
Lactic acid is an organic acid produced during anaerobic metabolism, and accumulation of this acid leads to a base deficit, the state of decreased buffer base (bicarbonate) in the fetal circulation. Severity of base deficit has been classified as [20, 24, 25]:

- Mild: 4–8 mEq/L
- Moderate: 8–12 mEq/L
- Severe: >12 mEq/L

Several studies have shown that base deficits >12 mEq/L are associated with many neonatal complications including encephalopathy and respiratory compromise. In addition, increasing values of base deficit beyond 12 mEq/L has been shown to lead to a progressively increased severity of neonatal complications [20, 25].

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Chapter 42
Anxious, Coughing, and Bound to Obstruct

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A 3-year-old-male presents for tonsillectomy and adenoidectomy, secondary to obstetric sleep apnea (OSA). The patient weighs 24 kg, and all vitals are within expectations. A brief history and physical reveals an extremely anxious child who is crying in his mother's lap. The mother reports that he has no allergies, takes no medications, and has no other medical issues. However, 4 days ago, he had a fever of 100.6 °F and developed coryza and a cough. Airway exam is unable to be performed secondary to noncompliance. Lung exam reveals stertor and mild rhonchi that clear with cough (**L-1**).

The decision is made not to premedicate the patient (**L-2**) but rather to allow his parent to accompany him into the operating room (OR) for induction. Despite this, the patient is still highly agitated. The inhalation induction is performed with sevoflurane and is remarkable for the combative nature of the child once the mask is applied. Mask ventilation is easy, and direct laryngoscopy reveals a grade one view. The patient is easily intubated with a 4.5-cuffed endotracheal tube. The tonsillectomy and adenoidectomy is without complication.

Once the procedure is completed, the decision is made to perform a deep extubation. However, once the endotracheal tube is removed, no end-tidal CO_2 is detectable and the patient is making obvious unsuccessful attempts to ventilate. The working diagnosis is laryngospasm (**L-3**). The mask is applied, 100 % O_2 is given, and a jaw thrust performed. The patient is now saturating 75 %. Continuous positive airway pressure (CPAP) of 10 cm H_2O is applied, but the patient still is not being adequately ventilated. The oxygen saturations are now 30 %, and the patient's heart rate decreases from 130 to 80. 1 mg/kg of succinylcholine is given, coincident with 0.5 mg of atropine. Ventilation is now easy, and heart rate increases to 166. Saturations increase as well and are soon 100 %. The patient is ventilated via mask

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until completely awake, and no further incidents occur. In the postanesthesia care unit (PACU), despite what is thought to be adequate analgesia, the patient exhibits extreme behavioral disturbances consisting of screaming, crying, and physically lashing out at parents and nurses. He is eventually discharged home after agitation subsides and all other PACU requirements are met.

Lessons Learned

L-1: Anesthesia in children with a URI

More than 200 viruses are associated with the common cold, the most common of which are rhino, corona, respiratory syncytial, influenza, and parainfluenza viruses [1]. Viral invasion into the respiratory epithelium and mucosa leads to airway inflammation, edema, and bronchoconstriction which sensitizes the airway to secretions and volatile agents [2]. Moreover, the viral infection can interact with the autonomic nervous system through the inhibition of cholinergic muscarinic M_2 receptors which is followed by an increased release of acetylcholine and consequent bronchoconstriction [3]. The virally induced liberation of tachykinin and neuropeptidases can lead to a constriction of smooth muscles in the respiratory tract that lasts for weeks. The resulting bronchial hyperreactivity can persist for up to 6 weeks, well beyond the disappearance of all clinical symptoms [4, 5]. Bronchial hyperreactivity can trigger severe complications in the perioperative period, particularly laryngospasm and bronchospasm, both of which can lead to fatal hypoxemia, which is the main cause of perioperative morbidity and mortality in children [6]. Typical adverse events in children with respiratory tract infection are laryngospasm, bronchospasm, breath holding, atelectasis, oxygen desaturation, bacterial pneumonia, and hospital admission [7, 8]. Independent risk factors for perioperative respiratory events can be divided into three categories: child-specific, anesthesia-specific, and surgery-specific risk factors (see Table 42.1 [7, 9–12]).

Table 42.1 Independent risk factors for perioperative respiratory events in children

Child-specific risk factors	Age below 6 years (especially infants below 1 year)
	Clinical signs of URI
	Primary pulmonary morbidity (asthma, bronchopulmonary dysplasia, cystic fibrosis, etc.)
	General infectious symptoms (fever, malaise)
	Passive smoke exposure
Anesthesia-related risks	Instrumental manipulation of the airway (endotracheal tube, bronchoscopy)
	Airway management (ETT > LMA > face mask)
	Anesthetic agents (desflurane > sevoflurane > propofol)
	Specialization of the anesthesiologist in pediatric anesthesia
Surgery-related risks	Airway surgery, ear-nose-throat surgery, eye surgery
	Upper abdominal surgery, cardiac surgery

The questions of whether to cancel surgery and anesthesia for a child with a URI and how long to delay the procedure are ones that do not presently have a succinct answer. There is a general consensus, however, that surgery does not need to be postponed for 6 weeks. Several studies suggest a delay of at least 2 weeks when acute clinical signs of infection are observed [8, 12, 13].

Given that the patient's surgery was an ear, nose, and throat (ENT) procedure, that the anesthetic (most commonly) involves the use of an endotracheal tube, and that the patient exhibited symptoms of an ongoing URI, it would be reasonable to cancel the case and reschedule it for 2 weeks later. Figure 42.1 outlines a decision making algorithm for a child with a URI presenting for anesthesia and surgery.

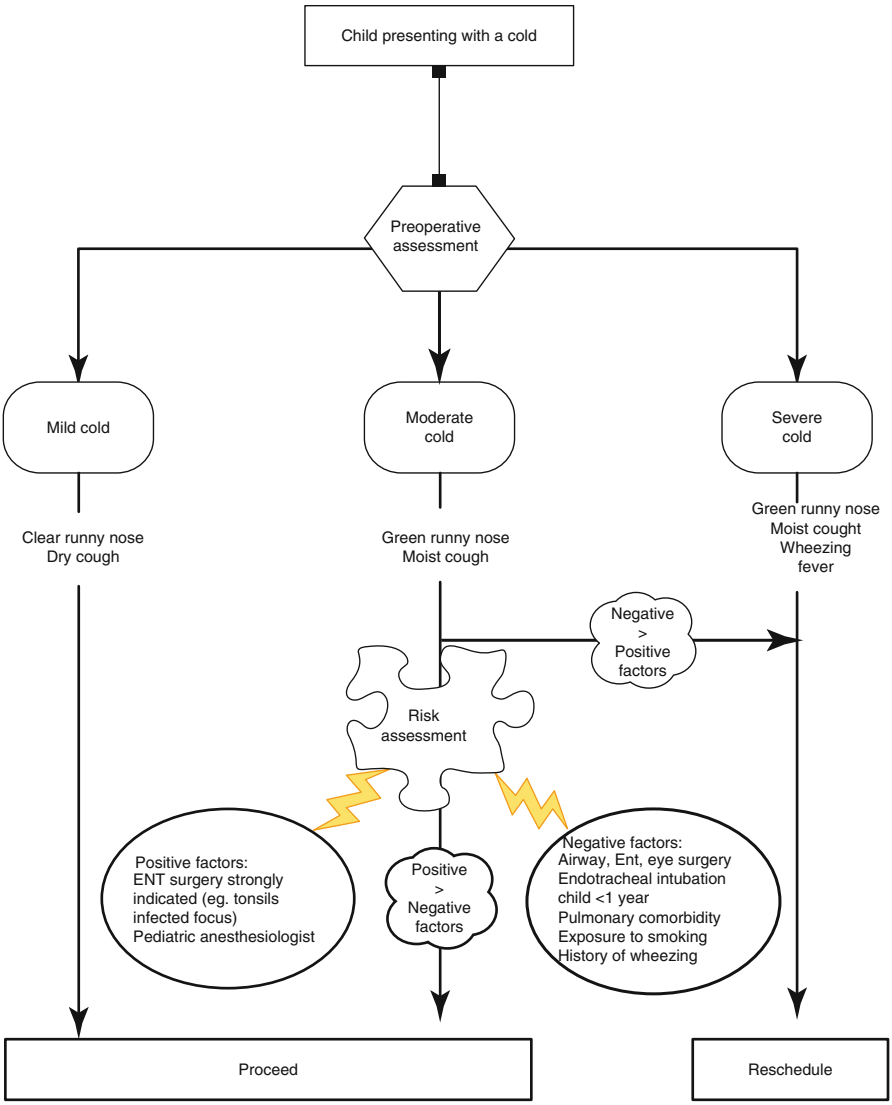

Fig. 42.1 Decision-making algorithm for a child with a URI presenting for anesthesia and surgery [7, 9, 14]

Table 42.2 Preferred premedications and routes

Drug	Mechanism of action	Route	Dose (mg/kg)	Time to onset (minutes)
Midazolam	GABA agonist	PO	0.25–1	10–20
		Intranasal	0.2	10
		IM	0.1–0.2	1–3
Clonidine	Alpha 2 agonist	PO	0.004	45–60
		Intranasal	0.003	30–45
Dexmedetomidine	Highly selective alpha 2 agonist	Intranasal	0.002	20–30
Ketamine	NMDA antagonist	IM	2–3	3–5
		PO	5	15–20

L-2: To premedicate or not premedicate?

It has been reported that up to 70 % of children experience stress and anxiety before surgery [14, 15]. Anxiety and stress before surgery can result in negative postoperative behavioral changes, such as separation anxiety, sleep disturbance, aggression toward authority, temper tantrum, and eating problems, and have been described in as much as 50–60 % of children [16–18]. These negative behavioral changes have been shown to persist postoperatively in 54 % of patients after 2 weeks, 20 % at 6 months, and 7.3 % at 1 year [18]. Children who are anxious during the induction of anesthesia are almost three times more likely to develop these postoperative negative behavioral changes [19]. Increased preoperative anxiety may also be associated with increased postoperative pain and may hinder recovery [16]. Emergence delirium occurs in 12–18 % of all children undergoing anesthesia and surgery, and Kain et al. showed that its incidence is directly related to the level of preoperative anxiety [16].

Unfortunately, the presence of a parent at induction does little to decrease the development of these postoperative sequelae, and while parental anxiety may be allayed, that of most children is not [20–22]. Other methods to decrease preoperative anxiety have been studied. One modality that combines parental presence and extensive cognitive preparation methods (ADVANCE) is as effective as midazolam but requires a preoperative visit with a nurse or psychologist with a behavioral background [23]. The presence of clowns at induction of anesthesia also decreases anxiety [24]. These alternative methods show some promise, and could be a part of an integrated approach, but are presently not as universally efficacious, available, and economically viable as sedative premedication for reduction of anxiety.

The major objectives of preanesthetic medication are to decrease the stress response with preservation of hemodynamic parameters, facilitate anesthesia induction, and produce amnesia. Sedative premedication with midazolam, clonidine, ketamine, or dexmedetomidine has been shown to decrease preoperative anxiety [15, 21, 22, 25, 26]. This decrease in preoperative anxiety decreases the incidence of behavioral changes seen at 1 week postoperatively [27].

This author is in favor of the treatment of preoperative anxiety and, given the above brief discussion, favors the use of sedative premedication to achieve this.

What can we give children as premedication? Presuming no IV access, the author's preferred premedications and routes are shown in Table 42.2 [25, 26, 28].

Note that it is rare that children under 6 months of age require premedication and that the most at risk age group is in the range of 2–5 years.

L-3: Laryngospasm

Laryngospasm occurs commonly in children and has an incidence that ranges from 1/1,000 up to 20/100 in high-risk surgery (as outlined in Table 42.1) [10, 12, 29]. The risk factors associated with an increased incidence of laryngospasm are outlined in Table 42.1. Laryngospasm is the number one cause of perioperative cardiac arrest of respiratory origin in children [6].

Laryngospasm is defined as a partial or complete airway obstruction concomitant with efforts to breathe against a closed glottis. Its diagnosis depends mostly on the clinical judgment of the anesthesiologist. Clinical signs are those of airway obstruction, suprasternal and infraclavicular retractions, tracheal tug, and paradoxical chest and abdominal movements. In complete laryngospasm, there will be no air movement or breath sounds, and if obstruction is not relived, oxygen desaturation, bradycardia, and cyanosis may occur [30].

The way in which an anesthetic is conducted influences the risk of laryngospasm. The risk factors outlined in Table 42.1 may help to serve as a guideline for the safe planning of an anesthetic in at risk children. Any type of airway manipulation or other untoward stimulation (such as IV access) at an insufficient depth of anesthesia is a major cause of laryngospasm. In the presence of a URI, the use of an endotracheal tube may increase the incidence of respiratory adverse events by 11-fold, in comparison to a facemask [7]. The use of a laryngeal mask airway (LMA) in children with a recent URI has been shown to increase the incidence of laryngospasm [8] but is associated with less of an increased risk than use of an endotracheal tube [12]. Induction and maintenance of anesthesia with propofol is associated with a decreased incidence of laryngospasm versus sevoflurane [12], a result that may be explained by the differences in laryngeal and respiratory reflex responses at similar depths of anesthesia between the two agents [31]. Presently, there is equivocal evidence supporting a deep extubation in order to decrease the incidence of laryngospasm [12, 13, 32]. It is the opinion of this author that in the absence of any significant concerns for the specific protection of surgical site, pulmonary or cerebral vascular beds, the safest way to extubate a pediatric patient is to wait until the patient is fully awake, at which point all airway protective reflexes are both present and coordinated.

Once laryngospasm is present, however, its effective management requires prompt diagnosis followed by appropriate and rapid management [33]. Most authors, including this one, suggest the application of airway manipulation maneuvers first, followed by the use of pharmacologic agents if necessary [33, 34] (Fig. 42.2). Airway manipulation most commonly involves removal of the irritant [33, 35], jaw thrust, chin lift [36], continuous positive airway pressure, and positive-pressure ventilation with 100 % oxygen [30, 37, 38]. These methods are easily performed, well known to anesthesia practitioners, and are known to increase the patency of the upper airway in the case of obstruction [39]. It should be noted that care should be exercised with the application of positive airway pressure, as it may result in significant gastric distention that in turn increases the

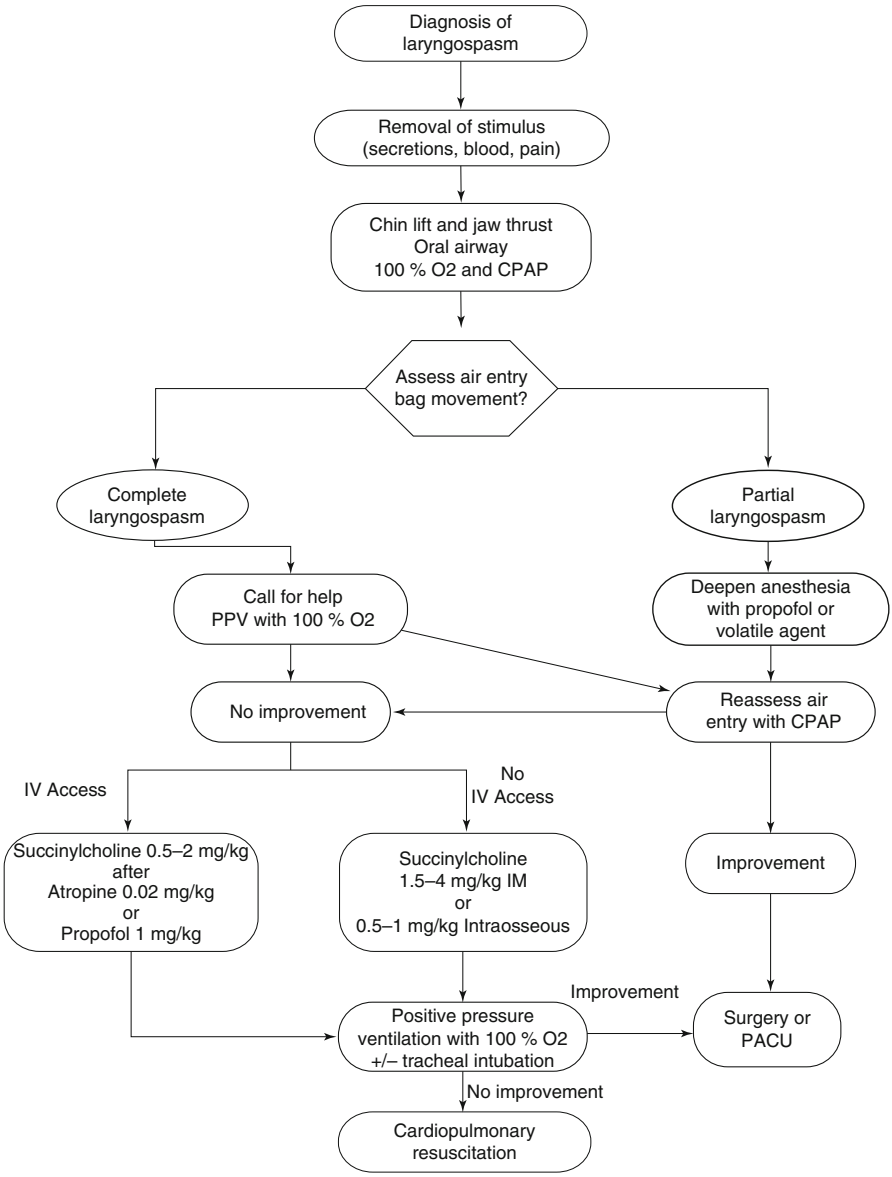

Fig. 42.2 Decision-making algorithm for the management of laryngospasm in pediatric patients (Adapted from [14, 30, 33, 34, 37])

risk of regurgitation. Ultimately, both hypoxia and hypercarbia lead to relaxation of the vocal cords, but this is a dangerous solution on which to rely. If the laryngospasm is complete and unable to be relieved, the use of intravenous agents is the next step in management.

Propofol depresses laryngeal reflexes [31] and is commonly used to treat laryngospasm in children [30]. Propofol (0.8 mg/kg), when given after gentle positive pressure with 100 % O_2 has failed, is able to relieve laryngospasm in 77 % of patients [40]. In this investigation, this dose of propofol was given if laryngospasm occurred after LMA removal, and its persistence resulted in an SpO_2 decrease to 85 % despite oxygen and positive-pressure ventilation [40]. Smaller doses of propofol can be effective but has only been proven as such in a small series of patients [41]. An important point of consideration is the use of propofol to relieve laryngospasm in the presence of bradycardia; its efficacy in the presence of complete laryngospasm and bradycardia is questionable [34] and may, by virtue of its myocardial depressant properties, accelerate the cardiovascular course down which the patient is descending.

The next class of intravenous agents to consider is muscle relaxants. The gold standard, by virtue of its rapidity of onset, is succinylcholine [34]. While there is some evidence to support the use of small doses of succinylcholine (0.1 mg/kg) [42], the lack of a dose response study makes such evidence difficult to rely upon. This author recommends dosing at least 0.5 mg/kg if IV access is available. If no access is available, succinylcholine can be dosed at; 1.5–4 mg/kg intramuscularly or 1 mg/kg via intraosseous line [43]. The injection of succinylcholine in a hypoxic patient may lead to severe bradycardia or arrest. As such, it is imperative to give IV atropine beforehand. In children in whom succinylcholine is contraindicated, rocuronium given at a dose two to three times the ED_{95} (0.9–1.2 mg/kg) may represent a reasonable substitute when a rapid onset is needed [44].

The decision as to which agent—propofol or succinylcholine—to administer first is a matter of timing. Severe laryngospasm and the presence of bradycardia may prompt the practitioner to first reach for atropine and succinylcholine.

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Part V
Special Diseases, Conditions, Situations

Chapter 43
Hypothermia During Laparoscopic Nephrectomy

Michael Bronson

The patient is a 38-year-old female (163 cm, 80 kg, body mass index [BMI] 30) presenting for laparoscopic nephrectomy for the purpose of donating a kidney to a relative. She had no significant past medical history and was taking no medications. Prior to induction, she received 800 mL of normal saline (NS) and 2 mg of midazolam while monitors were placed. Following preoxygenation, anesthesia was induced with fentanyl 500 mcg, lidocaine 100 mg, propofol 150 mg, and vecuronium 7 mg. Immediately after induction, the patient's blood pressure dropped from 130/80 to 85/45 mmHg which was treated with an intravenous fluid bolus of 500 ml NS and 200 mcg of phenylephrine. Intubation proceeded without incident, and the patient was placed in a lateral decubitus position.

Over the hour following induction, 4 L of NS and a total of 600 mcg of phenylephrine and 5 mg of ephedrine were given to maintain the blood pressure around 100/50 mmHg. The patient's temperature was also noted at this time to be 34.3 °C (**L-1**, **L-2**, **L-3**). Over the next hour, two more liters of NS were given, and the temperature gradually decreased to 33.6 °C (**L-1**, **L-2**, **L-3**). A forced-air warming blanket was placed on the patient, and the intravenous (IV) line was also connected to a fluid warmer at this time (**L-4**). At the end of the case the patient's temperature was 34.5 °C. The patient was transported to the postanesthetic care unit (PACU) intubated. Her temperature rose to 36.0 °C within 30 min and she was extubated uneventfully. Her postoperative course was uncomplicated.

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Lessons Learned

L-1: **In the absence of warming the patient, what is the expected change in temperature following induction of general anesthesia**?

In a nonanesthetized patient, a temperature gradient exists between the core (37 °C) and periphery (31–35 °C) due to thermoregulatory vasoconstriction [1, 2]. The induction of anesthesia has a dramatic effect on this gradient as vasodilation from volatile and intravenous anesthetics leads to a rapid redistribution of blood from the core compartments (thorax/abdomen) to the periphery (arms/legs), resulting in a subsequent decrease in core body temperature (Fig. 43.1) [1, 2]. Therefore, the initial drop in temperature over the first hour following the induction of general anesthesia is a function of the redistribution of body heat rather than the physical process of heat loss.

Following the initial period of redistribution, the patient's core temperature still gradually decreases over the next 2–3 h. This decline occurs as heat loss to the environment exceeds internal heat production. Four processes that contribute to this heat loss are (in order of most important to least important):

1. Radiation: all objects above absolute zero radiate heat
2. Convection: transfer of heat from object to air/fluid
3. Conduction: transfer of heat between objects
4. Evaporation: heat loss from water vaporization

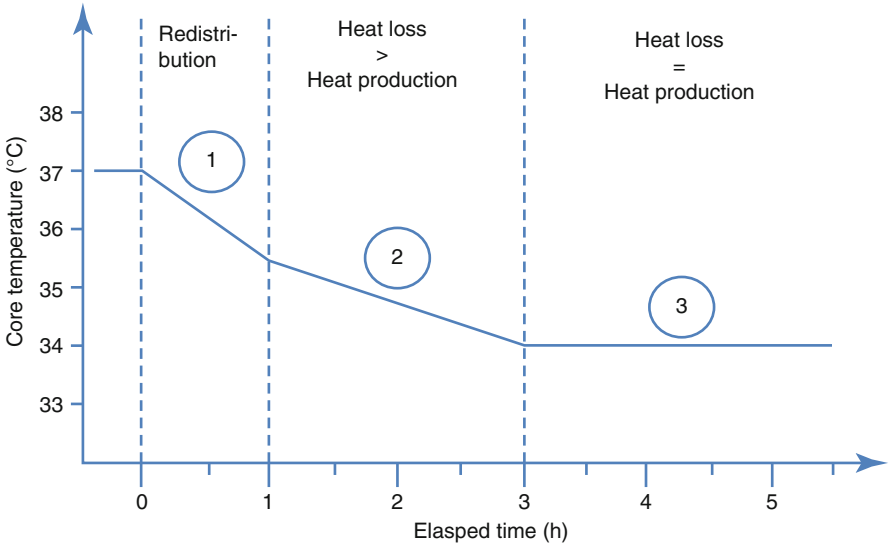

Fig. 43.1 Expected temperature changes following the induction of general anesthesia. Following induction, a temperature decrease of 0.5–1.5 °C occurs as blood is redistributed from the core components to the periphery from vasodilation (*1*). This is followed by a more gradual decline in temperature 1.0–1.5° over the next 2–3 h as heat loss remains greater than production (*2*). A state of equilibrium is achieved 3–4 h after the induction of anesthesia when heat loss becomes equal to heat production (*3*)

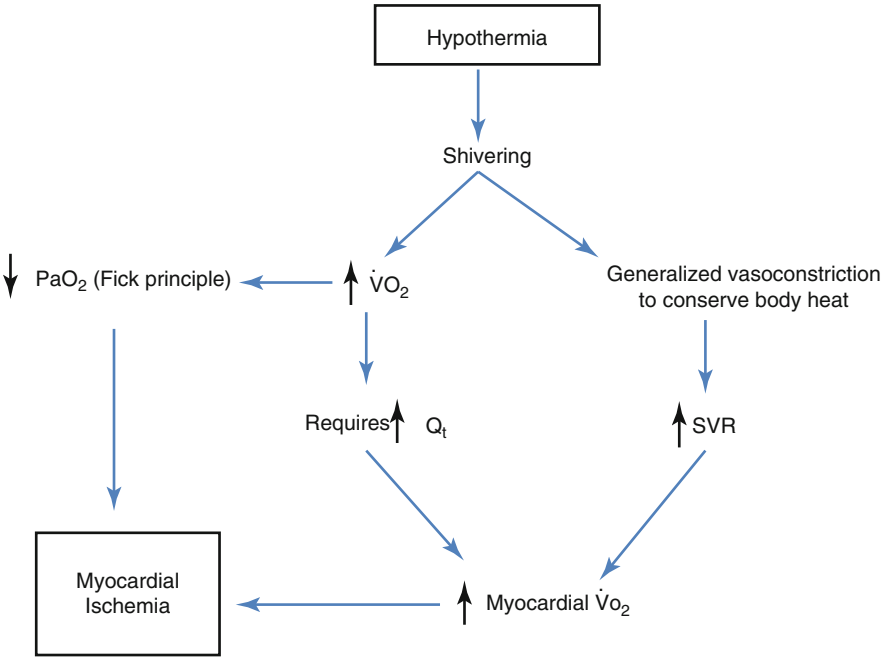

Fig. 43.2 Hypothermia can cause myocardial ischemia. Q_t cardiac output, VO_2 oxygen consumption, *SVR* systemic vascular resistance

Approximately 3–4 h following induction of anesthesia, heat production becomes equal to heat loss so the patient's core body temperature will remain relatively constant. This state of equilibrium occurs as a function of cutaneous vasoconstriction.

L-2: **What deleterious changes may occur if a patient becomes hypothermic**? Many adverse effects have been associated with hypothermia [1–3]:

1. Myocardial ischemia
2. Cardiac dysrhythmias
3. Wound infection/poor wound healing
4. Postoperative shivering
5. Peripheral vasoconstriction → increased systemic vascular resistance (SVR)
6. Coagulopathy
7. Increased blood loss and transfusion requirements
8. Decreased metabolism of drugs (muscle relaxants)
9. Increased PACU length of stay/decreased patient comfort

Myocardial ischemia is of particular concern (Fig. 43.2), especially in patients with known coronary artery disease (CAD) or risk factors for CAD. Postoperative shivering has been shown to increase oxygen consumption by up to 200 % resulting in a decreased PaO_2 and reducing overall oxygen supply to the myocardium [1–3].

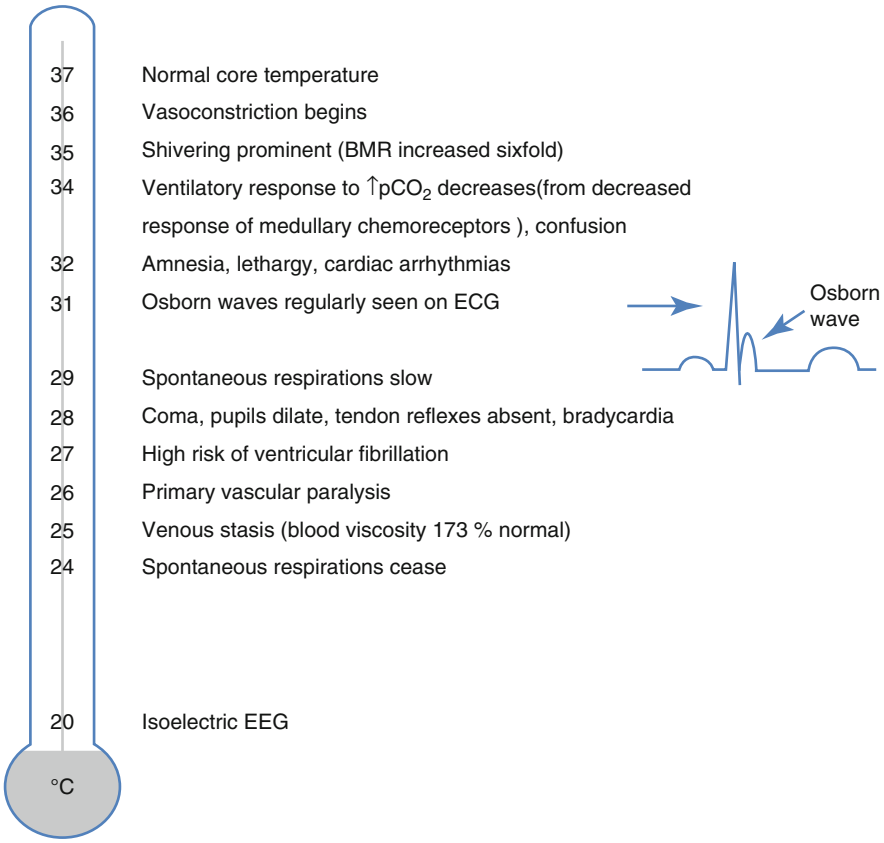

Fig. 43.3 Signs and symptoms of hypothermia (Adapted from [4])

This is coupled with generalized vasoconstriction that occurs as a physiologic response to conserve body heat. Consequently, an increased SVR places more afterload on the heart, which increases its demand. Therefore, both supply and demand of the myocardium become unfavorable during periods of hypothermia and as a result increase the risk of an acute coronary syndrome.

Many of the effects of hypothermia consistently occur at defined temperatures [4]. Figure 43.3 summarizes this correlation.

L-3: **What is the effect on patient temperature following the administration of 1 L of IV fluid at room temperature** (**20 °C**)?

It has been estimated that 1 L of crystalloid given at room temperature (20 °C) will result in a 0.25 °C decrease in the patient's core temperature [1]. As an example, in the case presented above, the patient received 6 L of crystalloid at room temperature before an IV warmer was connected; therefore, it is likely that the unwarmed IV fluids contributed to a 1.5 °C decrease in the patient's core temperature.

Table 43.1 Methods to warm a hypothermic patient (with reference to the patient)

1. Warm the OR to 23 °C (increases heat gain and decreases heat loss)
2. Place a forced-air warming blanket (increases heat gain and decreases heat loss)
3. Warm IV fluids (increases heat gain)
4. Humidify inspired gas (decreases heat loss)
5. Apply blankets/drapes to decrease surface area of exposed skin (decreases heat loss)

L-4: **What are the methods to warm a hypothermic patient**?
Methods aimed at warming a hypothermic patient are centered on two goals: (**1**) to transfer heat to the patient and (**2**) minimize heat loss from the patient (Table 43.1).

The most useful step in actively rewarming the patient is to increase the temperature of the operating room to donate radiant heat to the patient [1]. A forced-air warming blanket donates heat to the patient via convection currents. These methods of active rewarming also serve to minimize heat loss.

Intravenous administration of room temperature fluids contributes to ongoing heat loss (refer to **L-3**); therefore, connecting an IV warmer to the fluid line will help limit this. It is estimated that 10 % of heat loss occurs through respirations so applying an airway humidifier to the respiratory circuit should help decrease heat loss via this system [1]. Also, minimizing the exposure of the patient's skin to the environment is a key approach to maintaining body heat, so blankets and operating room drapes can be used to cover exposed areas.

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Chapter 44
Operating Room Management Case Scenarios

Leon Chang

All four cases presented below make the following assumptions:

1. You are the manager for anesthesiology resources ("floor runner") at your local hospital. Such a manager ordinarily has several priorities/objectives, namely,
   (a) Run the operating rooms (ORs) such that the cost to the hospital and anesthesia department is minimized.
   (b) Facilitate optimal clinical care.
   (c) Facilitate resident teaching.
   (d) Facilitate research.
2. The coverage is "mixed model"; attendings supervise Certified Registered Nurse Anesthetists (CRNAs) and residents and can provide solo coverage care.
3. An attending can supervise at most:
   - 2 residents
   - 1 resident and 1 CRNA
   - 3 CRNAs
4. Every case must have an attending assigned to either to supervise or staff solo.

In all cases presented below, the reader should understand that patient safety is directly related to how well planned and competently an OR suite is run. Specifically, a well-run OR suite results in:

1. The right personnel doing the right case
2. The right equipment being available for the right case
3. Anesthesia and surgical care being administered in a thoughtful and complete manner
4. *Decreases in complications and contributions to this book*

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Case 1. No One Completely "Free" and Urgent Case Scheduled

It is the beginning of the day and 11 operating rooms are in use. There is a 12th room which is unoccupied and reserved for emergency cases (e.g., trauma). You are called about an urgent exploratory laparotomy after a motor vehicle accident in the field, estimated time available (ETA) 10 min by ground.

The distribution of anesthesia providers is as follows:

OR 1—CRNA A, supervised by attending Smith
OR 2—resident A, supervised by attending Smith
OR 3—CRNA B, supervised by attending Jones
OR 4—CRNA C, supervised by attending Jones
OR 5—CRNA D, supervised by attending Jones
OR 6—resident B, supervised by attending Chen
OR 7—CRNA E, supervised by attending Chen
OR 8—attending Carter providing solo anesthesia
OR 9—CRNA F, supervised by you
OR 10—resident C, supervised by attending Wilson
OR 11—CRNA G, supervised by attending Wilson

(See Table 44.1 for a schematic representation.)

There is no one completely "free" to do the case. How can you rearrange coverage for the impending urgent case?

Discussion

Several options exist to rearrange existing coverage and optimize resources.

1. Determine if a case is ending in the next few minutes. If so, that team will be free to provide anesthesia for the impending urgent case. Note that this will require valid, real-time information. The case that is ending must truly finish within the limited time frame, and not just be "close." The floor runner will have to visit that room to physically verify facts for himself. This is an unlikely but possible option.

Table 44.1 Distribution of anesthesia providers

OR	Attending	CRNA	Resident
1	Smith	A	
2	Smith		A
3	Jones	B	
4	Jones	C	
5	Jones	D	
6	Chen		B
7	Chen	E	
8	Carter (solo)		
9	You (floor)	F	
10	Wilson		C
11	Wilson	G	

Table 44.2 Solution A: redistribution of anesthesia providers

OR	Attending	CRNA	Resident
1	You	A	
2	Smith		A
3	Jones	B	
4	Jones	C	
5	Jones	D	
6	Smith		B
7	You	E	
8	Carter (solo)		
9	You	F	
10	Wilson		C
11	Wilson	G	
12 (urgent case)	Chen (solo)		

2. Shuffle coverage such that attendings cover three CRNAs, freeing up another provider. In solution A (see Table 44.2), the floor runner could cover ORs 1 and 7, in addition to his original OR9, all of which have CRNAs in them. Smith would then be free to cover OR6 in addition to OR2 This would relieve Chen of all coverage responsibilities and enable him to do the urgent case solo. Solution B (see Table 44.3) would have Chen go into OR6 solo, freeing up resident B. Smith would take ORs 7 and 11 in addition to his original OR1. Wilson would then only have OR10 and would be able take OR2. You and resident B would then be able to do the urgent case together. Note in all of these examples supervision rules are not violated.
3. Violate supervision rules until resources become available. In this scenario, one would intentionally create a supervision violation for other, more important reasons (e.g., resident education, patient care). Suppose OR10, with resident C and Wilson are the most experienced trauma team. It may make sense to do the following (solution C): you (the floor runner) take ORs 1 and 2, covering three rooms with residents (violation) but freeing up Smith. Smith would then provide solo coverage to OR10, relieving Wilson and resident C. Chen would cover OR11 (violation), completely freeing up Wilson and allowing resident C and Wilson to do the urgent case (Table 44.4).

All of the above examples illustrate the importance of the floor runner having less supervision or workload requirements than other providers. This allows the floor runner the freedom to make real-time decisions as well as take on additional supervision should it become necessary. Utilizing every resource to its absolute maximum including the floor runner limits options in case of unexpected emergencies. At our institution, we attempt to schedule the floor runner slightly light on supervision and in fact consider the floor duties to be equivalent to covering an OR.

Table 44.3 Solution B: redistribution of anesthesia providers

OR	Attending	CRNA	Resident
1	Smith	A	
2	Wilson		A
3	Jones	B	
4	Jones	C	
5	Jones	D	
6	Chen (solo)		
7	Smith	E	
8	Carter (solo)		
9	You	F	
10	Wilson		C
11	Smith	G	
12 (urgent case)	You		B

Table 44.4 Solution C: redistribution of anesthesia providers, with the most experienced trauma team handling the urgent case (note violation of supervision rules)

OR	Attending	CRNA	Resident
1	You	A	
2	You		A
3	Jones	B	
4	Jones	C	
5	Jones	D	
6	Chen		B
7	Chen	E	
8	Carter (solo)		
9	You	F	
10	Smith (solo)		
11	Chen	G	
12 (urgent case)	Wilson		C

Finally, there may be several other realities that need to be taken into consideration. These include but are not limited to the following:

1. Some attendings may prefer to do solo anesthesia.
2. Some attendings may be better teachers than others.
3. Attendings have variable subspecialty strengths and weaknesses.
4. Experience level and abilities of the various CRNAs and residents.

Case 2. Maximizing Revenue and Minimizing Costs
You are writing the manpower distribution/schedule for the next day. Ten ORs need to be covered. You have the following providers available to you:

Residents A, B, and C
CRNAs A, B, C, D, E, F, G, and H
Attendings Smith, Jones, Carter, Chen, and Wilson

OR 1?
OR 2?
OR 3?
OR 4?
OR 5?
OR 6?
OR 7?
OR 8?
OR 9?
OR 10?

Assume the following financial situation:

1. Residents are "free" (in reality, they may cost an anesthesia department approximately 10K$/year but are largely paid for by the School of Medicine).
2. CRNAs cost 120K$/year.
3. Attendings cost 150K$/year.
4. Not using a resource saves the salary for that day for the department, institution, or both.
5. Daily salary savings directly decrease yearly cost.

What are some options to provide coverage for all the ORs while maximizing revenue return/minimizing costs?

Discussion

Clearly, all available residents should be utilized as they represent a nearly "free" resource. The only issue with residents is that they must be supervised and an attending can only supervise two rooms at once if a resident is involved.

Scenario 1.

Step 1—assume resident coverage of ORs 1, 2, and 3.

Next, examine the breakdown of attending solo coverage vs. attending coverage with CRNAs per room:

- Two room coverage with CRNAs and attending supervision = $2 \times 120\text{K} + 150\text{K} = 390\text{K}$ total, or 195K per room.
- Three room coverage with CRNAs and attending supervision = $3 \times 120\text{K} + 150\text{K} = 510\text{K}$, or 170K per room.

Note in both these examples it would be cheaper to run all rooms with solo attending coverage (150K/room). In actuality, individual practices may not consider this a feasible option.

Step 2—assign solo attending coverage and/or three CRNAs per attending wherever possible.

Our coverage now looks like this:

OR 1—resident A, attending Smith
OR 2—resident B, attending Smith
OR 3—resident C, attending Jones
OR 4——CRNA A, attending Jones
OR 5—CRNA B, attending Carter
OR 6—CRNA C, attending Carter
OR 7—CRNA D, attending Carter
OR 8—CRNA E, attending Chen
OR 9—CRNA F, attending Chen
OR 10—CRNA G, attending Chen

In this scenario, CRNA H and attending Wilson are not needed and can be written off the schedule.

Net cost = 4 × 150K/attd + 7 × 120K/CRNA = 1,440K.

Scenario 2.

Suppose only residents A and B were available; in that case, a possible schedule may look like this:-

OR 1—resident A, attending Smith
OR 2—resident B, attending Smith
OR 3—CRNA H (replaced resident C), attending Jones
OR 4—CRNA A, attending Jones
OR 5—CRNA B, attending Carter
OR 6—CRNA C, attending Carter
OR 7—CRNA D, attending Carter
OR 8—CRNA E, attending Chen
OR 9—CRNA F, attending Chen
OR 10—CRNA G, attending Chen

Total cost = 4 × 150K/attd + 8 × 120K/CRNA = 1,560K.

In this case, one could further optimize the schedule from a cost perspective by utilizing solo attending coverage:

OR 3 covered by attending Jones solo, OR 4 covered by attending Wilson solo. Net salary change = 1 attending (150K) – 2 CRNAs (240K) = −90K, 90K savings. Final coverage in this scenario would look like:

OR 1—resident A, attending Smith
OR 2—resident B, attending Smith
OR 3—attending Jones solo
OR 4—attending Wilson solo
OR 5—CRNA B, attending Carter

OR 6—CRNA C, attending Carter
OR 7—CRNA D, attending Carter
OR 8—CRNA E, attending Chen
OR 9—CRNA F, attending Chen
OR 10—CRNA G, attending Chen

Total cost = 5 × 150K/attd + 6 × 120K/CRNA = 1,470K.

The following conclusions should be drawn when considering resource utilization and financial optimization:

1. Utilize resident coverage whenever possible.
2. Maximize CRNA to attending coverage whenever possible.
3. Solo attending coverage in many instances can be cheaper than CRNA coverage with supervision.

Case 3. Case 2 Continued with Implications of Residents Working Late
Examining the first hypothetical planned schedule from case 2 above (reprinted below), what aresome implications of residents having to work into the late PM hours?
Hypothetical schedule from case 2:

OR 1—resident A, attending Smith
OR 2—resident B, attending Smith
OR 3—resident C, attending Jones
OR 4—CRNA A, attending Jones
OR 5—CRNA B, attending Carter
OR 6—CRNA C, attending Carter
OR 7—CRNA D, attending Carter
OR 8—CRNA E, attending Chen
OR 9—CRNA F, attending Chen
OR 10—CRNA G, attending Chen

Discussion

When residents work into the late PM hours, two major problems arise. First, violations of work-hour rules may result in loss of accreditation or other sanctions for residency programs. Secondly, residents routinely being required to work long hours can cut down on productivity and morale, resulting in a less effective anesthesia provider. Current ACGME/residency rules in fact require a resident to report if they are unduly fatigued and for the program to accommodate this should it occur. A chronically fatigued resident may in fact not be available in the workforce for the next day.

In addition, current resident work rules include a mandatory 10-h break between work shifts. Thus, if a resident stays until 10 pm on 1 day, they cannot be asked to work until 8 am the next day. This may have schedule implications for the next day. Consider the following scenario:

The hypothetical schedule is as above. Resident C unfortunately worked until 10 pm the night before. Cases start at 7 am the following day. For all intents and purposes, resident C *cannot* work until 8 am the following day, and thus the schedule must be rewritten to have another provider in C's place.

Also note the financial implications of a resident working past duty hours as in the above example. Two possible schedule rewrites as from case 2 are:

Schedule rewrite 1
OR 1—resident A, attending Smith
OR 2—resident B, attending Smith
OR 3—CRNA H (replaced resident C), attending Jones
OR 4—CRNA A, attending Jones
OR 5—CRNA B, attending Carter
OR 6—CRNA C, attending Carter
OR 7—CRNA D, attending Carter
OR 8—CRNA E, attending Chen
OR 9—CRNA F, attending Chen
OR 10—CRNA G, attending Chen

Total cost = 4 × 150K/attd + 8 × 120K/CRNA = 1,560K.

Schedule rewrite 2
OR 1—resident A, attending Smith
OR 2—resident B, attending Smith
OR 3—attending Jones solo (replacing resident C)
OR 4—attending Wilson solo
OR 5—CRNA B, attending Carter
OR 6—CRNA C, attending Carter
OR 7—CRNA D, attending Carter
OR 8—CRNA E, attending Chen
OR 9—CRNA F, attending Chen
OR 10—CRNA G, attending Chen

Total cost = 5 × 150K/attd + 6 × 120K/CRNA = 1,470K.

Note both of these schedules are more expensive than had resident C been available (total cost = 1,440K). Additional CRNAs and/or attendings will have to be utilized to make up for resident C's absence. Loss of resident manpower due to any factor including work-hour violations has deleterious consequences for any anesthesiology program and should be avoided wherever possible.

Case 4. Turnover and Production Pressure

Reducing turnover and other forms of production pressure have become a daily reality for almost every anesthesiologist. These pressures are relevant for examination because a rushed and pressured anesthesia provider is predisposed to make errors and create cases for M&M.

The Case

You are asked by the hospital OR executive committee to help address the "turnover problem." Many of the surgeons feel that turnover between cases is too slow and that anesthesiology is a major source of this delay.

Question 1: **What data may be useful**, **and what information do you need to properly examine this issue**?

The following pieces of information will be helpful:

Precise definitions used to determine turnover time. Everyone must be on the same page, using the same metrics. Does turnover begin when the surgeon is done with the case, when the anesthesia provider leaves the room with the patient, or when the cleaning crew actually enters the room? Similarly, does the next case begin when the anesthesia provider picks the patient up, when the patient enters the room, or when incision is made? Most institutions including UCSD use "time patient leaves the OR, until the next patient enters" as turnover time. It is important that all parties understand the definition.

National and/or local standards of turnover time for similar practices. Benchmark data for comparison will be helpful. This will help frame the discussion and place the debate in objective, rather than subjective terms.

Turnover time data from the ORs. Once the definition of turnover time is agreed upon, only the objective data should be used. Subjective impressions may be powerful, but they should not be given credibility in lieu of the actual data.

Turnover time expectations of the surgeons, anesthesiologists, and hospital and OR staff. It is important to know what all vested parties' expectations are. If one group of the OR team is operating under completely different assumptions than another team, it is a recipe for misalignment. In addition, all parties' expectations should be reasonable for what the OR is capable of achieving, e.g., an expectation of a 5-min turnover, while ambitious, is unrealistic.

Question 2: **Suppose you learn the following**:

- Turnover time is defined as "surgical end" to "next patient in OR."
- Average turnover time for your hospital is 45 min; the comparable local average is 30 min.
- The hospital and surgeons expect the ORs to achieve the local average.

Is the use of "surgical end time" a reasonable start to measure turnover time? Why might a surgeon assume this marks turnover time?

In the author's opinion, "surgical end time" is not a good metric to use to measure turnover time. For one, the case isn't over until the patient actually leaves the OR! While the patient is in the room, the cleaning crew/turnover team cannot physically begin the turnover process. Additionally, the time between surgery end and when the patient leaves the OR can be quite variable. Emergence from anesthesia remains an event with an unpredictable duration, and the time taken is primarily influenced by patient safety. As such, the only reasonable metric to mark the beginning of turnover time should be "time patient physically leaves the OR," not "surgical end time" or even "anesthesia end time" (which usually occurs in the postanesthesia care unit [PACU], after the patient has left the OR).

Although the surgeon's involvement in the case may be over after "surgical end time," this has no bearing on the actual turnover time between cases. Some amount of disagreement may arise from the fact that different parties are finished with the case at different times, leading to the perception of delays.

Question 3: **You are questioned why** "**anesthesia end time**" **is often after the** "**time patient leaves the OR**". **Some administrators feel this may be the source of slow turnovers**. **How can you respond**?

Unfortunately, there is a fair amount of misunderstanding and even ignorance concerning the various metrics in use in the perioperative theater. "Anesthesia end time" marks the time when the anesthesia provider passes off care to another trained provider (e.g., PACU or intensive care unit [ICU] nurse). Necessarily, this is typically after the patient leaves the OR. The anesthesia provider's job is not finished simply by leaving the OR; they must safely get the patient to the next step in recovery, and then turn over the patient's care. While a long time period between "patient leaves the OR" and "anesthesia end time" may indeed slow the room turnover, the mere fact that "anesthesia end time" comes after "time patient leaves the OR" is meaningless.

Question 4: **Suppose the metrics are reexamined**. **Turnover time is now defined as** "**time patient leaves the OR**" **to** "**time next patient arrives in OR**". **Your hospital's turnover times are now an average of 35 min**, **with the local average being 30 min**. **What are some possible areas of improvement**?

The following broad categories all influence turnover time:

1. Surgical—all paperwork/consent must be in order, and patient concerns addressed.
2. Anesthesia—the next patient must be seen, preoperative evaluation done, and room and anesthesia machine setup/turnover complete.
3. Nursing—paperwork must be in order; surgical equipment must be ready.
4. Turnover crew—the room must be physically cleaned and organized, special equipment needs addressed.

Each category should be examined for systemic problems that may be causing delay. A good general approach would be to assess the cause and frequency of each delay to understand which area could most be improved (e.g., if turnover crew is causing 80 % of delays, it makes sense to start there). Addressing problems in the system invariably yields higher results than addressing individual personnel; thus, systemic issues should be sought out aggressively.

Question 5: **Does reduction of turnover time by 5 min really even matter**?

From a practical standpoint, no. Suppose a hypothetical OR was open for 10 h/day, with an average case length of 2 h, including a turnover time of 30 min. In this scenario, the OR can handle five cases/day. Reducing each turnover by 5 min would yield a time savings of $5 \times 5 = 25$ min, which does not represent enough time to do another case of average length. There is a small case to be made for a reduction in the duration the OR is functioning, which may reduce the cost of nursing personnel to a small degree.

However, as the perioperative theater depends on every team (e.g., surgery, anesthesia, nursing) working together efficiently and in harmony, it does make sense for each team to appropriately address all concerns. The anesthesia teams should always been seen as willing participants in any effort to improve OR efficiency and surgical/patient satisfaction.

Chapter 45
Anaphylaxis Reactions

Sun Choe Daly

Case A: A Case of Intraoperative Anaphylaxis to Isosulfan Blue

The patient is a 76-year-old male with multiple medical problems who presents for wide excision of T4a melanoma (>4-mm thickness without ulceration) from the left lateral chest wall and sentinel lymph node biopsy of the left axilla. His past medical history was significant for coronary artery disease, hypertension, hyperlipidemia, gastroesophageal reflux disease, degenerative joint disease in the left hip, and chronic low back pain. His past surgical history was significant for coronary artery bypass grafting, total hip arthroplasty, and excisional biopsy of his melanoma. The patient reported multiple allergies to medications including penicillin, clindamycin, and tetracyclines, which caused hives and rash. He reported that five different statin medications caused throat swelling, shortness of breath, and muscle pain. Morphine and vicodin caused hallucinations, and he reported nausea and vomiting as reactions to tramadol and oxycodone.

The patient was induced with etomidate 20 mg and propofol 250 mg, and a laryngeal mask airway was placed easily. Maintenance anesthesia was provided with a mixture of 50 % nitrous oxide and 1 % sevoflurane. The patient was hemodynamically stable with blood pressure (BP) ~140/90 mmHg, heart rate (HR) ~60 beats per minute (bpm), and oxygen saturation (SpO_2) 98–100 % while spontaneously ventilating with tidal volumes ~500 mL.

Three minutes after subcutaneous injection with 5 mL of 1 % isosulfan blue, the patient's SpO_2 was noted to be 81 % (**L-1**) and the patient's breathing appeared to become labored with end-tidal CO_2 values decreasing to ~20 mmHg (**L-2**). The pulse oximetry monitor was replaced due to possible monitoring artifact (**L-3**) without

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change in saturation. Concomitantly the patient became progressively hypotensive with blood pressure decreasing to ~70/40 mmHg and ephedrine 10 mg was given. The patient was intubated for definitive airway management, and despite aggressive vasopressor support with phenylephrine, epinephrine, and positioning in Trendelenburg position, his hypotension worsened and progressed to pulseless electrical activity. Chest compressions were initiated, and epinephrine, vasopressin, diphenhydramine, ranitidine, and hydrocortisone were administered for presumed diagnosis of anaphylaxis (**L-4**). A raised erythematous rash (**L-5**) was noted on the patient's chest and neck. After 2 min of chest compressions, the patient's hemodynamics improved with return of palpable pulses. A second intravenous line and a right femoral arterial line were placed. The patient received 2 L of normal saline, and an epinephrine infusion was initiated (**L-6**). Arterial blood gas examination demonstrated metabolic acidosis with pH of 7.28, and a tryptase level (**L-7**) was drawn. The patient was transported to the intensive care unit sedated and intubated. Another tryptase level was drawn 3 h later. The patient was extubated later in the same day once extubation criteria were met. The patient had no apparent sequelae from his arrest and was discharged to home on postoperative day 1. Fifteen days later the tryptase levels returned and the initial value was elevated at 43 ng/mL and the second value was 30 ng/mL (normal range is 2–13 ng/mL).

Case B: A Case of Anaphylactoid Reaction to Cefazolin

The patient is a 25-year-old female seen for open reduction and internal fixation of her left ankle. She weighed 73 kg and was 69 in. tall. Her past medical history was significant for occasional migraine headaches and *Clostridium difficile* colitis. Her surgical history was significant for wisdom teeth extraction with local anesthesia and sedation. She denied any drug allergies.

Prior to arriving in the operating room, the patient received a continuous popliteal nerve block with catheter placement. The patient was induced with fentanyl 100 mcg, lidocaine 100 mg, and propofol 200 mg, and a laryngeal mask airway (LMA) was placed easily. Maintenance anesthesia was provided with 50 % nitrous oxide and 0.95 % sevoflurane. Cefazolin 1 g was administered prior to incision. The patient was mildly hypotensive with blood pressure ~80/40 and heart rate (HR) ~80 beats per minute (bpm). Phenylephrine was administered without change in blood pressure (BP) over the next 15 min. Over the next 5 min, her hypotension worsened to ~70/30 mmHg and her heart rate increased to ~100 bpm despite colloid bolus administration (**L-8**). Vasopressin two units were administered and had a mild effect, returning her BP to ~80/40 mmHg, but the patient continued to be tachycardic. A diffuse rash was noted, and on auscultation of lung fields, clear breath sounds were noted. The patient was intubated for definitive airway management, and she was administered diphenhydramine, famotidine, hydrocortisone, and epinephrine for presumed diagnosis of anaphylaxis vs anaphylactoid reaction (**L-9**, **L-10**).

Lessons Learned

L-1: Why does decreased cardiac output cause a decrease in arterial oxygen content? See Figs. 45.1 and 45.2.

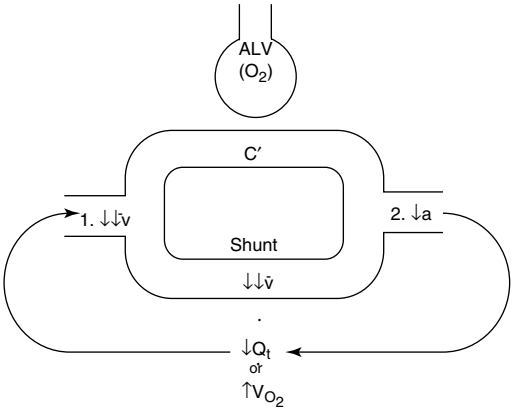

Fig. 45.1 Effect of a decrease in cardiac output or an increase in oxygen consumption on mixed venous and arterial oxygen contents. Mixed venous blood ($\bar{v}$) perfuses either ventilated alveolar (ALV 0_2) capillaries and becomes oxygenated end-pulmonary capillary blood (C′) or perfuses whatever true shunt pathways exist and remains the same in composition (desaturated). These two pathways must ultimately join together to form mixed arterial (a) blood. If the cardiac output ($\dot{Q}_t$) decreases and/or the oxygen consumption ($\dot{V}_{O_2}$) increases, the tissues must extract more oxygen per unit volume of blood than under normal conditions. Thus the primary effect of a decrease in $\dot{Q}_t$ or an increase in $\dot{V}_{O_2}$ is a decrease in mixed venous oxygen content. The mixed venous blood with a decreased oxygen content must flow through the shunt pathway as before (which may remain constant in size) and lower the arterial content of oxygen. Thus, the secondary effect of a decrease in $\dot{Q}_t$ or an increase in $\dot{V}_{O_2}$ is a decrease in arterial oxygen content

L-2: Why does decrease in cardiac output cause a decrease in arterial $P_{ET}CO_2$?

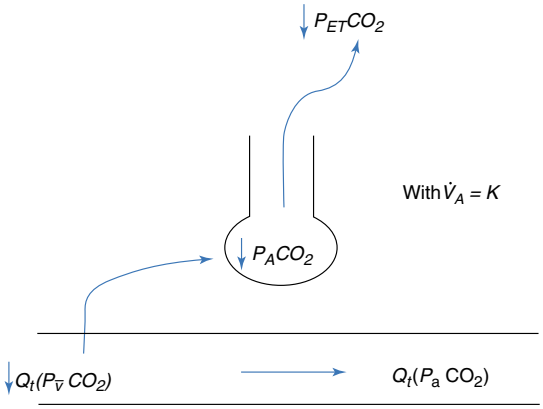

Fig. 45.2 Cause of decreased end-tidal CO_2 with decreased cardiac output. A sudden decrease in cardiac output causes a decrease in the supply of CO_2 to alveolar space. With alveolar ventilation maintained at a constant, acutely there is 1:1 decrease in partial pressure of end-tidal CO_2 with a given decrease in cardiac output. $P_{ET}CO_2$ partial pressure of end-tidal carbon dioxide, Qt cardiac output, $P_{\bar{v}}CO_2$ partial pressure of mixed venous carbon dioxide, P_aCO_2 partial pressure of arterial carbon dioxide, $\dot{V}_A$ alveolar ventilation, K constant [1]

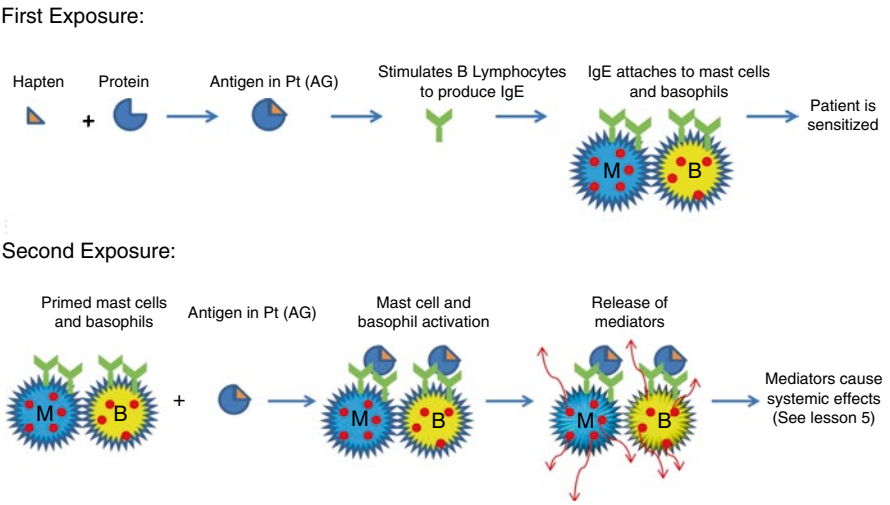

Fig. 45.3 Mechanism of anaphylaxis

L-3: What are the causes of erroneous SpO_2 readings?

1. Motion
2. Position
3. Color confusion
   (a) Nail color
   (b) Dyes
   (c) Probe placed over very dark skin (black or deep yellow)
4. Electrical radiation and external light
5. Hemoglobinopathy
   (a) Carboxyhemoglobin—pure solution reads 100 %.
   (b) Methemoglobin—pure solution reads 85 %.
6. Decreased pulse for any reason

L-4: What is an anaphylactic reaction?

Anaphylaxis is a type I immunoglobulin E (IgE)-mediated hypersensitivity reaction. There are two stages that are necessary to have this response. In the first exposure, a hapten, a small molecule, becomes an antigen after binding with a protein. The body will mount an immune response and form IgE antibodies to this antigen. In the next phase, the patient will have re-exposure to the same hapten, which will cause a cross-linking of IgE antibodies expressed on mast cells and basophils, causing an explosive release of mediators with systemic effects. The mediators released are prostaglandins, leukotrienes, histamine, proteoglycans, and proteases (Figs. 45.3 and 45.4).

L-5: What is the pathophysiology of anaphylactic and anaphylactoid reactions?

See **L-10** for discussion.

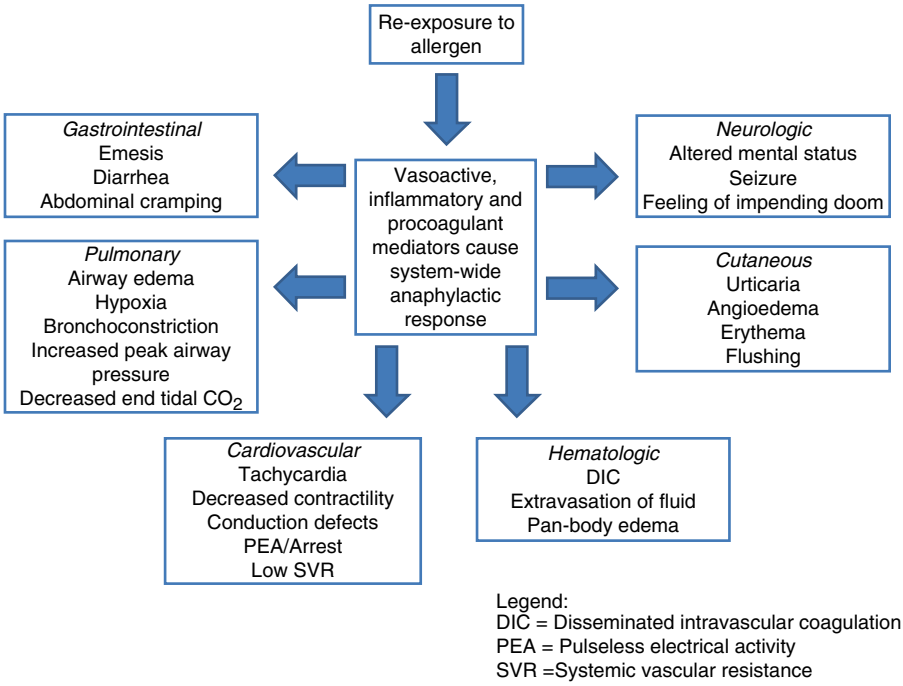

Fig. 45.4 Classic signs and symptoms of anaphylaxis [2]

L-6: **What is the treatment for anaphylaxis?**
See Table 45.1

L-7: **What do tryptase levels mean?**
Tryptase is a mast cell protease that is released by mast cells when activated and may serve as a marker of anaphylaxis. This can be measured in plasma 30 min after the initial signs and symptoms of anaphylaxis. It has a half-life of 2 h and can be elevated for hours or days, depending on the duration of the anaphylactic reaction. The tryptase will remain elevated, and the plasma levels correlate with the severity of the response of mast cells. Although this is a good marker and may possibly aid in the diagnosis of anaphylaxis, it may be misleading since direct mast cell activation (i.e., anaphylactoid reaction) will also cause an increase in tryptase levels. You may also have IgE-mediated anaphylaxis with an absence in rise of tryptase levels according to some reports. You may not use tryptase levels to distinguish between anaphylaxis and anaphylactoid reactions. Measurement of IgE to certain antigens with *in vitro* studies such as radioallergosorbent test (RAST) may be performed to diagnose anaphylaxis (see Table 45.1 [2]).

L-8: **What is the fluid deficit in anaphylaxis?**
In a recent case report of a patient who experienced anaphylaxis while on cardiopulmonary bypass, it was estimated that 73 % of her intravascular volume was lost in 15 min [3]. Other older studies report a more modest with loss of 35 % loss of intravascular volume in the first 10 min of anaphylaxis [4].

Table 45.1 Treatment guide for perioperative anaphylaxis

Management	Action
Antigen exposure	Discontinue offending drug
Airway	Endotracheal intubation
Hypoxia	Provide 100 % oxygen
Vasodilation and extravasation	IV fluids, 35–75 % of intravascular volume may be needed
Hypotension	Epinephrine, vasopressin
Bronchoconstriction	Epinephrine, albuterol, ipratropium bromide
Cardiac arrhythmias	Defibrillation, antiarrhythmics, i.e., amiodarone 300 mg IV
Block histamine receptors	Diphenhydramine (H1) 25–50 mg and ranitidine (H2) 150-mg bolus or cimetidine (H2) 400-mg bolus,
Prevent late and delayed symptoms	0.5–1.0-mg/kg methylprednisolone, 1–5-mg/kg hydrocortisone
DIC	FFP and/or pRBCs as needed

Adapted from Hepner and Castells [2]

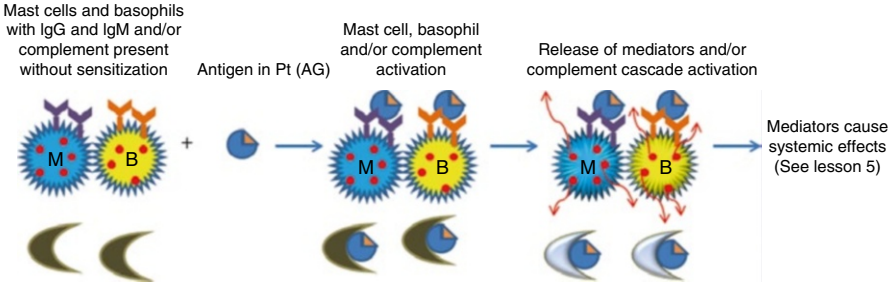

Fig. 45.5 Mechanism of anaphylactoid reaction

L-9: **What is an anaphylactoid reaction?**
An anaphylactoid reaction is a reaction that appears similar to anaphylaxis, but the mechanism is not driven by IgE antibodies. A sensitization phase is not necessary since it is not driven by IgE cross-linking. It is caused by direct mast cell and/or basophil degranulation and/or complement activation (Fig. 45.5).

L-10: **Can you tell the difference between anaphylaxis and anaphylactoid reactions based on signs and symptoms?**
No, these two syndromes are clinically indistinguishable.

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Chapter 46
Autonomic Dysreflexia

Christopher Edwards

The patient is a 46-year-old male with a past medical history of a T3–T4 paraplegia scheduled to have an elective colostomy. The original injury was secondary to a motorcycle accident. After the accident, he had a T1–L1 posterior spinal fusion. He subsequently developed neurogenic bowel and bladder and significant lower extremity spasticity. His history was also significant for an episode of autonomic dysreflexia (**L-1**) secondary to constipation. The patient was brought to the operating for the scheduled colostomy. The anesthetic plan was to perform a spinal anesthetic (**L-2**) with 15 mg of 0.75 % hyperbaric Marcaine supplemented with light sedation including midazolam and fentanyl. The spinal anesthetic was placed at the L4–5 interspace without incident. The patient was then positioned; an appropriate level (write-up does not specify a pre-incision level) was achieved and surgery started. Initial blood pressures were noted to be 90–100 mmHg/60–70 mmHg; after incision, blood pressure started to rise, peaking at 180–220 mmHg/100–120 mmHg. The heart rate also started to fall, initially 75–80 bpm, to a low of 60 bpm. Initial measures to control blood pressure consisted of cessation of all surgical stimulation: placement of a 1-in. patch of nitroglycerin paste and small divided 20 mcg boluses of nitroprusside. These measures had little effect on the blood pressure, and despite the fact that the patient was asymptomatic, it was decided to convert to a general anesthetic. The patient was intubated without difficulty; high-dose desflurane and propofol were used to control blood pressure, which had slowly drifted back to 100/60 mmHg. When the surgery was finished, the patient was taken to the postanesthesia care unit extubated and following commands. His only complaint was a mild headache.

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Lessons Learned

L-1: **What is autonomic dysreflexia**?
To fully understand what autonomic dysreflexia (AD) is and why it occurs, a firm understanding of the anatomy and function of the autonomic nervous system is required. The autonomic nervous system is comprised of two separate systems, the sympathetic and the parasympathetic nervous system. The sympathetic nervous system (SNS) is responsible for the fight and flight bodily functions. The SNS is mediated by the sympathetic chain which is primarily thoracolumbar. The sympathetic chain typically runs from the first or second thoracic level to the second or third lumbar level. The main function is to prepare the body to respond to a stressful stimulus such as pain; common responses are tachycardia, hypertension, and bronchodilation. The parasympathetic nervous system is anatomically arranged in a cranial-sacral fashion. Cranial nerves III, VII, IX, and X, along with sacral nerves, mediate the parasympathetic response. The vagus nerve (cranial nerve X) mediates roughly 75 % of the parasympathetic response. The parasympathetic response typically involves bradycardia and bronchoconstriction. These two systems integrate to maintain homeostasis.

A stimulus such as pain from a surgical incision sends signals via afferent pain fibers to the spinal cord (see Figs. 46.1 and 46.2). The signal then ascends in the spinal cord, integrates in the brainstem, and sends out an appropriate efferent response via the autonomic nervous system. This normal arch of afferent and efferent fibers maintains appropriate hemodynamic parameters from various somatic and visceral stimuli. In a patient with a spinal cord injury, the development of autonomic dysreflexia is highly dependent on the level of injury. Injury to the spinal cord above the level of T6 typically correlates with an 85 % chance of development of AD. The physiologic explanation lies in the innervation of the splanchnic circulation and in part secondary to adrenal gland innervation. The splanchnic circulation typically receives 20–25 % of cardiac output and at any given time roughly holds 25 % of total blood volume. The splanchnic bed typically receives innervation from T5–T7 (greater splanchnic nerve), T10–T11 (lesser splanchnic nerve), and T12 (least splanchnic nerve). When the level of injury is above T6 and the patient experiences a noxious stimulus below T6, the afferent stimulus is blocked from ascending in the spinal cord. A spinal reflex arch below the level of spinal cord injury predominates and an unopposed sympathetic response leads to vasoconstriction. The sympathetic response below the spinal cord lesion is unopposed because the descending parasympathetic inhibitory modulation is anatomically blocked (see red X in Fig. 46.2). In addition, the sympathetic nervous system outflow below the level of spinal cord injury also stimulates the adrenal medulla to release excess catecholamines (see 2c in Fig. 46.2c). The unopposed vasoconstriction leads to systemic hypertension, which is sensed by the carotid and aortic baroreceptors. Both a reflex bradycardia and vasodilation above the spinal cord lesion occur in an attempt to control blood pressure. In addition to the hypertension, other common history and physical exam findings during an episode of AD are as follows: sweating above the level of spinal cord injury, nasal congestion, headaches, visual

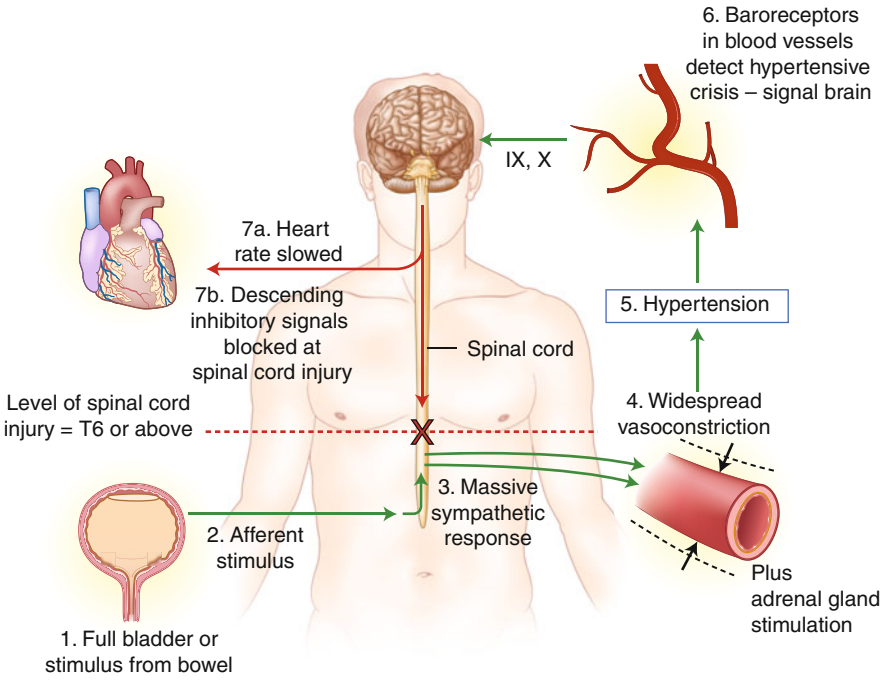

Fig. 46.1 Schematic showing response during autonomic dysreflexia (Adapted from Blackmer [3])

disturbances, and flushing of the skin. If left untreated, catastrophic outcomes including cerebral hemorrhage, myocardial infarction, seizures, arrhythmias, and even death have occurred.

L-2: **Why did autonomic dysreflexia occur in this case**?
As mentioned above, AD occurs from a stimulus such as incisional pain or visceral organ distention and that is the reason why patients with spinal cord injuries still require anesthesia despite having no sensation. Why did AD occur in a case where a spinal anesthetic was used?

(a) *Inadequate anesthetic level*: Determining the level of a spinal anesthetic in a patient with a spinal cord injury can be difficult. Nonetheless achieving an adequate level is necessary in all patients. There are a multitude of reasons why failure to achieve a proper level may occur. The principal reason why it may be difficult to assess a proper level in a patient with spinal cord injury is the *gray zone*. The *gray zone* is a dermatomal area where normal sensation and function may merge with areas of decreased or no sensation or function. This makes determining an adequate anesthetic level, which may also be associated with another superimposed gray zone challenging (Table 46.1).
(b) *Connective tissue septae*: Both the epidural space and subarachnoid space are known to have trabeculations and septae that can on occasion "compartmentalize" the respective spaces. These connective tissue components have been studied in

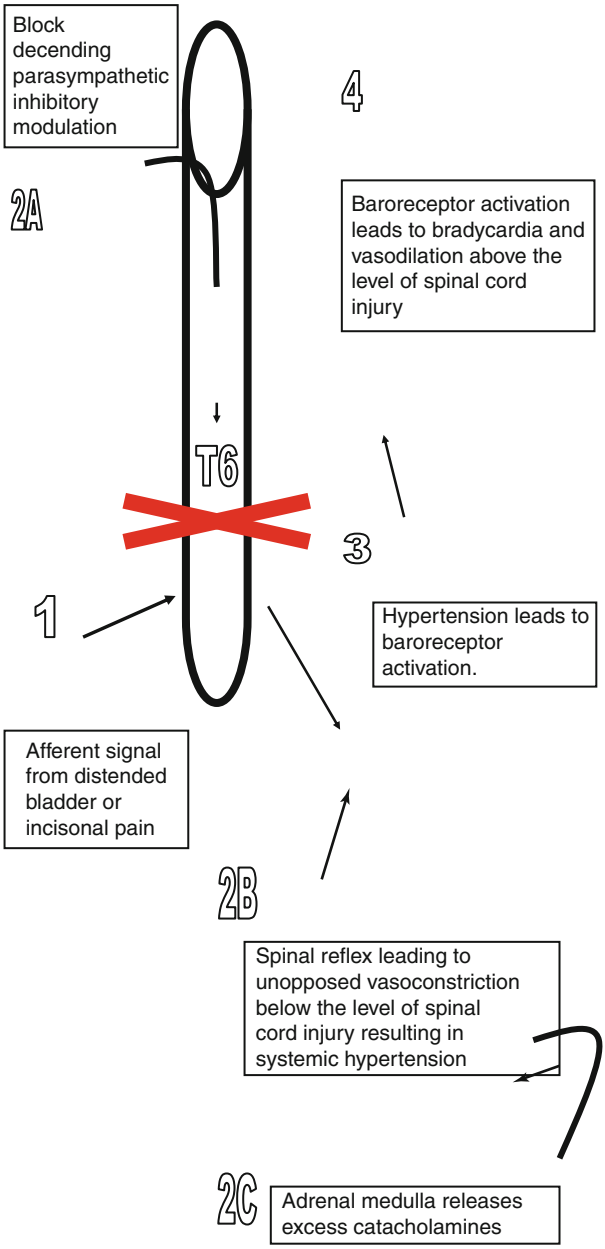

Fig. 46.2 Schematic of events that lead to the development of autonomic dysreflexia (note that "2" has three components: 2A, 2B, 2C)

Table 46.1 Factors that affect the height and length of a spinal anesthetic

Factor	Explanation of factor
Injury related	*Gray zone of partial loss of function*
Patient-related characteristics	Height, weight, gender, age, intra-abdominal pressure (pregnancy), anatomic variations in the spinal column (see B)
Technique	Site of injection, direction of needle, location of needle, rate of injection
Spinal fluid	Volume of spinal fluid, CSF pressure, CSF density
Anesthetic drugs	*Gray zone of anesthetic level*, density, mass of drug, concentration, volume, addition of vasoconstrictors, bad batch of drug (outdated or possible drug swap)

Adapted from Birnbach and Browne [2]

Table 46.2 Antihypertensive agents (all doses are intravenous form except volatile agent)

Agent	Onset	Duration	Dose	HR	Afterload
Nitroglycerin	1 min	5 min	50–100 mcg	↑	↓↓
Nitroprusside	1 min	5 min	50–100 mcg	↑↑	↓↓↓
Hydralazine	10 min	2–4 h	5–20 mg	↑↑↑	↓↓↓
Phentolamine	2 min	15 min	1–5 mg	↑↑	↓↓
Volatile agent	Rapid	Varies	1–2 mac	↑↑	↓↓↓
Propofol	1–2 min	10–15 min	Varies	↑↑	↓↓↓

Adapted from Birnbach and Browne [2]

human cadavers and are fairly inconsistent between humans. The connective tissue in the subarachnoid space has fenestrations allowing fluid to flow freely between compartments. This may be one reason why spinal anesthetics are more reliable than epidural anesthetics. The leading theory as to why humans have these septae is that they provide spinal cord protection, may limit the spread of infection, and structurally support nerves and blood vessels. At present time, there does not appear to be a reliable or practical test to detect these septae. History of a failed, patchy, or one-sided spinal anesthetic may help to guide future neuraxial anesthetics. [1]

L-3: **Treatment of autonomic dysreflexia**

The treatment goal of AD is to return blood pressure to a normal range. This needs to be done in a controlled fashion as these patients have very labile blood pressures during these episodes. The first measure should be to stop the inciting event, whether that is a painful incision or a distended bladder. If feasible, placing the patient in the upright position can help to lower the blood pressure; however, pharmacologic intervention will likely be required. There are many options for blood pressure control, but the overriding theme should be *slow titration of rapid-onset and short-duration medications*. There are many drugs that satisfy these goals; some are anesthetics while others are hypotensive agents. The choice of drugs will depend on the clinical context in which AD develops. If it occurs in the operating room, there are far more options than if the episode occurs in clinic. Table 46.2 provides a list of commonly used medications that are used to treat AD.

Other drugs that have fallen out of current clinical practice include the alpha-receptor antagonist phenoxybenzamine and ganglionic blocking drugs trimethaphan and mecamylamine. These medications have varying half-lives but in general are not ideal for rapid titration on and off. Understanding these medications and how to properly administer them in a timely manner during an episode of AD can be the difference between a good and disastrous outcome.

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Chapter 47
Porphyrias

Bahareh Khatibi

The patient was a 62-year-old female with pulmonary hypertension scheduled for pulmonary thromboendarterectomy (PTE). Medical history was also significant for acute intermittent porphyria **(L-1, L-2)** manifested by abdominal pain.

General anesthesia was induced with 2,500 mcg fentanyl, 100 mg lidocaine, and 10 mg vecuronium. Approximately 30 s later, the patient had what appeared to be a tonic-clonic seizure. Mask ventilation continued and seizure activity abated. After intubation, 1.5 % isoflurane was started, which normalized the patient's initial tachycardia and hypertension. Electrolyte and EEG analysis were normal. The remainder of the case was uneventful.

Lessons Learned

L-1: **What are porphyrias**? **What is the cause**? **What are the signs and symptoms**?

(a) Porphyrias are a group of inborn errors of metabolism characterized by the overproduction of porphyrins and their precursors. Porphyrins are involved in a wide range of reactions related to oxygen utilization, transport, and storage [1].
(b) Any enzyme deficiency in the pathway of porphyrin production leads to the accumulation of pathway intermediaries, causing a porphyria. See Fig. 47.1. For example, heme is a porphyrin which binds proteins to form compounds that include hemoglobin and cytochrome P-450. Any increase in heme requirements stimulates the synthetic pathway of heme. If there is an enzyme deficiency in

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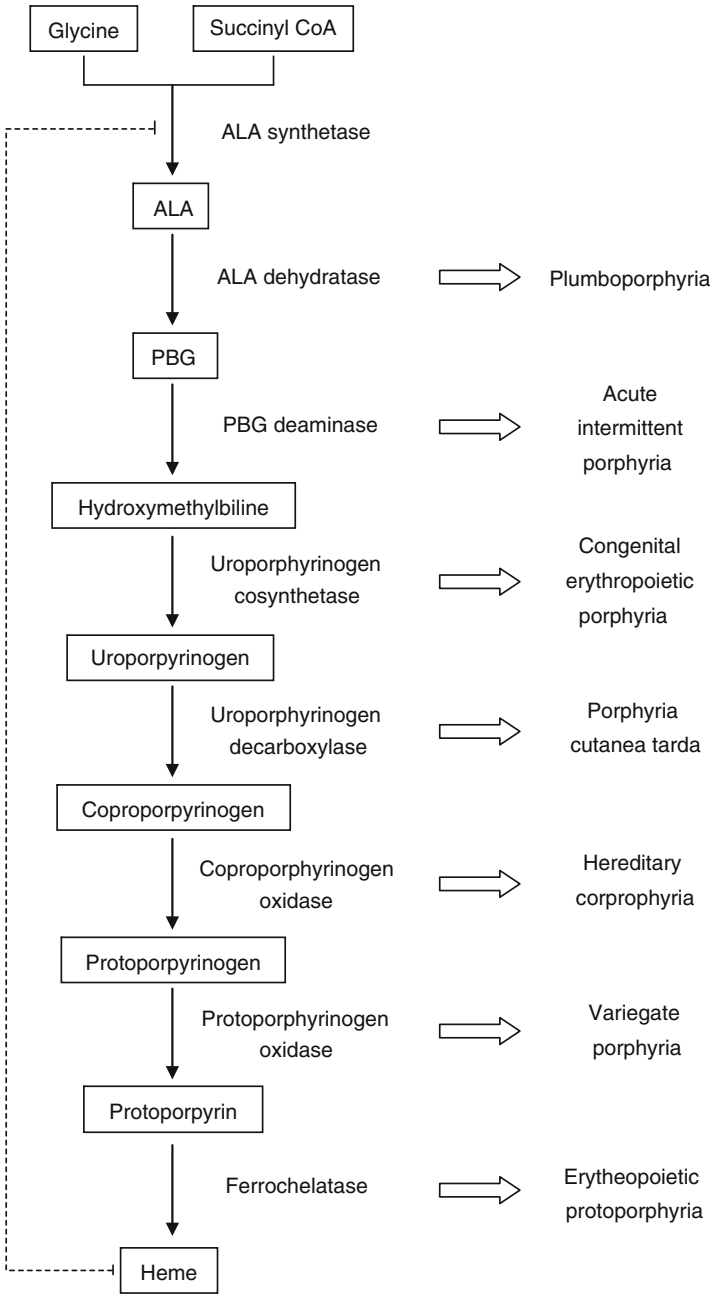

Fig. 47.1 Metabolic pathway for heme synthesis. The type of porphyria associated with the specific enzyme deficiency is noted at the right. *ALA* **δ**-aminolevulinic acid, *PBG* porphobilinogen (Adapted from James and Hift [1])

Table 47.1 Porphyrias and their disease-specific symptoms

Acute porphyrias (acute attacks can be precipitated; see text 1c)	
1. Acute intermittent porphyria	Hypertension and renal dysfunction; symptoms are usually severe
2. Variegate porphyria	Neurotoxicity and cutaneous photosensitivity
3. Hereditary coproporphyria	Neurotoxicity and cutaneous photosensitivity; symptoms are less severe than variegate porphyria
4. Plumboporphyria	Only neurological symptoms
Non-acute porphyrias (acute attacks do not occur)	
1. Porphyria cutanea tarda	Photosensitivity, hepatocellular necrosis
2. Erythropoietic uroporphyria	Hemolytic anemia, bone marrow suppression, and splenomegaly
3. Erythropoietic protoporphyria	Photosensitivity, vesicular cutaneous eruptions, urticaria, and edema

the pathway, intermediary substrates accumulate acutely causing an acute attack of porphyria.

(c) The signs and symptoms of porphyrias include severe abdominal pain, autonomic system instability, electrolyte disturbance, and neuropsychiatric manifestations, ranging from skeletal muscle weakness to seizures. Acute attacks may be precipitated by fasting, dehydration, stress, infection, menstruation, and certain drugs. Aside from the above signs and symptoms common in most porphyrias, each porphyria has specific signs and symptoms. See Table 47.1.

L-2: **What are the anesthetic considerations of porphyria**?

(a) Anesthesia has been implicated in triggering acute attacks of porphyria in susceptible patients. Only the acute forms of porphyria are affected by anesthetic drugs. Patients with active porphyria or a history of acute crisis must be considered increased risk for morbidity.

(b) Preoperatively

1. If an acute porphyria exacerbation is suspected preoperatively, it is important to note skeletal muscle strength and cranial nerve function to assess for the risk of respiratory failure and aspiration.
2. Minimize fasting if possible. Consider intravenous infusions of saline with glucose in the preoperative period if prolonged fasting is necessary.

(c) Intraoperatively

1. It is not possible to predict which drugs will precipitate an attack, but drugs have been labeled as safe or unsafe based on anecdotal reports of triggering acute attacks. Safety information continues to be updated.
2. Specific drugs

All barbiturates, including thiopental, thiamylal, and methohexital, are considered unsafe in patients with porphyria.

Halothane and enflurane are considered unsafe.

Animal studies suggest that etomidate is a trigger for acute porphyria, despite its reported safe use in patients.

Propofol has been used safely for induction, although safety of prolonged infusions is unknown.

Succinylcholine and pancuronium are considered safe. Other muscle relaxants including rocuronium, vecuronium, and cisatracurium are unlikely to provoke an acute attack, but their safety has not been conclusively established.

3. General principles

Short-acting drugs are presumed to be safe because their rapid elimination limits the exposure time.

Exposure to multiple potential triggering agents is likely more dangerous than any single agent.

Regional anesthesia should probably be avoided in the setting of an acute porphyric attack since it may cloud the differentiation between regional anesthetic onset and porphyric neuropathy.

4. In this case, fentanyl induction is reasonable given that narcotics are regarded as safe in patients with porphyria. Propofol for induction is also a possibility if the patient could tolerate it hemodynamically. Lidocaine should be omitted given that there is conflicting data about the safety of various local anesthetics. Vecuronium is a reasonable choice for muscle relaxation, but pancuronium may be safer since there is more data on its safe use in patients. Maintenance with isoflurane is reasonable choice since modern inhaled anesthetics like sevoflurane, desflurane, and nitrous oxide have not been implicated in causing porphyric attacks.
5. Given the above, it would be speculation to opine on what caused the seizure in this case; theoretical candidates would be lidocaine and vecuronium.

(d) Postoperatively

1. Mechanical ventilation may be required during an acute crisis.

(e) Treatment of acute porphyric crises includes removal of the triggering factors. Ensure adequate hydration and carbohydrate intake. Should a seizure occur during an attack, treat with benzodiazepines or propofol, given that traditional anticonvulsants are inducers of ALA synthetase and may be triggering agents. Treatment of acute porphyria attacks with hematin (3–4 mg/kg iv over 20 min) is thought to suppress ALA synthetase activity, thus inhibiting the overproduction of pathway intermediaries in Fig. 47.1. Cimetidine is also recommended since it may decrease heme consumption and inhibit ALA synthetase activity.

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Chapter 48
Monitored Anesthesia Care (Medical Implications) and Wrong-Sided Operations (Legal Implications)

Erica K. Stary

A 77-year-old man presented to an outpatient surgical center for corneal transplant, cataract extraction, and intraocular lens implant of the left eye, under regional block plus monitored anesthesia care (MAC) (**L-1**, **L-2**). Past medical history included hiatal hernia, gastric reflux, and osteoarthritis of many joints, including the cervical spine. Medications included Hytrin, Prilosec, and aspirin (which was discontinued 1 week prior to surgery). Airway exam was remarkable for a Mallampati class III view, with extremely limited mobility of the cervical spine.

The patient was placed on the operating room bed, where an intravenous catheter and monitors were placed and oxygen was delivered by nasal cannula. Intravenous sedation was titrated to moderate sedation (**L-1**, **L-2**, **L-3**, **L-4**) to prepare for the infraorbital block and subsequent surgery and included fentanyl 50 mcg, midazolam 1 mg, and diphenhydramine 25 mg (**L-3**). The anesthesiologist begin to chart vital signs and when she looked up at the ophthalmologist, she noticed that he was injecting the right eye, although the consent read for surgery on the left (**L-5**). The anesthesiologist attempted to verbally stop the ophthalmologist, but it was too late. The surgery was aborted for that day, and the error was explained to the patient once he fully recovered from the intravenous medications given (**L-6**).

Lessons Learned

L-1: **Sedation levels and definitions**
Sedation and analgesia include a continuum of states of consciousness ranging from minimal sedation (anxiolysis) to general anesthesia (GA). See Table 48.1 for some of their differences [1].

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Table 48.1 Definitions of levels of sedation

	Minimal sedation (anxiolysis)	Moderate sedation (conscious sedation)	Deep sedation/ analgesia	General anesthesia
Response to stimulation	Normal response	Purposeful[a] response to verbal or tactile stimulation	Purposeful response after repeated or painful stimulation	Not arousable, even w/ painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous ventilation	Unaffected	Adequate	May be impaired	Frequently inadequate
Cardiovascular function	Unaffected	Usually maintained	Usually maintained	May be impaired
Who is allowed to perform?	MD, CRNA, AA, SN[b]	MD, CRNA, AA, SN[b]	MD, CRNA, AA[b]	Anesthesiologist, CRNA, AA[b]
Capnography needed?[c]	No	Yes	Yes	Yes

Defined and adopted by the American Society of Anesthesiologists [1]
MD medical doctor, *CRNA* certified registered nurse anesthetist, *AA* anesthesiologist assistant, *SN*/*SPA* sedation nurse/physician assistant (see Lesson 4 for description of these providers)
[a]Reflex withdrawal from painful stimulus is NOT considered a purposeful response
[b]Under the supervision of preferably an anesthesiologist but in some states any licensed MD
[c]See ASA basic monitoring standards below

From a medicolegal point of view, it is clear from Table 48.1 that a patient in deep sedation is hard to arouse, may have impaired spontaneous ventilation, must be monitored by capnography, and may need airway intervention, which only an anesthesiologist, certified registered nurse anesthetist (CRNA), or in some states an anesthesiologist assistant (AA) can successfully perform. For these reasons, a sedation nurse *cannot* perform deep sedation (or GA).

The American Society of Anesthesiologist's standards for basic anesthetic monitoring state that:

- Carbon dioxide capnography and pulse oximetry should be continuously monitored in moderate, deep, and general anesthesia.
- Every patient receiving anesthesia should have the electrocardiogram continuously displayed from the beginning of anesthesia until preparing to leave the anesthetizing location.
- Every patient receiving anesthesia should have arterial blood pressure and heart rate determined and evaluated at least every 5 min.
- Level of consciousness should be assessed at regular intervals throughout the sedation process, for all levels of sedation. Verbal stimuli should be used for moderate sedation, with more profound stimuli used for deep sedation [2].

L-2: **Monitored anesthesia care**

The American Society of Anesthesiologist's position on monitored anesthesia care is that it is clearly distinct from moderate sedation due to the expectations and

Table 48.2 Modified observer's assessment of alertness/sedation scale

Responsiveness	Score
Agitated	6
Responds readily to name spoken in normal tone (alert)	5
Lethargic response to name spoken in normal tone	4
Responds only after name is called loudly and/or repeatedly	3
Responds only after mild prodding or shaking	2
Does not respond to mild prodding or shaking	1
Does not respond to deep stimulus	0

qualifications of the provider who must be able to utilize all anesthesia resources to support life and to provide patient comfort and safety during a diagnostic or therapeutic procedure [3]. It includes all aspects of anesthesia care, including a preprocedure visit, intraprocedure care, and post-procedure anesthesia management. During monitored anesthesia care, the anesthesiologist *provides or medically directs* a number of specific services, including but not limited to:

- Diagnosis and treatment of clinical problems that occur during the procedure
- Support of vital functions
- Administration of sedatives, analgesics, hypnotics, anesthetic agents, or other medications as necessary for patient safety
- Psychological support and physical comfort
- Provision of other medical services as needed to complete the procedure safely

Monitored anesthesia care may include varying levels of sedation, analgesia, and anxiolysis as necessary. The provider must be *prepared and qualified* to convert to general anesthesia when necessary. If the patient loses consciousness and the ability to respond purposefully, the anesthesia care is a general anesthetic, irrespective of whether airway instrumentation is required.

L-3: **Sedation scales used outside the operating room** (**e.g.**, **ICU**)
There are several sedation scales and scoring systems that are used outside the operating room, which describe level of consciousness. The modified observer's assessment of alertness and sedation (MOAA/S) and the Ramsey Scale are currently used most often in clinical research and in the intensive care unit. However, these are not interchangeable with the ASA's definitions of levels of sedation, as they do not take into account cardiorespiratory status and there is some subjectivity as to what MOAA/S levels constitute moderate or deep sedation. For reference, these two scales are included below (Tables 48.2 and 48.3).

L-4: **Definitions of nonphysicians who provide sedation**
The American Society of Anesthesiologists (ASA) states, "All anesthetics should be delivered by or under the medical direction of an anesthesiologist." In the ASA's "Statement on Qualification of Anesthesia Providers in the Office-Based Setting" from 2009, they do acknowledge that Medicare and individual state's laws recognize this is not always possible. For this reason, they state that at minimum, a licensed physician must supervise anesthesia providers [4].

Virtually all states require direct physician participation in the care provided by nonphysician anesthesia providers, because they are not primarily trained to make

Table 48.3 Ramsey sedation scale

Responsiveness	Score
Patient is anxious and agitated or restless, or both	1
Patient is cooperative, oriented, and tranquil	2
Patient responds to commands only	3
Patient exhibits brisk response to light glabellar tap or loud auditory stimulus	4
Patient exhibits sluggish response to light glabellar tap or loud auditory stimulus	5
Patient exhibits no response	6

medical judgments. However, each state has its own set of regulations dictating the specific requirements and scope of practice of nurse anesthetists. States that license anesthesiologist assistants and sedation nurses require direct anesthesiologist participation in the care provided by them. Sedation nurses are only allowed to perform minimal and moderate sedation under any circumstance.

Nurse anesthetist – a registered nurse who has satisfactorily completed an accredited nurse anesthesia training program.

Anesthesiologist assistant (AA) – a health professional who has satisfactorily completed an accredited anesthesiologist assistant training program granting a master's degree, has been certified by the National Commission for Certification of Anesthesiologist Assistants, and has been credentialed by the institution.

AAs currently work in 18 states.

The states in which AAs work by a license, regulation, and/or certification are Alabama, District of Columbia, Florida, Georgia, Kentucky, Missouri, New Mexico (university hospital settings), Ohio, Oklahoma, South Carolina, Vermont, and North Carolina.

The states in which AAs are granted practice privilege through physician delegation (meaning the anesthesiologist can delegate specific anesthesia tasks to an AA) are Colorado, Michigan, New Hampshire, Texas, and West Virginia.

Sedation nurse and sedation physician assistant – a licensed registered nurse, advanced practice nurse, or physician assistant (PA) who is trained in compliance with all relevant local, institutional, state, and/or national standards, policies, or guidelines to administer prescribed sedating and analgesic medications and monitor patients during minimal sedation ("anxiolysis") or moderate sedation ("conscious sedation"), but *not deeper levels of sedation* or general anesthesia. Sedation nurses and sedation physician assistants may only work under the direct supervision of a properly trained and privileged medical doctor (MD or DO). While a patient is sedated, the responsible doctor must be physically present and immediately available in the procedure suite.

L-5: **Wrong-side/wrong-site surgery**

The magnitude and potential implications of wrong-side/wrong-site (WSS) surgery should not be understated. They can be devastating and life-altering for the patient and damaging to the provider's career as well. State licensure boards are imposing penalties on surgeons for WSS, and some insurers have decided to no longer pay providers for WSS or wrong-person surgery nor for leaving a foreign object in a

patient's body after surgery. According to the Ophthalmic Risk Management Digest and the American Academy of Orthopedic Surgeons, as of 2006, 79 % of wrong-site eye surgery and 84 % of wrong-site orthopedic claims resulted in malpractice awards.

It is difficult to ascertain how often these events actually occur, especially since the Joint Commission historically states reporting these events is voluntary. Overall, the Joint Commission reports a low incidence, although there is a wide variety in the numbers they report: 1 out of 27,686 cases, or 1 out of every 112,994 surgeries, or 1 in 5 hand surgeons during their career, or 1 out of 4 orthopedic surgeons with 25 years' experience. In an article published by the Joint Commission in February of 2012, they write that the national incidence rate, which includes wrong-patient, wrong-procedure, wrong-site, and wrong-side surgery, is as high as 40 incidents per week in the United States [5].

The Joint Commission has identified risk factors for these preventable occurrences, which include emergency operations, unusual time pressures to start or complete the procedure, and involvement of multiple surgeons or multiple procedures in a single surgical visit. However, this list is not all encompassing, and there have been errors made during cases where none of the above variables came into play.

In response to the occurrence of these errors, the Joint Commission implemented the "Universal Protocol for Preventing Wrong Site, Wrong Procedure and Wrong Person Surgery" in 2004, with revisions in 2010, for all accredited hospitals, ambulatory care centers, and office-based surgery facilities [6]. This protocol has three components: a preprocedure verification, site marking, and a "time-out."

1. **Preoperative verification process**

   Verification of the correct person, procedure, and site should occur (as applicable):

   - At the time the surgery/procedure is scheduled
   - At the time of admission or entry into the facility
   - Anytime the responsibility for care of the patient is transferred to another caregiver
   - With the patient involved, awake, and aware, if possible
   - Before the patient leaves the preoperative area or enters the procedure/surgical room

   A preoperative verification checklist may be helpful to ensure availability and review of the following, prior to the start of the procedure:

   - Relevant documentation (e.g., history and physical, consent)
   - Relevant images, properly labeled and displayed
   - Any required implants and special equipment

2. **Marking the operative site**

   - Make the mark at or near the incision site. *do not* mark any nonoperative site(s) unless necessary for some other aspect of care.

- The mark must be unambiguous (e.g., use initials; "X" may be ambiguous).
- The mark must be positioned to be visible after the patient is prepped and draped.
- The mark must be made using a marker that is sufficiently permanent to remain visible after completion of the skin prep. Adhesive site markers should not be used as the sole means of marking the site.
- The method of marking and type of mark should be consistent throughout the organization.
- At a minimum, mark all cases involving laterality, multiple structures (fingers, toes, lesions), or multiple levels (spine). Note: In addition to preoperative skin marking of the general spinal region, special intraoperative radiographic techniques are used for marking the exact vertebral level.
- The person performing the procedure should do the site marking.
- Marking must take place with the patient involved, awake, and aware, if possible.
- Final verification of the site mark must take place during the "time-out."
- A defined procedure must be in place for patients who refuse site marking.

Exceptions:

- Single organ cases (e.g., Cesarean section, cardiac surgery)
- Bilateral surgeries (i.e., tonsils, ovaries)
- Interventional cases for which the catheter/instrument insertion site is not predetermined (e.g., cardiac catheterization)
- Teeth
- Mucosal surfaces or perineum
- Premature infants, for whom the mark may cause a permanent tattoo

3. **"Time-out" immediately before starting the procedure**

To be conducted in the location where the procedure will be done (i.e., operating room), just before starting the procedure. A procedure is not started until all questions or concerns are addressed. It must involve the entire operative team, use active communication, be briefly documented, and must, at the least, include:

- Correct patient identity.
- Correct side and site.
- Correct procedure to be done.
- If the person performing the procedure changes, another time-out needs to be performed before starting each procedure.

A comprehensive research study has not been executed in which the effects of the implementation of the "universal protocol" have been examined. Current studies that look at WSS are retrospective in nature and include only one to two types of surgical disciplines. One study done by Kwaan et al. looked at the incidence, characteristics, and causes of wrong-site surgeries from 1985 to 2005 at insured

institutions in the United States, then examined the presence and characteristics of site verification protocols [7]. Among 2,826,367 operations during the study period, 13 nonspine WSS cases were identified, which also had records available. They state that under optimal conditions, the Joint Commission's "universal protocol" might have prevented 8 (62 %) of 13 cases. In this study, the authors concluded that the "universal protocol" would not have prevented the remainder of the cases because of errors started in weeks before the surgery (e.g., wrong documentation, inaccurate labeling of radiological reports).

The "universal protocol" (i.e., preoperative verification, marking the operative site, and "time-out") has been in place for almost 20 years. Perhaps now that more cases of WSS are being reported since there are more avenues in which to do so, more comprehensive research could be carried out to examine the incidence of WSS and near misses and whether there is anything that can or should be added to the "universal protocol" to ensure even fewer errors.

L-6: **Requirements for a lawsuit to be judged in favor of the patient**
In order for a plaintiff patient to prevail in a medical malpractice lawsuit against a physician, the patient must show all of the following four requirements were met:

1. The medical doctor has a duty that the doctor owed to the patient.
2. The duty was breached.
3. The patient has suffered a significant injury.
4. The breach of duty caused the significant injury.

In the present case, the medical doctor had the duty to perform the regional anesthesia and surgery on the correct eye, as dictated by the consent. This duty was breached when the physician performed an infraorbital block on the incorrect eye. However, it can be argued that the patient did not suffer a *significant* injury from this error. He regained sensation and function of the eye, which was incorrectly blocked, and the medical error did not lead to long-term deficits or an inability of him to receive the correct procedure in the future. Thus, since one of the four requirements was missing, this patient would probably not prevail in a WSS lawsuit.

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Chapter 49
Diabetic Ketoacidosis in the Urgent Anesthesia Setting

Sameer J. Shah

The patient was a 53-year-old man with a history of hypertension, type II diabetes, hyperlipidemia, and a history of left below-the-knee amputation (BKA) who presented to the emergency department complaining of left knee pain for 3 days. He denied fevers or chills but had been experiencing nausea which he attributed to hydrocodone use. His medications also included insulin 70/30, atenolol, aspirin 81 mg, atorvastatin, metformin, and lisinopril. He had no allergies. He had no other surgical history other than the BKA. He also had a history of methamphetamine, alcohol, and marijuana abuse.

Physical examination revealed a normally developed man weighing 95 kg, 5 ft 11 in. tall with a body mass index of 29.3. Intake vitals showed a BP of 98/58 mmHg, HR of 94 bpm, respiratory rate of 18 breaths per minute, temperature of 98.1 °F, and oxygen saturation (SpO_2) of 98 % breathing room air. The rest of the exam was suspicious for a septic joint. Preliminary labwork at 17:53 included a metabolic panel, which revealed the serum sodium (Na^+) to be 123 mEq/L, serum potassium (K^+) to be 3.6 mEq/L, serum chloride (Cl^-) to be 91 mEq/L, serum bicarbonate (HCO_3^-) to be 13 mEq/L, blood urea nitrogen (BUN) to be 10 mg/dL, serum creatinine (Cr) to be 0.44 mg/dL, serum glucose to be 388 mg/dL, serum calcium (Ca^{++}) to be 8.5 mg/dL, serum magnesium (Mg^{++}) to be 1.6 mg/dL, and serum phosphate (PO_4^{3-}) to be 1.4 mg/dL. Serial dilution of the patient's blood sample was positive for ketones at a ratio of 1:16. Serum lactate was found to be 5.8 mg/dL (see Table 49.1) (**L-1**).

The patient was started on vancomycin 1 g by the emergency room staff. A second set of labs including an ABG at 20:44 was drawn which showed a persistent metabolic anion gap (AG) acidosis and worsening hyperglycemia (**L-2**). An erythrocyte sediment rate and C-reactive protein returned elevated at 78 mm/h (normal = 0–20 mm/h) and 38.1 mg/dL (normal < 0.5 mg/dL), respectively. Given this

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Table 49.1 Normal laboratory values and units (*italics*); patient data over time

Time	Na^+ *135–145 mEq/L*	K^+ *3.5–5.0 mEq/L*	Cl^- *97–107 mEq/L*	HCO_3^- *22–29 mEq/L*	AG[a] *8–16 mEq/L*	BUN *8–18 mg/dL*	Cr *0.4–1.2 mg/dL*	Glucose *70–100 mg/dL*	Ca^{++} *8.5–10.4 mg/dL*
17:53	123	3.6	91	13	23	10	0.44	388	8.5
20:44	124	3.9	91	14	23	9	0.48	434	
00:05	127	3.0		13				360	
00:52	129	2.9	104	15	13				
01:00	127	3.1	97	14	19	7	0.40	272	7.6
01:34	127	2.8	108	18	4			224	

Time	Mg^{++} *1.7–2.6 mg/dL*	PO_4^{3-} *2.5–4.5 mg/dL*	Ketones negative	Lactate *4.5–19.8 mg/dL*	pH *7.35–7.46*	pCO_2 *36–46 mmHg*	pO_2 *74–109 mmHg*	BE *−3.3–1.2 mmol/L*	iCa^{++} 1.13–1.32 mmol/L
17:53	1.6	1.4	1:16	5.8					
20:44				5.8	7.29	26	82	−12.8	
00:05					7.33	26	243	−11.2	1.08
00:52					7.31	29	174	−10.3	1.02
01:00	2.0	2.3							
01:34					7.36	32	220	−7.0	1.00

[a]Anion gap (AG) = $(Na^+ + K^+) - (Cl^- + HCO_3^-)$

new data, the orthopedics team performed a joint aspiration, isolating 40,000 white blood cells per mm^3 and 26,000 red blood cells per mm^3 in the sample. The decision was made to take the patient to the operating room for an irrigation and debridement of the septic knee, and anesthesiology was called.

Up to this point, the patient had received a 500 cc bolus of normal saline (at 20:52), an insulin infusion at 10 units was started (at 21:47), piperacillin/tazobactam 4.5 g were given in addition to the vancomycin (at 21:53), and a normal saline infusion had been started at 150 cc/h (at 22:58). The patient was taken to the operating room at midnight. He was aggressively volume resuscitated and given supplemental magnesium 2 g and potassium phosphate 20 mEq. He was administered regular insulin 2 units IV and then started on an insulin infusion at 10 units/h (**L-3**). An arterial line and second IV were placed. A spinal anesthetic was chosen over general anesthesia given that the patient had recently eaten; 1.2 cc of bupivacaine 0.75 % administered intrathecally at the L3–L4 level resulted in a T10 level of dense anesthesia. Upon the start of the case, a baseline intraoperative arterial blood gas (ABG) was drawn. ABGs and repeat venous labwork were then sent approximately 60 and 90 min later (see Table 49.1).

The surgery proceeded smoothly; with episodic hypertension to systolic BP in the 160s mm Hg being treated with metoprolol and labetalol. A total of 2,500 mL of normal saline was given. In the recovery area, additional potassium phosphate 40 mEq was given. He was eventually admitted by the medicine service for management of diabetic ketoacidosis (DKA) and continued on an insulin infusion overnight.

Table 49.1 shows the normal ranges for the laboratories performed in the case, as well as our patient's values for comparison and trending over time.

Lessons Learned

L-1: **Stress in diabetics**: **the fundamental pathophysiology of DKA and HHS**
Acute complications in diabetic patients include diabetic ketoacidosis (DKA) and hyperglycemic, hyperosmolar states (HHS) such as hyperglycemic, hyperosmolar nonketotic coma. Simplistically, type I diabetics exhibit a propensity for DKA when their insulin requirement exceeds their exogenous insulin intake, and Type II diabetics are prone to HHS but are fairly ketosis resistant (see Table 49.2). There also exists a condition known as ketosis-prone type II diabetes mellitus, one of several

Table 49.2 Comparison chart for DKA and HHS

	DKA	HHS
Primarily affects?	Type I diabetic	Type II diabetic
Ketones?	+	–
Acidosis?	+	±
Hyperglycemia?	+	++
Hyperosmolality?	±	++

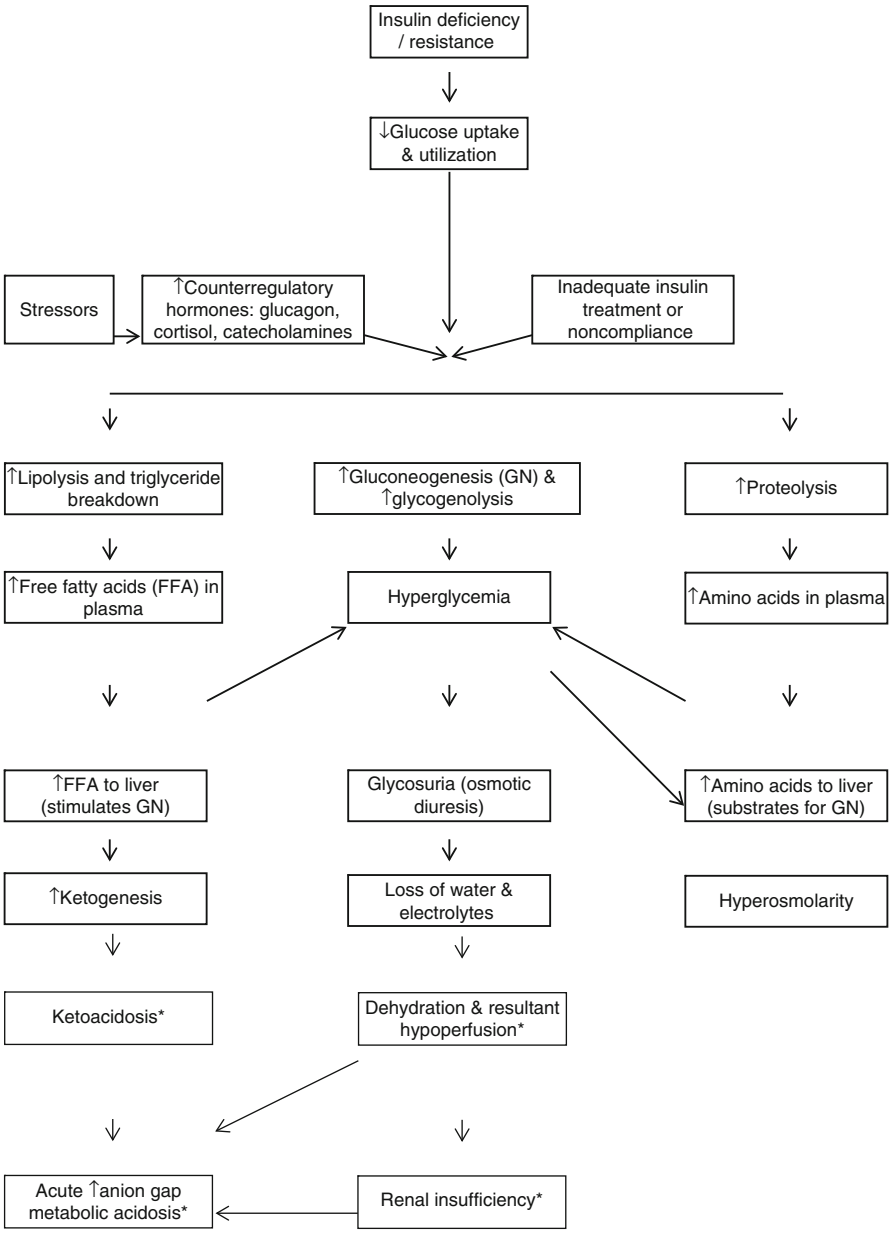

Fig. 49.1 The pathogenesis of DKA and HHS. Stressors result in increased counter regulatory hormones, which when added to baseline insulin deficiency or resistance result in ketoacidosis (in DKA), hyperglycemia, and hyper osmolarity

diabetes mellitus subtypes that does not fall neatly into the traditional dichotomy. Type II diabetics can be pushed into DKA during times of extreme stress (see Fig. 49.1).

Table 49.3 Diagnostic criteria in DKA and HHS

Finding	Mild DKA	Moderate DKA	Severe DKA	HHS
Plasma glucose (mg/dL)	>250	>250	>250	>600
Arterial pH	7.25–7.30	7.00–7.24	<7.00	>7.30
Serum HCO_3^- (mEq/L)	15–18	10–15	<10	>15
Serum or urine ketones	Positive	Positive	Positive	Small
Beta-hydroxybutyrate	High	High	High	Normal or high
Effective serum osmolality[a] (mOsm/kg)	Variable	Variable	Variable	>320
Anion gap[b]	Upper limits of normal or > normal	>Normal	Significantly > normal	Variable
Sensorium	Alert	Alert or drowsy	Stupor/coma	Stupor/coma

[a]Effective serum osmolality = 2 × measured Na^+ (mEq/L) + BUN (mg/dL)/2.8 + glucose (mg/dL)/18, >320–330 mOsm/kg associated with neurologic deterioration

[b]Anion gap = $(Na^+ + K^+) - (Cl^- + HCO_3^-)$

Common predisposing or precipitating factors in HHS and DKA include iatrogenic or patient-related causes (inadequate insulin treatment, noncompliance), a host of acute illnesses (a joint infection in our case), decompensated endocrine conditions (the most common of which is undiagnosed or new-onset diabetes), and myriad drugs.

*See **L-2**. Figure 49.1 illustrates how potentially three sources of acid (lactic acid, ketoacids, azotemic acids) can depress HCO_3^- and cause an increased anion gap.

L-2: Explanation and interpretation of laboratory values and anion gap in DKA

Patients in DKA or HHS present with a host of laboratory abnormalities. Total body stores of Na^+, Mg^{++}, Ca^{++}, PO_4^{3-}, and K^+ are generally depleted, with variable serum concentrations. Typical findings and treatment guidelines for electrolytes are covered in L-3 below. Table 49.3 summarizes diagnostic criteria in DKA and HHS.

Three ketone bodies are produced in DKA: two ketoacids (beta-hydroxybutyric acid and acetoacetic acid) and one neutral ketone (acetone). The production of ketoacids is one of the major factors leading to the hallmark anion gap metabolic acidosis seen in DKA and present in our patient. These patients can also have a metabolic acidosis from lactic acid production secondary to hypoperfusion and buildup of organic acids from renal insufficiency. Figure 49.2 reviews the approach to a patient with an acute metabolic acidosis.

L-3: The anesthesiologist's approach to the acutely uncontrolled diabetic patient

Several measures must be taken to see the decompensated diabetic patient through emergent surgery. Treating the intravascular volume depletion and metabolic perturbations associated with DKA and HHS can greatly reduce the incidence of intraoperative hemodynamic collapse and cardiac arrhythmias. Table 49.4 sums up the

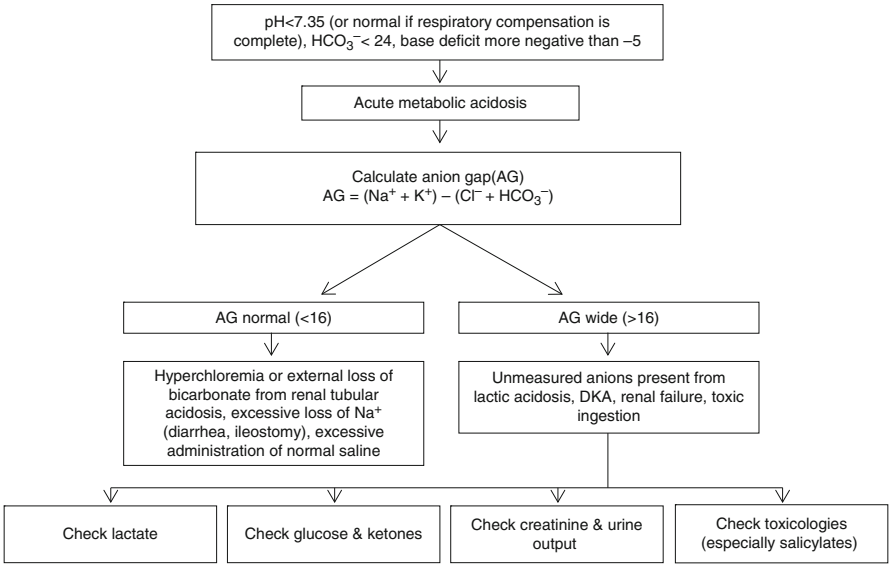

Fig. 49.2 Stepwise approach to diagnosing etiology of acute metabolic acidosis

Table 49.4 Basic treatment guidelines for perioperative HHS and DKA

General derangement	Specific guidelines
Hypovolemia	Use 0.9 % NS (if serum osmolality↑↑, consider .45 % NS)
	Rapid bolus followed by infusion at 250–1,000 cc/h
	Titrate to hemodynamic stability and UOP
	Caution in patients with depressed cardiac function
Electrolytes	Expect ↓Na^+, Mg^{++}, Ca^{++}, PO_4^{3-}, K^+
	Monitor q30–60 min
	Replace K^+ once UOP adequate and serum K^+ <5 mEq/L
Hyperglycemia	Start with regular insulin 10 units IV
	Infusion rate = (last serum glucose/100) units/h
	Add dextrose 5 % to fluids once glucose <250 mg/dL
Acidosis	Treat underlying DKA, hypovolemia, and renal failure
	Consider HCO_3^- if pH <7.15, hypotension refractory to volume

NS normal saline, *UOP* urine output

treatment guidelines. Treatment of hypovolemia should commence immediately. Fluid loss is from glycosuria and osmotic diuresis, and the patient generally loses free water in excess of electrolytes. While overall body Na^+ reserves are likely low, the serum Na+ concentration can be elevated because of this. A pseudohyponatremia from hyperglycemia (the plasma sodium concentration decreases by about

1.6 mEq/L for every 100-mg/dL increase in plasma glucose above normal) can also be seen, as with our case. Calculating free water deficit is of limited benefit. Volume replacement should begin rapid administration of normal saline (usually ~1 L) followed by a normal saline infusion at a rate of 250–1,000 mL/h, adjusting for observed degree of hypovolemia and the patient's cardiac function. The rate and duration of the infusion should be titrated to normalization of vital signs, urine output, and other measures of volume resuscitation.

Volume expansion and increased urine output will worsen the patient's deficiencies in Na^+, Mg^{++}, Ca^{++}, PO_4^{3-}, and K^+, and frequent monitoring of these values (every 30–60 min) is important. Electrolytes should be replaced as warranted. Routine PO_4^{3-} administration does not improve outcomes and may result in hypocalcemia, so generally PO_4^{3-} should be replaced if the serum level falls below 1 mg/dL. Of all these disturbances, hypokalemia is this most dangerous. Patients in DKA or HHS can initially present with hyperkalemia, though the serum K^+ will fall rapidly with hydration, insulin therapy, and correction of acidosis. Deficits in total body K^+ range from 3 to 10 mEq/kg body weight. K^+ should be started as soon as adequate urine output is confirmed and the serum K^+ level less than 5 mEq/L. Typically 20–30 mEq of K^+ is given for each liter of fluid replacement. If the serum K^+ is less than 3.3 mEq/L, K^+ replacement should be given immediately and insulin should be started only after the K^+ level is above 3.3 mEq/L.

Insulin treatment is initiated with a 10-unit (u) bolus of regular insulin IV, followed by an infusion of regular insulin IV. The rate can be calculated for a high-risk patient (patient with BMI >35 kg/m^2, on steroids, with active infection) by the following formula: (last serum glucose/100)=# u/h. For patients that do not qualify as "high risk," the following formula can be applied: (last serum glucose/150)=# u/h. An alternative approach is to start the infusion at 0.1 u/kg/h. Frequent monitoring (every 30–60 min) is important as the serum glucose may fall rapidly in the first 2 h of the infusion. Dextrose 5 % should be added to the IV solution once the serum glucose reaches 250 mg/dL.

Diabetics in crisis can have metabolic acidosis from multiple etiologies (see **L-2**). Generally, buffers such as HCO_3^- are not routinely administered unless the pH is less than 7.15 (some sources advise holding off even with pH as low as 6.9), the serum HCO_3^- is less than 10 mEq/L, or hypotension does not respond to rehydration. Ketoacidosis should improve with insulin therapy and decreasing glucose levels, and lactic acidosis from hypoperfusion should improve with volume resuscitation. Renal insufficiency and accumulation of organic acids, if primarily caused by a prerenal dehydration state, should also improve with volume resuscitation.

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Chapter 50
Fever, Altered Mental Status, and Rigidity in the Perioperative Course

Sameer J. Shah

The patient was a 67-year-old man who presented for a scheduled right above-the-knee amputation (AKA). The patient has a history of hypertension, peripheral vascular disease, chronic renal insufficiency, recent gastrointestinal bleed, 60-pack-year history of smoking, and recent onset of fine tremors and dementia. He had recently had a surgical site infection after a below-the-knee amputation (BKA). His medication list included aspirin, lisinopril, atorvastatin, bupropion, citalopram, Colace, heparin, hydrochlorothiazide, risperidone, acetaminophen, hydrocodone, ondansetron, and piperacillin/tazobactam. He was allergic to lorazepam, which caused psychosis.

Physical exam revealed a 98 kg, 5′10″ male. His airway exam showed a Mallampati class III airway, a thyromental distance of greater than 4 cm, full range of motion of the neck, and very poor dentition. He had a history of a difficult intubation requiring the use of the fiber-optic bronchoscope (FOB), but no other intraoperative complications. His preoperative labs were remarkable for a baseline hematocrit of 26.6 % and a serum creatinine of 1.51 mg/dL. Electrocardiogram (EKG) showed sinus tachycardia with occasional premature ventricular contractions.

The patient was taken to the operating room (OR) where routine monitors were placed. The patient was preoxygenated adequately, and an IV induction with etomidate and cisatracurium was performed. An asleep fiber-optic intubation through a laryngeal mask airway was performed. Anesthesia was maintained with a combination of nitrous oxide, sevoflurane, and fentanyl. The surgery proceeded uneventfully and the patient was extubated after full reversal of paralysis and return of spontaneous ventilation and consciousness.

The patient was taken to the recovery room at 13:30. At 14:50, he had a right femoral nerve and right sciatic nerve block placed for postoperative pain. Catheters were placed using ultrasound guidance, and infusions of ropivacaine were started. For the next several hours, he received multiple doses of promethazine for nausea.

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Table 50.1 Temperature and blood gas values over time

Time	Lab	pH	pCO_2 (mmHG)	pO_2 (mmHG)	BE (mmol/L)	Temp (°F)
18:35	VBG	7.28	36	38	−9.3	99.9
19:50	ABG	7.34	43	93	−2.9	102.8
21:40	ABG	7.34	45	113	−1.9	101.8
23:22	ABG	7.30	48	124	−3.2	101.5
03:00	ABG	7.39	37	115	−1.2	99.5
05:40	ABG	7.41	37	115	−1.2	100.1
08:00	ABG	7.43	34	124	−1.6	99.0

He also started become progressively more and more agitated with worsening tremors. Two doses of haloperidol 2.5 mg IV and one dose of midazolam 2 mg IV at 18:00 did not significantly improve the agitation or tremors.

At 18:30, the patient was noted to have muscle rigidity along with worsening agitation and tremors (**L-1**). A venous blood gas (VBG) was sent, along with a complete blood count and set of chemistries (see Table 50.1). An arterial line (A-line) was placed in the radial artery. The patient's blood pressure (BP) on the A-line tracings was in the 140s/90s mm Hg, his heart rate was 106 beats per minute (bpm), respiratory rate was in the high 20s breaths/min, oxygen saturation was 96 % on 2 L of oxygen per minute via nasal cannula, and his temperature was 99 °F at that time. The patient received diphenhydramine 100 mg IV, diazepam 5 mg IV, and acetaminophen 650 mg PR. Diazepam would only temporarily alleviate the tremors. At 19:50 an ABG was sent; pCO_2 was noted to be rising despite the tachypnea. The patient's temperature was now 102.1 °F, with a respiratory rate of 36 breaths/min. The patient was uncovered, a cooling fan was placed at the bedside, and a cooled IV saline infusion was started. Despite these cooling measures, the patient remained febrile with a temperature of 102–103 °F. A third ABG revealed persistent hypercapnia and acidosis. At this time, roughly 22:00, a bolus of dantrolene 1 mg/kg IV was given, followed by a dantrolene infusion at 1 mg/kg/h (**L-2**, **L-3**). The patient became calmer shortly after the administration of dantrolene, though still remained tachypneic, with a respiratory rate of 30 breaths/min. The decision was made to intubate him given the risk of respiratory failure from exhaustion. The patient was reintubated successfully after an IV induction with propofol and rocuronium and then taken to the intensive care unit (ICU).

In the ICU, the patient again appeared in extremis around 03:00, with a BP of 210/100, a heart rate in the 120s BPM, and temperature of 99.5 °F. Labetalol 10 mg IV was given and a repeat ABG was sent. CO_2 on the ABG was normal, however, and remained normal on surveillance ABGs throughout the rest of the morning. The patient's vitals normalized after labetalol administration, and he remained calm and stable up until extubation the late morning of the following day.

Lessons Learned

L-1: **Differential diagnosis for muscle rigidity and hyperthermia**
Several entities can mimic the constellation of symptoms seen in malignant hyperthermia (MH), which are detailed further in **L-2**. While MH was certainly suspected in our patient's particular case, it is useful for the anesthesiologist to have a working differential when muscle rigidity, hyperthermia, and markers of a hypermetabolic state are encountered. Table 50.2 compares two rare but fulminant conditions that can be confused for MH.

L-2: **Pathophysiology of malignant hyperthermia**
Malignant hyperthermia (MH) is a rare pharmacogenetic disease, passed down in an autosomal dominant fashion. Fifty to seventy percent of cases are due to a mutation of the ryanodine receptor in the sarcoplasmic reticulum of skeletal muscle cells. In normal patients, ryanodine receptors mediate release of calcium ions from the sarcoplasmic reticulum, a key step in the process of muscle contraction. A nerve impulse arrives at the motor end plate and triggers the release of acetylcholine (Ach) into the neuromuscular junction. Ach causes the depolarization of the muscle membrane; this in turn activates voltage sensors which then activate the ryanodine receptors. Ryanodine receptors allow the release of calcium ions stored within the sarcoplasmic reticulum, increasing the intracellular concentration of calcium. Calcium then initiates actin-myosin coupling and muscle contraction. This whole process, called excitation-contraction coupling, is dysregulated in MH. The mutated ryanodine receptors in MH allow the release of excessive amounts of calcium and are resistant to inactivation. Exposure to MH triggers (volatile agents, succinylcholine, or, rarely, high environmental heat or strenuous exercise) results in flooding of the skeletal muscle cell with high concentrations of calcium through the faulty ryanodine receptors. Pumps and exchangers work overtime to correct the increase in calcium, consuming ATP and producing heat. The inability of these mechanisms to lower the intracellular calcium contraction below the contractile threshold manifests as muscle rigidity and eventual breakdown from sustained contraction. Figure 50.1 illustrates the pathophysiology of MH in a stepwise fashion.

L-3: **Treatment algorithm for malignant hyperthermia**
The Malignant Hyperthermia Association of the United States (MHAUS) distributes a simple treatment algorithm, as shown in Table 50.3.

The Association of Anaesthetists of Great Britain and Ireland (AAGBI) expands on these steps in their treatment algorithm, as shown in Table 50.4.

Table 50.2 Comparison of conditions that result in muscle rigidity and hyperthermia

Disease/condition	Malignant hyperthermia	Neuroleptic malignant syndrome (NMS)	Malignant (lethal) catatonia
Pathophysiology	Dysregulated excitation-contraction coupling due to defective ryanodine receptors	Likely central dopamine blockade in hypothalamus and nigrostriatal pathways	Unknown; some correlation with ↓activity at GABA and D2 receptors and ↑activity at NMDA receptors in pathways that connect basal ganglia to thalamus and cortex
Triggers	Halogenated anesthetics, succinylcholine	Typical high-potency antipsychotics (e.g., haloperidol, fluphenazine) > atypical antipsychotics (e.g., olanzapine, risperidone, clozapine), antiemetics (e.g., metoclopramide, promethazine)	Unknown
Risk factors	Myopathies (central core, multicore, King-Denborough syndrome, Native American), history of heat- or stress-induced rhabdomyolysis, family history of MH	Certain psychiatric disorders, acute catatonia, extreme agitation, dehydration	Comorbid mood disorders or schizophrenia > other medical conditions
Presenting sign or symptom	Rise in $etCO_2$	Altered mental status	Usually preceded by behavioral prodrome of several weeks characterized by psychosis, agitation, catatonic excitement
Prominent signs and symptoms	Hypercapnia, mixed respiratory/metabolic acidosis, sustained muscle contraction including trismus, tachycardia, hyperthermia, hyperkalemia with arrhythmias, rhabdomyolysis	Muscle rigidity with superimposed tremor, hyperthermia, autonomic dysfunction with profound diaphoresis, ↑CK, leukocytosis, rhabdomyolysis, ↓ serum iron	"Positive" motor phenomena (dystonic posturing, repetitive movements), hyperthermia, rigidity, autonomic instability, ↑ CK, leukocytosis, ↓ serum iron (lab abnormalities generally less pronounced than NMS)

$etCO_2$ end-tidal CO_2, *CK* creatine kinase, *D2* dopaminergic type 2

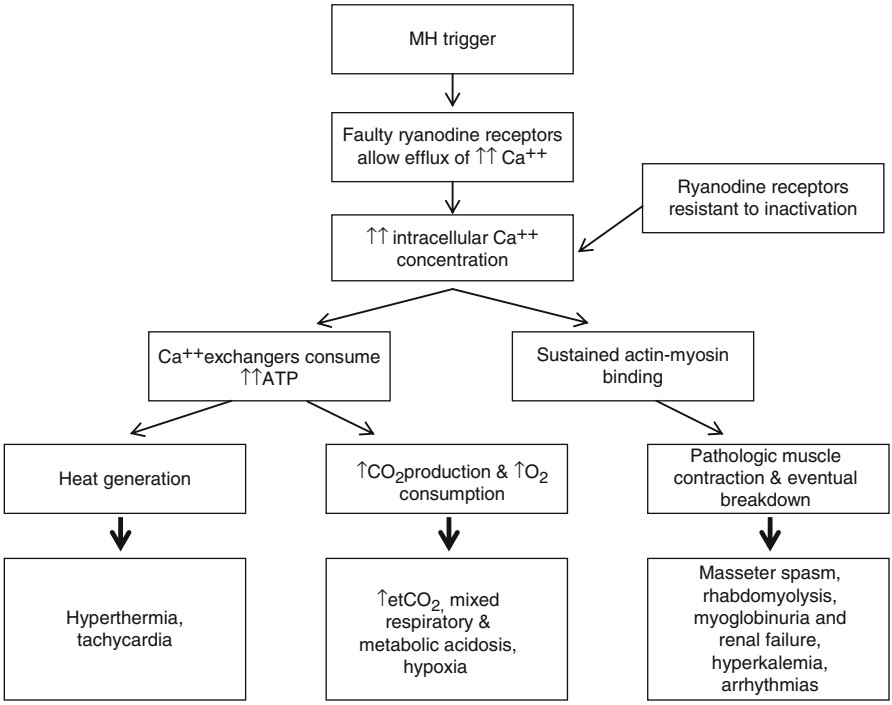

Fig. 50.1 Dysregulated excitation-contraction coupling in MH stems from exaggerated release and increased intracellular concentration of Ca^{++}, which ultimately leads to hyperthermia, a hypercatabolic state, muscle rigidity, and muscle breakdown

Table 50.3 MHAUS treatment algorithm for MH

Call the MH 24-h hotline (for emergencies only)
Start emergency therapy for MH acute-phase treatment
1. Get help. Get dantrolene. Notify surgeon
2. Dantrolene sodium for injection 2.5 mg/kg rapidly IV (through large-bore IV if possible)
3. Bicarbonate for metabolic acidosis
4. Cool the patient
5. Dysrhythmias: usually responds to treatment of acidosis and hyperkalemia
6. Hyperkalemia
7. Follow: end-tidal CO_2, electrolytes, blood gases, CK, serum myoglobin, core temperature, urine output and color, and coagulation studies

Table 50.4 AAGBI treatment algorithm for MH

1. Call for help
2. Diagnosis – consider MH if:
   (a) Unexplained, unexpected increase in end-tidal CO_2 together with
   (b) Unexplained, unexpected tachycardia together with
   (c) Unexplained, unexpected increased in oxygen consumption
3. Take measures to halt the MH process
   (a) Remove trigger drugs, turn off vaporizers, use high fresh gas flows (oxygen), use a new clean non-rebreathing circuit, and hyperventilate. Maintain anesthesia with intravenous agents such as propofol until surgery is complete
   (b) Dantrolene; 2–3 mg/kg IV initially and then 1 mg/kg PRN
   (c) Use active body cooling but avoid vasoconstriction. Convert active warming devices to active cooling and give cold intravenous infusions, cold peritoneal lavage, extracorporeal heat exchange
4. Monitor
   ECG, SpO_2, $etCO_2$, invasive arterial BP, CVP, core and peripheral temperature, urine output and pH, arterial blood gases, potassium, hematocrit, platelets, clotting indices, creatine kinase (CK) (peaks at 12–24 h)
5. Treat the effects of MH
   (a) Hypoxemia and acidosis: 100 % O_2, hyperventilate, sodium bicarbonate
   (b) Hyperkalemia: sodium bicarbonate, glucose and insulin, calcium chloride IV (if in extremis)
   (c) Myoglobinemia: forced alkaline diuresis (aim for urine output >3 mL/kg/h, urine pH >7.0)
   (d) Disseminated intravascular coagulation: fresh frozen plasma, cryoprecipitate, platelets
   (e) Cardiac arrhythmias: procainamide, magnesium, amiodarone (avoid calcium channel blockers because of interaction with dantrolene)
6. ICU management
   (a) Continue monitoring and symptomatic treatment
   (b) Assess for renal failure and compartment syndrome
   (c) Give further dantrolene as necessary (recrudescence can occur for up to 24 h)
   (d) Consider other diagnoses (see Table 50.2)
7. Late management
   (a) Counsel patient and/or family regarding implications of MH
   (b) Refer patient to MH unit, counseling

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Part VI
Neuro/Neuromuscular-Related Cases

Chapter 51
Emergent Craniotomy for Evacuation of Epidural Hematoma

Michael Bronson

The patient is a 30-year-old male who was brought emergently to the operating room for evacuation of a right-sided epidural hematoma secondary to a motor vehicle accident. On presentation, the patient was already intubated and quick neurologic exam showed he was obtunded with a Glasgow Coma Scale (GCS) of 3 (**L-1**) and pupils were dilated to 5 mm and nonreactive. He was 71 in. tall and weighed approximately 80 kg (body mass index [BMI] 24.6), and his vital signs were temperature 37.2 °C, pulse 82 beats per minute (bpm), respiratory rate 14 breaths/min (on ventilator), blood pressure 150/80 mmHg (**L-2**, **L-3**), and SpO_2 98 %. Past medical history was unknown. The patient was induced with 1 mg fentanyl, 50 mg propofol, and 10 mg vecuronium. Following induction, he was placed on Nitrous oxide contradictst some of the learning points to follow a propofol infusion and isoflurane for anesthetic maintenance. A second IV (14 gauge) was also placed at this time in the left hand, and an arterial line was placed in the left dorsalis pedis artery. Baseline arterial blood gas (ABG) showed pH 7.31, $PaCO_2$ 41 mmHg, PaO_2 213 mmHg ($FiO_2 = 0.5$), hemoglobin (Hgb) 9.8 g/dL, and hematocrit (HCT) 29 %. The patient received 12.5 g mannitol, 100 mg furosemide, and 250 mL of 3 % NaCl. In addition, the patient was hyperventilated to end-tidal CO_2 ($etCO_2$) of 28 mmHg throughout most of the case (**L-4**).

Following evacuation of the hematoma, the surgeons noted a considerable amount of active arterial bleeding, which was eventually controlled. A repeat ABG at this time revealed pH 7.21, $PaCO_2$ 30 mmHg, PaO_2 157 mmHg ($FiO_2 = 0.5$), Hgb 7.5 g/dL, and HCT 22 %. Total estimated blood loss for the case was 2 L, and the patient received a total of 6 units of packed red blood cells (PRBCs), 4 units of fresh frozen plasma (FFP), 1 unit of platelets, and 5 L of crystalloid. An intracranial pressure (ICP) monitor and ventricular drain were placed at the end of the case, and initial ICP was noted to be 20 mmHg. A central venous catheter was placed by the

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anesthesia providers in the right subclavian vein using sterile precautions. The patient was transported to CT scan and then taken to the intensive care unit. At the end of anesthesia care, the patient's vital signs were temperature 35.5 °C (**L-5**), pulse 104 bpm, respiratory rate 12 breaths/min (on ventilator), blood pressure 108/75 mmHg, and SpO_2 96 %. Blood-gas analysis revealed a HCT of 22 %.

On postoperative day #1, the patient's ICP ranged from 65 to 135 mmHg on the monitor (**L-6**, **L-7**). Repeat head CT showed increased contusions and edema. A radionuclide angiogram was done which revealed absence of flow and uptake in the cerebrum and cerebellum, consistent with the diagnosis of brain death. The patient was maintained on the ventilator with pressor support as needed for eventual organ harvesting.

Lessons Learned

L-1: **What is the Glasgow Coma Scale**?

The Glasgow Coma Scale (GCS) was introduced by Teasdale and Jennett in 1974 as a means of unifying the assessment for level of consciousness [1]. They sought to create a clinical tool that was simple to use and could produce consistent results when performed by physicians and nurses caring for patients with diminished level of consciousness. The scale is divided into three parts: motor, verbal, and eye opening which the authors felt reflected three different areas contributing to the patient's overall level of consciousness:

1. Motor – testing the integrity of the central nervous system (six items; see Table 51.1)
2. Verbal – signaling recovery to consciousness (five items; see Table 51.1)
3. Eye movement – assessing whether arousal mechanisms were intact (four items; see Table 51.1). The 15-point Glasgow Coma Scale (Table 51.1) has become the most universally applied scale for assessing level of consciousness [2, 3].

There are three major uses for the Glasgow Coma Scale [4–7]:

1. To stratify the severity of head injury:
   (a) Mild (GCS 13–15)
   (b) Moderate (GCS 9–12)
   (c) Severe (GCS ≤8)

Table 51.1 The Glasgow Coma Scale

Motor response	Verbal response	Eye opening
6 Follows commands	5 Oriented	4 Spontaneous
5 Localizes to pain	4 Confused	3 To speech
4 Withdraws to pain	3 Inappropriate words	2 To pain
3 Abnormal flexion	2 Incomprehensible sounds	1 None
2 Abnormal extension	1 None	
1 No movement		

Range of scores: 3–15. Verbal is unable to be assessed if the patient is intubated

2. To define coma and likely need for intubation (GCS ≤8)
3. To use as a prognostic indicator

L-2: **What are the determinants of cerebral perfusion pressure**?
Cerebral perfusion pressure (CPP) is defined as the pressure difference between mean arterial pressure and intracranial pressure or central venous pressure (CVP), whichever of these two back pressures is greater:

$$\text{CPP} = \text{MAP} - \left[\text{ICP or CVP, whichever is greater}\right]$$

Normal cerebral perfusion pressures range from 80 to 100 mmHg and are largely dependent on MAP in the patient with normal ICP (5–15 mmHg). Cerebral blood flow (CBF) is usually preserved within a wide range of mean arterial pressures (70–150 mmHg) because of autoregulation (see below) [8, 9]. Outside of this range, changes in cerebral perfusion pressure are directly related to changes in cerebral blood flow. To best estimate cerebral perfusion pressure as a function of MAP and ICP, the transducer for the ICP monitor and arterial line should be zeroed to the level of the ear.

L-3: **What is cerebral autoregulation**?
Normal cerebral blood flow is about 50 mL/100 g of brain tissue/min. Eighty percent of CBF supplies gray matter of the brain, whereas the other 20 % supplies white matter [8]. Cerebral blood flow is maintained relatively constant over a wide range of blood pressures, MAPs 70–150 mmHg, because of cerebral autoregulation, which is accomplished via vasoconstriction and vasodilation of cerebral arterioles (Fig. 51.1) [8, 9]. Outside of the range of autoregulation, CBF is directly related to MAP. For that reason, as MAP falls below the lower limit of autoregulation, a subsequent decline in CBF occurs. The opposite effect is seen at the other end of the curve with MAPs beyond the upper limits of autoregulation leading to hyperemia.

The range of MAPs over which autoregulation occurs has commonly been cited as 50–150 mmHg. However, the lower limit of autoregulation has recently been disputed and is thought to be around 70 mmHg [10]. With this in mind, in patients with normal ICP(5–15 mmHg) and without chronic hypertension, the lower limits of autoregulation as it pertains to cerebral perfusion pressure are in the range of 55–65 mmHg (i.e., 70 − (5–15) = 65 − 55). In patients with chronic hypertension, the autoregulation curve is shifted right; therefore the lower limits of autoregulation will be higher in this group [9].

L-4: **What is the relationship of cerebral blood flow to $PaCO_2$**?
Cerebral blood flow can be altered by shifting the $PaCO_2$ in either direction, via hyperventilation or hypoventilation, which effectively adjusts the pH of the extracellular fluid around the brain [8]. A linear relationship exists between a $PaCO_2$ of 25–75 mmHg in that an increase in $PaCO_2$ will result in an increase in CBF and vice versa (Fig. 51.2). It has been estimated that for every change of 1 mmHg in $PaCO_2$, CBF will change by 1–2 mL/100 g/min in the same direction.

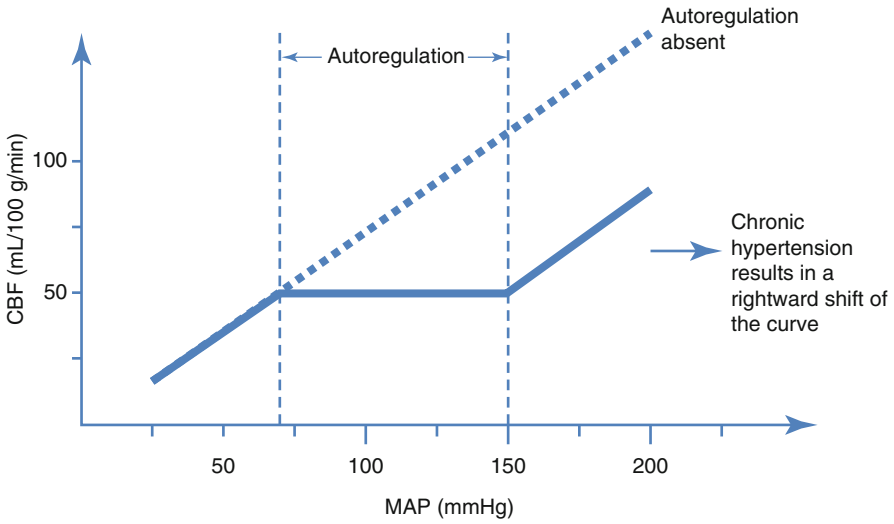
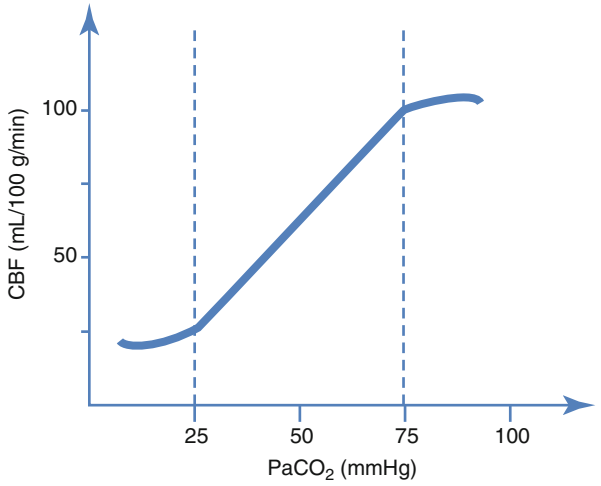


Fig. 51.1 Autoregulation of cerebral blood flow. Within MAPs of 70–150 mmHg, autoregulation occurs so that changes in blood pressure inside of this range result in minimal changes to CBF. Outside of the autoregulatory region, CBF is dependent on MAP. Autoregulation occurs at higher MAPs in patients with chronic hypertension

Fig. 51.2 Relationship of CBF to $PaCO_2$. Within a $PaCO_2$ of 25–75 mmHg, CBF increases 1–2 mL/100 g/min for every increase in $PaCO_2$ by 1 mmHg



With sustained hyperventilation, bicarbonate production within the extracellular fluid begins to decrease, and the pH begins to normalize, so CBF-lowering effects by hyperventilation are temporary, lasting roughly 6–12 h [9]. In addition, rapid normalization of $PaCO_2$ after a period of hyperventilation can lead to a transient acidosis within the extracellular fluid, creating a state of cerebral vasodilation and resulting in increases in ICP.

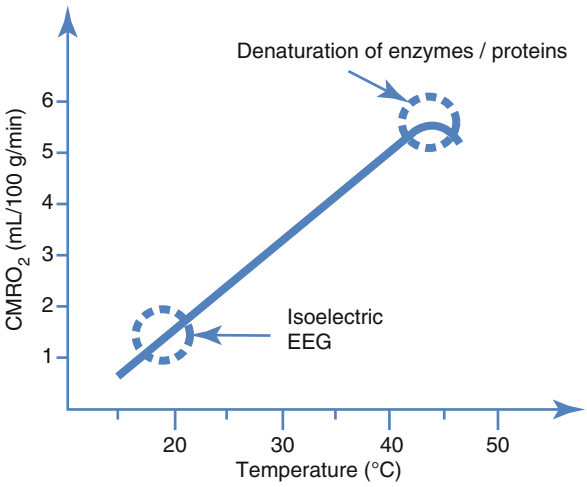


Fig. 51.3 Relationship of $CMRO_2$ to temperature. For every 1 °C change in temperature, $CMRO_2$ changes 6–7 % in the same direction. Further reductions in $CMRO_2$ occur beyond an isoelectric EEG. Above 42 °C, $CMRO_2$ begins to decline as denaturation of enzymes and proteins begins to occur

It should be noted that CBF appears to be significantly decreased within the first 24 h following a traumatic brain injury (TBI) and further decreases in CBF by hyperventilation can have deleterious effects, most importantly contributing to cerebral ischemia [11]. For that reason it is recommended that hyperventilation be avoided in the first 24 h following TBI, unless absolutely necessary to facilitate surgical intervention and prevent herniation.

L-5: **What is the relationship of cerebral blood flow to temperature**?
The effects of temperature on cerebral blood flow have been well described [8, 9, 12]. For every 1 °C decrease in temperature, cerebral metabolic rate of oxygen consumption ($CMRO_2$) will decrease by 6–7 % (Fig. 51.3). $CMRO_2$ is coupled to CBF in that as the metabolic rate decreases, the blood flow to the area will also decrease to achieve an appropriate supply/demand balance. Therefore, hypothermia results in a lower CBF via a decreased $CMRO_2$. At around 18–20 °C, the EEG becomes isoelectric, yet further decreases in temperature still cause a continuing decrease in CBF as opposed to the administration of intravenous anesthetics which cannot achieve further CBF reductions beyond an isoelectric EEG.

A direct relationship between hyperthermia and CBF holds true as well. Between 37 and 42 °C, increasing temperature results in an increase in CBF. Above 42 °C, it has been found that $CMRO_2$ actually begins to decrease, which is thought to be a result of denaturation of enzymes and proteins, signifying brain cell damage/death.

L-6: **What is the relationship of intracranial pressure to brain volume**?
The cranium is a rigid structure with limited ability to increase the volume of intracranial components, mainly brain tissue (80 %), blood (10 %), and cerebrospinal fluid (10 %). This was first established in the Monro-Kellie doctrine or hypothesis which laid the foundation for the intracranial pressure volume relationships that we use today [13]. Since the cranial vault is a rigid, unyielding structure, an increase in the volume of one component must be compensated by the decrease in the volume

of the other components. In view of the fact that displacement of brain tissue is essentially the disastrous state of herniation, physiologically acceptable compensatory mechanisms are limited to an extracranial movement of blood and/or CSF. Accordingly, an increase in the size of brain tissue (i.e., edema, tumors) must be compensated for by a displacement of blood and/or CSF extracranially to maintain a relatively constant intracranial pressure (Fig. 51.4) [9].

When intracranial volume reaches a level in which compensatory mechanisms are exhausted, there is a brisk rise in ICP for any further increases in intracranial volume (Fig. 51.5). There are two potentially devastating consequences of continued rise in ICP as a result of increasing intracranial volume: herniation of brain tissue and diminished cerebral perfusion pressure leading to ischemic injury [9].

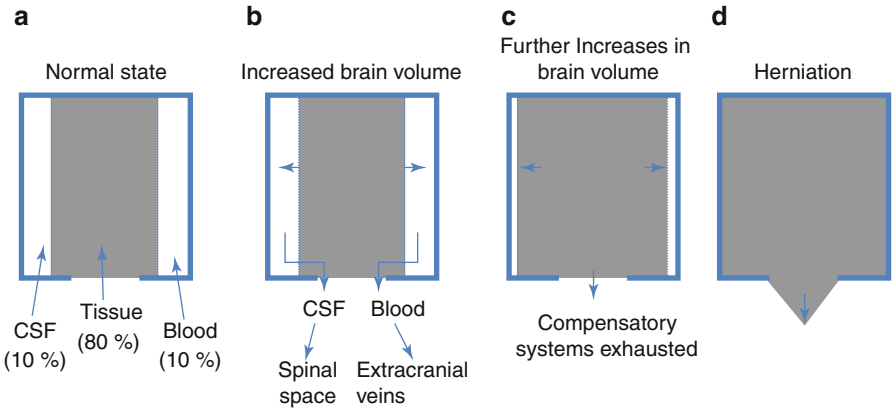


Fig. 51.4 (**a**) Normal relationship of brain tissue, blood, and cerebrospinal fluid within cranial vault. (**b**) Increased size of brain volume, such as with acute cerebral edema, results in displacement of blood and/or CSF to extracranial veins and spinal space, respectively. (**c**) Once compensatory systems are exhausted, the rigid cranial vault cannot accommodate further increases in brain volume. (**d**) Herniation occurs as brain continues to expand and tissue is displaced caudally through the foramen magnum

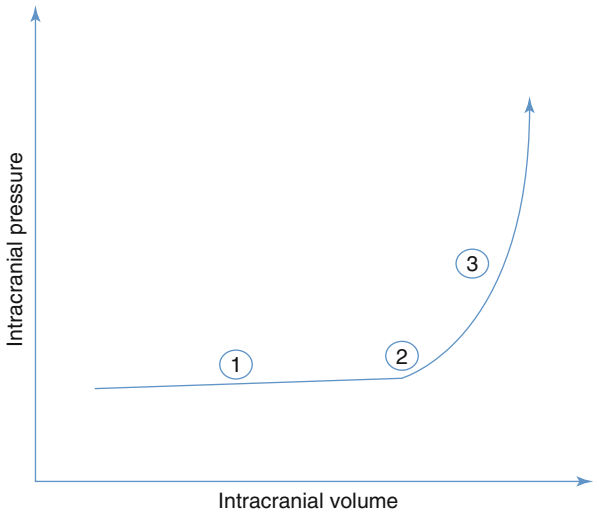


Fig. 51.5 Relationship of intracranial volume to intracranial pressure. (*1*) Volume compensation by displacement of blood and/or CSF helps to maintain a relatively constant intracranial pressure. (*2*) Once these mechanisms are exhausted, there is a (*3*) rapid increase in ICP for further increases in intracranial volume leading to herniation and diminished cerebral perfusion pressure

L-7: **What can be done to help lower intracranial pressure**?
There are multiple methods that have shown to be effective in lowering intracranial pressure (Table 51.2 and Fig. 51.6). Note that not all of the methods listed will be useful in every setting of elevated intracranial pressure, so clinical judgment in coordination with discussions with the surgical team should guide therapy.

Table 51.2 Methods to decrease intracranial pressure

Category of therapy	Maneuver	Mechanism
Positioning	1. Head elevation to 30°	Improves venous drainage resulting in lowered cerebral blood volume. Should be performed so as not to compromise CPP via decreases in MAP
	2. Avoid excessive head flexion/rotation	Prevents obstruction to venous outflow
Pharmacology	3. Intravenous anesthetics	With the exception of ketamine, IV anesthetics result in a decrease in $CMRO_2$ which is coupled with a decrease in CBF
	4. Mannitol 5. 3 % NaCl 6. Furosemide	Decreases the volume of intracellular and extracellular fluid within the brain via raising the serum Osm (4) and (5). This creates an osmotic gradient to promote flow of fluid to the intravascular space, subsequently removed by kidneys via diuretic effect (mannitol [4] and furosemide [6] only)
	7. Steroids	Have shown to be effective in decreasing cerebral edema if present
	8. Avoid vasodilators	Vasodilators, importantly volatile anesthetics, will increase CBF via its dilatory effects on cerebral arterioles, thusly increasing ICP
Respiratory	9. Hyperventilation	It has been well established that a decrease in $PaCO_2$ results in a decrease in CBF by alkalinizing cerebral intracellular fluid. Note that this is not recommended in the acute setting of TBI unless absolutely necessary
	10. Avoid hypoxia ($PaO_2 < 60$ mmHg)	Above a PaO_2 of 60 mmHg, CBF is relatively unaffected by changes in PaO_2. Under a PaO_2 of 60 mmHg, however, there is an abrupt rise in CBF
	11. Avoid increases in intrathoracic pressure	Increases in intrathoracic pressure, such as during straining and coughing, leads to increases in central venous pressure which diminishes cerebral venous outflow. PEEP also exerts a similar effect so should be kept to a minimum
Temperature	12. Hypothermia	$CMRO_2$ and CBF can be lowered by decreasing temperature. However, there are conflicting reports to the actual benefits, so it is not currently recommended in the setting of TBI

(continued)

Table 51.2 (continued)

Category of therapy	Maneuver	Mechanism
Surgery	13. Ventriculostomy	Decreasing the volume of CSF decreases total intracranial volume, lowering ICP
	14. Surgical decompression	Decompression of masses within the cranial vault (hematoma, tumor, etc.) results in lower total intracranial volume and lower ICP

These should only be performed within the context of the clinical situation

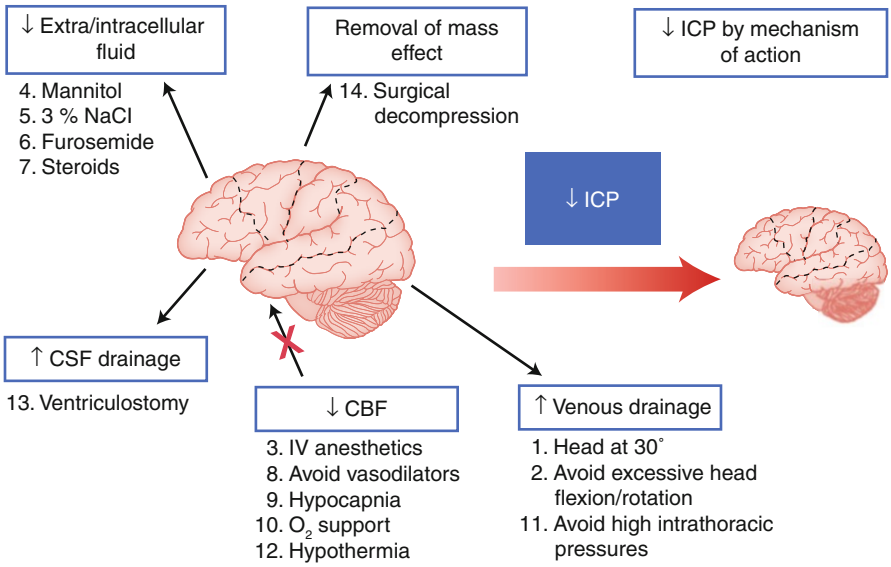


Fig. 51.6 Illustration of methods to decrease intracranial pressure

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Chapter 52
Hyperkalemia and Residual Neuromuscular Blockade After Kidney Transplantation

Geoffrey Langham

A 55-year-old woman, 52 kg and 62 in. tall, was brought to the operating room (OR) for deceased-donor kidney transplantation for stage five chronic kidney disease, the etiology of which was polycystic kidney disease (**L-1**). The patient had never been on hemodialysis and had no dialysis grafts or fistulae. The past medical history was also significant for secondary hyperparathyroidism, anemia, and rheumatoid arthritis. The preoperative laboratory studies were significant for hematocrit (HCT) of 31.8 %, platelets 255,000/μL, serum potassium concentration ($[K^+]$) 5.3 mEq/L (upper limit of normal 5.2 mEq/L), serum CO_2 20 mmol/L, and creatinine 6.1 mg/dL (**L-2**).

The patient underwent successful kidney transplantation without complication. Intraoperatively, the patient received methylprednisolone 500 mg, furosemide 100 mg, and mannitol 12.5 g intravenously (IV). Adequate, clear urine output was noted at the end of surgery, and hemostasis was adequate. The patient emerged from anesthesia, was extubated, and was taken to the postanesthesia care unit (PACU) for recovery.

After 4 h in the PACU, the patient became hypotensive with blood pressure (BP) 74/46 mmHg and HCT 17.6 %, which was treated by the surgical team with 1 L of albumin IV and 2 units of packed red blood cells (PRBCs). Preliminary interpretation of an abdominal ultrasound revealed perinephric fluid. Pertinent laboratory studies included $[K^+]$ 5.3 mEq/L, serum CO_2 15 mmol/L, and creatinine 4.9 mg/dL. After 1 h, the surgical team notified the anesthesia service that the patient needed to return to the OR for urgent surgical exploration to evaluate possible hemorrhage.

The patient was brought to the OR, where preoxygenation and induction of anesthesia proceeded with lidocaine, etomidate, and cisatracurium, and an endotracheal tube (ETT) was placed. The patient's BP remained stable during anesthesia and surgery, with systolic BP 110–130 mmHg and diastolic BP 50–70 mmHg. Two additional units of RBCs and 1 L of normal saline (NS) were given. The surgeons

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Table 52.1 Arterial blood gas data

Event	pH	pCO_2, mmHg	pO_2, mmHg	Base excess, mmol/L	HCT, %	$[K^+]$, mEq/L	Glucose, mg/dL
During somnolence	6.94	79	340	−15.2			
After reintubation	7.24	38	458	−11	37	6.5	225

noted 250 mL of clotted blood around the graft kidney, but no active bleeding, and proceeded to close the incision. At the end of surgery, a peripheral nerve stimulator ("twitch monitor") on the facial nerve showed a train-of-four (TOF) of 2/4. Neostigmine 2.5 mg and glycopyrrolate 0.3 mg IV were given. Within 5 min, the spontaneous respiratory rate (RR) was 18, and tidal volumes (V_t) were about 150 mL (**L-3**, **L-4**, **L-5**). The twitch monitor was placed over the ulnar nerve, where the TOF was 4/4, but 5 s of 100 Hz tetany showed fade. Neostigmine 1 mg IV was given, without change in the neuromuscular or respiratory status. The patient was taken to PACU intubated.

Shortly after arriving in PACU, the patient began to "buck" on the ETT and followed commands (**L-6**, **L-7**). The patient was extubated but within 10 min became progressively somnolent. An arterial blood gas (ABG) showed a profound acidosis, as seen in Table 52.1.

The patient was reintubated and manually bag-ventilated. An arterial line was placed in the left dorsalis pedis artery. A repeat ABG, after reintubation, is shown in Table 52.1.

Following this ABG, sodium bicarbonate 50 mEq, furosemide 20 mg, insulin 10 units, and normal saline 1 L were given quickly IV to correct acidosis and hyperkalemia. A repeat blood draw showed $[K^+] = 5.7$ mEq/L.

An additional 20 units of insulin with 10 % dextrose were given IV. The patient was transported intubated and sedated to the intensive care unit, where the $[K^+]$ was 4.2 mEq/L. A subsequent evaluation by the surgical team and blood bank ruled out hemolytic transfusion reaction as a cause of the patient's hyperkalemia.

Lessons Learned

L-1: **What is the pathophysiology of chronic kidney disease**?
Chronic kidney disease (CKD) is characterized by progressive loss of renal function and classified into Stages 1 through 5 based on the estimated or measured glomerular filtration rate. CKD most commonly results from chronic arterial hypertension, diabetes mellitus, or polycystic kidney disease. Renal dysfunction affects most of the body's organ systems including the central nervous, circulatory, hematologic, and musculoskeletal systems.

As such, the pathophysiology of CKD is complex. It can best be understood by separating the kidney's functions into the distinct processes of (1) glomerular filtration, affecting intravascular volume status and elimination of nitrogenous waste

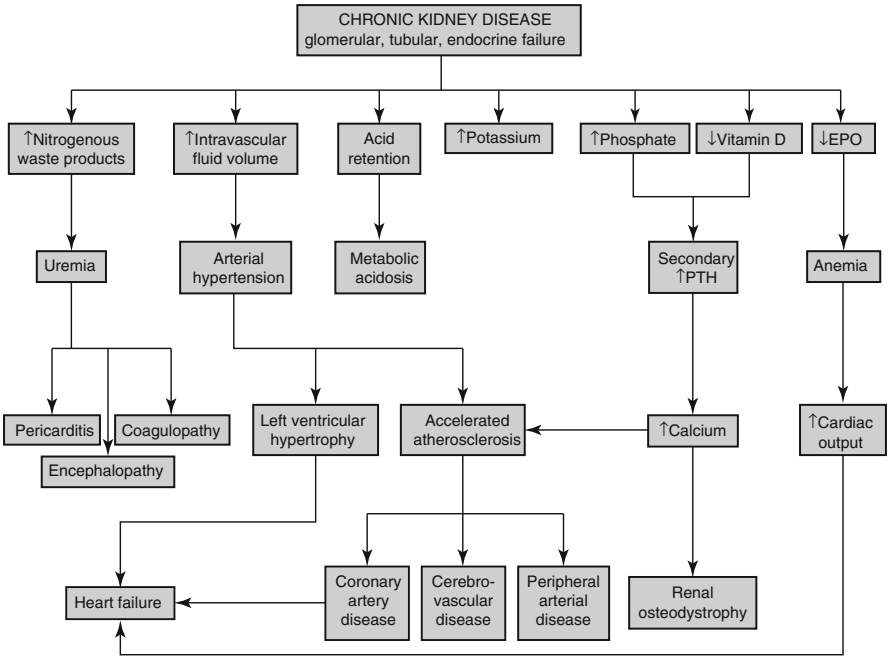

Fig. 52.1 Pathophysiology of chronic kidney disease. *PTH* parathyroid hormone, *EPO* erythropoietin

products; (2) tubular secretion and reabsorption, affecting electrolytes and acid-base balance; and (3) renal endocrine function, affecting erythropoietin and Vitamin D metabolism. See Fig. 52.1.

L-2: **How should this patient's preoperative hyperkalemia be managed**?
The approach to hyperkalemia begins with an assessment of its etiology and chronicity. Initial steps should always include ruling out artifactual causes of the elevated [K^+] such as laboratory error, hemolysis, and pseudohyperkalemia (spurious serum [K^+] elevation due to leukocytosis or thrombocytosis). With most laboratory abnormalities, the presence or absence of end-organ effects guides clinical decision-making regarding treatment and management. However, hyperkalemia is unusual in that it is usually asymptomatic, but its primary effects (on the cardiovascular system) can be sudden and life threatening.

Because hyperkalemia is usually asymptomatic, assessment of the electrocardiogram (ECG) is critical to determine aggressiveness of therapy and patient readiness for surgery. ECG abnormalities related to hyperkalemia classically progress from peaked T waves to loss of P waves, QRS widening, ventricular arrhythmias, and "sine wave" or asystole. This analysis is complicated by the understanding that ECG changes seem to be related more to acuity of the change in [K^+] rather than the absolute value and that the first sign of cardiac dysfunction from hyperkalemia may be ventricular fibrillation.

Table 52.2 Classes of therapy for hyperkalemia

Cardiac membrane stabilization	Intracellular shift of K^+	Elimination of K^+
IV calcium chloride or calcium gluconate	IV insulin (with glucose)	Sodium polystyrene sulfonate (Kayexalate) by mouth or per rectum
	β_2 agonists, IV or inhaled (albuterol, epinephrine)	Hemodialysis
	Correction of acidosis, if present: IV sodium bicarbonate, hyperventilation	Kaliuresis induced by aggressive hydration, IV loop diuretics (furosemide), or urinary alkalinization (bicarbonate)

See dosing in Fig. 52.2

Treatment of hyperkalemia relies on three strategies: (1) stabilization of cardiac cell membranes, (2) shift of potassium (K^+) from the blood into cells, and (3) elimination of K^+ from the body. Table 52.2 shows common therapies for elevated [K^+].

It is generally recommended that the [K^+] be below 5.5 mEq/L prior to elective surgery; this is, of course, a recommendation, and the relationship of this value to the upper limit of normal varies from lab to lab. It also recommended that any [K^+] greater than 6.5 mEq/L, even if asymptomatic, requires emergent treatment, because most patients with a [K^+] greater than 6.7 mEq/L will have ECG changes [1].

Hyperkalemia is particularly dangerous in the perioperative period due to the common and potentially large swings in [K^+] and serum pH in the setting of respiratory and metabolic derangements like mechanical ventilation, succinylcholine use, hemorrhage, transfusion, tourniquet release, or transplanted organ reperfusion.

Figure 52.2 shows an algorithm for the approach to preoperative hyperkalemia.

In this case, the preoperative hyperkalemia was mild, without symptoms, and without documented ECG changes. Factors pertinent to the preoperative management include (1) the urgent nature of the surgery related to donated kidney availability and viability, (2) the likelihood that a successfully transplanted kidney could itself correct the hyperkalemia, and (3) the lack of immediate vascular access for hemodialysis. Given the nonemergent nature of both the hyperkalemia and surgery, a number of treatment strategies would be reasonable, including watchful waiting, administration of calcium, induction of intracellular shift with insulin and glucose, Kayexalate administration per rectum, or aggressive hydration with furosemide administration after implantation of the graft kidney.

L-3: **What is the clinical utility of common bedside tests to assess recovery from neuromuscular blockade**?

Residual postoperative neuromuscular blockade is common. In the absence of routine antagonism of neuromuscular blockade or routine neuromuscular monitoring, the incidence has been reported to be as high as 40–45 % [3, 4]. The most common monitoring technique to assess depth of neuromuscular blockade during anesthesia is the TOF, while assessing recovery from neuromuscular blockade at the end of anesthesia is commonly done with the TOF and response to 100 Hz tetany.

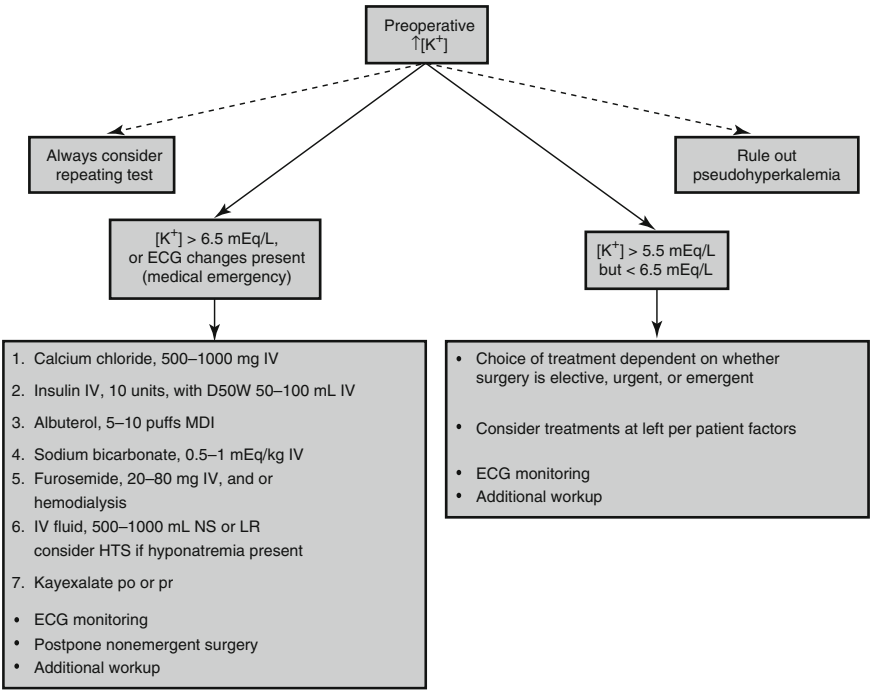

Fig. 52.2 Approach to preoperative hyperkalemia [1, 2]. The treatments at left are in order of priority: cardiac membrane stabilization with calcium (#1), followed by therapies to induce intracellular shift of potassium (#2–4), followed by therapies to remove potassium from the body (#4–7). [K^+] serum potassium concentration, *ECG* electrocardiogram, *IV* intravenous, *MDI* metered-dose inhaler, *D50W* 50 % dextrose in water, *NS* normal saline, *LR* lactated Ringer's solution, *HTS* hypertonic (3 %) saline, *po* by mouth, *pr* per rectum

The TOF ratio is defined as the strength of the fourth twitch divided by the strength of the first twitch and is used as a convenient assessment of recovery from neuromuscular blockade. Historically, a TOF ratio ≥0.7 measured at the adductor pollicis muscle was considered to be consistent with adequate recovery from neuromuscular blockade. However, visual and tactile assessment of the TOF ratio is prone to operator error [5], and many patients demonstrate significant weakness at TOF ratios between 0.7 and 0.9 [6]. Therefore, the current recommendation is to use the more stringent endpoint of a TOF ratio of 0.9 as an indicator of adequate recovery from neuromuscular blockade.

Selection of a site for peripheral nerve monitoring must be done carefully because of the variable resistance of different muscles to neuromuscular blockade and because the clinician is concerned with the full recovery of all muscle groups. The diaphragm and orbicularis oculi are resistant to neuromuscular blockade, requiring up to twice as much pancuronium as the adductor pollicis for an equal degree of blockade [7, 8]. The muscles of the pharynx and the adductor pollicis are relatively sensitive, while the muscles of the larynx show intermediate sensitivity [9, 10].

Weakness of the pharyngeal muscles threatens the patency of the upper airway and the patient's ability to swallow and clear secretions. In addition, weakness of the laryngeal muscles threatens respiratory function in at least three ways: (1) by poorly protecting the airway from fluid and particles via weak spasm, (2) by impairing the forceful glottic closure needed to generate an effective cough, and (3) by causing paradoxical vocal cord movement.

Taken together, peripheral nerve monitoring at a muscle that is sensitive to neuromuscular blockade is more likely to detect residual low-degree blockade. Therefore, monitoring of the ulnar nerve at the more sensitive adductor pollicis, rather than the facial nerve at the more resistant orbicularis oculi, is recommended at the end of anesthesia.

One bedside test consistent with a TOF ratio equal to or greater than 0.9 is the "tongue depressor test," in which the patient is instructed to oppose the incisor teeth against a wooden tongue depressor, which the clinician attempts to remove from the patient's mouth [6]. Patients with TOF ratios above 0.85–0.9 can generally retain the depressor despite significant force applied by the tester.

Figure 52.3 demonstrates the ranges of TOF ratios over which common clinical parameters and tests are likely to be normal.

L-4: **What is the role of assessing train-of-four prior to administration of a nondepolarizing neuromuscular blocker when evaluating recovery from neuromuscular blockade**?

Assessment of the TOF prior to administration of a nondepolarizing neuromuscular blocker (NDNB) is vitally important for the safe practice of anesthesia. Tactile and visual assessment of the amplitude of the TOF and the TOF ratio (if fade is present) is prone to error due to operator inexperience and the limitations of this qualitative method [5]. There is significant interpatient variation in the magnitude of response to TOF at a given current. There is also variation for one patient during a single anesthetic, as the resistance to the applied current may change due to skin temperature or sweating or the leads of the twitch monitor may be moved or changed to a different monitoring site during anesthesia.

Therefore, a "baseline" TOF prior to NDNB is crucial for TOFs obtained during and at the end of the anesthetic to have adequate accuracy for the safe practice of anesthesia. It is not sufficient simply to assess the TOF at the conclusion of surgery and use this one-time information to guide selection and dosing of agents to antagonize neuromuscular blockade. Figures 52.4 and 52.5 demonstrate how interpatient and intrapatient variability can mislead the clinician.

L-5: **What is the rapid shallow breathing index (RSBI)**? **How is the RSBI used to evaluate readiness for extubation**?

The RSBI, also known as the f/V_t ratio or the Tobin index after an author who popularized the parameter, is simply the respiratory rate divided by the tidal volume (in L). A healthy subject may have an RSBI around 30, whereas the patient in this case had an RSBI = (18/min/0.15 L) = 120/L/min.

The RSBI was shown to have clinical relevance in a landmark study by Yang and Tobin from 1991, in which the authors observed respiratory parameters and

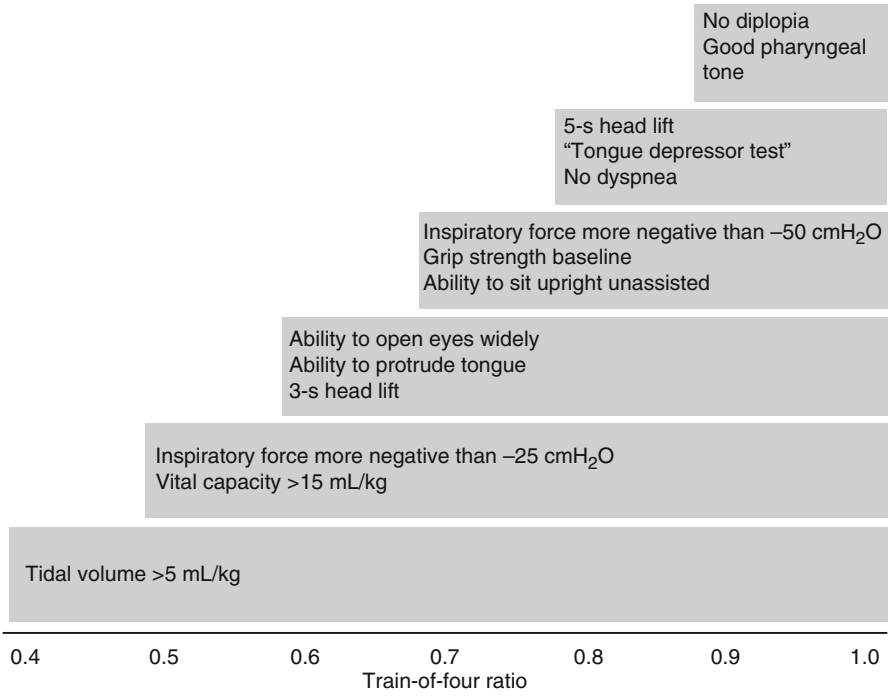

Fig. 52.3 Train-of-four (*TOF*) ratios and clinical parameters used to assess degree of neuromuscular blockade. Each bar represents the range of TOF ratios in which the clinical parameters and tests that contained the bar are likely to be normal. For example, a patient with baseline grip strength and a tidal volume greater than 5 mL/kg may have a TOF ratio as low as 0.8. Good pharyngeal tone = coordinated swallowing and no inspiratory upper airway collapse [11]. *Tongue depressor test* opposition of incisor teeth on a wooden tongue depressor forceful enough to prevent removal by the tester

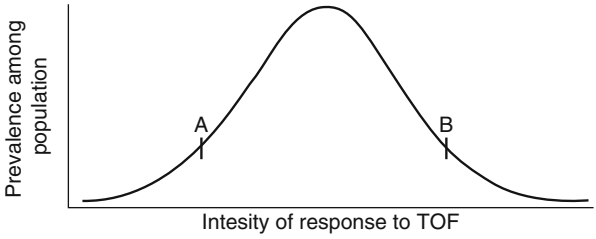

Fig. 52.4 Normal variation in intensity of response to train-of-four (*TOF*) in a population of patients. If the clinician is caring for a patient with baseline response *B*, but the clinician does not know what the magnitude of the baseline response is, and the patient has test response *A* at the end of anesthesia, the clinician will underestimate the degree of neuromuscular blockade. This may potentially lead to inappropriate administration or omission of antagonists of neuromuscular blockade or inappropriate extubation

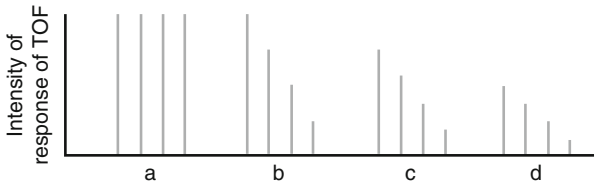

Fig. 52.5 Normal variation in intensity of response to train-of-four (*TOF*) within a single patient. TOF (**a**) represents a normal response prior to neuromuscular blockade. TOFs (**b–d**) represent increasing depth of neuromuscular blockade. The first twitch in TOF (**b**) shows 100 % of the baseline intensity and represents a state in which up to 70–80 % of receptors are blocked. The first twitch in TOF (**d**) shows 50 % of the baseline intensity and represents a state in which up to 95 % of receptors are blocked. Each of TOF (**b–d**) has a TOF ratio of 0.25, and without the "baseline" that TOF (**a**) provides, they may be confused for one another. If, at the end of anesthesia, the clinician mistakes TOF (**d**) for TOF (**b**), the degree of neuromuscular blockade will be underestimated, potentially leading to inappropriate administration or omission of antagonists of neuromuscular blockade or inappropriate extubation

reintubation rates in 100 critically ill patients who were weaned from mechanical ventilation [12]. The authors observed that an f/V_t ratio greater than 105 demonstrated a 97 % sensitivity, and a 95 % negative predictive value (NPV), in predicting the need for reintubation within 24 h. Stated differently, the study concluded that an f/V_t ratio less than 105 predicted with 95 % certainty that a patient would remain extubated 24 h after extubation.

There are several important limitations of the RSBI in clinical practice. First, the Yang and Tobin study is somewhat dated, and many aspects of critical care medicine, including weaning protocols and respiratory care techniques, have changed significantly since its publication. Second, the high sensitivity and NPV of the RSBI make it an excellent screening test of readiness for extubation, but not a confirmatory test. Even so, the sensitivity and NPV of the RSBI have been found to be lower than initially reported; one study in 1994 failed to reproduce the results of the 1991 study [13], and a 2006 meta-analysis [14] reported an average sensitivity of 87 %. Third, the RSBI is only one metric used to assess readiness for extubation and only takes into account ventilatory parameters (RR and V_t). Last, this landmark study was performed in patients with medical critical illness, whose similarity to patients undergoing brief mechanical ventilation for anesthesia and surgery is superficial.

L-6: **What are appropriate extubation criteria**?

Commonly cited extubation criteria are shown in Table 52.3. Patients who are being considered for extubation must meet all the criteria for discontinuation of two related but distinct components: (1) endotracheal intubation and (2) mechanical ventilation. There are two important caveats when considering extubation criteria. First, most studies on extubation criteria are based on intensive care unit patients, not patients undergoing anesthesia for surgery. Second, the specific values and thresholds of most extubation criteria are a matter of opinion or consensus; they represent relative, rather than absolute, goals prior to extubation.

Table 52.3 Extubation criteria

Criterion category	Specific aspects
Correction of indication for intubation	Has the disease process requiring intubation, or the indication for intubation, been corrected?
Neurologic status	Patient arousable to voice
	Patient following commands (preferred)
	Absence of neuromuscular blockade or neuromuscular disease (see ventilation below and text above)
Upper airway obstruction	Intact cough and gag reflexes, indicating ability to maintain airway patency
	Absence of upper airway masses, edema, etc.
	Presence of leak around ETT cuff
	If known difficult intubation, appropriate equipment and personnel available for possible reintubation
Oxygenation	F_IO_2 0.4–0.5
	PEEP 5–8 cmH_2O
	P_aO_2 >60 mmHg on above
	P_aO_2/F_IO_2 ratio >300
Ventilation [15]	f/V_t ratio less than 60–105/L
	Minute ventilation less than 10–15 L/min
	V_t greater than 4–6 mL/kg
	Forced vital capacity greater than 15 mL/kg
	RR less than 30–38 breaths/min
	Maximum inspiratory pressure more negative than −15 to −30 cmH_2O
	$P_aCO_2 < 50$ mmHg with above
Pulmonary toilet	Absence of excessive secretions
	Adequate cough
	Patient cooperative with suctioning and toilet maneuvers
Systemic illness	Absence of dynamic systemic processes, e.g., hypotension, fever, electrolyte abnormalities

L-7: **What is the role of an airway exchange catheter in the extubation of a patient with a known difficult airway or potential upper airway obstruction**?
An airway exchange catheter (AEC) is a long, hollow, semirigid catheter that can be passed through an in situ ETT prior to extubation of the trachea. AECs come in various outer diameters and lengths, generally have centimeter markings along their length, and usually have adapters to facilitate jet ventilation.

Extubation "over" an AEC is done for two reasons: (1) to facilitate reintubation or (2) to provide oxygen directly to the trachea. The AEC's effectiveness as a conduit for reintubation is greatly increased if the clinician also uses a laryngoscope to displace the pharyngeal soft tissues (or other upper airway obstruction) that could provide resistance to passage of the new ETT. The largest available AEC that fits inside the in situ ETT is most likely to provide a smooth passage of a new ETT through the larynx should reintubation be required. Jet ventilation through an AEC, as with transtracheal jet ventilation, must be done with meticulous attention to short inspiratory times, long expiratory times, and facilitation of air exit from the trachea.

With appropriate attention to the potential risks of tracheal/bronchial tissue trauma, barotrauma, and correct sizing, the AEC can be an effective and safe airway management device [16].

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Chapter 53
A Defasciculating Dose of Nondepolarizing Neuromuscular Blocker

Geoffrey Langham

A 44-year-old man, 127 kg and 72 in. tall, was brought to the operating room for open reduction and internal fixation of his left ankle following an all-terrain vehicle accident. The past medical history was significant only for severe obesity (body mass index = 38 kg/m^2). Last oral intake was 11 h prior to arrival in the preoperative area. Examination revealed an extremely anxious man in no apparent distress, wearing a cervical collar, with normal vital signs except heart rate = 102 beats/min. Airway examination revealed a Mallampati class 2, normal thyromental distance, intact dentition, and adequate mouth opening. Auscultation of the chest was unremarkable.

The patient was premedicated with 2 mg midazolam and 150 μg fentanyl intravenously (IV) and transferred to the operating room. Monitors were placed and preoxygenation with 100 % oxygen was done for more than 5 min. The patient remained anxious and complained the mask fit too tightly. Another 3 mg midazolam IV was given. Vecuronium 2 mg IV was given as a "defasciculating dose" prior to the succinylcholine to be used for rapid-sequence induction. The patient was warned that he may feel weak, and preoxygenation continued for another 4 min (**L-1**, **L-2**).

Suddenly, the patient became agitated and started to flail his limbs. He pulled the mask off and yelled "I can't breathe!" The anesthesia team attempted to keep the mask over his mouth and nose while calming him. The pulse oximeter showed a mild desaturation, decreasing from 100 to 92 % during this time. When the S_pO_2 reached 86 % and the patient remained agitated and uncooperative, the decision was made to proceed with induction. The anterior part of the cervical collar was quickly removed and the surgeon held in-line cervical spine stabilization. Lidocaine 100 mg, propofol 200 mg, and succinylcholine 200 mg IV were given in rapid succession while the nurse held cricoid pressure.

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The hypoxemia rapidly worsened, with S_pO_2 now 40 % (**L-3**). Direct laryngoscopy showed a Grade 1 view, a 7.0 mm endotracheal tube was placed easily, and manual bag-ventilation quickly restored the S_pO_2 to 100 %. Anesthesia was maintained with sevoflurane and nitrous oxide throughout the case, which concluded uneventfully. Emergence, extubation, and the postoperative course proceeded uneventfully, and the patient was discharged to home on postoperative day #1.

Lessons Learned

L-1: **What is the proper timing and dose of a nondepolarizing neuromuscular blocker if used as a "defasciculating dose"?**

The optimal timing of a defasciculating dose of nondepolarizing neuromuscular blocker (NDNB) is 3 min before succinylcholine [1]. Allowance of up to 5 or 7 min before succinylcholine provides equivalent abolition of fasciculation but has an increased risk of side effects. The most commonly studied doses of NDNB range from 10 to 30 % of the ED_{95} for the drug being studied, with the ED_{95} defined as the dose needed to reduce twitch strength by 95 % from baseline. Some authors [2] advocate a dose no higher than 10 % of an ED_{95}.

In this case, the interval between vecuronium administration and induction was at least 4 min, and the 2-mg dose selected represents 37 % of the ED_{95} for this 127-kg patient. Clearly, both the timing and the dose of vecuronium selected for this patient were inappropriate, and the patient developed partial but significant weakness, dyspnea, worsened anxiety, and hypoxemia as a result.

L-2: **What are the pros and cons of a defasciculating dose of nondepolarizing neuromuscular blocker prior to succinylcholine?**

In clinical medicine, the decision to proceed with any intervention is determined by the balance of its relative risks and benefits. Such decisions may be supported by the evidence available in the literature, the balance of the clinician's experience, and the clinician's assessment of the present situation. Figure 53.1 shows the "balance" of the positive effects (pros) and negative effects (cons) of using a defasciculating dose of NDNB prior to succinylcholine. In the author's opinion, the cons significantly outweigh the pros.

Although the relationship between succinylcholine-induced fasciculation and myalgia is not fully understood, the evidence does suggest a role of a defasciculating dose of NDNB in preventing myalgia. A 2005 meta-analysis of 52 randomized trials [2] found that the incidence of succinylcholine-induced fasciculation was 94 % but that of succinylcholine-related myalgia was only 51 %. There was no apparent graphical relationship of fasciculation to myalgia, as shown in Fig. 53.2. Nonetheless, this study found that a defasciculating dose of NDNB did provide a benefit in prevention of myalgia. Considering all NDNBs together, a defasciculating dose made the absence of myalgia at 24 h approximately 1.36–1.75 times more likely in the NDNB-treated groups than the control groups. The number of patients needed to treat with a defasciculating dose to prevent one case of myalgia was quite low, between 3 and 6; see Fig. 53.3. Interestingly, this meta-analysis found that the

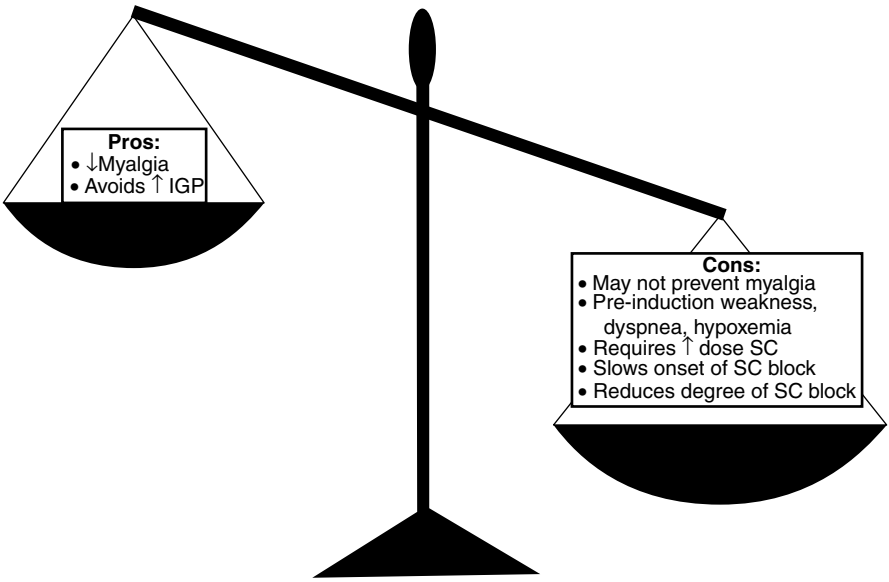

Fig. 53.1 The "balance" of the pros and cons of a defasciculating dose of nondepolarizing neuromuscular blocker prior to succinylcholine. The cons significantly outweigh the pros. *IGP* intragastric pressure, *SC* succinylcholine

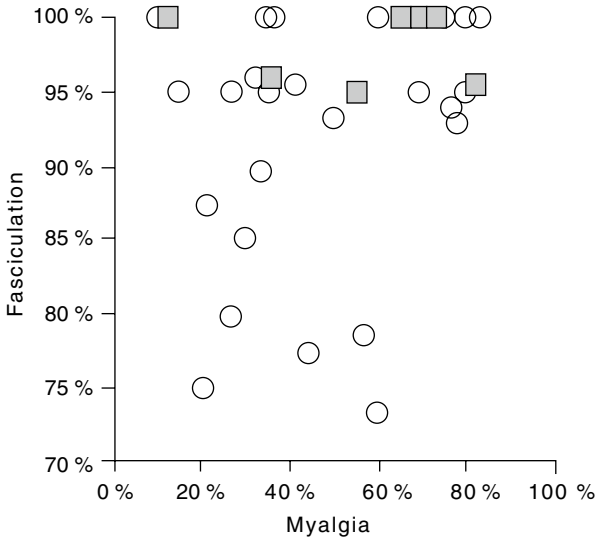

Fig. 53.2 Incidence of fasciculation and of myalgia in patients who received no pretreatment prior to succinylcholine [2]. Each symbol represents one trial in the meta-analysis performed by Schreiber et al. Data are shown for 35 trials that reported on both fasciculation and myalgia and where the dose of succinylcholine was 1 mg/kg (*gray squares*) or 1.5 mg/kg (*clear circles*)

use of IV lidocaine prior to succinylcholine was at least as effective as a defasciculating dose of NDNB in preventing myalgia.

Succinylcholine administration is associated with undesirable increases in intracranial pressure (ICP), intraocular pressure (IOP), and intragastric pressure (IGP). Increases of ICP and IOP with succinylcholine are known to be unrelated to muscle

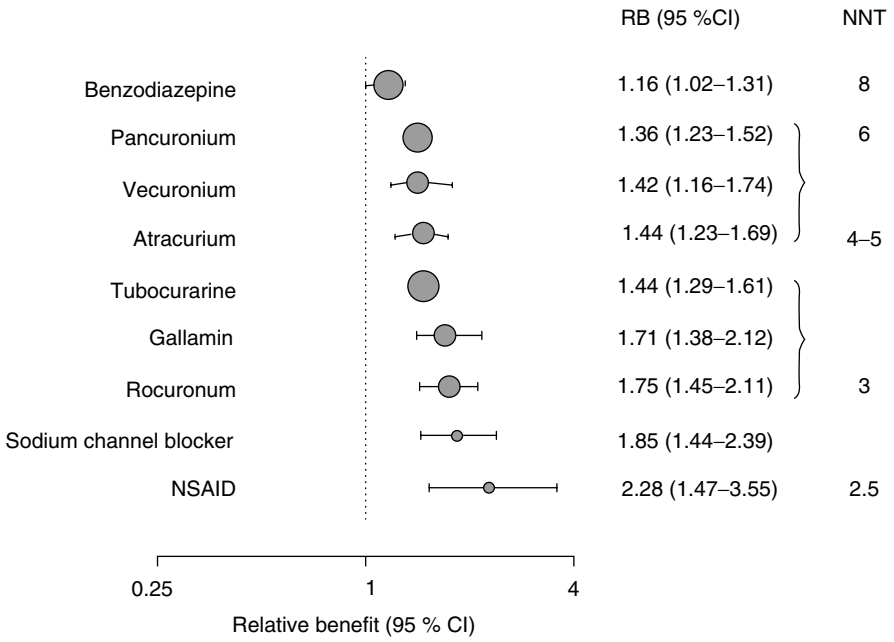

Fig. 53.3 Pharmacologic techniques to prevent succinylcholine-related myalgia [2]. For each intervention that was tested in at least three trials, a meta-analysis was performed by Schreiber et al. Symbol sizes are proportional to the number of analyzed patients. Relative benefit is a statistic that refers to the change in likelihood of a favorable outcome. In this case, a relative benefit greater than one favors the use of the listed intervention to prevent succinylcholine-related myalgia. All tested interventions have a relative benefit greater than 1. *CI* confidence interval, *NNT* number needed to treat, *NSAID* nonsteroidal anti-inflammatory drug, *RB* relative benefit

activity and fasciculation because these increases are not blocked by a defasciculating dose of NDNB [3]. On the contrary, increases of IGP with succinylcholine are indeed blocked by a defasciculating dose [4], and thus, avoiding an increase in IGP is another potential benefit of a defasciculating dose.

On the contrary, the risks of use of a defasciculating dose are numerous and draw on our understanding of pharmacology in this setting, the available data, and clinical experience with the adverse effects. Clearly, in some patients the use of a defasciculating dose will not prevent postoperative myalgia, which may be of another etiology or simply "resistant" to this technique. In addition, use of a defasciculating dose causes resistance to succinylcholine [5], necessitating a higher dose. The defasciculating dose may also slow the onset of succinylcholine [6] or reduce the degree of blockade induced by succinylcholine [7]. Duration of succinylcholine-induced block is influenced by the choice of NDNB used for pretreatment; block duration is prolonged with pancuronium but shortened with vecuronium [7].

Importantly, the use of a defasciculating dose predisposes the patient to clinically significant weakness prior to induction [2]. This may manifest as blurred vision, diplopia, difficulty swallowing, difficulty vocalizing, or dyspnea. Patients with anxiety or tenuous respiratory status may poorly tolerate these changes. Such weakness

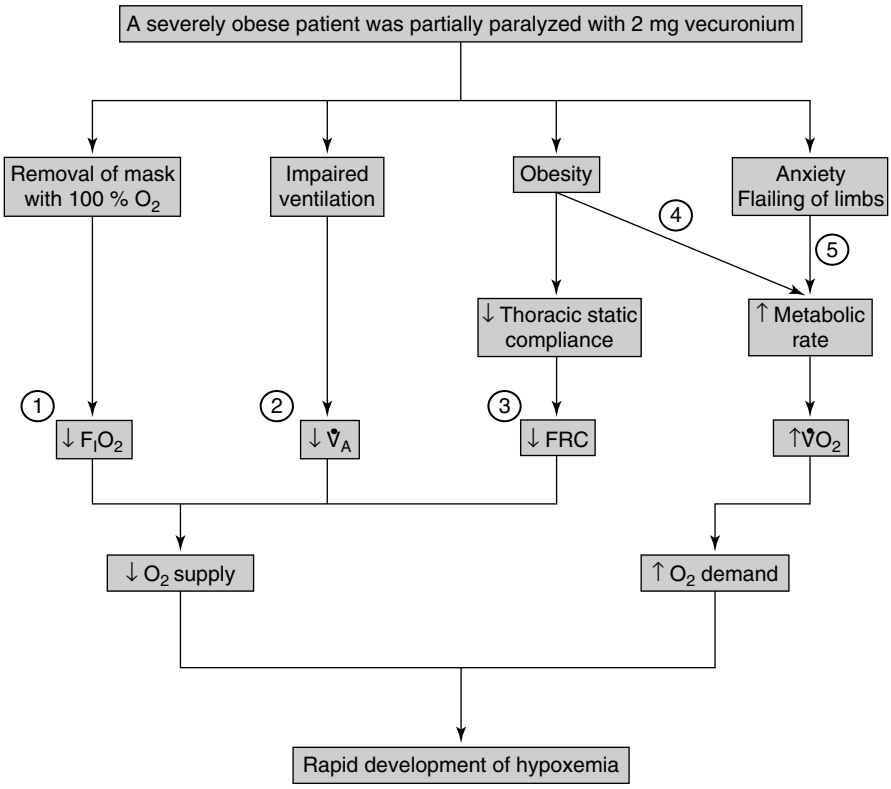

Fig. 53.4 Factors contributing to rapid development of hypoxemia in this patient. The five factors described in the text are labeled. F_IO_2 inspired fraction of oxygen, $\dot{V}_A$ minute alveolar ventilation, *FRC* functional residual capacity, $\dot{V}O_2$ minute consumption of oxygen

may also compromise airway reflexes, which is clearly undesirable in patients who are at high risk for regurgitation of stomach contents or aspiration.

In light of the above, we believe the "balance" of the pros and cons strongly favors avoiding a defasciculating dose of NDNB prior to succinylcholine, as shown in Fig. 53.1.

L-3: **What factors contributed to this patient's rapid oxyhemoglobin desaturation following the administration of vecuronium**?

Once this patient removed the mask with 100 % oxygen from his face, he rapidly developed oxyhemoglobin desaturation, which can be attributed to five factors. See Fig. 53.4.

First, the patient's inspired fraction of oxygen (F_IO_2) rapidly fell from 1.0 to 0.21 once the mask was removed and the patient inspired room air.

Second, partial neuromuscular blockade almost certainly reduced the patient's minute ventilation ($\dot{V}_E$, which likely contributed to alveolar hypoventilation ($\downarrow \dot{V}_A$).

Third, the patient has severe obesity, which is associated with adipose tissue deposition on the chest wall and which reduces thoracic static compliance and functional residual capacity (FRC), as shown in Fig. 53.5.

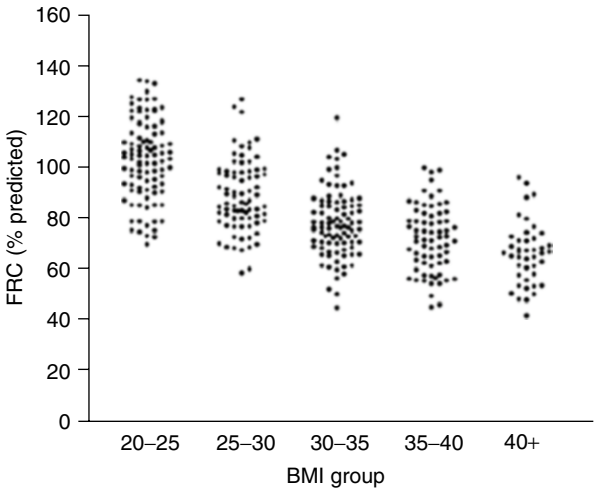

Fig. 53.5 Effects of body mass index (*BMI*) on functional residual capacity (*FRC*) [8]. Each dot represents data from one patient. FRC is shown as percent of predicted FRC, with this prediction being based on measured FRC of height-matched normal volunteers. BMI is shown grouped into 5-kg/m^2 intervals

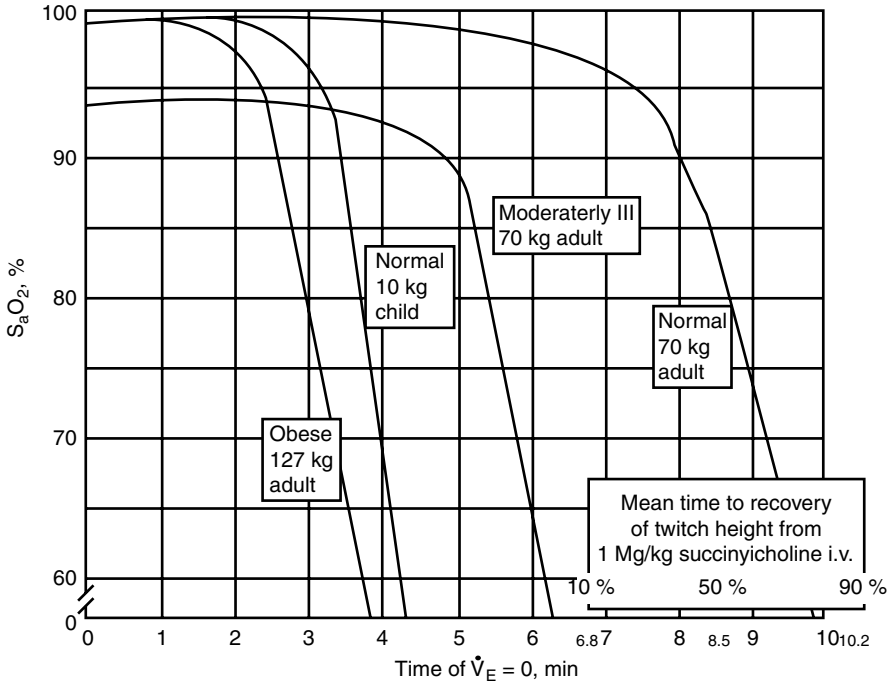

Fig. 53.6 S_aO_2 versus time of apnea for various types of patients [9]. The data were produced by a computer model of apnea. $\dot{V}_E$ ventilation, S_aO_2 arterial oxyhemoglobin saturation

Fourth, the patient's severe obesity is associated with increased minute oxygen consumption ($\dot{V}O_2$). Obese patients simply have more tissue that is metabolically active than normal-weight patients and thus have a higher basal metabolic rate and $\dot{V}O_2$ at rest. In light of the third and fourth factors described here, it is no surprise that obese patients experience oxyhemoglobin desaturation more quickly than normal-weight patients, as shown in Fig. 53.6.

Fifth, the patient's metabolic rate and $\dot{V}O_2$ were raised further by his anxiety, flailing movements of the extremities, and recent trauma. Other systemic causes of elevated $\dot{V}O_2$ include pain, fever, burns, or sepsis.

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Chapter 54
Postoperative Monocular Vision Loss

Engy T. Said and Bishoy Said

The patient is a 31-year-old female scheduled for nasal endoscopy for severe persistent epistaxis. She is 5′5″ tall and weighs 85 kg with a body mass index (BMI) of 31. She has a past medical history of hereditary hemorrhagic telangiectasia (HHT), also known as Osler-Weber-Rendu syndrome (**L-1**), two cerebral arteriovenous malformations (AVMs) in the occipital region, multiple hepatic AVMs, chronic migraines with visual aura, and persistent severe epistaxis requiring, on one occasion, a blood transfusion. Her surgical history is significant for craniotomy in 1997 for removal of multiple AVMs from the right occipital lobe, embolization of pulmonary AVMs in 1997 and 2006, a cesarean section in 2003, and nasal photocoagulation in 2010. She has no known drug allergies and only takes iron for anemia and vicodin as needed for migraines. She denies any use of tobacco, alcohol, or drugs. Family history is only significant for HHT in her mother and maternal aunt. Laboratory analysis showed electrolytes within normal limits. Her starting hematocrit (HCT) was 35.9 % and international normalized ratio (INR) 1.1. Baseline vitals in preoperative area were temperature 98.9 F, blood pressure (BP) 113–123/69–73 mmHg with a mean arterial pressure (MAP) of 83–88 mmHg, heart rate (HR) 74 beats per minute (bpm), respiratory rate (RR) 16 breaths/min, and oxygen saturation (SpO_2) of 100 % on room air (**L-2**). Airway exam was unremarkable, with a class I Mallampati score, >6 cm thyromental distance, full range of motion of her neck, good dentition, and the ability to prognath.

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The patient was brought back to the operating room, American Society of Anesthesiologists (ASA) standard monitors were placed, and she was preoxygenated with 100 % oxygen via face mask. Intravenous anesthesia induction was uneventful with bed turned 180° and attending anesthesiologist present, using 100 μg of fentanyl, 80 mg of lidocaine, and 100 mg of propofol. Easy ventilation via mask was confirmed, and baseline twitches were noted prior to giving 40 mg of rocuronium and another 150 mg of propofol just before intubation. Direct laryngoscopy was atraumatic with a grade 1 view. A 7.0 flexible endotracheal tube (ETT) passed easily between the vocal cords with subsequent end-tidal CO_2 and bilateral breath sounds. ETT was secured, and the patient was connected to ventilator, and 10 mg of dexamethasone were given per surgeon's request. Propofol drip was started at 25 mcg/kg/min for antiemetic purposes. Surgical site was then prepped, draped, and surgery began.

The ear, nose, and throat (ENT) surgeon requested that systolic blood pressures be kept around 80 mmHg for decreased bleeding in the surgical field (**L-3**). Accordingly, the patient's systolic blood pressures were maintained in the mid-80s/50s mm Hg with MAPs of 60–65 mmHg throughout the case with inhalational anesthesia. No intravenous pharmacological intervention was required for BP maintenance. The patient was stable throughout and surgery was complete within 45 min. The patient was then given 4 mg of ondansetron, noted to have 3/4 twitches back and was then reversed with 5 mg of neostigmine and 0.8 mg of glycopyrrolate. On emergence, she was spontaneously ventilating, following commands, and was then extubated uneventfully and taken to the postanesthesia care unit (PACU).

Operative findings included multiple telangiectasias in bilateral nasal cavities, several of which were cauterized. Intraoperatively, the patient received injections of gold solution (0.5 % ropivacaine and 2 % lidocaine + epi 1:50,000) bilaterally into the nasal turbinates, sphenopalatine bones, and septum. She also received bilateral injections of bevacizumab/Avastin (vascular endothelial growth factor [VEGF] inhibitor) 100 mg diluted in 8 mL of normal saline.

Approximately 40 min after arrival to PACU, she complained of difficulty seeing out of her left eye. The attending anesthesiologist was called to the bedside and physical exam revealed blurry left eye vision and intact right eye vision. Bilaterally light perception and extraocular motion were intact. The right pupil was normally reactive to light, whereas the left pupil was sluggishly reactive. No photophobia or proptosis was observed. Stat neurology and ophthalmology consults were obtained. Visual acuity was OD 20/20 OS 20/200. "OS vision preserved in small area to the left of center of visual field, but patient cannot see light peripherally to that area." "Both direct and indirect afferent pupillary reflexes are diminished. Fundoscopic exam showed pale optic disc and blurred retinal vessels." Neuro exam was otherwise normal. Both services concluded that the exam was consistent with postoperative ischemic optic neuropathy of unclear mechanism (**L-4**, **L-5**, **L-6**, **L-7**). Their differential diagnoses included prolonged vasospasm of central retinal artery status post-epinephrine or post-Avastin injection versus small particulate matter in central retinal artery via collateral vessel versus carotid artery dissection. Subsequent discussions by ENT, radiology, and ophthalmology attendings suggested that the

etiology of the incident could also be secondary to injection into the sphenopalatine artery with resultant reverse flow and spasm of a retinal artery with retinal ischemia. CT angiogram was ordered, and patient was admitted for observation (**L-4**, **L-5**, **L-6**, **L-7**).

Interestingly, upon further questioning, she reported that she did have postoperative vision loss of a similar nature after her craniotomy in 1997. She was unable to recall which eye was affected at that time but did report spontaneous resolution of vision loss within 24 h.

Computed tomography (CT) angiogram of the head was performed later that night. In addition to old postcraniotomy changes, it revealed "small pocket of air at the inferior orbital fissure on the left" with "no evidence of abnormal CT density in the left optic nerve" and "normal carotid and vertebral arteries." On postoperative day (POD) #1, exam revealed moderate improvement in visual acuity and peripheral vision in the left eye. Brain and orbit magnetic resonance imaging (MRI) obtained approximately 24 h later on the night of POD#1 revealed "normal size and appearance of optic nerves," "no evidence of infarction to orbits," and "no apparent evidence of gas seen at left orbital apex on prior CT scan."

Lessons Learned

L-1: **What is Osler-Weber-Rendu syndrome**?
Hereditary hemorrhagic telangiectasia (HHT), also known as Osler-Weber-Rendu syndrome, is an autosomal dominant genetic disorder that leads to abnormal blood vessel formation in many organs including lungs, liver, brain, skin, and mucous membranes. The pathophysiology of the disease consists of disorders in the growth and migration of endothelial cells, which leads to telangiectasias and arteriovenous malformations (AVMs) [1]. The exact mechanism by which the HHT mutations influence this process is not yet clear, but it is likely that they disrupt a balance between pro- and antiangiogenic signals in blood vessels. Small vascular malformations, telangiectasias, usually occur in the skin and mucosal linings of the nose and gastrointestinal tract. For this reason, the most common problem is nosebleeds, which affects 90–95 % of people with HHT. The wall of telangiectasias is unusually friable, which explains the tendency of these lesions to bleed. AVMs occur predominantly in the lungs (50 %), liver (30–70 %) and the brain (10 %).

L-2: **The importance of determining a control blood pressure of a patient, especially when deliberate hypotension is going to be used. How do you decide what is the control blood pressure**? **What % decrease intraoperatively for deliberate hypotension is prudent**?
Determining an accurate control blood pressure is considered vital especially in cases of planned hemodynamic manipulation, because with set limits on percentage decrease in BP, where you end up depends on where you start (i.e., control BP). A study done here at the University of California San Diego (UCSD) sought to

Table 54.1 Rare risks of noninvasive blood pressure monitoring [3]

Pain, petechiae, and ecchymoses
Venous stasis, limb edema, and thrombophlebitis
Peripheral neuropathy and compartment syndrome

determine the influence of day-of-surgery anxiety on the preop BP measurements (the "white coat" effect) [2]. The accuracy of these values was compared to baseline BP and clinic BP (serving as two "control" BPs). Baseline BP was defined as medical/surgical clinic BPs over the preceding 7 months, whereas clinic BP is the value obtained in anesthesia preop clinic within 30 days of scheduled surgery. Results have shown a marked increase in blood pressure on day of surgery in comparison to baseline BP, especially in moderately to severely hypertensive patients. In fact, patients with severe hypertension, the first in OR BPs, were found to have a MAP increase by 16.4 mmHg in comparison to baseline BP. Likewise, patients with moderate hypertension, the first in OR BPs, were found to have a MAP increase by 7.4 mmHg from baseline. When first in OR, BP is normotensive; that BP usually reflects baseline BP. For this reason, it is highly recommended to take into account the patient's prehospitalization or clinic BP findings when estimating a control blood pressure, even more so for the moderately to severely hypertensive patient.

Deliberate hypotension, also known as induced hypotension or controlled hypotension, is generally defined as a 20–30 % reduction of baseline MAP. While this may be considered safe in an ASA 1 patient, it may not be well tolerated by patients with other comorbidities.

L-3: **Should deliberate hypotension be used without invasive blood pressure monitoring**? **And how often should a noninvasive blood pressure (NIBP) be checked**?

The standards for basic anesthetic monitoring, as outlined by the ASA, require that every patient receiving a general anesthetic have an arterial blood pressure determined and evaluated at least every 5 min. These standards may be exceeded at any time based on the judgment of the anesthesiologist. Table 54.1 outlines the rare risks of NIBP monitoring, which include pain, edema, and venous stasis [3]. These complications have been associated with prolonged periods of excessively frequent cycling of the cuff and especially in patients with preexisting peripheral neuropathies, diabetes mellitus, impaired limb perfusion, or anticoagulation therapy [4]. And although we do not have a defined minute value beyond which the risk of complications significantly increases, there are case reports of complications with prolonged and frequent cycling of the cuff, including one of rhabdomyolysis in a 6-h procedure where the BP cuff was cycling every 3 min [5]. However, most anesthesiologists would agree that the benefits of capturing twice as much data by cycling a BP cuff every 2.5 min far outweigh the rare risks outlined.

In cases of hemodynamic manipulation, as in deliberate hypotension, it is vital to know the blood pressure at all times. Even if a noninvasive blood pressure is set to cycle every 1 min, prudent information regarding perfusion can be missed. For this reason, the author believes an arterial line, for beat-to-beat monitoring, is necessary in this case. In addition, the general indications for an arterial line (A-line) are as

Table 54.2 Optic nerve perfusion pressure

When Pv > IOP	PP = Pa – Pv
When IOP > Pv	PP = Pa – IOP

Pa Mean arterial pressure, *Pv* mean venous pressure, *IOP* intraocular pressure

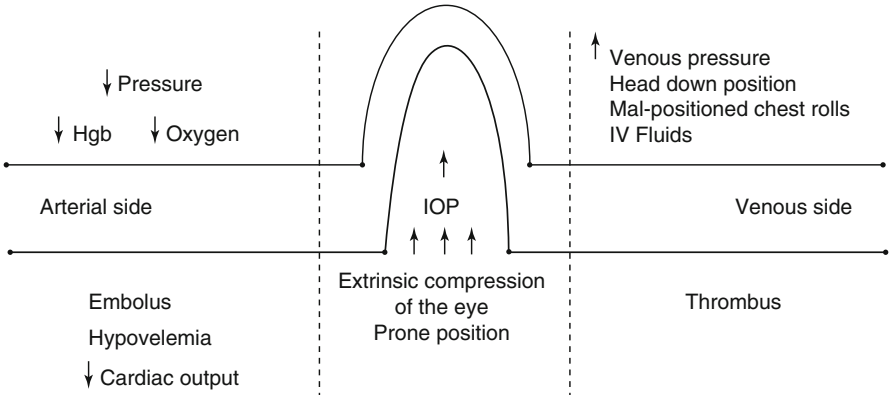

Fig. 54.1 Factors affecting the optic nerve's well-being. Any perioperative decrease in pressure, hemoglobin, or oxygenation would compromise perfusion to the optic nerve. Likewise, heart failure, hypovolemia, or an embolus can lead to ischemic optic neuropathy. The same applies for any increase in intraocular pressure (*IOP*). On the other hand, an increase in venous pressure can lead to impaired venous drainage and "compartment syndrome"

follows. First there are many diseases, conditions, and situations wherein it is desirable to mandatory to know the blood pressure beat-to-beat; deliberate hypotension is one of them. Second, there are many diseases, conditions, and situations where it is desirable to mandatory to be able to repeatedly serially sample blood for numerous laboratory analyses. Third, an arterial line is necessary for pulse pressure and stroke volume variation monitors. All indications for an A-line monitoring fall into one of these three categories.

L-4: **What are the determinants of the optic nerve well-being**?

Perfusion pressure (PP) is an important determinant of optic nerve well-being and is defined as mean arterial blood pressure (Pa) minus intraocular pressure (IOP) or mean venous pressure (Pv), whichever is greater. When IOP is greater than Pv, then PP = Pa – OP. Likewise, if Pv is greater than IOP, then PP = Pa – Pv (Table 54.2).

Blood flow to the optic nerve depends on many factors, as illustrated in Fig. 54.1. These factors play a major role in affecting perfusion pressure. In general, however, moderate changes in IOP and BP have little effect on optic nerve blood flow secondary to autoregulatory mechanisms, which maintain flow under a wide range of these variables.

Ischemic optic neuropathy has also been reported in cases of massive fluid resuscitation. The central retinal vein exits out of the optic nerve, so fluid administration could result in increased IOP or accumulation of fluid in the optic nerve. This may lead to an in internal "compartment syndrome." Likewise, this is the case with impaired venous drainage as in head-down positions, and malpositioned chest rolls.

Table 54.3 Further considerations beyond Fig. 54.1, with regard to factors leading to ischemic optic neuropathy

No autoregulation
Normal amount of edema + small optic nerve
Normal size optic nerve + large amount of edema
Direct trauma from nearby surgical site

Embolism is also a differential for ischemia. The part of the ophthalmic artery that enters the eye at the optic nerve is called the central retinal artery. Any occlusion, whether embolic, thrombotic, inflammatory, or traumatic will lead to decreased blood flow in the retinal blood vessel. Central retinal artery occlusion (CRAO) is discussed in detail in **L-6**. Generally, these patients have predisposing factors including atherosclerosis and coagulopathies.

There are also other factors, aside from those shown in Fig. 54.1, which can contribute to the development of ischemic optic neuropathy (Table 54.3). First, studies have shown, however, that some individuals do not show optic nerve blood flow autoregulation but exhibit a decline in blood flow linearly related to increasing IOP [6]. These patients, including otherwise healthy ones with no known vascular disease, have been shown to have "watershed" areas which predisposes them to optic nerve damage when perfusion pressure is decreased, either after systemic blood pressure decreases or IOP is elevated [7]. At present, however, no clinical technique can reliably detect such patients.

Second, patients have variable optic nerve size, and those with a small nerve size, and a normal amount of edema, will have a higher interstitial pressure and decreased blood flow. A small optic disc is more susceptible to ischemic optic neuropathy, because axons of the optic nerve pass through a narrower opening as they exit the eye and are therefore prone to injury in the presence of edema or decreased blood flow [3].

Third, patients may differ with respect to the amount of edema formed. Some patients will form a significantly larger amount of edema than normal and, even in the setting of a normal-sized optic nerve, will develop ION. This may be due to increased interstitial pressure.

Lastly, nasal and paranasal sinuses surgery pose a special risk of ocular damage. Given the fragility of the bones in that area, a retrobulbar hemorrhage may follow surgical damage leading to indirect nerve compression [3]. Similarly, direct surgical damage to the optic nerve is a consideration given its proximity to the surgical area.

L-5: **Differential diagnosis for the loss of vision in this case**

Postoperative vision loss in non-ophthalmic surgery is usually caused by ischemic optic neuropathy (90 %). Ischemic optic neuropathy (ION) can be divided into anterior and posterior ION. Anterior ischemic optic neuropathy is far more common than posterior. Anterior ION is usually unilateral and presents with nerve findings of swelling on fundoscopic exam [8]. Posterior ION is bilateral and shows no fundoscopic changes (unless the ischemic process extends anteriorly) [9]. Table 54.4 summarizes the findings common to anterior and posterior ION.

Table 54.4 Findings in anterior and posterior ischemic optic neuropathy (ION) [8, 9]

Clinical exam	Anterior ION	Posterior ION
Pupillary exam	Abnormal reflex	Abnormal reflex
Optic nerve head exam	Optic nerve edema (usually sectoral)	Normal nerve appearance
Cup-to-disc ratio[a]	Small cup-to-disc ratio	Normal cup-to-disc ratio
Laterality	Unilateral	Bilateral
Etiology	DM, HTN, collagen vascular disease	Anemia, hypotension, medications, surgical blood loss, GI bleed, trauma, dialysis

[a]The cup is the central pale portion of the nerve. The cup-to-disc ratio is the ratio of the size of the cup to the overall diameter of the nerve

Table 54.5 Ophthalmic findings in central retinal vein occlusion (CRVO) and central retinal artery occlusion (CRAO) [10, 11]

Clinical exam	CRVO	CRAO
Visual acuity	Normal to poor (20/20 – hand motion)	Markedly decreased (hand motion)
Pupillary exam	Abnormal reflex	Abnormal reflex
Optic nerve head exam	Nerve swelling	Normal
Retinal vessels exam	Tortuous and dilated	Attenuated (narrowed)
Retinal exam	Flame-shaped hemorrhages, macular edema	Cherry red spot
Prognosis	Depends on the extent of occlusion (range from 20/20 to no light perception)	Poor after 90 min of nonperfusion
Laterality	Unilateral	Unilateral
Etiology	Usually embolic (not ischemic)	Usually embolic (not ischemic)

This patient presented with acute, unilateral painless loss of vision. She had decreased visual acuity, abnormal pupillary reflex, and abnormal fundoscopic exam of the nerve head and vessels. These findings are consistent with anterior ischemic optic neuropathy.

L-6: **Discuss retinal findings in retinal ischemia**

Central retinal vein occlusion (CRVO) and central retinal artery occlusion (CRAO) are usually unilateral phenomenons that are caused by embolic events, not ischemic causes. Fundoscopic findings are more pronounced in CRVO because the occlusion occurs in the vein resulting in backup of blood and fluid in the eye [10, 11]. This presents on fundoscopic exam with dilated tortuous veins, bleeding in the retina, and swelling of the optic nerve. Table 54.5 highlights similarities and differences between these two disease states.

Although this patient's exam showed abnormal pupillary reflexes (which can be consistent with ION or CRAO/CRVO), the fundoscopic exam showed blurred retinal vessels, rather than flame-shaped hemorrhages and/or tortuous and dilated vessels, as in CRVO, or the classic "cherry red" spot of CRAO. Likewise, this patient does not have medical conditions that would predispose her to an embolic

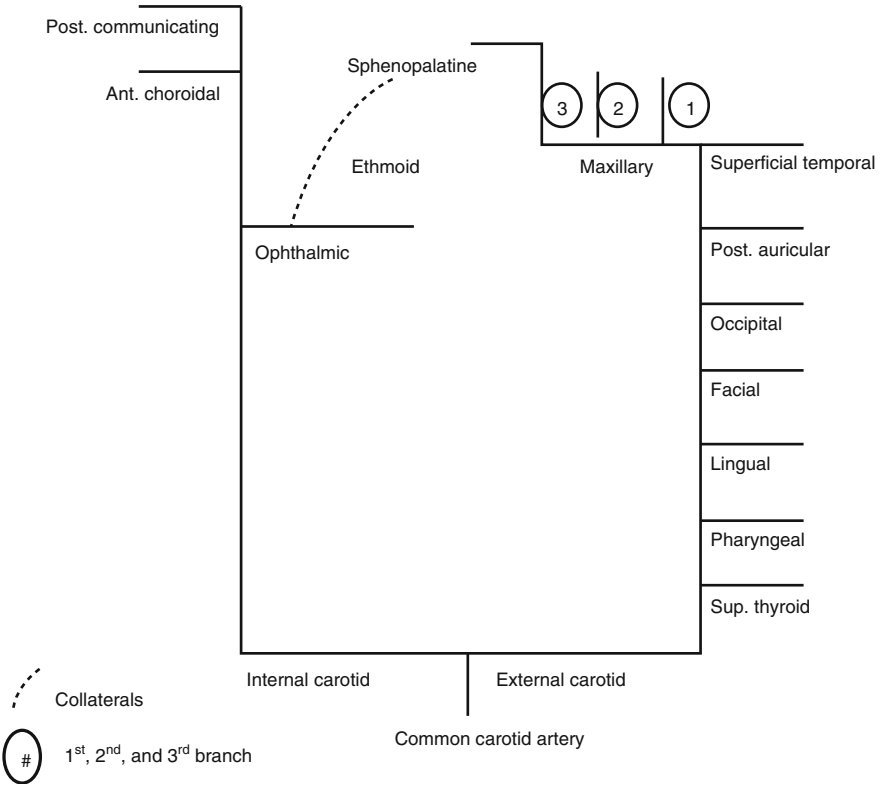

Fig. 54.2 Schematic diagram of the common carotid artery and its major branches. The eye is supplied by the ophthalmic artery, which is the first branch of the internal carotid artery. There are multiple sources of collateral circulation connecting the internal and external carotid arteries including the ethmoidal arteries in the medial orbit

phenomenon leading to CRAO/CRVO. It is also unlikely for vision to spontaneously improve following a CRAO. For these reasons, it is unlikely she suffered CRAO/CRVO, yet it is important to consider these possibilities as part of the differential for monocular postoperative vision loss.

L-7: **Where is the sphenopalatine artery, what does it supply, and how could it have caused vision loss in this case**?

The sphenopalatine artery (Fig. 54.2), also known as the nasopalatine artery, is a branch of the maxillary artery, which passes through the sphenopalatine foramen into the cavity of the nose giving off posterior lateral and posterior septal nasal branches [12]. It is responsible for the most serious posterior nosebleeds.

In this case, there may have been reverse flow through the sphenopalatine artery of the external carotid to the ethmoidal arteries of the internal carotid back to the ophthalmic artery supplying the optic nerve. This reverse flow may have carried high concentrations of vasoconstictor epinephrine and thereby causing ischemic optic neuropathy.

However, given her history of a similar monocular loss of vision that occurred following a craniotomy in 1997, it is less likely that reverse flow is the etiology in this case. The recurrence of this rare monocular vision loss, after two different surgical procedures, makes it more likely that she suffered anterior ischemic optic neuropathy secondary to a persistent factor that compromised optic nerve perfusion, rather than two variable etiologies leading to the same outcome. As discussed in **L-4**, there are several determinants of optic nerve well-being, including a few that predispose certain patients to ION more than the general population. These factors, listed in Table 54.3, include lack of autoregulation, a small optic nerve size, or increased edema in the setting of a normal optic nerve size. There are unfortunately no clinical techniques that differentiate between these predisposing factors, and despite an anesthesia provider's best efforts to minimize any compromise to the optic nerve perfusion, it may be inevitable that these patients suffer a form of ION.

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Chapter 55
Delayed Emergence After Aneurysm Clipping

Sun Choe Daly

The patient is a 59-year-old male, weighing 82 kg and measuring 74 in. tall, who presents for elective clipping of his left anterior communicating artery aneurysm. His aneurysm was discovered incidentally during a work-up of his lung adenocarcinoma with his only complaint being mild headaches. The magnetic resonance brain angiogram indicated the aneurysm to be located at the junction of the anterior communicating artery and the A1 segment of the left anterior cerebral artery (**L-1**) without evidence of subarachnoid hemorrhage (SAH) (**L-2, L-3**). The aneurysm was lobulated and measured one centimeter in diameter (**L-4**).

The patient's past medical history is significant for adenocarcinoma of the lung status post radiation treatment, coronary artery disease, congestive heart failure New York Heart Association (NYHA) Class I, hyperlipidemia, hypertension, and cocaine abuse (**L-5**). The patient's past surgical history is significant for appendectomy. The patient's baseline vital signs were heart rate of 80 beats per minute (bpm), blood pressure (BP) of 160/100 mmHg, and respiratory rate of 12 bpm, and oxygen saturation was 98 % while breathing room air. His laboratory examination showed hemoglobin of 12.1 mg/dL and a sodium level of 140 meq/L and showed no evidence of coagulopathy.

After proper preparation, the patient was brought to the operating suite for the aneurysm clipping (**L-6**). The patient had a radial arterial line placed prior to induction with good subcutaneous local anesthetic infiltration. He was given a large narcotic load with titrated amounts of fentanyl until his respiratory rate was less than 10 and the patient appeared somnolent. He was then induced with propofol, relaxed with rocuronium, and intubated without any hemodynamic disturbances. The patient was given an additional bolus of propofol during head pinning to avoid any alterations in blood pressure. His state of anesthesia was maintained with a balance

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of nitrous oxide in oxygen and a propofol infusion. His blood pressure was controlled with his mean arterial BP ranging between 70 and 80 mmHg. During attempted clipping of the aneurysm, there was rupture of the aneurysm (**L-7**). There was copious hemorrhage, estimated around 200–300 cc, while the surgeons struggled to gain control. The BP was maintained during this event, and there were no episodes of hypotension. The aneurysm was successfully clipped, and the rest of the case proceeded uneventfully.

At the end of the case, the patient was breathing spontaneously but was unresponsive to tactile or verbal stimuli (**L-8**). The patient remained intubated and was transported to the postanesthesia care unit. The patient's pupils were pinpoint, and small increments of naloxone were given. After 30 min, the patient was noted to move his right upper and lower extremities but no spontaneous movement was noted on his left side.

The patient was taken urgently to angiogram (**L-9**)**.** The computed tomography (CT) angiogram demonstrated decreased flow in the right A2 portion of the right anterior cerebral artery. The patient was deemed to have most of his blood supply to his right A2 artery from the left side due to a congenital defect with little native blood flow from his right side. The patient was extubated on postoperative day 3 with moderate weakness of his left upper and lower extremities.

Lessons Learned

L-1: What are the anatomical segments of the circle of Willis?
The circle of Willis is where the majority of aneurysms are located, particularly at branch points of the circulation. There are nine segments that make up this vital circle, and they are often referred to in neurosurgery to provide a detailed description of the location of the aneurysm. Figure 55.1 illustrates the segments [1].

L-2: What are common systemic complications in a patient with a subarachnoid hemorrhage?
There are many complications that are associated with SAH. The major ones are listed by system and what the treatments are if any [2].

Neurologic

Cerebral vasospasm causing cerebral ischemia is the major cause of morbidity and mortality from SAH. Onset is usually 3–12 days after initial hemorrhage, and the risk is present for an average duration of 14 days. The incidence of vasospasm in patients with SAH can be up to 60–70 % as can be seen with diagnostic studies, but the clinical presentation becomes apparent in 20–30 % of patients [3]. The current strategy to prevent vasospasm is to start patients on oral nimodipine 60 mg orally every 4 h for 21 days [4] and employing hemodilution, hypervolemia, and hypertension (HHH) therapy [5], which can be ramped up once vasospasm occurs [6]. Once there is vasospasm, additional strategies that may be employed are balloon angioplasty and intra-arterial injection of papaverine, nicardipine [7], or a variety of vasodilators as well

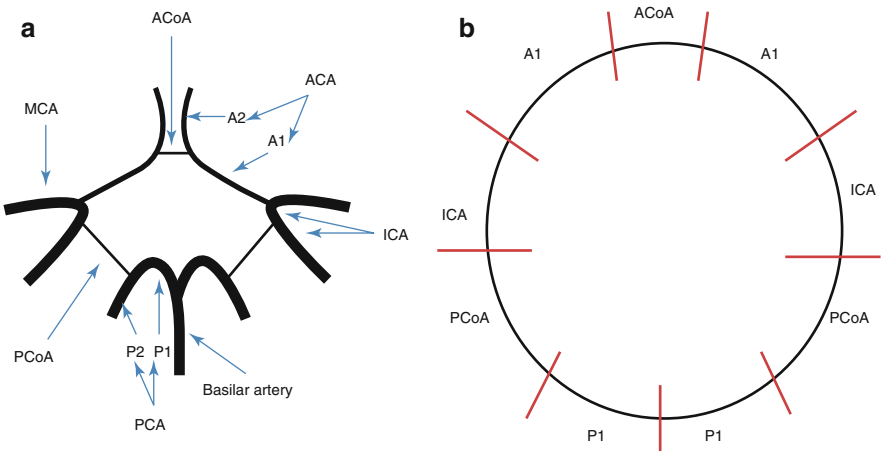

Fig. 55.1 (**a**) Graphical scheme of the segments of the circle of Willis: *ACA* anterior cerebral artery which is made up of both A1 and A2 segments, *ACoA* anterior communicating artery, *ICA* internal carotid artery, *PCoA* posterior communicating artery, *PCA* posterior cerebral artery which is made up of both P1 and P2 segments [1]. (**b**) Complete circle of Willis demonstrated with highly schematized segments

Table 55.1 Practical recommendations on prophylaxis of vasospasm after SAH [6]

Before aneurysm is secured
Avoid antihypertensive medications (unless systolic blood pressure >140, diastolic blood pressure >90 mmHg.)
Avoid diuretics
Nimodipine 60 mg orally every 4 h for 21 days
After aneurysm is secured
Maintenance with crystalloid intravenous fluid at 100–150 cc/h
5 % albumin (250 cc IV three times daily if no cardiac or pulmonary comorbidities)
Keep central venous pressure 10–12 mmHg
Monitor serum sodium and maintain above 135 meq/L with NaCl tabs or 3 % NaCl intravenously

as instillation of intracisternal vasodilators such as papaverine [8]. Table 55.1 shows some recommendations on how to prevent vasospasm [6]. Table 55.2 shows a more aggressive approach to the treatment of vasospasm [6].

Pulmonary

Neurogenic pulmonary edema and pulmonary hypertension are possible complications, and incidence ranges from 2 to 49.5 % after SAH [9].

Cardiovascular

One can see various nonspecific electrocardiogram changes such as T wave inversions, nonspecific ST segment depressions or elevations, prolonged QT intervals, and Q waves [10]. The patient may suffer from a hypotensive shock state due to the resulting injury or myocardial stunning from a large catecholamine release. The more severe the neurologic damage, the more severe the alteration may be in cardiac function,

Table 55.2 Practical recommendations on treatment of vasospasm after SAH [6]

Place central venous pressure catheter (CVP)
Consider pulmonary artery catheter placement for monitoring cardiac indices and pulmonary artery wedge pressures (PAWP) every 4–6 h
Target CVP or PAWP to 12–16 mmHg with crystalloid or albumin fluid boluses
Keep systolic blood pressure >160 mmHg
If mean arterial blood pressure (MAP) is used, keep MAP >120 mmHg
Vasopressors such as phenylephrine, dopamine, and dobutamine to keep cardiac index 5–6
Avoid antihypertensive drugs
Consider endovascular therapy

Table 55.3 Hunt and Hess grading scale for SAH [12]

Grade	Clinical description
I	Asymptomatic or minimal headache and slight nuchal rigidity
II	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
III	Drowsiness, confusion, or mild focal deficit
IV	Stupor, moderate to severe hemiparesis, and possibly early decerebrate rigidity and vegetative disturbance
V	Deep coma, decerebrate rigidity, and moribund appearance

although reversible. This phenomenon is likened to Tako-tsubo cardiomyopathy due to the fact that both are associated with left ventricular ballooning and dysfunction from excessive catecholamine release. Due to the similar appearance, some suggest a more encompassing term to describe this cardiac state, "acute ballooning cardiomyopathy" [11]. This may pose a problem in a patient who would normally be placed in HHH therapy since these goals would stress the heart further. In a patient with severe cardiac dysfunction and shock, an intra-aortic balloon pump may be beneficial.

Metabolic

There are some common electrolyte abnormalities associated with SAH: hyponatremia, hypokalemia, hypocalcemia, and hypomagnesemia. Hyponatremia is the most concerning electrolyte abnormality due to its association with vasospasm and increased brain swelling. The differential diagnoses consist of cerebral salt wasting syndrome, syndrome of inappropriate antidiuretic hormone secretion (SIADH), or iatrogenic causes. The treatment for SIADH would be fluid restriction and for cerebral salt wasting is administration of normal saline or hypertonic saline. In patients who are suffering from SAH, normal or hypervolemia is preferable to hypovolemia, which could worsen vasospasm; therefore, both possible etiologies are usually treated with administration of saline.

L3: What are some classifications of subarachnoid hemorrhage and what do they tell us?

The most commonly used scales for grading SAH are Hunt and Hess, the World Federation of Neurologic Surgeons Grade, and the Fisher Scale [12–14]. These scales are used as guidelines as to when surgery should be performed and as predictors of morbidity and mortality. According to authors Hunt and Hess (Table 55.3), surgery for patients in Grade I and II was performed without delay. In patients with

Table 55.4 World Federation of Neurological Surgeons Grading Scale for aneurysmal SAH. GCS, Glasgow Coma Scale [13]

Grade	GCS score	Motor deficit[a]
I	15	Absent
II	13 or 14	Absent
III	13 or 14	Present
IV	7–12	Present or absent
V	3–6	Present or absent

[a]Excludes cranial neuropathies, but includes dysphasia

Table 55.5 Fisher grading scale of cranial computed tomography (CCT) [14]

Grade	Findings on CCT
1	No subarachnoid blood detected
2	Diffuse or vertical layers ≤1 mm
3	Localized blot and/or vertical layer >1 mm
4	Intracerebral or intraventricular clot with diffuse or no subarachnoid hemorrhage

Table 55.6 Surgical mortality and major morbidity of subarachnoid hemorrhage according to clinical grades [15]

Grade (Hunt and Hess)	Mortality (%)	Morbidity[a] (%)
0	0–2	0–2
I	2–5	0–2
II	5–10	7
III	5–10	25
IV	20–30	25
V	30–40	35–40

[a]Morbidity defined as neurologic dysfunction

higher grades, the patients were treated conservatively, and surgical intervention was performed when the patients could be reclassified as Grade I or II. The World Federation of Neurological Surgeons Grading Scale for aneurysmal subarachnoid hemorrhage (Table 55.4) uses the Glasgow Coma Scale (GCS) score and the presence of a motor deficit to determine the severity [13]. The Fisher Scale (Table 55.5) is based on the radiologic severity of the subarachnoid hemorrhage on computerized tomography scan [14]. Table 55.6 shows the correlation between the severity of subarachnoid hemorrhage and the morbidity and mortality of the patients who undergo surgical clipping [15].

L4: What are the general preoperative considerations in a patient who presents for an aneurysm clipping?

There are several considerations for a patient who presents for an aneurysm clipping. One must assess the patient's neurologic condition and history, complete with an independent neurologic exam to verify that the patient's neurologic status is stable. One must be certain to know about any specific neurologic deficits, symptoms of increased intracranial pressure, nausea or vomiting, decreased mentation, visual changes, headaches, or seizure history. You must review the available radiologic studies to evaluate the location of the aneurysm and the clinical grade of subarachnoid hemorrhage (SAH) if any present (see **L-3**). These studies will also indicate any signs of increased intracranial pressure (ICP) such as diminished ventricle size, patency of the fourth ventricle, midline shift, or any herniation.

Table 55.7 Mean flow velocity values in the middle cerebral artery [19–21]

	Below normal	Normal	Above normal	Severely elevated
Mean flow velocity in cm/s	<50	62 ± 12	>74	>120

Table 55.8 New York Heart Association classification [25]

Class	New York Heart Association functional classification
I	Patients have cardiac disease but without resulting limitations of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or angina pain
II	Patients have cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or angina pain
III	Patients have cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitation, dyspnea, or angina pain
IV	Patients have cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency or of the angina syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased

Some patients will have intraventricular catheters in situ (normal limits for intraventricular pressure are 1–15 mmHg) [16].

Some patients may have had transcranial Doppler (TCD) examinations to evaluate ICP, and if so the reader should be familiar with the parameters of TCD. TCD measures the blood flow velocities of the middle cerebral artery. These velocities are then used to calculate a pulsatility index, which has been shown to correlate with increases in ICP. In a patient with increased ICP, the pulsatility index is elevated [17]. The normal value for pulsatility index is 0.7 ± 0.3 and is calculated from the difference in the systolic and diastolic flow velocity divided by the mean flow velocity [18]. Table 55.7 shows the normal and abnormal values for mean flow velocity as measured by TCD [19–21].

One must communicate with the surgical team for positioning and any need for special monitors or lines such as a multi-orifice catheter for venous air embolism retrieval, central lines, or lumbar cerebral spinal fluid drain. You must be prepared to manipulate the patient's physiology to aid the surgeons if necessary such as having barbiturates to induce a barbiturate coma, having adenosine for a period of cardiac standstill [22], inducing hypotension, and inducing hypertension.

One must optimize the patient's physiologic disturbances if time allows such as correcting electrolytes and coagulopathy prior to starting the procedure [23].

L-5: What is the NYHA classification for heart failure?

The NYHA classification for heart failure was originally published in 1928 to categorize patients with heart failure based on symptoms with activity; the classification is a subjective clinical tool that has proven to be very accurate in predicting mortality [24]. Table 55.8 shows the NYHA classifications [25].

L6: What are intraoperative considerations for aneurysm clipping?
The most important goal is to avoid changes in the transmural pressure by maintaining stable hemodynamic parameters. One may want to place the arterial line prior to induction and intubation, under controlled conditions with good local analgesia. Avoiding sympathetic responses to painful stimuli is important such as during head pinning and direct laryngoscopy. The surgeons may request that measures be taken to reduce intracranial pressure by use of diuretics, switching to total intravenous anesthesia, and utilizing hyperventilation strategies. Ensure complete muscle relaxation during the procedure to avoid bucking or coughing. Avoidance of hyperthermia and hyperglycemia are also key tenets to the management of these patients.

L-7: What are the anesthetic implications when you have intraoperative aneurysm rupture?
One must be in close communication with surgical team in order to facilitate resuscitation and establish hemodynamic goals during the hemorrhage. Initially, the goal is to maintain perfusion of the distal parts of the brain with mean arterial pressures that are near baseline or slightly elevated. In some instances, the surgeons will require induced hypotension or cardiac standstill for short periods of time in order to gain control of the ruptured vessel. You must ensure adequate volume status with repletion of blood loss as necessary.

L-8: What are the differential diagnoses for delayed awakening in a patient after aneurysm clipping?
One must consider all the causes of delayed awakening after general anesthesia, which are beyond the scope of this chapter. The specific causes inherent to aneurysm clipping cases are tension pneumocephalus, ruptured aneurysm, stroke, vasospasm, and hyponatremia. One must consider rare causes; such as in this case of anomalous anatomy where the patient was dependent on collateral flow from the contralateral side to perfuse the right anterior cerebral arterial territory.

L-9: What are the diagnostic steps to delayed emergence in a patient after aneurysm clipping?
After the anesthetic or metabolic causes are ruled out, one must visualize vessels and the brain parenchyma. The quickest method to scan the intracranial contents is via CT scan, which can be combined with IV contrast to visualize the vessels to rule out re-rupture, vasospasm, or pneumocephalus. A bedside test to rule out vasospasm is via transcranial Doppler if available.

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Part VII
Pain and Regional Anesthesia-Related Cases

Chapter 56
Unintentional Dural Puncture in a Patient with Severe Preeclampsia

Michael Bronson

The patient is a 25-year-old gravida 2 para 0 at 33 weeks gestation with past medical history significant for depression and diabetes mellitus type 1 who was admitted for severe preeclampsia (**L-1**). The obstetric (OB) anesthesia team was notified of planned cesarean section, and the patient was taken back to the operating room for placement of neuraxial blockade (**L-2**, **L-3**). The initial plan was to place a combined spinal-epidural for analgesia using the standard epidural kit containing a 17-g Tuohy needle; however, the dura was breached at 4 cm with flow of cerebrospinal fluid (**L-4**). In efforts to reduce the chance of a postdural puncture headache (PDPH), the catheter was threaded intrathecally and left at 8 cm at the skin (**L-5**). Appropriate analgesia to T4 dermatome level was obtained with an intrathecal injection of 1.4 mL of 0.75 % bupivacaine (10.5 mg), 10 μg of fentanyl, and 0.1 mg of morphine. The cesarean section was uneventful, and the spinal catheter was discontinued 24 h later. The patient was asymptomatic except for minor back pain. The following day the anesthesia service was asked to evaluate the patient again as it was noted that clear fluid was leaking at the site of catheter insertion. On examination, the site was clean without erythema, swelling, or tenderness, and she remained asymptomatic. She continued to have persistent leakage at the site over the next few hospital days, and on postoperative day #4, neurosurgery was consulted. Neurosurgery proceeded to place a subcutaneous figure-of-eight stitch using 3-0 nylon, and the patient was discharged home with instructions to have her primary care physician remove the stitch in 10–14 days or return to the emergency department if leakage persisted (**L-6**). The patient's leakage ceased following stitch placement, and she remained asymptomatic.

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Table 56.1 Criteria for the diagnosis of mild to severe preeclampsia

	Blood pressure	Proteinuria	Other signs/symptoms
Mild	≥140 mmHg systolic OR ≥90 mmHg diastolic	≥0.3 g/24 h	Peripheral edema Decreased urine output (By definition, >500 ml/24 hr is NOT oliguria)
Severe	≥160 mmHg systolic OR ≥110 mmHg diastolic	≥5 g/24 h	Oliguria of <500 mL/24 h Headaches/visual disturbances Pulmonary edema/cyanosis Epigastric/right upper quadrant pain Impaired liver function Thrombocytopenia HELLP syndrome Fetal growth restriction

Diagnosed after 20 weeks gestation in a woman with previously normal blood pressure
HELLP syndrome: Hemolysis, elevated liver enzymes, and low platelets

Lessons Learned

L-1: What is the definition of mild and severe preeclampsia?
Preeclampsia is defined by hypertension and proteinuria after 20 weeks gestation with a reported incidence of 5–8 % [1, 2]. Additional signs and symptoms may be present including oliguria, headaches, vision disturbances, and laboratory abnormalities (Table 56.1). It should be noted that only one positive indicator is needed for the diagnosis of preeclampsia; thus, treatment should not be delayed for the results of a 24-h urine analysis. Preeclampsia is categorized as either mild or severe based on the most recent practice bulletin from the American Congress of Obstetricians and Gynecologists (ACOG) reviewing the diagnosis and management of preeclampsia and eclampsia [1]. A very broad review of the literature by this author did not uncover clear diagnostic criteria for "moderate preeclampsia," as it is often discussed as "mild-moderate" or "moderate-severe" without definitive boundaries.

L-2: Should a preeclamptic patient receive an intravascular volume load if an epidural or spinal is going to be performed?
Hypotension following neuraxial anesthesia is one of the most common complications experienced in obstetric anesthesia. Sympathectomy leading to decreased systemic vascular resistance in addition to increased venous capacitance and pooling of a large portion of the patient's blood volume are thought to be the major causes of the drop in blood pressure [3]. To combat some of these effects, it has been recommended to bolus intravenous fluids prior to or at the time of neuraxial blockade. Patients with preeclampsia, especially severe preeclampsia, may be extremely hypovolemic due to extravascular losses of fluid and proteins in addition to vasoconstriction, so intravascular volume expansion is essential in this patient population [4, 5] (However, to be complete, see Aya et al. in **L-3**). Not surprisingly, some studies have suggested volume deficits of 600–800 mL/m^2 in preeclamptic patients [6]. With this in mind, based on the average height of a United States female of approximately

162 cm as reported by the Centers for Disease Control and Prevention (CDC), preeclamptic patients can be estimated to have a volume deficit of 1,575–2,100 mL, further highlighting the importance of a volume load at the induction of neuraxial blockade [7]. Several investigations into spinal anesthesia for preeclamptic patients undergoing cesarean delivery have used fluid boluses consisting of 1,500–2,000 mL of crystalloid 20 min prior to spinal anesthesia, which have shown to be an adequate volume load in terms of minimizing hypotension from the resulting sympathecomy [8, 9]. Additionally, from what we know about intravenous fluid kinetics, it is highly likely that a colloid preload and a crystalloid or colloid coload would also minimize decreases in blood pressure following the induction of spinal anesthesia [10–13].

L-3: Which anesthetic technique is preferred for cesarean delivery in a patient with severe preeclampsia?

The selection of anesthetic is of great importance, as patients with preeclampsia often require cesarean delivery. General anesthesia in the obstetric population carries significant risks with respect to airway management and fetal depression, so general anesthesia is therefore used mostly when regional methods are not an option such as in the preeclamptic patient with severe coagulopathy. It has previously been suggested that epidural anesthesia be the preferred method compared to a spinal technique due to the ability to slowly titrate up the level of anesthesia and sympatholysis [4]. More recently however spinal anesthesia for cesarean delivery, even in patients with severe preeclampsia, has been shown to be an acceptable approach [8, 9, 14]. In a retrospective study, Hood and Curry looked at severely preeclamptic patients receiving either a spinal or epidural technique for cesarean delivery [14]. They identified 103 patients receiving a spinal and 35 receiving an epidural and found that the lowest mean blood pressures, use of ephedrine, APGAR scores, and incidence of intensive care unit (ICU) admission were similar for both groups, although the patients receiving a spinal technique did require significantly more intraoperative crystalloid.

Given the vasoconstricted and hypovolemic state of patients with preeclampsia, it would be expected that this patient population would have an extreme response to the rapid sympatholysis from spinal anesthesia compared to non-preeclamptic patients. In spite of this, recent studies have suggested that this may not be the case. Aya and coauthors investigated the incidence and severity of hypotension following spinal anesthesia in patients diagnosed with severe preeclampsia [8]. In this prospective cohort study, they compared 30 patients with severe preeclampsia to 30 healthy patients and found that patients with severe preeclampsia had a lower incidence of clinically significant hypotension, defined as a systolic blood pressure less than 100 mmHg or a 30 % decrease in mean blood pressure, following induction with spinal anesthesia(16.6 % vs. 53.3 %; $P = 0.006$).

L-4: What is the incidence of postdural puncture headache (PDPH) when a large-bore (17 or 18 g) Tuohy needle causes unintentional dural puncture?

Unintentional dural puncture during routine placement of a Tuohy needle into the epidural space occurs in 0.4–0.6 % of patients [15, 16]. After the dura has been breached with a large-bore Tuohy epidural needle, the incidence of PDPH ranges from 76 to 85 % [17].

L-5: Are there methods to decrease the chance of the patient developing PDPH following unintentional dural puncture?

Normal cerebrospinal fluid (CSF) volume is about 150 mL and is produced at a rate of approximately 0.35 mL/min [4]. It is thought that loss of CSF as a result of dural puncture leads to a diminished cushioning effect on the brain from the decreased amount of CSF around the brain. The lack of CSF around the brain creates traction on the meninges, meningeal vessels, and cranial nerves and is responsible for many of the consequences, including PDPH (Fig. 56.1a) [4]. Consequently techniques aimed to reduce the incidence of PDPH attempt to maintain CSF volume by replacing losses, decreasing the rate of loss, and increasing production through hydration.

Many maneuvers have been trialed to decrease the incidence of PDPH following inadvertent dural breach (Fig. 56.1b). No single technique has been shown to be entirely effective. Kuczkowski and Benumof found in a series of seven patients that after unintentional dural puncture, (1) injecting the CSF in the glass syringe back into the subarachnoid space, (2) inserting an intrathecal catheter and leaving it in place for 12–20 h, (3) injecting 3–5 mL of normal saline into the intrathecal space, and (4) administering a local anesthetic bolus followed by continuous intrathecal labor analgesia, that PDPH only occurred in one of the seven patients (14 %) [18]. Other studies claim to have reduced the incidence of PDPH from 0 to 32 % [19–22]. Since epidural blood patch is considered the "gold standard" for treatment of a PDPH, many anesthesiologists will place a prophylactic epidural blood patch following delivery and resolution of sensory and motor blockade. A recent review article by Agerson and Scavone evaluated six different studies, which included one randomized, double-blinded control trial [23]. The authors concluded that a prophylactic blood patch does not decrease the incidence of PDPH, but it may decrease the intensity of headaches and/or duration of symptoms.

L-6: What are the longterm implications of unintentional dural puncture?

A study by MacArthur and coauthors found that in patients that experienced an unintentional dural puncture during epidural placement, 23 % of them experienced a headache lasting longer than 6 weeks [24]. Long-term follow-up was not reported in this study, but further investigations into the subject have indicated that there is in fact an increased incidence of chronic headache in these patients. Indeed, a more recent study by Webb and coauthors followed 40 patients who were identified as receiving an unintentional dural puncture for up to 24 months following delivery [25]. Compared to a matched control group, the incidence of chronic headaches as determined from their questionnaires was 28 % in the study group vs. 5 % in the matched control ($P<0.05$). Almost 20 % of the patients suffering from chronic headaches reported that they were significantly disabled as a result. The incidence of chronic backache was also significantly greater in the study group, 43 % compared to the matched controls, 15 % ($P<0.05$).

Chronic intracranial subdural hematoma is a rare complication from unintentional dural puncture. It is postulated that the decrease in intraspinal/intracranial pressure as the result of a CSF leak causes traction on and tearing of delicate dural vessels in the cranium [26]. Presenting symptoms of chronic subdural hematomas

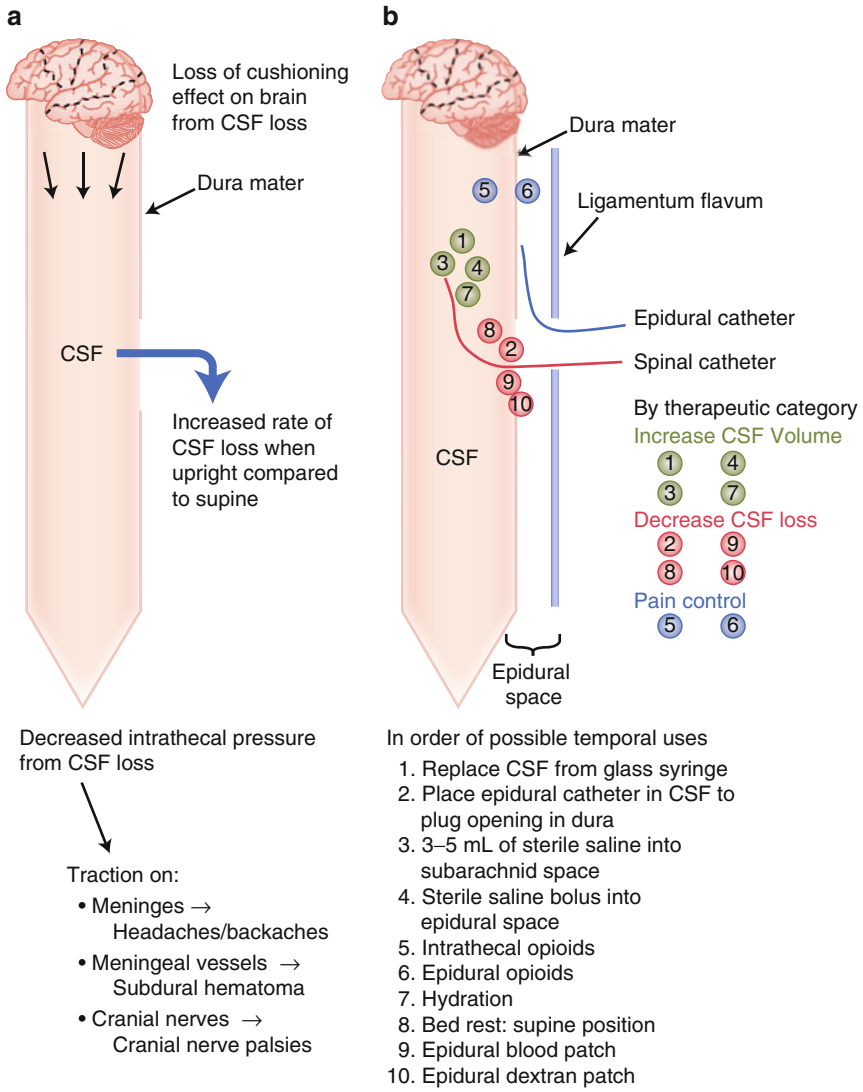

Fig. 56.1 (**a**) Pathophysiology of symptoms following unintentional dural puncture. (**b**) Techniques for the prevention of a postdural headache following unintentional dural puncture

are often headaches which are commonly thought to be postdural puncture headaches and treated with supportive care and/or epidural blood patch. Further investigation with CT/MRI of the head would be needed to further investigate chronic subdural hematoma as a possible cause of the headache when it is suspected as a possible etiology. A case series by Zeidan and coauthors reported 21 cases of intracranial subdural hematomas following dural puncture during epidural placement [27]. Of the 21 cases, 12 were determined to be chronic in nature (lasting >3 weeks).

There have also been many reports of cranial nerve palsies as a result of unintentional dural puncture. The proposed pathophysiology is a pressure imbalance intracranially as a result of CSF leakage leading to traction on various cranial nerves. Most reports have involved the abducens, facial, and vestibulocochlear nerves [28]. Cases of transient hearing loss as a result of dural puncture have been described with resolution following placement of epidural blood patch [29]. In a study by MacArthur and coauthors using questionnaires to follow up patients who suffered an unintentional dural puncture, it was found that 2 of 74 (2.7 %) patients complained of visual disturbances long term and 1 of the 74 patients (1.4 %) complained of long-term auditory impairment [24].

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Chapter 57
Complex Regional Pain Syndrome

Timothy Furnish

The patient is a 52-year-old female with a past medical history of complex regional pain syndrome (CRPS) type I (**L-1, L-2, L-3**) who is scheduled for left needle localization lumpectomy for a breast mass of unknown type. Her preoperative ultrasound and mammogram were not consistent with tumor or cyst. The suspicion was that this mass represents a retained foreign body from prior port-a-cath removal. Her CRPS began in 1994 after a left thumb sprain (**L-3**) but is now present in all four extremities but worst in left upper extremity. Additional past medical history includes severe pain flairs with prior surgery (**L-5**), which have all required inpatient admission, depression, cervical epidural abscess, and subsequent decompression.

Outpatient medication includes:

Hydromorphone 32 mg orally (PO) every 4 h (q4h) averaging 4/day (**L-5**)
Diazepam 50 mg at bedtime (qHS)
Ibuprofen 800 mg every 6 (q6) h
Rizatriptan 10 mg as needed (prn) migraine
Flector (diclofenac) patch prn
Lidoderm patch prn
Methocarbamol 750 mg 2 tabs prn (rarely uses)
Metaxalone 800 mg 2 tabs prn (rarely uses)
Bupropion 150 mg twice daily (BID)
Citalopram 40 mg PO qday
Lidocaine compounded to 20 %, self-administered subcutaneous injections prn in various painful areas

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Allergies: meperidine and morphine (N/V and hives), rifampin (hives)

Preoperatively intravenous (IV) access (**L-6**) was obtained in the right upper extremity with the assistance of ultrasound guidance. The patient was given a one-time dose of pregabalin 150 mg PO (**L-5, L-7**) 1 h prior to surgery. A left supraclavicular nerve block (**L-5, L-6, L-7**) was then placed under ultrasound guidance to prevent flair of pain from intraoperative positioning in the extremity most severely affected by CRPS. The block was initiated with 20 mL of 0.5 % ropivacaine. Paravertebral nerve blocks (**L-5, L-6, L-7**) were then placed at T3, T4, and T5 under ultrasound guidance with infiltration of 0.5 % ropivacaine for postoperative pain control in the surgical field. The patient was taken back to the operating room. Induction of anesthesia proceeded with intravenous ketamine 50 mg (**L-6, L-7**) supplemented by lidocaine 100 mg, midazolam 5 mg, fentanyl 50 μg, and propofol 100 mg. Laryngeal mask airway placement was uneventful, and anesthesia was maintained with propofol/ketamine mixture (100 mL of propofol 1 % with 50 mg ketamine) infusion at 100 μg/kg/min of propofol and intermittent bolus doses of fentanyl totaling 250 μg with inhaled oxygen, nitrous oxide, and sevoflurane. Total operative time was 45 min. In the recovery room the patient reported 8–10/10 pain primarily in her extremities, and additional fentanyl 200 mcg along with hydromorphone 10 mg was administered intravenously in divided doses over 1 h. She was subsequently transferred to the floor. Her home dose of oral hydromorphone 32 mg PO q4 (**L-5**) hours scheduled around the clock was resumed with patient-controlled analgesia (PCA) hydromorphone 1 mg (**L-5**) with a 15-min lockout for additional postoperative pain. She was transitioned off IV opioids and discharged home on POD #2 with pain scores near her baseline preoperative pain level.

Lessons Learned

L-1: What is CRPS?

Complex regional pain syndrome is a chronic neuropathic pain disorder that involves one or more extremities and presents with a constellation of sensory, motor, and vascular changes along with severe pain. The pain characteristics include intense burning, hyperalgesia, and allodynia. CRPS is associated with prominent autonomic features including edema, altered sweating, skin temperature, and skin color (Fig. 57.1). The condition often starts with an injury, sometimes minor, with pain that persists well beyond and is out of proportion to the inciting injury [1, 2]. The incidence is estimated to be 5–20/100,000, with females outnumbering males by more than 4:1 [3, 4].

L-2: What are the diagnostic criteria for CRPS?

CRPS type I (formerly known as reflex sympathetic dystrophy) [5]:

- Continuing pain disproportionate to the inciting event.
- No other diagnosis better explaining the signs and symptoms.
- Report 1 symptom in 3 of 4 categories.

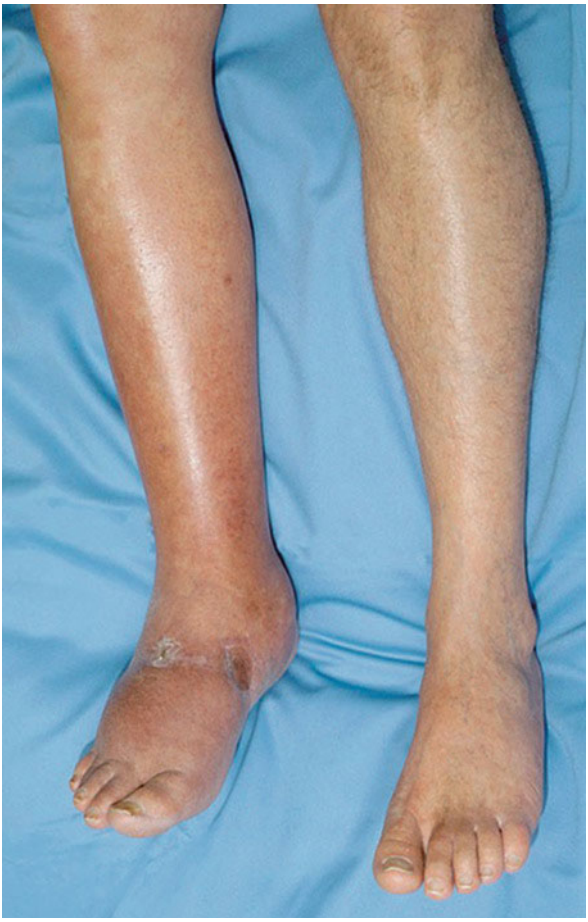

Fig. 57.1 Typical presentation of CRPS, not the patient in this case description (Reprinted with permission from Schmidt and Willis [12])

  - Sensory: hyperesthesia and/or allodynia
  - Vasomotor: temperature asymmetry and/or skin color changes
  - Sudomotor/Edema: edema and/or sweating changes
  - Motor/Trophic: decreased range of motion (ROM), and/or motor dysfunction and/or trophic changes
- Display 1 sign in 2 or more categories.
  - Sensory: hyperesthesia; allodynia (mechanical, temperature); deep somatic pressure/joint movement
  - Vasomotor: temperature asymmetry (>1 °C); skin color changes
  - Sudomotor/Edema: edema; sweating changes
  - Motor/Trophic: decrease ROM; motor weakness; tremor; dystonia; trophic changes

CRPS type 2: same as CRPS Type 1 but with a history of nerve injury (formerly known as causalgia) [5].

Table 57.1 Inciting injuries in CRPS type 1

Inciting event (%)
None (10.8)
Fracture (44.1)
Sprain (17.6)
Elective surgery (12.2)
Other (8.8)

Data from de Mos et al. [4]

L-3: What causes CRPS?

An inciting injury is often present but may not always be recalled. These injuries may include sprains, fractures, or surgery [3] (Table 57.1).

The pathophysiology of CRPS is poorly understood. The prevailing theory for many years has been that a dysfunction of the sympathetic nervous system causes hyperactive sympathetic outflow. This is believed to account for the temperature changes, edema, and skin discoloration seen in CRPS and results in "sympathetically maintained pain." Sympathetic nerve blocks have been used to treat CRPS, and the efficacy of these blocks has been held up as evidence of sympathetic dysfunction. However, not all patients respond to sympathetic blocks, and some symptoms are more consistent with a hypoactive sympathetic system or inflammation. An alternative theory holds that deep tissue microvascular damage due to ischemia-reperfusion injury with persistent ischemia and inflammation may account for the signs and symptoms seen in CRPS [1, 2].

L-4: How is CRPS treated?

There are few large well-controlled studies of CRPS treatments. Several widely used treatments have little more than case series to support them including sympathetic nerve blocks. The mainstay of chronic rehabilitation for CRPS remains physical therapy. Other treatment options include [6–9]:

(a) Spinal Cord Stimulation: There is one randomized controlled trial that found significant pain reduction persisting 2 years after implantation.
(b) Opioids: No studies in CRPS and few studies in chronic neuropathic pain, but they are often used.
(c) Gabapentin: Two studies with CRPS patients which found small but significant benefit.
(d) Other Anticonvulsant Drugs: None have been studied in CRPS, but several have been found to be beneficial for other neuropathic pain disorders.
(e) Tricyclic Antidepressants: Have not been studied in CRPS but have been found to be beneficial for other neuropathic pain disorders.
(f) Steroids: Two small studies showed mild improvement with use early in the disease when given pulsed dose over a few weeks.
(g) Ketamine: Low-dose infusions (0.3 mg/kg/h). Two small studies of 4-h outpatient infusions done 3 days per week for 4 weeks with significant improvement in pain but not function. Rarely done in the USA.
(h) Ketamine Coma: 5 days in ICU on ketamine 3–7 mg/kg/h. One case series found significant reductions in pain intensity up to 6 months. Not done in the USA.

L-5: Perioperative management of the opioid-dependent patient

(a) Patients on large doses of opioids should continue those drugs up to and including the morning of surgery in order to prevent withdrawal [10].
(b) Postoperatively they will need their regular outpatient dose of opioid plus additional opioid for postsurgical pain. This increased dose may be anywhere from 30 to 100 % of their preoperative dose.
(c) Patients on chronic opioids may have delayed gastric emptying.

L-6: What are some perioperative concerns for CRPS patients?

(a) Placement of peripheral IV should be in a non-CRPS affected limb if possible. For multiple affected limbs ultrasound guidance may be useful.
(b) Placement of a blood pressure cuff should be on a non-CRPS affected limb.
(c) Manipulation of the affected extremity may cause a flair of pain even under general anesthesia. Extra padding and careful positioning may be required. A plexus or peripheral nerve block may be useful for either the CRPS affected limb or the surgical limb. If the surgical site is different than the CRPS affected limb, doing two separate nerve blocks may place the patient at risk of local anesthetic systemic toxicity. Surgery performed on the CRPS affected extremity is extremely high risk for causing a flair and difficult-to-control postoperative pain.

L-7: What are some non-opioid options for minimizing pain flares after surgery for this patient?

The concept of preventive or preemptive analgesia suggests that the administration of a drug before a surgical stimulus or under anesthesia during surgery will have an analgesic effect that extends into the postoperative period beyond the expected half-life of the drug. The benefits include decreased pain and decreased opioid requirements. Several drugs and techniques have been employed to reduce pain and opioid requirements in patients at high risk of difficult to control post-operative pain:

(a) Regional anesthetic peripheral, neuraxial, or plexus blockade of the surgical site or the CRPS affected limb.
(b) Preoperative dose of gabapentin or pregabalin. Gabapentin and pregabalin bind to the α_2-δ subunit of voltage-gated calcium channels, reducing Ca^{2+} influx during depolarization. This reduces release of the neurotransmitters, glutamate, norepinephrine, and substance P. Several studies for postoperative pain and opioid tolerant patients have shown reductions in pain and opioid requirements after various surgeries. Recommended doses are pregabalin 150–300 mg or gabapentin 600–1,200 mg 1–2 h before surgery.
(c) Ketamine infusions. Ketamine is a noncompetitive *N*-methyl-D-aspartate (NMDA) receptor antagonist blocking glutamate's excitatory action on Ca+ channel influx. The antagonism of spinal cord NMDA receptors postsynaptically is theorized to decrease dorsal horn cell central sensitization during surgery. Ketamine infusions under general anesthesia have been shown to reduce pain and opioid requirements in various surgeries and especially in opioid tolerant patients. Typical infusion dose is 5–10 μg/kg/min with or without a preincisional bolus dose of 0.3–0.5 mg/kg [11].

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Chapter 58
Vascular Absorption of Local Anesthetic Producing Systemic Toxicity

Jackie Phan and Preetham Suresh

A 60-year-old female anesthesiologist was scheduled for a left thumb base interposition arthroplasty for osteoarthritis. She was 5′4″ and 57.9 kg, with a past medical history significant for hypertension, hyperlipidemia, and remote tuberculosis s/p treatment. She had had multiple general anesthetics in the past with a history of narcotic sensitivity. Her medications included amlodipine, Celebrex, benazepril, Crestor, Actonel, calcium, and aspirin. Her physical exam was notable for a Mallampati class I airway, full range of motion of her neck, intact dentition, and the ability to prognath. Regional anesthesia was discussed with the patient preoperatively, and she opted for regional with no sedation, as she stated that she was "very sensitive" to narcotics—she required Narcan after 50 mcg fentanyl for a colonoscopy in the past. The patient was placed supine on a gurney, monitors placed, and her left infraclavicular area was prepped and draped. The brachial plexus along with surrounding vessels was visualized clearly on ultrasound. The block was performed uneventfully with the Tuohy needle in view at all times, without any vascular puncture seen on ultrasound. Negative aspiration was confirmed prior to infiltrating all three cords with a total of 35 mL of 1.5 % mepivicaine with 1:200,000 epinephrine. The patient had a motor block in the distribution of all three cords within 5 min of performing block, but shortly after testing, the patient complained of tongue numbness. Vital signs at this time were within normal limits, with the systolic blood pressures (SBPs) in the 160s mmHg (baseline), heart rate (HR) within normal limits (WNL), and without electrocardiogram (EKG) changes. The presumptive working clinical diagnosis at this time was vascular absorption of local anesthetic producing systemic toxicity (LAST) (**L-1, L-2, L-3**).

The patient was given 2 mg of midazolam. She became somnolent after the injection but was able to answer questions appropriately. Shortly thereafter her

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tongue numbness resolved, and her vital signs remained stable. She was taken to the operating room (OR) (without the pump connected), monitors placed, and was observed to have tonic-clonic movements of the upper extremities and face that lasted less than 1 min, again it was presumed that she had LAST from vascular absorption of local anesthetic (**L-1, L-2, L-3**). Her vital signs remained stable during this seizure activity.

Immediately after, the patient was awake and answering questions and did not appear postictal. The regional team was called to the OR, and a discussion was had with the OR anesthesia team about the need for intralipid, but given that the patient remained hemodynamically stable, it was not given. The procedure was performed under regional anesthesia without further sedation. Postoperatively in the postoperative anesthesia care unit (PACU), approximately 3 h postblock her motor function began to return, but she continued to have good pain control. A discussion was had as to whether the patient's pump should be connected and run at what dose given her excellent pain control and adverse reaction to narcotics. The course of events was discussed at length with patient, and she was adamant about testing the catheter prior to making any decisions about what to do with it (**L-4, L-5**). At this time, the patient's catheter was bolused with 10 mL 2 % lidocaine with bicarbonate, without epinephrine, with return of her motor block. Within 5 min of the bolus, the patient again complained of tongue numbness, with stable vital signs. One milligram midazolam was given at this time. Under ultrasound visualization, 10 mL of normal saline was bolused through catheter and was seen to be extravascular and well posterior to the axillary artery. One milliliter of air was injected as well with the same observation. No hematoma was noted at injection site or visualized under ultrasound. It was decided to remove the catheter, and the patient was discharged home after an extended stay in PACU without VS/EKG changes or other signs of local anesthetic (LA) toxicity.

Lessons Learned

L-1: What is the relationship of vasculature to the brachial plexus?
The brachial plexus arises from the C5 to T1 nerve roots between the anterior and middle scalene muscles; then divides into upper, middle, and lower trunks; followed by multiple divisions; then the lateral, posterior, and medial cords; and finally into branches (Fig. 58.1) [1]. With an infraclavicular block, local anesthetic is placed at the level of the cords, which closely surrounds the axillary artery. Care must be taken to avoid inadvertent vascular puncture and injection of local anesthetic systemically as this can lead to rapid rise in plasma local anesthetic and possibly, toxicity, which was explored as a possible cause of this patient's LAST symptoms. *Ultimately, in this case, we speculated that very small arterial and venous vessels in the area of the cords and axillary artery absorbed the local anesthestic, as opposed to direct intravascular injection into the axillary artery itself.*

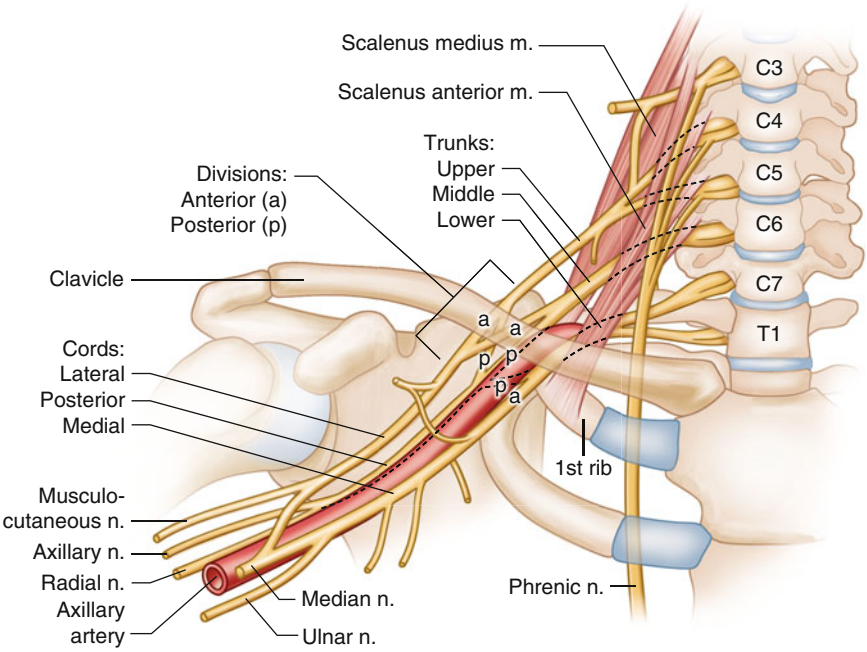


Fig. 58.1 The brachial plexus and its anatomical relationship to the axillary artery, scalene muscles, and bony structures (Adapted from Edmonton Academy of Regional Anesthesia [1])

L-2: What are the signs and symptoms of local anesthetic toxicity?

(a) Central nervous system (CNS) symptoms are usually a premonitory sign of impending local anesthetic toxicity, especially complaints of perioral numbness. However, a large percentage of patients demonstrate local anesthetic toxicity by presenting with seizure activity. As seen in Fig. 58.2, the presenting symptoms of LAST also relate to the plasma concentration of the LA itself, with higher plasma concentrations leading to more severe symptoms, including cardiovascular compromise [2]. The rate of rise of plasma concentration also determines whether or not the patient will experience symptoms such as perioral numbness, prior to developing seizures or cardiovascular (CV) symptoms. As seen with this patient, perioral numbness preceded seizure activity, suggesting a gradual rise in plasma concentration of local anesthetic as opposed to a direct large volume intravascular load.

(b) Cardiovascular symptoms require approximately three times the dose that generates CNS toxicity, although it varies for different local anesthetics, as seen in Fig. 58.3 [3]. This is particularly true for bupivicaine, where the toxic dose for seizures versus CV symptoms is much more narrow compared with lidocaine. Thus, if symptoms of toxicity present in a patient who was given bupivicaine, especially if seizure activity develops, this patient should be aggressively treated

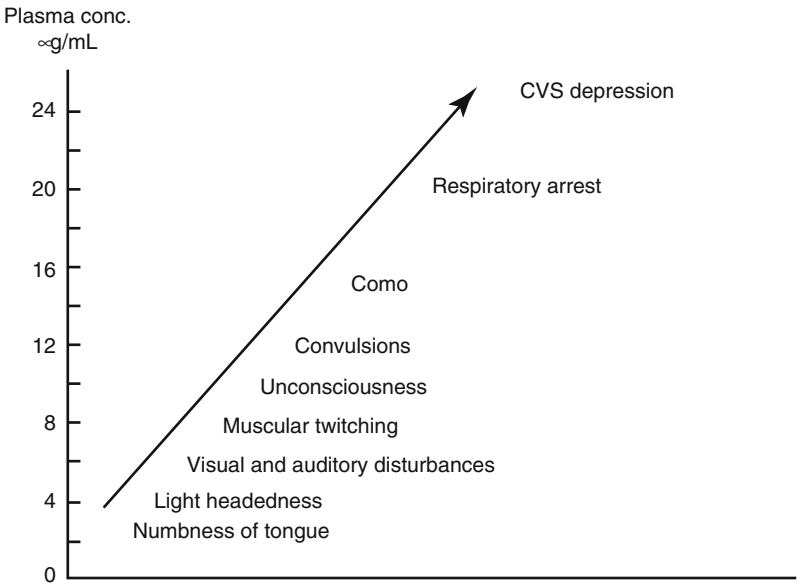
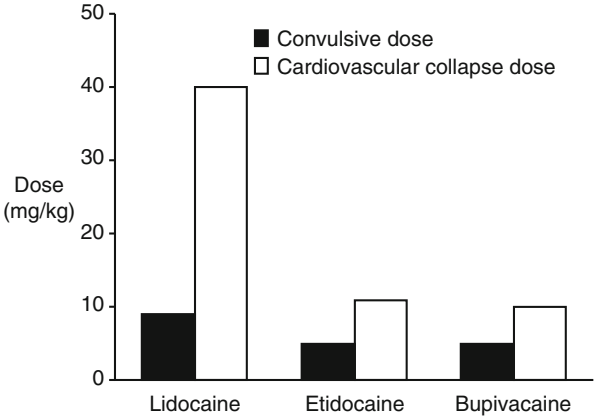


Fig. 58.2 Relationship of escalating LAST symptoms with increasing plasma concentration (Adapted from Scott [2])

Fig. 58.3 The dose of local anesthetic needed to produce CNS versus CV symptoms is much narrower for bupivacaine compared with lidocaine (Adapted from Brown [3])



with intralipid. This is in contrast to the patient in this case who demonstrated LAST after a block with mepivicaine, a less cardiotoxic local anesthetic, and was subsequently not treated with lipid therapy.

L-3: What factors affect plasma concentration of local anesthetics?

Many factors contribute to plasma concentration of local anesthetics, including the dose of local anesthetic given, the location of where it is being administered, the absorption of it into the plasma from the block site, and the metabolism and excretion of it; the relationship of which is illustrated in Fig. 58.4.

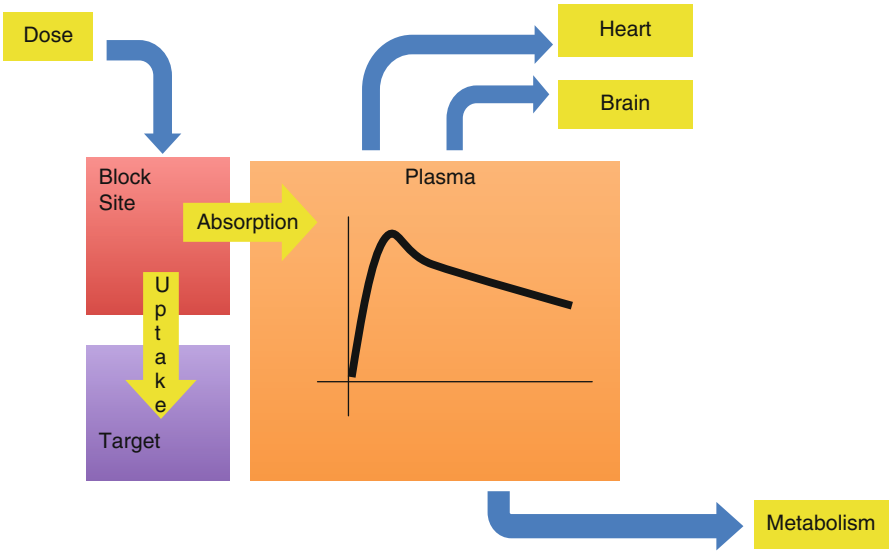


Fig. 58.4 Multiple factors contribute to the plasma concentration of local anesthetic

(a) Dose

Different studies have examined absolute dosing versus weight-based dosing to determine the maximum safe doses of local anesthetics; however, neither has been shown to be more accurate in predicting plasma concentrations or the likelihood of toxicity than the other. When considering the maximum allowable dose, one must consider the block site in addition to patient's weight and the type of local anesthetic being used. The dose of mepivicaine used in this case was more than the calculated maximum dose per kg (7 mg/kg or 405 mg), but less than the maximum absolute dose of mepivicaine (525 mg in the US [4]), demonstrating that other factors, aside from dose, play a role in the development of local anesthetic toxicity [4].

Plasma concentration is linearly related to the dose given at a particular site (Fig. 58.5) [5]. However, although there may be a linear correlation in dose and plasma concentration at a particular site, the impact of the other factors needs to be considered in calculating the maximum dose.

(b) Block Site

The site of injection and the relative vascularity of that site is a large determinant of the eventual plasma concentration of local anesthetic. Intercostal blocks have relatively high-peak plasma concentrations given the proximity to the nerves to the intercostal artery and veins compared with the lower plasma levels after spinal blocks, as this is a relatively unvascular site (Fig. 58.6). The brachial plexus block performed in this case is considerate intermediate in terms of vascularity of the site itself and resultant blood level of LA after infiltration [4] (Fig. 58.7).

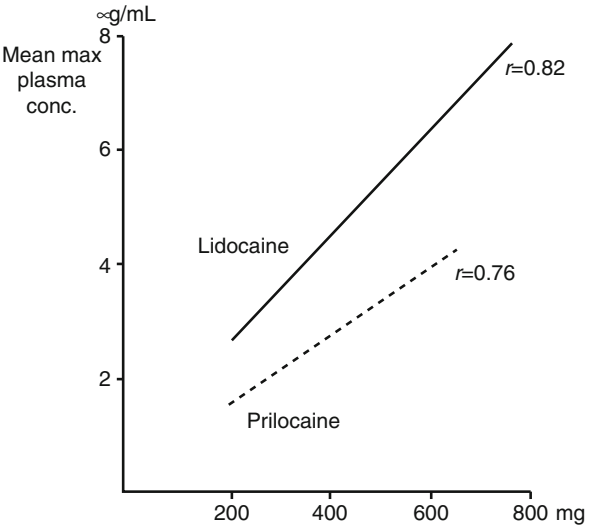


Fig. 58.5 Plasma concentration of local anesthetic linearly increases with increasing dose (Adapted from Cousins [5])

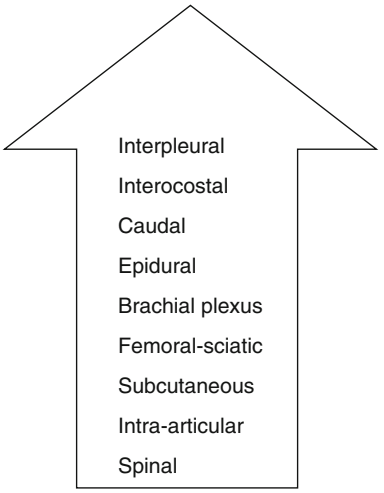


Fig. 58.6 Various regional anesthesia blocks are listed in order of increasing plasma level of local anesthetic from bottom up (Adapted from Brown [3])

(c) Epinephrine

The presence of epinephrine in the local anesthetic administered, most commonly 1:200,000 or 1:400,000, produces vasoconstriction at the block site, decreasing the amount of local anesthetic taken up into the plasma and increases the amount of local anesthetic that can be safely used.

(d) Concentration

The concentration of local anesthetic injected also plays a role in time to maximum plasma level, with higher concentrations of local anesthetic leading to shorter time to maximum plasma concentration as seen in Fig. 58.8. In this case we used 35 mL of 1.5 % mepivicaine (525 mg) which is a relatively large

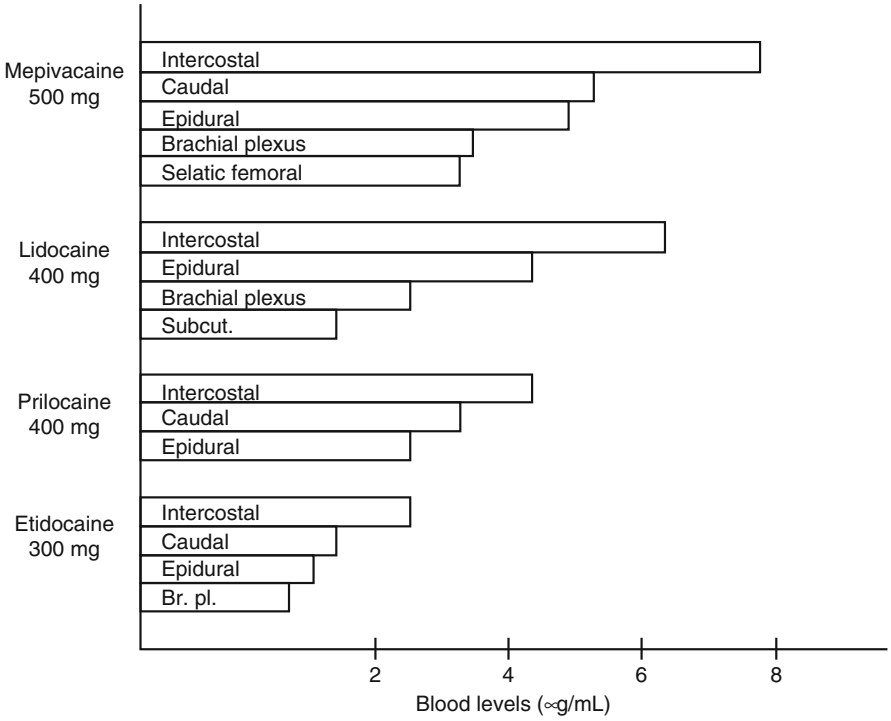


Fig. 58.7 The blood level of standard doses of various local anesthetics after different types of blocks, demonstrating that certain blocks have greater vascular absorption than others (Adapted from Rosenberg et al. [4])

dose, administered over a short period of time, which leads to rapid development of LAST and presumably rapid rise of plasma LA concentration (Fig. 58.8) [6].

(e) Absorption/Uptake

Peak concentration ∝ dose/cardiac output × time of injection

More rapid infusions, including bolus doses versus a continuous infusion, will lead to higher CNS concentrations. Similarly with low cardiac output, local anesthetic concentration in plasma is greater, and the amount of drug reaching the brain is increased. In our case, we cannot say exactly at what rate the local anesthetic entered the vascular space (i.e., whether the local anesthetic entered as a bolus, rapid infusion, or a slower drip).

The intrinsic properties of the local anesthetic including potency, onset, and duration also play a large role in the uptake and eventual plasma concentration of local anesthetic. In this case, we used mepivicaine, a low potency, moderate onset local anesthetic.

(f) Potency

Potency is the amount of local anesthetic needed to exert its effect, with higher potency drugs producing an effect at lower concentrations. Potency of

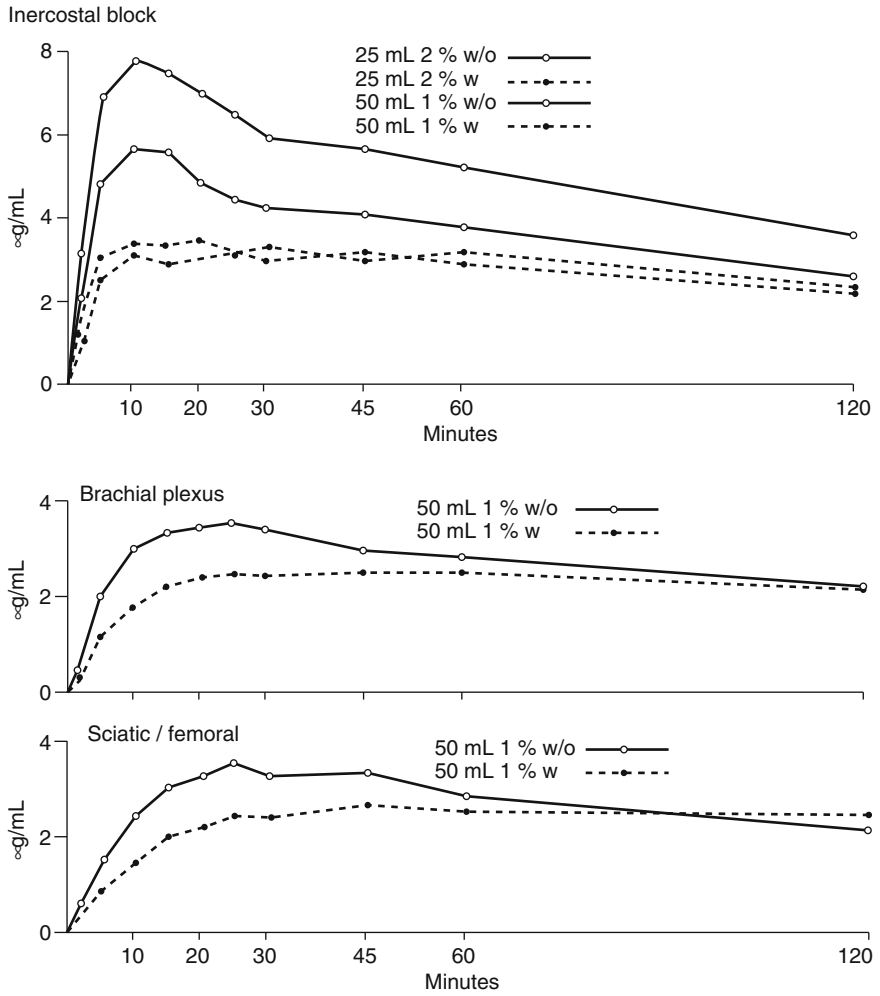


Fig. 58.8 Mean arterial plasma levels of mepivicaine after intercostal vs brachial plexus vs sciatic nerve blocks. w/o = plain solution. W = plus 1:200,000 epinephrine (Adapted from Tucker et al. [6])

Table 58.1 Potency of local anesthetics in relation to their lipid solubility

	Drug	Lipid:H_2O coefficient
Low potency	Chloroprocaine	0.14
	Mepivicaine	0.8
	Lidocaine	2.9
High potency	Ropivicaine	2.9
	Tetracaine	4.1
	Bupivicaine	27.5

Table 58.2 Onset of local anesthetics in relation to their pK_a

	Onset	pK_a	% ionized (pH 7.4)
Mepivicaine	Moderate	7.6	61
Lidocaine	Rapid	7.9	76
Ropivicaine	Moderate	8.1	83
Bupivicaine	Slow	8.1	83
Tetracaine	Slow	8.5	93
Chloroprocaine	Rapid	8.7	95

Table 58.3 Duration of local anesthetic in relation to its protein binding

	Duration (h)	%Protein binding
Bupivicaine	1.5–8	95
Ropivicaine	1.5–8	94
Tetracaine	1.5–6	94
Mepivicaine	1–2	77
Lidocaine	0.75–2	64
Chloroprocaine	0.5–1	n/a

LA is related to its lipophilicity (in Table 58.1, lipophilicity is referred to as the lipid:H_2O coefficient). The more lipophilic a drug is, the more potent it is as it more readily crosses the lipid bilayer.

(g) Onset

Onset is related to concentration and lipid solubility, with lower solubility leading to a faster onset. Onset however is mostly dependent upon pK_a, which is the pH at which the amount of unionized drug is equal to the ionized form.

The acid dissociation constant is $K_a = [H][A]/[HA]$ with the pK_a being the – log K_a. Local anesthetics with more physiologic pK_as have a faster onset, as there is less of the molecule in its ionized form, as seen in Table 58.2. The exception to this is 2-chloroprocaine, which has a high pK_a, but a very fast onset, owing to its high dose [4] compared with other local anesthetics.

Acidosis also increases the proportion of ionized to non-ionized local anesthetic in the plasma. Thus, adding bicarb to local anesthetics speeds the onset of action of the LA. One point to consider when performing blocks is that sedation with benzodiazepines if often given to minimize discomfort during placement of the block, which may cause hypoventilation leading to an increase in $PaCO_2$, which in turn leads to an increase in cerebral blood flow and increased delivery of local anesthetic to the brain. In this case, midazolam was given to the patient after she complained of perioral numbness to raise the seizure threshold; however, she became quite somnolent afterward and shortly thereafter developed seizure activity which was perhaps in part aided by her sensitivity to the benzodiazepine and subsequent hypoventilation and development of respiratory acidosis.

(h) Metabolism and Elimination

Duration of local anesthetics (Table 58.3) is related to both its lipid solubility and its plasma protein binding. An increased lipid solubility and increase plasma protein binding lead to longer duration of action. 2-chloroprocaine for

Table 58.4 Checklist for management of LAST

Get help
Initial focus
Airway management: ventilate with 100 % oxygen
Seizure suppression: benzodiazepines are preferred; avoid propofol in patients having signs of cardiovascular inability
Alert the nearest facility having cardiopulmonary bypass capability
Management of cardiac arrhythmias
Basic and ACLS will require adjustment of medications and perhaps prolonged effort
Avoid vasopressin, calcium channel blockers, beta-blockers, local anesthetic
Reduce individual epinephrine doses to <1 μg/kg
Lipid emulsion (20 %) therapy (values in parentheses are for 70-kg patient)
Bolus 1.5 mL/kg (lean body mass) intravenously over 1 min (≈100 mL)
Continuous infusion of 0.25 mL/kg/min (≈18 mL/min; adjust by roller clamp)
Repeat bolus once or twice for persistent cardiovascular collapse
Double the infusion rate to 0.5 mL/kg/min if BP remains low
Continue infusion for at least 10 min after attaining circulatory stability
Recommended upper limit: ≈10 mL/kg lipid emulsion over the first 30 min

Adapted from Neal et al. [7]
ALCS Advanced Cardiac Life Support, *BP* blood pressure

instance is an unbound drug, leading to its very short duration of action. Duration of action is also determined by the metabolism and excretion of the drug.

Ester local anesthetics are metabolized by pseudocholinesterase, while amides are extensively metabolized the P450 enzymes in the liver prior to undergoing renal excretion.

Studies have shown that the plasma concentration of local anesthetics are similar for healthy patients compared with patients with compromised renal function, as shown with mepivicaine. However, the current recommendation is to reduce the dose of LA by 10–20 % for severely renally compromised patients and more importantly to reduce the number of repeat doses as clearance of the local anesthetic is decreased. There is no need for reduced dosing in hepatic failure and heart failure patients, except if there is also a question of compromised renal function in these patients. Reduced dosing in the elderly is recommended because of increased sensitivity of nerves to LA blockade with increased age, secondary to changes in nerve morphology. The patient in this case had no history of compromised cardiac, renal, or hepatic function that would suggest that the dose of local anesthetic for her needed to be reduced, but she clearly demonstrated an increased sensitivity to local anesthetic doses that were within the recommended range.

L-4: How do you treat local anesthetic toxicity?

Table 58.4 is the ASRA recommended steps for treatment of local anesthetic toxicity.

(a) How does intralipid work?

Intralipid, the main component of which is soya oil, helps treat LAST by creating a large lipid sink that the lipophilic local anesthetic will bind to, effectively drawing the local anesthetic out of the systemic circulation.

(b) Risks and benefits of intralipid

Intralipid has been shown to improve the success of cardiopulmonary resuscitation in cases of cardiovascular collapse from LAST compared with ACLS performed on patients who did not receive the infusion. There appears to be very little risk associated with lipid therapy, although some possible adverse events include allergic reaction, nausea, shivering, and increased temperature.

L-5: What are the steps for prevention of local anesthetic toxicity?

There are no hard and fast rules that can be followed that will always prevent LAST; however, there are precautions that can be undertaken to reduce the chance of a patient developing LAST. Recommendations to decrease the incidence of LAST are listed in below.

- Use the lowest dose of LA possible to achieve the desired effect.
- Administer the LA in small increments, 3–5 mL, at a time with a 15–30 s pause in between injections.
- Aspirate the needle before each injection.
- Use an intravascular marker, such as epinephrine. Ten to 15 mcg/mL of epinephrine should produce a 10+ beat increase in HR and/or a 15+ mmHg increase in the SBP.
- Use of ultrasound guidance.

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Chapter 59
Inadvertent High Spinal in Parturient

Zakir Rangwala

The patient is a Spanish-speaking-only 36-year-old female, gravida 7 para 3 at 38 weeks and 4 days, who presented for a scheduled repeat cesarean section. The patient is 271 lb, 5′2″, body mass index (BMI) 49.9, presenting with vital signs of blood pressure (BP) 134/61 mmHg, heart rate (HR) 81 beats per minute (bpm), respiration rate (RR) 20/min, temperature 97.7 °F, and O_2 saturation 94 % on room air. Past medical history includes gastroesophageal reflux disease (GERD) and diabetes mellitus type II. Medications include glyburide, neutral protamine Hagedorn (NPH), Humalog, and prenatal vitamins. Past surgical history includes three previous C-sections under spinal anesthesia without any complications. The patient had an unremarkable airway exam and nothing by mouth (NPO) status was confirmed for greater than 8 h. The anesthesia team decided on a combined spinal-epidural (CSE) technique for the procedure.

After premedication for aspiration prophylaxis, the patient was brought to the operating room (OR) and CSE was placed. An 18-G Tuohy needle was passed into the epidural space with loss of resistance at 7.5 cm. Next, a 25-G pencil-point needle was passed through the Tuohy into the intrathecal space with return of clear cerebrospinal fluid (CSF). 1.6 cc of 0.75 % hyperbaric bupivacaine with 25 μg fentanyl and 0.1 mg morphine was injected intrathecally. After the spinal needle was removed, a 19-G epidural catheter was threaded easily into the epidural space and secured at 12 cm at the skin.

After laying the patient supine with left uterine displacement, the anesthetic level was assessed with temperature to a level of T3 (**L-1**). The patient denied any difficulty with respirations and had strong bilateral hand grip strength (**L-1, L-2**).

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She was given supplemental oxygen at 6 L via facemask with saturations remaining in the 97–100 % range. BPs remained stable but HR decreased from 80 to 50 bpm (**L-2**) which responded to 0.4 mg atropine and 5 mg of ephedrine. Surgery commenced at 1,245.

At 1,258, the patient complained of pain, so 3 cc of 2 % lidocaine with 1:200,000 epinephrine was administered into the epidural catheter to verify epidural catheter placement before administering a larger dose. One minute later, an additional 2 cc of the same solution was administered. Shortly afterwards, the patient's HR slowed to 50 bpm and subsequent noninvasive blood pressure (NIBP) was 85/43 mmHg. The patient's voice grew quieter and she was unable to move her upper extremities (**L-2**). The obstetric team was notified to temporarily halt procedure. Pulse oximetry rapidly dropped to 50 % and ventilation via facemask with 100 % O_2 was initiated without difficulty, bringing the saturations back up to 100 %. Hypotension was treated successfully with boluses of phenylephrine and ephedrine. Additional intravenous (IV) access was obtained and an arterial line was placed. During ventilation via facemask, the patient remained unresponsive for approximately 12 min (**L-2, L-3**), then began to initiate spontaneous breaths but required assistance with ventilation. Surgery continued while patient was assisted with ventilation and hypotension was supported with medication. Vital signs remained stable and O_2 saturations were within 93–100%. After 30 min, hemodynamics stabilized and the patient awoke. She reported upper extremity weakness but no recollection of the previous hour's events. Once the C-section was completed, the patient was transported to the postanesthesia care unit (PACU) with stable vital signs.

Lessons Learned

L-1: How does one assess an adequate level of surgical anesthesia when using a spinal anesthetic?

(a) It is important to note that spinal anesthetics exhibit differential blockade in terms of sympathetic, somatic, and motor block. A somatic blockade at the T4 (nipple) level provides reliable, adequate surgical anesthesia for C-section (see Fig. 59.1) [1].
(b) The three modalities used to measure somatosensory blockade is light touch, pinprick, and temperature sensation. Studies have shown that light touch is the most resistant to blockade, while temperature is the most easily blocked [2].
(c) On average, the level of blockade for temperature is two dermatomes higher than pinprick, which is two dermatomes higher than light touch sensation [2].
(d) At our institution, we find that decreased sensation to pinprick at the T4 level is the most reliable indicator of adequate somatic blockade and anesthesia for C-section.

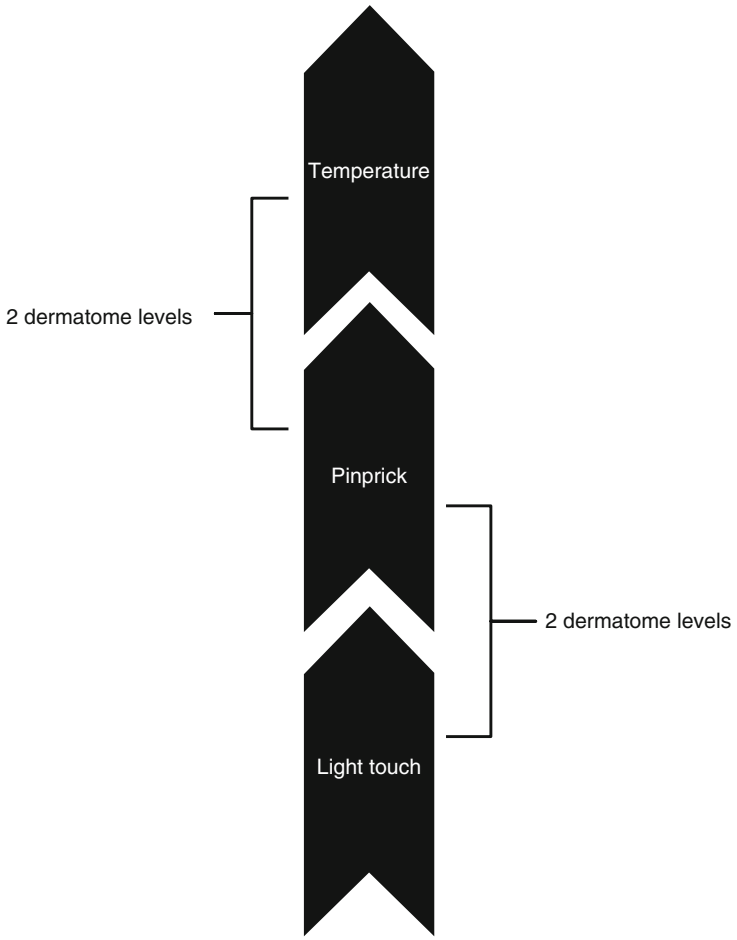

Fig. 59.1 Differential sensory blockade in spinal anesthesia

L-2: How does a spinal anesthetic ascend the CSF space in terms of signs and symptoms?

(a) T5–L1. Sympathetic blockade of these nerve roots will cause venodilation in the lower extremities and splanchnic circulation. This vascular pooling will lead to decreased venous return and lower CO and BP. Sympathetic blockade will also affect the GI tract in the form of a contracted gut and active peristalsis.

(b) T1–T4. Unopposed vagal activity after sympathetic blockade of the cardiac accelerator fibers will result in bradycardia and decreased contractility. The heart is also unable to respond to hypovolemia with an increased heart rate as the baroreceptor reflex arc has been blocked (Fig. 59.2).

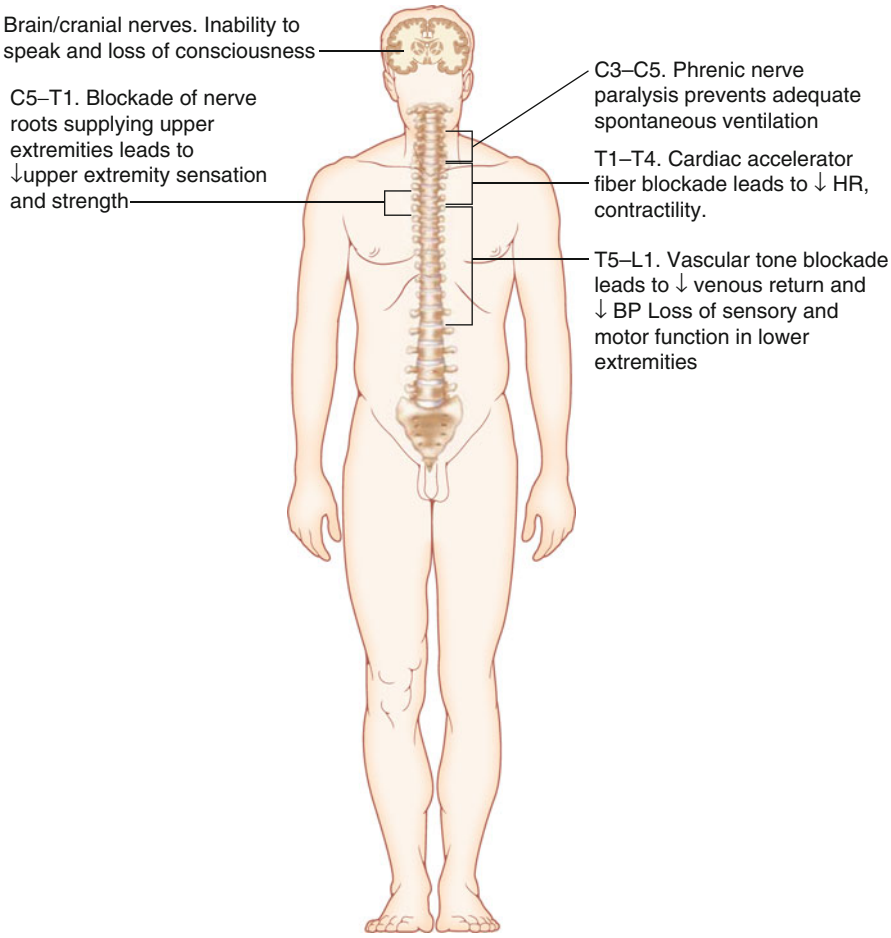

Fig. 59.2 Schematic diagram detailing symptoms of ascending sympathetic blockade during spinal anesthesia

(c) C5–T1. Blockade of the brachial plexus nerve roots will result in sensory loss and weakness and eventual paralysis of the upper extremities.
(d) C3–C5. Diaphragmatic paralysis can affect ventilation in healthy patients and will have a more pronounced effect in patients with little pulmonary reserve who rely on accessory muscles for respiration.

1. Patients with a C3–C5 blockade will present in 1 of 3 clinical manners (Table 59.1):

The patient is not in distress; respiratory mechanics appear to be within normal limits (top row Table 59.1).
The patient is in distress despite respiratory mechanics appearing to be within normal limits. The respiratory distress is due to the loss of proprioception of the chest wall muscles and the inability of the patient to "feel" their own breathing,

Table 59.1 Respiratory distress and mechanics in high spinal (C3–C5) anesthesia: three possible presentations

	Respiratory distress: i.e., feel good/bad	Respiratory mechanics: i.e., looks good/bad	Physician intervention
1	Good	Good	Observe
2	Good	Good	Reassure patient
3	Bad	Bad	Secure airway

leading to the assumption that they "can't breathe." Patients can be reassured by placing their own hand on their chest and feeling the respiratory mechanics functioning (middle row Table 59.1).

The patient is in distress and respiratory mechanics appear abnormal (Table 59.1 bottom row). This scenario is usually not tolerated by the patient and the airway needs to be secured.

2. C3–C5 blockade will also prevent the ability to cough and clear airway secretions.

(e) Brainstem/cranial nerves. Blockade of cranial nerve X will prevent function of the recurrent laryngeal nerve and superior laryngeal nerve. The patient will be unable to control the muscles in the larynx and thus be unable to phonate.

(f) Brain. A total spinal ascending above the foramen magnum will cause unconsciousness in a patient.

L-3: Should the patient have been intubated? Why?

(a) When an inadvertent total spinal occurs, loss of consciousness occurs and implies the inability to protect one's own airway, so we believe that securing the airway is the most prudent course of action.

(b) Additionally, in the parturient, there is an increased risk of aspiration because of increased intra-abdominal pressures from the gravid uterus, delayed gastric emptying, and high circulating progesterone levels. This would further support the need to secure the patient's airway.

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Part VIII
Outpatient Surgery-Related Cases

Chapter 60
Plastic Surgery at a Surgeon's Office

Luis M. Rivera

The patient is a healthy 70 kg, 49-year-old female scheduled for an extensive cosmetic procedure at her plastic surgeon's office. The 6-h procedure includes a face- and neck lift, endobrow, upper and lower blepharoplasties, a chemical peel to her neck, and a flank and buttock liposuction. Based on information provided by the surgeon's office, her past medical history is significant for anxiety and postoperative nausea and vomiting (PONV), requiring an unscheduled overnight admission for uncontrollable PONV following a prior cosmetic surgery (**L-1**). The patient was unable to be reached by phone the night before surgery, and this is the first time that our group has provided anesthesia services at this facility.

On arrival to the surgeon's office the day of surgery, it is noted that the anesthesia machine is comprised of a vaporizer circuit and lacks a ventilator. There is a crash cart available with the basic Advanced Cardiovascular Life Support (ACLS) drugs, and an old-model uniphasic defibrillator, but the only airway equipment available at this facility are the basic endotracheal tubes and laryngoscope handles and blades. Further, upon meeting and speaking to the patient for the first time on the day of the scheduled surgery, she reveals that she has had a prior chin implant to cosmetically correct her retrognathia. On exam, her true thyromental distance is less than 3 cm, and her airway is a Mallampati class 3 with somewhat limited mouth opening. Securing the airway with an endotracheal tube (ETT) will be necessary due to the length of the procedure and the need for a prone and/or lateral position during portions of the case. After obtaining a more detailed past medical history and carefully examining the patient, a challenging intubation is anticipated.

The risk is assessed, and consideration is given to the possibility of rescheduling the case at an inpatient facility with equipment more suitable for the management of a difficult airway. The surgeon and the patient are not pleased with the prospect

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of a cancellation nor is the anesthesia group that is motivated by the establishment of a new business relationship with this surgeon's office. The patient is relatively young, healthy, nonsmoker, and physically fit. After weighing all the options and discussing the risks with the surgeon and the patient, it is anticipated that the patient should be relatively easy to ventilate via mask, although intubating her may be difficult, if not impossible, with the equipment available. There is also a perceived sense of urgency to make a prompt decision, as the case start has been delayed by almost an hour already. Therefore, the decision is made to proceed with the case by attempting an asleep endotracheal intubation via direct laryngoscopy, with the understanding that if the airway cannot be secured safely, the patient would be allowed to emerge and the surgery rescheduled at a facility that can provide backup airway equipment (**L-2**).

The patient is placed in optimal sniffing position and preoxygenated with 100 % O_2 using a tight-sealing face mask for a full 5 min. Anesthesia is induced with midazolam 2 mg IV, lidocaine 80 mg IV, and propofol 120 mg IV. She is paralyzed with succinylcholine 80 mg IV and is relatively easy to ventilate via mask, but direct laryngoscopy with a Macintosh 3 blade reveals a Grade IV view. A second laryngoscopy with a Miller 2 blade attains the same view. Repeated attempts to locate and gently mobilize the epiglottis with the tip of a laryngeal tracheal anesthetic (LTA) kit and a bougie bridged by ventilation by mask do not improve the view either. After another three attempts bridged by ventilation by mask, a bougie is finally advanced blindly into the patient's trachea. However, by now, the paralyzing agent is beginning to wear off, and attempting a blind passage of the ETT while the vocal cords might have not been fully abducted could prove harmful. Therefore, additional propofol 100 mg IV and Succinylcholine 60 mg IV are administered. After waiting for the drugs to take effect, the ETT is advanced into the trachea without resistance, as the oxygen saturation levels dip into the low 80s. Proper placement of the ETT is confirmed by auscultation and CO_2 return. An additional 50 mg of IV propofol is infused, and the patient is ventilated by bag with 100 % O_2 via the ETT until the O_2 saturations return to 100 % before the volatile agent and N_2O are initiated (**L-3**).

Prophylactic antiemetics are administered (metoclopramide 10 mg IV, dexamethasone 8 mg IV, and ondansetron 4 mg IV) (**L-4**), and the case proceeds uneventfully with the patient having returned to spontaneous ventilation on 0.8–1 % sevoflurane, 50 % oxygen (O_2), and 50 % nitrous oxide (N_2O) (**L-5**), along with a subhypnotic propofol infusion to aid in PONV prevention, running at approximately 20 mcg/kg/min throughout the 6.5-h duration of the case (a total of 550 mg of propofol). Since there are not any infusion pumps at this facility, a drip was prepared by emptying an IV fluid bag, putting 600 mg (60 mL) of propofol into it, and "eyeballing" the drip rate to target the above infusion rate (**L-6**). At the end of the case, a prophylactic dose of Phenergan 12.5 mg IV is given; the patient emerges and is extubated awake.

She spends 70 min in recovery and goes home without complications.

Table 60.1 Patient's perspective: order of importance in controlling anesthesia side effects

Side effect	Overall rank	% Ranking factor as least desirable
Vomiting	1	24
Gagging on the tracheal tube	2	22
Incisional pain	3	21
Nausea	4	6
Recall without pain	5	20
Residual weakness	6	7
Shivering	7	1
Sore throat	8	0
Somnolence	9	0

Adapted from Macario et al. [1]

Lessons Learned

L-1: **How important is the prevention of PONV**?
Particularly in the ambulatory surgery environment, control of PONV is possibly one of the most important factors in what otherwise should be a straightforward, uneventful anesthetic on a relatively healthy patient. There are various components to achieving this goal: avoiding or minimizing the use of triggering agents, proper use and timing of antiemetic prophylactic agents, hydration, oxygenation, and temperature control.

While it is highly impractical if not impossible to avoid all proemetic agents unless one relies solely on regional techniques, most offending agents are dose dependent. Therefore, if they cannot be avoided, their use should be minimized. In general, using the lowest amount of narcotics while providing adequate analgesia is advisable. The same is true of N_2O and the volatile agents: running a deeper anesthetic than required may increase the likelihood of undesirable side effects. Avoiding a non-depolarizing muscle relaxant has the advantage of not requiring exposure to neostigmine, long considered a known proemetic. Keeping the patient from moving and the surgeon happy requires lots of vigilance and anticipation by the anesthesiologist.

Recent studies have revealed that, of all likely anesthesia side effects, including pain, shivering, gagging, choking, residual weakness, and PONV, PONV was ranked higher in order of importance by most patients (Table 60.1).

Therefore, proper control and, better yet, proper prophylaxis of PONV is imperative. Most studies support the use of multiple prophylactic agents, where the number of different drugs used increases as the risk of PONV increases. Antiemetic efficacy has often been expressed as a number needed to treat (NNT), where NNT represents the number of high-risk patients receiving prophylaxis in order to prevent one emetic event that would have occurred had the patient not received the intervention. While it is important to stratify patients in order to establish what their

Table 60.2 PONV risk stratification

Number of risk factors	0	1	2	3	4
PONV probability (%)	10	21	39	61	79

pre-op risk is, it may not be justifiable to withhold prophylaxis solely based on statistical values. For instance, there is evidence that the NNT for both dexamethasone, and the 5-Hydroxytryptamine-3 Serotonin Receptor (5-HT3) inhibitors such as ondansetron, and dexamethasone ranges between five and eight patients [2]. Although the philosophy to provide prophylaxis to this many patients in order to prevent a single PONV episode may vary from facility to facility, one must take into account cost, patient satisfaction, and the potential for adverse effects such as pulmonary aspiration, wound dehiscence, and other complications.

Although there is a lack of conclusive evidence and different studies have shown mixed results over several years, a good estimate is that even a patient with zero risk factors may have a 10 % probability of PONV without adequate prophylaxis. A patient with one risk factor has an approximately 21 % probability, two risk factors yield a 39 % probability, three risk factors yield a 61 % probability, and 4+ risk factors a 79 % probability (Table 60.2). Well-established predisposing risk factors are female gender, young age, nonsmoking status, history of PONV, and motion sickness. Other potential risk factors may include anxiety, obesity, preexisting gastric disorders, and paradoxically American Society of Anesthesiologists (ASA) physical status I or II.

In this case, the patient is a relatively young, nonsmoking female, with a significant history of PONV, and therefore has a 79 % probability of developing PONV if not provided with adequate prophylaxis. The fact that the surgery is taking place at a self-standing surgeon's office adds to the importance of this issue, as an unscheduled admission to a hospital is highly undesirable and not devoid of logistic complexity. Therefore, an aggressive PONV prevention approach was taken by administering metoclopromide, ondansetron, and dexamethasone at the beginning of the case, followed by a continuous subhypnotic propofol infusion throughout the case and Phenergan at the end of the case (See **L-4**).

L-2: **What added production pressures are unique to outpatient cosmetic procedures**?

Cosmetic plastic surgery brings us a unique cohort of patients. The surgery is 100 % elective, the patients pay a significant amount of money for it, and they expect perfection. This includes the anesthetic, which is rarely presented clearly to the patient by the surgeon.

It has been estimated that OR time comes at a cost of $50 per minute in the main OR of a large medical center. Although outpatient surgicenter rates may not be this high, other logistic issues are usually at play. Even at private surgeons' offices, inefficient management of OR time will erode into the practice's profit margin. Some facilities may charge surgeons by the minute for OR time. Therefore, expect production pressures and incentives to be expeditious in the use of OR time. Some facilities have a limited number of beds and nurses, so a patient with a prolonged

PACU stay will consume resources often needed in the pre-op area. Most outpatient surgicenters pay their staff on an hourly rate. Again, a prolonged PACU stay will result in unscheduled overtime. At most facilities, an anesthesiologist must remain on the premises until the last patient is discharged at the end of the day. A complicated patient may keep you in the PACU for hours after you thought your day was done.

At this surgeon's office, as it is often the case, there is only one suite that serves the dual purposes of being an operating room as well as a recovery room. Although this case was very lengthy requiring over 6 h and no cases were scheduled to follow, this may often not be the case. A significant amount of production pressure may be the norm at some facilities. This office offered the opportunity to start IV access in another room, while the OR was prepared. In some cases, IV access must be established as soon as the patient lies down in the operating table, while a nurse applies the monitors.

Preoperatively, there is usually the expectation that every case will proceed expeditiously and that all relevant information necessary to proceed has been sorted out so that the patient is ready for the beginning of surgery a few minutes after arrival. Late findings do not contribute to this goal.

Similar production pressures are present intraoperatively. Some surgeons may proceed to position the patient, prep the skin, stimulate the patient, and begin the surgery as you are still securing the airway. Although occasionally a surgeon must be told to wait, one must strive to maximize efficient use of time.

The end of the case is usually where most unnecessary time is spent. Delayed reversal of neuromuscular blockade, return to spontaneous ventilation, and delayed emergence can add a significant amount of time to any case. In order to have the patient to wake up soon after the surgeon is done, early down titration of volatile agents is essential towards the end of the procedure.

In this case, the patient was allowed to breath spontaneously throughout the entire procedure, and the liberal use of local anesthetics by the surgeon allowed keeping the patient anesthetized with less than 1 total MAC (0.8–1 % sevoflurane plus 50 % N_2O). The anesthetic gases were turned off about 15 min before it was anticipated that the surgeon would finish applying all the dressings, and the patient emerged promptly thereafter. Two 20 mg boluses of propofol were given during this period, as the patient began slowly turning her head as the bandages were applied. There was no recall experienced.

This case's start time was delayed by almost 1 h due to unanticipated patient difficult airway anatomy and lack of backup airway management equipment. A significant portion of this delay may have been avoided by proper communication between the anesthesiologist, the surgeon, and the patient prior to surgery. The fact that the patient could not be reached by phone the night before surgery, combined with the anesthesiologist's lack of familiarity with this facility, caused a significant amount of delay. Conversely, proper management at the end of the case, and an uneventful PACU stay, allowed for the patient to be safely discharged home and the staff to conclude their work for the day in a timely fashion.

L-3: **Additional challenges of office-based anesthesia: airway equipment and management**

Many cosmetic procedures will be done, not in a surgicenter, but at the surgeon's office. Some offices have only a skeleton crew and basic electronic monitors and life support equipment. Most of these offices lack a gas analyzer (other than CO_2 and O_2). Some, like this one, do not have a ventilator. The choices are to do the case as monitored anesthesia care (MAC), ventilate by hand by repeatedly squeezing a bag for hours, or let the patient breath spontaneously under general anesthesia. For safety reasons, when working at a surgeon's office, it is always advisable to carry one's own laryngoscope and blade, 2 ETTs, a #4 laryngeal mask airway (LMA), an endotracheal tube introducer (bougie), and a nerve stimulator. Checking the crash cart and defibrillator on arrival in the morning is always a must.

A patient requiring an unscheduled hospital admission must be taken to a nearby hospital's emergency room (ER) via a commercial ambulance service and, in some cases, be accompanied by the anesthesiologist. It is not uncommon for many places with only one operating room that the only other physician in the facility may be an ophthalmologist or a plastic surgeon, rarely comfortable with ACLS procedures and almost never trained in airway management. The difficult airway kit often consists of a single #4 disposable LMA, and any life-threatening complications get handled by dialing 911. Moreover, remote locations like this one do not lend themselves to help from anesthesia colleagues. As you can see, delays and complications and unplanned admissions must be avoided in a successful practice.

Patients with a difficult airway pose a unique challenge. As opposed to an academic institution or a large hospital, there may be little opportunity to meet the patient in person before the day of surgery. Occasionally, significant medical issues may be underestimated or even unrecognized until you perform your own history and physical examination. More often, one must rely on a phone call to the patient the night before surgery.

In this case, the patient could not be reached after several calls to the phone number provided by the surgeon's office, and the first indication of a potentially difficult intubation was not identified until during the pre-op discussion with the patient the morning of surgery.

A careful plan was put in place taking into consideration all the clinical factors, and the decision was made to proceed with the case. Since there was no difficult airway equipment available at this facility, the plan included the potential use of a bougie that is routinely carried in the anesthesiologist's bag for emergencies. Preparations were made for a challenging intubation, and key decisions and actions were taken at every step.

The patient was placed in optimal sniffing position, and preoxygenated with 100 % O_2 with a tight-sealing face mask for a full 5 min. Anesthesia was then induced with midazolam 2 mg IV, lidocaine 80 mg IV, and propofol 120 mg IV and the patient was paralyzed with Succinylcholine 80 mg IV. Although she was relatively easy to mask ventilate, successfully intubating the trachea required numerous attempts and different techniques bridged by ventilation by mask. Patient safety was

maintained throughout the entire process, and a successful intubation was accomplished by following a carefully designed plan with the following key events:

1. Agreement was reached with the surgeon to wake up the patient and postpone the surgery if intubation proved impossible or potentially harmful to the patient.
2. A well-practiced alternate plan was put in place to utilize a bougie (a simple, portable, inexpensive device well known to anesthesia providers) if direct laryngoscopy failed.
3. The patient was meticulously positioned in an optimal sniffing position.
4. The patient was adequately preoxygenated with high-flow 100 % O_2 using a tight-fitting mask for a full 5 min.
5. Induction of anesthesia and neuromuscular blockade was accomplished with the minimum amount of drugs necessary to attain the desired results, promoting rapid return to spontaneous ventilation, should ventilation and intubation proved impossible.
6. Bridging ventilation by mask was done between all airway interventions, maintaining an adequate oxygen saturation level throughout.
7. Adequate hypnosis and muscle paralysis was ensured at critical junctions by administration of supplemental doses of the required agents.

L-4: **How can the use of the commonly available antiemetics be optimized**?
Even some patients with minimal or zero-risk factors often feel sick after an anesthetic. Since PONV is a multifactorial issue involving multiple receptors, combination therapy has become the gold standard. With the cost of a dose of ondansetron and other 5-HT3 inhibitors having decreased from over $20 to less than $2, and its side-effect profile usually being minimal, in the absence of contraindications, it may be cost-effective to prophylax most patients with ondansetron 4 mg IV, along with dexamethasone 8 mg IV, and metoclopromide 10 mg IV, regardless of their number of risk factors. With the favorable profile and low cost of the 5-HT3 inhibitors, acting proactively and providing prophylaxis to most, if not all patients, may be advisable (Table 60.3).

Metoclopromide is a very weak Dopamine-2 (D2) receptor antagonist at this low dose, and its antiemetic properties have been questioned repeatedly. However, its prokinetic properties promote gastric emptying, tightens lower esophageal sphincter tone, may prevent aspiration at the end of the case, and has been shown to be as effective as a 5-HT3 inhibitor when used in combination with dexamethasone in some studies.

Metoclopromide must be given at the beginning of the surgery. There is no conclusive evidence quantifying the effectiveness of dexamethasone based on timing of administration, although it is believed that early administration is preferred. Recent articles postulate that 5-HT3 antagonists seem to work better when administered towards the end of surgery. But since most outpatient procedures last less than 2 h, and there is some evidence that providing receptor blockade at the chemoreceptor trigger zone *before* exposure to the proemetic agents may be a factor, all the antiemetics may be administered during induction without compromising effectiveness.

Table 60.3 Commonly used antiemetic agents and their characteristics

Agent	Typical dose	Mechanism of action	Significant side effects	Time of administration
Metoclopromide	10 mg IV	D2 antagonist	Extrapyramidal, dysphoria, QT-prolongation	Early
Ondansetron	4 mg IV	5HT-3 antagonist	QT prolongation, migraines	Late
Dexamethasone	4–10 mg IV	Unclear	Wound healing, adrenal insufficiency, mental status changes	Early
Scopolamine	1.5 mg TD patch	Antimuscarinic	Anticholinergic	4 h before surgery
Phenergan	6.25–12.5 mg IV/IM	D2/H1 antagonist	Extrapyramidal, dysphoria, sedation	Late, rescue
Droperidol	0.625 mg IV	D2 antagonist	Extrapyramidal, dysphoria, QT-prolongation FDA warning, sedation	Either early or late
Haloperidol	1 mg IV/IM	Same as droperidol	Same as droperidol	Either early or late
Propofol	10–25 mcg/kg/min	GABA agonist	Minimal at subhypnotic dose	Continuous infusion
Ephedrine	25 mg IM	Sympathomimetic	Mild tachycardia, HTN	Rescue

Those patients with identified history of motion sickness may benefit from a Scopolamine patch (antimuscarinic) the night before surgery or on arrival the morning of the procedure. While it takes up to 4 h for the drug to reach consistent therapeutic plasma concentrations via a transdermal patch, a patch applied immediately after the patient presents the morning of surgery should release enough drug to reach therapeutic concentrations by the time the patient reaches the PACU.

Those patients with recalcitrant PONV, like this one, may benefit from an intraoperative propofol drip (GABA agonist), at 10–25 μg/kg/min started immediately after induction, and lasting until after the anesthetic gases are turned off and the patient is on 100 % O_2. Intermittent doses of propofol have not been shown to be effective, as a minimal plasma concentration must be maintained throughout the case (*see* **L-6**).

In the rare case of PONV following the above technique, Phenergan 12.5 mg IV (Dopamine-2/Histamine-1 receptor antagonist) may be used as a rescue agent, as the D2 receptor is inadequately blocked by metoclopromide, and the H1 remains unblocked. Occasionally, there may be reluctance to giving Phenergan by the PACU nurses, claiming that Phenergan makes patients sleepy and prolongs PACU stay. Ironically, this is precisely why it is wise to use it right away. Sedation from Phenergan given soon after arrival in the PACU will usually wear off within the 45–60 min that the patient would have spent in the PACU anyway. Phenergan given

after the patient has been in the PACU for, say, 30 min will likely result in a stay over 60 min, as one waits for the sedation from this drug to wear off.

A common mistake is to perseverate attempting to block the same receptor repeatedly. A second dose of ondansetron is no better than the first. Likewise, ondansetron in the PACU is not going to be effective if Dolasetron or other 5-HT3 antagonist agent had been given earlier and proved ineffective. If all else fails, one may consider using ephedrine 25 mg intramuscularly (IM) (the exact mechanism of action is unknown, but the net effect is greater than what would be expected from correction of hypotension alone) on patients that can tolerate potential mild hypertension and tachycardia. Sublingual lorazepam has been used by our Oncology colleagues. Diphenhydramine and the other antihistamines, as well as Toradol, and others may also be effective.

Haloperidol 1 mg IV is also a very effective rescue agent. Unfortunately, it is rarely available in most outpatient facilities. Droperidol has essentially been removed from most anesthesia carts due to the 2001 US Food and Drug Administration (FDA) black box warning regarding the risk of QT interval prolongation. Ironically, other butyrophenones as well as all the 5HT-3 antagonists carry the same risk. NK1 antagonists may not be routinely used anytime soon. They are too expensive and the triple-therapy prophylactic technique presented above (metoclopromide 10 mg IV, ondansetron 4 mg IV, dexamethasone 8 mg IV) has been shown to provide adequate results for most procedures.

Of the nonpharmacologic factors, proper hydration and oxygenation are probably the most important in preventing PONV. The average 70-kg patient, after fasting overnight for 8 h, will present with an 880-mL fluid deficit the morning of surgery. In a case where the estimated blood loss (EBL) is 150 mL and the surgery lasts 2 h, this patient will require at least an additional 670 mL of crystalloid just to break even. Afternoon cases will require more fluids. In young, healthy patients that can tolerate the potential of a slight degree of fluid overload and in surgeries where edema is not a significant factor, aiming for euvolemia, and even a small degree of hypervolemia, may be advisable. Longer surgeries without bladder cannulation may require waiting until later in the procedure before infusing the rest of the IV fluid in order to avoid a full bladder for a prolonged period of time. A good marker of proper hydration is that the patient can easily void in the PACU. It is advisable to have all patients void immediately before going into the OR.

Proper oxygenation is also presumed to be a contributing factor in controlling PONV. Administering supplemental O_2 to all patients in the PACU, even if their O_2 saturation is 100 %, and keeping it at 100 % until they are fully awake may be helpful.

Finally, keeping the patients warm by using condensers and making sure that air warmers such as Bair Huggers are in direct contact with the skin or that there is not more than a thin gown between the patient and the warmer may be helpful. An air warmer placed on top of two blankets will only serve to warm up the top blanket, not the patient. Contrary to common belief, a properly applied air-warming blanket

without any tears will not cause any burns. A light blanket over the air-warming blanket will ensure adequate contact between the air-warming blanket and the skin.

Warming up IV fluids will not make much of a difference. One to two liters of crystalloid at room temperature constitutes a very small percentage of total body water, and therefore will have an insignificant effect on core temperature. However, once the patient is awake, room temperature IV fluids at high rates may be uncomfortable and contribute to shivering.

Nasogastric tubes (NGTs) are essential in nonelective cases with a full stomach, and may be helpful in cases where the stomach may be insufflated due to prolonged or forceful ventilation via mask. However, inserting an NGT and suctioning the stomach has been shown to be ineffective as a prophylactic maneuver.

In this lengthy surgery lasting over 6 h, ondansetron 4 mg IV, dexamethasone 8 mg IV, and metoclopromide 10 mg IV were administered immediately after induction, and a second dose of ondansetron was given at the end of the procedure. Additionally, a subhypnotic propofol infusion running at 15–25 μg/kg/min was used for the duration of the procedure. Finally, in view of this patient's recalcitrant history of PONV, a dose of Phenergan 12.5 mg IV was administered at the end of the surgery, as the H1 receptor had yet to be blocked and the receptor-blocking properties of metoclopromide at the D2 receptor at this low dose are considered marginal at best.

This plastic surgeon's office did not stock urinary bladder catheters. Moreover, the surgeon insisted on minimizing the IV fluids in order to avoid facial edema. Therefore, the total IV fluid was limited to 1,500 mL of normal saline, 500 mL given at the beginning of surgery, 700 mL during the intraoperative course, and the remaining 300 mL at the end. An air warmer was used intraoperatively after the liposuction was concluded.

The patient did not exhibit PONV. She went home in satisfactory condition after a 70-min PACU stay.

L-5: **How can the use of volatile agents and nitrous oxide be optimized**?

Selection of an anesthetic gas for an outpatient procedure of short duration is often quite simple: use the least soluble agent available. In our practice, sevoflurane is usually preferred over isoflurane. Desflurane (though rarely available at small outpatient facilities) is marketed based on its rapid onset and time to emergence. However, except in very long cases involving morbidly obese patients, the general consensus is that there is not a significant difference between desflurane and sevoflurane. Because of its pungency, desflurane is also associated with an increased incidence of laryngospasm in patients with LMAs when its concentration is diminished rapidly—i.e., when you want the patient to promptly wake up at the end of the case. Because of its sympathomimetic effects, it is also associated with tachycardia when its concentration is augmented rapidly—i.e., when you want the patient to go to sleep at the beginning of a case. For these reasons, it has fallen out of favor. Some facilities may only offer one choice.

A long case done with isoflurane requires that one starts titrating down earlier than a case done with another less-soluble agent. A good rule of thumb is that it

takes approximately 4 min to wake up per MAC-hr of sevoflurane and about 7 min per MAC-hr of isoflurane. Although these are only approximations, they do not take away from the fact that the wake time is almost doubled with the latter.

Turning off the volatile agent and increasing the fresh gas flow as the surgery is reaching the end and the patient is not being significantly stimulated, and using small boluses of propofol (10–30 mg IV) if it is anticipated that the patient may move while the skin is being closed, or may emerge before all the dressings are in place, will aid in prompt awakening after long-lasting procedures. Vigilance, anticipation, and planning are the keys to timely emergence.

A MAC of inhaled anesthesia is a MAC of proemetic gases, any way it is achieved. Nitrous oxide has been shown to be proemetic, especially at high concentrations (70 %) and during certain types of surgeries, particularly those involving significant bowel manipulation. But so have been implicated the volatile agents. Therefore, 50 % N_2O in conjunction with 1.0–1.5 % sevoflurane may constitute a good compromise between the potential for PONV and a prolonged awakening and delayed discharge resulting from using a higher concentration of a volatile agent. Ultimately, this combination may yield a similar risk of PONV as using 2 % sevoflurane alone.

Recent studies have postulated that one of the mechanisms whereby N_2O causes PONV is by expansion of the middle ear. For this reason, it may not be advantageous to increase N_2O to 70 % at the end of the procedure. On the contrary, turning off the N_2O 5–10 min before the end of the procedure may result in a lesser risk of PONV.

Avoiding N_2O concentrations greater than 50 % may be helpful for the above reasons. Allowing the patient to breathe spontaneously towards the end of the case has added advantages. First, the respiratory rate and end-tidal CO_2 ($etCO_2$) are good indicators of how much analgesic the patient will require upon emergence. Second, there is no time wasted by waiting for the patient to begin breathing spontaneously as the $etCO_2$ builds up. If this is not practical, adjusting the ventilator settings to let the $etCO_2$ to slowly climb into the upper 40s range towards the end of the case will allow gentle hyperventilation of the patient after turning off the volatile agent, expediting its elimination while ending with an $etCO_2$ in the low mid-40s, which usually results in an adequate ventilatory drive.

In the case being discussed, the patient had significant risk factors for PONV. The unavailability of any kind of infusion pump plus the limited amount of propofol stocked at the facility prevented the use of a total intravenous anesthesia (TIVA) technique. Since the surgeon used local anesthetic liberally and the patient was breathing spontaneously, she was maintained with slightly less than 1 MAC of anesthesia with 0.8–1 % sevoflurane, 50 % O_2, and 50 % N_2O. The case required a total of 6.5 h of anesthesia. Therefore, the volatile agent was turned off 15 min before the end of the procedure (0.5 MAC of sevoflurane × 6.5 h = 13 min), immediately after the surgeon finished closing the skin, and as the patient's hair and face were washed and the bandages applied. Two boluses of 20 mg of propofol were used during this period. The patient emerged and was extubated promptly at the conclusion of the procedure.

Table 60.4 Approximate continuous propofol drip rates by patient weight required to maintain prophylactic PONV prevention using a 15 Gtt/mL gravity flow administration IV set

Patient weight (kg)	50	60	70	80	90	100
Infusion rate (Gtt/min)	1.5	1.8	2.1	2.4	2.7	3.0
Approximate time interval between drops (s)	40	33	30	25	22	20

L-6: **How to use propofol as an effective antiemetic**

For propofol to work as an effective antiemetic, a minimum plasma concentration must be maintained throughout the case. Intermittent doses are ineffective and the so-called sandwich technique, where one induces anesthesia with propofol and infuses a small bolus near the end of the procedure, has not been proven to be reliable. There is evidence in support that maintaining a minimum plasma concentration of 343 ng/mL provides an adequate antiemetic effect. Recent multicompartmental simulation studies suggest this can be achieved with a continuous infusion at 10–15 μg/kg/min, following an initial 10–20 mg bolus dose [3]. At this subhypnotic dose, propofol does not accumulate and will not delay emergence significantly.

The surgeon's office where this case took place was not equipped with any kind of IV pumps. Therefore, a drip was made by emptying an IV fluid bag, putting 600 mg (60 mL) of propofol into it and "eyeballing" the drip rate to target the above infusion rate. Using a standard 15-Gtt/mL gravity flow administration IV set, a constant 20 μg/kg/min rate of propofol in a 70-kg patient may be accomplished by a continuous drip at approximately 2 Gtt/min, that is, one drop every 30 s. Table 60.4 shows approximate drip rates for different patient weights. Drip rates for other weights can be easily calculated by multiplying the patient's weight in kilograms by 0.03.

Calculating an exact rate is usually not necessary. Any rate in a range between the minimum 10 μg/kg/min necessary for propofol to act as an antiemetic and 25 μg/kg/min, a rate at which propofol typically will not cause significantly delayed emergence, should be adequate.

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Chapter 61
Orthopedic Surgery at an Ambulatory Surgicenter

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The patient is a 68-year-old, 82-kg male who presents to an outpatient surgicenter for a scheduled shoulder arthroscopy and rotator cuff repair. His past medical history is significant for osteoarthritis treated with indomethacin and occasional hydrocodone/APAP (Vicodin); hypertension (HTN) controlled with atenolol, losartan, and hydrochlorothiazide (doses unknown); 40 pack-year remote history of smoking; asymptomatic coronary artery disease (CAD) with a bare-metal stent placement 5 years ago; and poorly controlled gastroesophageal reflux disease (GERD) with occasional diet-related nocturnal symptoms, despite self-treatment with over-the-counter omeprazole and calcium carbonate (Tums) periodically. His laboratory work, recent echocardiogram, and electrocardiogram (EKG) are essentially normal. His vital signs are blood pressure (BP) 138/72 mmHg, heart rate (HR) 65 BPM, room air oxygen saturation (O_2 Sat) 96 %, and respiratory rate (RR) 16. In spite of having been directed otherwise, he continued to take all his antihypertensive medications as originally prescribed, including taking all three antihypertensives on the morning of surgery. The procedure is scheduled for 2.5–3 h, and the surgeon requests controlled hypotension with a systolic blood pressure (SBP) of about 100 mmHg. The surgeon also emphatically rejects the idea of placing a brachial plexus interscalene block (ISB) (**L-1**).

The patient is prehydrated with 500 mL of normal saline (NS) and anesthesia is induced using midazolam 2 mg IV, lidocaine 100 mg IV, fentanyl 250 mcg IV titrated slowly, propofol 100 mg IV, and rocuronium 120 mg IV and intubated using a modified rapid sequence induction (RSI) with cricoid pressure (**L-2**). Of note, this facility does not carry sodium citrate or other oral nonparticulate antacids. He is intubated easily but requires several IV boluses of ephedrine, 5–10 mg, and phenylephrine, 50–100 μg, in order to treat postinduction hypotension, totaling 600 μg of

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phenylephrine and 20 mg of ephedrine over approximately 15 min. He is positioned in a beach chair position, as his BP is maintained with periodic boluses of phenylephrine 50–100 μg IV.

The surgeon begins the arthroscopy and the patient's SBP stabilizes within the target range of 100–110 mmHg. A few minutes later, in response to surgical stimulation, the patient's SBP further increases to the 140s and his heart rate to the mid 80s. In order to bring his pressure back into the desired range, an additional fentanyl 250 mcg, propofol 50 mg, and labetalol 15 mg IV are titrated over the next 5–10 min. The surgery proceeds uneventfully with a SBP in the desired range of 100–110 mmHg and a HR in the low 50s.

Fifteen minutes later, the surgeon suddenly announces that the rotator cuff tear is not repairable, injects local anesthetic into the joint, and proceeds to close. He requests a quick operating room (OR) turnover, as there are several cases scheduled to follow. The patient has no twitches on train of four (TOF) and neuromuscular blockade cannot be reversed at this point (**L-3**). Further, the patient becomes hypotensive after the surgical stimulus has ended. He is placed in a supine position and then in Trendelenburg, but this time the hypotension is unresponsive to ephedrine and phenylephrine. The blood pressure slowly returns to an acceptable range after several boluses of vasopressin and phenylephrine IV, plus an additional 1.5 L of normal saline (**L-4**). Since there are no ventilators in the postanesthesia care unit (PACU) and the PACU staff at this facility is not comfortable caring for intubated patients, the patient is kept in the OR under anesthesia and maintained with 0.5 % sevoflurane and 70 % N_2O until neuromuscular blockade can be reversed and the patient can be extubated safely.

Seventy-five minutes after that, the patient develops one twitch on TOF. His muscle paralysis is reversed with neostigmine 5 mg IV and glycopyrrolate 0.9 mg IV. The anesthetic gases are then turned off, and he begins breathing spontaneously several minutes later at a rate of 5–6 breaths per minute. Although his O_2 saturation is in the mid 90s while on 100 % oxygen, his expired end-tidal carbon dioxide ($etCO_2$) concentration is 72 mmHg and does not yet meet extubation criteria (**L-5**). He is extubated 20 min later once he becomes somewhat responsive, now with an $etCO_2$ of 51 mmHg. He is taken to the PACU, breathing spontaneously with an oral airway in place.

In the PACU, the patient's vital signs are in the acceptable range, but he is very somnolent and requires a 2.5-h stay before meeting discharge criteria. The total surgical time was 26 min, but the total OR time and anesthesia time were both well over 2 h. Including pre-op and post-op time, the patient spent almost 6 h at the facility.

Lessons Learned

L-1: **Controlled hypotension and the advantages of combined anesthetic techniques using regional nerve blocks**

Shoulder surgeries tend to be significantly more stimulating than other peripheral orthopedic procedures. Moreover, during shoulder arthroscopies, it is commonplace for the surgeon to request a controlled hypotensive technique in order to reduce

Table 61.1 Advantages of supplementing a general anesthetic with an ISB[a] for shoulder surgeries

Allows delivery of controlled hypotension at a lower level of general anesthesia
Leads to reduced amount of tissue edema by decreasing amount of irrigation fluid required
Leads to increased tissue visibility by surgeon
When used in combination with an LTA, facilitates a lower level of general anesthesia towards the end of the case

[a]Regional anesthesia usually takes more time in an outpatient private setting that does not have a dedicated regional anesthesia team

intra-articular bleeding and improve their visibility. Higher systemic blood pressures are undesired by most surgeons for two reasons: First, it forces the surgeon to increase the flow rate of the irrigation solution, often resulting in a greater amount of soft tissue edema. Second, the impaired visibility may result in an increased surgical degree of difficulty and, ultimately, a lengthier surgical procedure (Table 61.1).

ISBs may be advantageous during most shoulder surgeries. Although these blocks may not provide a sufficiently dense level of anesthesia immediately, if placed before the surgery, they will start to provide analgesia within about 30 min or less. Therefore, although this block is often done as a means of providing postoperative pain control, it is extremely useful in surgeries where the surgeon requests a controlled hypotensive technique. With an ISB in place immediately before the beginning of the surgical procedure, a deeper level of anesthesia and narcotics are required only during the initial 30-min period, while the block sets. Further, once it sets, the block may serve as the sole mechanism to deliver controlled hypotension with a moderate level of general anesthesia, often not requiring supplementation with narcotics or beta-blockers.

Fentanyl, 2–3 μg/kg, given before laryngoscopy and intubation to blunt their hemodynamic effects, may be all the narcotic that is necessary. Transiently, the patient may require a deep level of anesthesia in order to maintain the BP in the desired range before the block becomes fully effective. This may be accomplished by transiently increasing the volatile agent concentration and supplementing with boluses or even a continuous infusion of propofol while the block sets. As the block starts to work, the anesthetic requirements will gradually diminish, and the block may be all that is required to maintain the SBP in the desired range along with a moderate level of general anesthesia. Towards the end of the case, with the patient breathing spontaneously on less than 1 MAC of volatile agent, all that may be needed is an adequate anesthetic for the patient to remain in a hypnotic state and tolerate the endotracheal tube. Using a local tracheal anesthetic (LTA) before intubation will further diminish the general anesthetic requirements by suppressing the cough and gag reflexes, allowing the case to be finished with a minimal volatile agent concentration. This translates into fewer narcotic- and volatile agent-related side effects, a quicker emergence, and usually a much shorter PACU stay.

Conversely, unlike in the scenario commonly seen at academic centers and large hospitals, where a separate regional team does all the blocks ahead of time, most regional anesthesia procedures at surgicenters are done after the patient enters the OR and IV access is established. This requires delicate orchestration with the OR

circulating nurse and staff in order to position and prep the patient, apply and cycle the monitors, provide supplemental O_2, gather and prepare all the necessary supplies, sedate the patient, and do the block expeditiously. Some surgeons do not like regional procedures because of the added time it requires. On average, about one fourth of the surgeons prefer to have one, about one fourth leave it up to the discretion of the anesthesiologist, and about half vehemently oppose it for different reasons, mostly because of the additional required time. While it is unadvisable to argue with the surgeon and patient refusal is an absolute contraindication, a large proportion of both patients and surgeons may be agreeable to having the procedure done, once the risks and benefits are clearly presented. If they are still apprehensive or unsure, I find it counterproductive to attempt to persuade them. The last thing an anesthesiologist wants is a complication on a patient resulting from a procedure the surgeon did not want to begin with.

Most outpatient surgicenters will offer only two choices of local anesthetic: lidocaine and bupivacaine. A 50:50 mixture of preservative-free 1/4 % bupivacaine and 1/2 % bupivacaine with 1:200 K to 1:400 K epinephrine usually provides about 24 h of analgesia and minimal motor blockade. Some patients may subjectively feel that the block is not working unless they feel a "numb and heavy" arm. The resulting 0.375 % concentration with the above recipe usually provides a high enough concentration to provide some, but not total, motor blockade. Patients must also be warned about phrenic nerve blockade (100 % incidence) and subjective shortness of breath, as well as potential transient hoarseness, facial numbness, and Horner's syndrome.

Invariably, in spite of the benefits of an ISB, some surgeons and patients will refuse it. Sometimes an alternative is a suprascapular block. This is a field block that takes less than 1 min to do and, as opposed to an ISB, can be done safely with the patient already asleep. While clearly not as effective as an ISB, it provides some analgesia in the posterolateral aspect of the shoulder and the joint capsule. This is useful in blunting the pain resulting from trocar insertion and some posterior shoulder work. This block will not be effective with the anteromedial work required for subacromial decompressions and clavicle surgeries. Although it is usually insufficient as the sole mechanism to provide controlled hypotension, an ISB may facilitate this goal and significantly reduce the overall anesthetic requirements.

In cases where regional anesthesia techniques are not used, a third option is for the surgeon to inject the joint with local anesthetic, followed by the placement of an intra-articular catheter by the surgeon for a continuous infusion postoperatively. Continuous infusions had become very popular among the surgeons in the past few years. However, their use has diminished dramatically due to reports of the chondrotoxic effects of continuous infusions of local anesthetics, especially bupivacaine at the higher concentrations. Most surgeons usually inject one dose of local anesthetic (usually 1/4–1/2 % bupivacaine) at the end of the procedure.

However, if the surgeon is willing to inject local anesthetic at the *beginning* of the procedure, the local anesthetic will serve to lower the general anesthetic requirements and will also make it easier to provide controlled hypotension. Most surgeons

Table 61.2 Disadvantages of using a supraglottic airway (LMA) during shoulder surgery

The anesthesiologist may lose control of the patient's airway
The anesthesiologist has decreased access to the patient's airway
The LMA introduces a tracheal aspiration risk
An LMA is relatively contraindicated in cases where prolonged positive pressure ventilation may be required

are amenable to the use of ketorolac at the end of the case. Thirty milligrams IV has been shown to be as effective as 10 mg of morphine.

In this case, the surgeon emphatically rejected the use of regional techniques. Therefore, rather than relying on the hemodynamic benefits of an ISB, a controlled hypotensive technique was provided by the anesthesiologist by resorting to the use of large amounts of narcotics and beta-blockers. Once the surgeon suddenly stopped the noxious stimulus, ended the procedure, and injected local anesthetic, the patient became profoundly hypotensive, necessitating the use of vasopressin while aggressive fluid resuscitation was simultaneously done (see **L-4**).

Additionally, the large amount of narcotic used (500 μg fentanyl IV) significantly delayed emergence and prolonged the time needed to meet extubation criteria, as well as PACU stay.

In summary, while there is always a risk of complications, an ISB may have facilitated the provision of a controlled hypotensive technique without the need for a high dose of anesthesia, opiates, and beta-blockers.

L-2: **Does an ETT provide an advantage over a supraglottic airway (LMA) in ahoulder surgeries**?

Some anesthesiologists will use a laryngeal mask airway (LMA) or an equivalent supraglottic airway device for most shoulder surgeries, including lengthy procedures done in a lateral position. Although in a young, lightweight patient with high chest compliance, an adequate airway, and a good LMA seal, an LMA is usually adequate for most of these surgeries; there are three distinct disadvantages by using an ETT (Table 61.2).

First, most shoulder arthroscopies are done in the lateral position, where LMAs may dislodge and become problematic on a patient breathing spontaneously, while the arm is manipulated and moved during the procedure. Even in a seated position, typically used by surgeons for an open procedure, if an airway complication occurs, there is no easy access to the airway without having to interrupt the surgery and break the sterile field to secure the airway. Both positions also require patient repositioning after the LMA has been placed, risking mobilization of the LMA and degradation of the seal during repositioning. Although there is controversial data from several studies quantifying the risk of airway complications with a supraglottic airway versus an endotracheal tube and these complications are rare, recent studies list turning the table and having no direct access to the patient's airway as potential risk factors for airway complications [1]. Therefore, it may be advantageous to secure the airway with an ETT up front in these procedures, especially if one expects a lengthy surgery where an LMA may end up causing mucosal edema.

Second, most surgeons will ask for controlled hypotension during shoulder arthroscopies. Rather than resorting to the use of large amounts of narcotics and beta-blockers, an alternate way to provide controlled hypotension is to use an adjunct propofol drip and supplemental propofol boluses as required to provide some vasodilation and maintain the patient's SBP in the target range. However, the high doses of propofol necessary during periods of peak stimulation will invariably result in hypopnea or even apnea, requiring positive-pressure ventilation. In the absence of a pressure-control ventilator, at times this may become difficult, may result in excessive hypercapnia, and may end up causing an insufflated stomach. A poor LMA seal may make positive-pressure ventilation impossible.

Third, whereas an LMA usually results in a lesser hemodynamic response to airway manipulation, a well-planned, gentle intubation is usually a simple procedure, and one should be able to do this while providing as good hemodynamic control as when using an LMA. An option to facilitate positive-pressure ventilation is to use a Pro-Seal LMA, if one is available.

There is yet another reason that may favor the use of endotracheal tubes over supraglottic airways. Many surgeons will start manipulating joints, lysing capsular adhesions, and otherwise noxiously stimulating the patient soon after anesthesia has been induced but before a deep level of volatile agent anesthesia has been achieved. Alternatively, circulating nurses may begin prepping the skin, inserting urinary bladder catheters, repositioning, and stimulating patients before the anesthesia level is deep. A light patient with a supraglottic airway may buck, cough, go into laryngospasm, vomit, and aspirate following noxious stimulus, even from something as seemingly innocuous as applying room-temperature (65 °F) Betadine to the skin. It is sometimes difficult to avoid having these members of the staff refrain from manipulating the patient during this transient but critical period, especially when a slow induction with limited amounts of anesthetic is being done on a fragile patient that cannot tolerate deep anesthetic excursions. Securing the airway with an ETT and a neuromuscular blocker allows the staff to proceed with these manipulations without risking the above complications, thus expediting OR flow.

In this case, the use of an ETT was mandated by the patient's history of symptomatic GERD. It is worth pointing out that this facility, as is typical of most outpatient surgicenters, does not stock any oral nonparticulate antacids such as sodium citrate (Bicitra). In retrospect, even if the patient did not have any pulmonary aspiration risk factors, intubating the trachea turned out to be a fortunate choice. The large amount of narcotic used during this surgery (fentanyl 500 μg IV) would have caused significant hypopnea, if not apnea, as evidenced by an $etCO_2$ of 72 mmHg when the patient started to breathe spontaneously 75 min after the conclusion of the procedure (See **L-5**). The degree of respiratory depression induced would have required prolonged positive-pressure ventilation via a supraglottic airway with its associated risks. Also, the patient could have been intubated with the aid of a depolarizing agent (succinylcholine) and allowed to return to spontaneous ventilation in order to use the $etCO_2$ and RR parameters as a guide as to the amount of narcotic required and tolerated (See **L-3**).

Table 61.3 Advantages and disadvantages of depolarizing neuromuscular blockers

Advantages	Disadvantages
Rapid onset of action	May cause fasciculation myalgias
Short duration of action	Increases intracranial and intraocular pressure
Does not require reversal of blockade	Promotes intracellular potassium release
Provides a dense blockade	May cause prolonged block in patients with unknown pseudocholinesterase deficiency
Inexpensive	May trigger malignant hyperthermia

L-3: **Muscle relaxants: advantages and disadvantages of depolarizing versus nondepolarizing agents**

With the possible exception of laparoscopies and some spine surgeries, there are few procedures done at outpatient surgicenters that truly require absolute muscle relaxation. Most times, muscle relaxation is needed only to facilitate intubation. Succinylcholine is an excellent choice in these cases. The main drawback is a high incidence of myalgias, particularly in ambulatory procedures. Whether or not young age, muscular build, or male gender constitutes risk factors remains controversial. Whereas a defasciculating dose of a nondepolarizing agent will not prevent hyperkalemia, elevated intracranial, or intraocular pressure, it has been shown to decrease the incidence of myalgias [2]. Succinylcholine also has the advantage of not requiring reversal with neostigmine (a known proemetic drug) and glycopyrrolate, often causing HR excursions (Table 61.3).

If muscle relaxation is required intraoperatively, the shorter acting agents are preferred. Mivacron (mivacurium), possibly the most used agent in outpatient surgery, has unfortunately been removed from the market. Its closest substitute (Nimbex, cisatracurium) has a much slower onset and longer duration of action. At most outpatient surgicenters, the only nondepolarizing agent stocked is rocuronium. In cases of short duration where muscle relaxation is required in order to facilitate intubation or surgical conditions, rocuronium may be used in small doses (0.3–0.4 mg/kg). Although it takes longer in order to achieve adequate (although perhaps not ideal) intubating conditions (2–3 min onset vs. 90 s when using 0.6 mg/kg), the onset may sometimes be accelerated somewhat by using a priming dose and potentiating its effect with a volatile agent. Maintaining at least one twitch out of four in TOF ensures that the agent is readily reversible immediately thereafter. Frequent titration rather than large boluses is the key to avoid having to wait in order to reverse neuromuscular blockade at the end of the case.

In this case, the patient's poorly controlled GERD mandated taking precautions against pulmonary aspiration. On the other hand, his CAD and other cardiovascular risk factors (male, over 50, history of smoking, HTN) mandated the need to prevent hypertension and tachycardia during laryngoscopy and intubation. The anesthesiologist chose to do a modified RSI with 1.5 mg/kg of rocuronium, while maintaining tight hemodynamic control. This large dose of rocuronium required waiting over 75 min for the patient to develop one twitch on TOF before reversal could be

Table 61.4 Commonly prescribed oral angiotensin-converting enzyme inhibitors

Generic name	Common trade names	Usual dose
Benazepril	Lotensin	20–40 mg po qd
Captopril	Capoten	25–50 mg po bid-tid
Enalapril	Vasotec	10–40 mg po qd
Fosinopril	Monopril	20–40 mg po qd
Lisinopril	Prinivil, Zestril	10–40 mg po qd
Moexipril	Univasc	7.5–30 mg po qd
Perindopril	Aceon	4–8 mg po qd
Quinapril	Accupril	20–80 mg po qd
Ramipril	Altace	2.5–20 mg po qd
Trandolapril	Mavik	2–4 mg po qd

attempted. A similar modified RSI with adequate hemodynamic control may have been achieved with the use of an LTA, a short-acting beta-blocker such as Esmolol, and succinylcholine for muscle relaxation. If fasciculation myalgias are a concern, a small dose of rocuronium (3–5 mg IV) may be used, followed by an increased dose of succinylcholine (2 mg/kg). It has been shown that the risk of fasciculation myalgias is not increased as the dose of succinylcholine is increased [2].

L-4: **Preoperative use of antihypertensives: which and when is it advisable to withhold them**?

There is near universal agreement in the anesthesia community that diuretics should be held the morning of surgery. Regarding angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs), there exists controversy, but there is growing justification and support of recommendations to withhold these drugs as well, especially when used in combination with diuretics.

These families of drugs, especially when combined, may create a physiological state in which some patients precipitously vasodilate and lose their vascular tone upon induction of general anesthesia, which is almost always unresponsive to ephedrine but also resistant to phenylephrine, often necessitating the use of vasopressin and relatively large amounts of IV fluids. Although with gentle hydration and a well-planned, controlled induction, one should be able to provide adequate hemodynamic control in most cases, it is widely accepted that it is easier and safer to treat perioperative hypertension than postinduction hypotension. Recent reports seem to indicate that the incidence of perioperative hypertension is not statistically higher when withholding these drugs prior to surgery.

Unlike large hospitals and academic centers, outpatient surgicenters usually do not have a pre-op clinic, and patients are not routinely seen by an anesthesiologist before their scheduled surgery. Therefore, the preanesthetic instructions rely on a brief phone call to the patient by a surgicenter's nurse the night before surgery. This necessitates that the instructions be kept simple and straightforward. The ARBs can cause refractory hypotension of a magnitude even greater than the ACEIs. It may therefore be advisable to withhold these medications preoperatively in patients scheduled to receive a general anesthetic. See Tables 61.4 and 61.5 for a list of the commonly prescribed oral ACEIs and ARBs.

Table 61.5 Commonly prescribed oral angiotensin receptor blockers

Generic name	Common trade names	Usual dose
Azilsartan	Edarbi	40–80 mg po qd
Candesartan	Atacand	8–32 mg po qd
Eprosartan	Teveten	400–800 mg po qd
Irbesartan	Avapro	75–300 mg po qd
Losartan	Cozaar	25–100 mg po qd
Olmesartan	Benicar	20–40 mg po qd
Telmisartan	Micardis	20–80 mg po qd
Valsartan	Diovan	80–320 mg po qd

In the case being discussed, it is customary for the staff at this facility to instruct the patient to withhold all diuretics, ACEIs, and ARBs on the morning of surgery and also the night before surgery. However, no formal policy exists, and it is unclear whether or not the instructions were conveyed correctly to the patient by the surgicenter. Moreover, the surgeon's office does not have a policy or any written material with patient instructions on this subject either. The patient continued to take all his antihypertensives, including atenolol, losartan, and hydrochlorothiazide, as prescribed. To complicate matters, the patient did not know the doses and did not bring the medicine vials with him, the surgeon's notes did not indicate the dosages in question, and the primary care physician prescribing the medications could not be reached before the scheduled surgical time.

Based on the information available at the time, the patient's history and physical exam, the anesthesiologist chose to proceed with the surgery by conducting a well-planned induction with tight hemodynamic control. In spite of this, the patient lost some vascular tone, requiring rescue with significant amounts of phenylephrine (a total of 600 μg) immediately after induction. Further into the procedure, the patient responded to surgical stimulus with his SBP further increasing to the 140s and his HR to the upper 80s. He did not appear to be adequately beta-blocked. Therefore, the anesthesiologist chose to increase the level of anesthesia, analgesia, and beta-blockade in order to decrease the SBP to the level requested by the surgeon. However, once the surgical stimulus was terminated, the patient became profoundly hypotensive, this time necessitating more aggressive rescue maneuvers.

This case was brought to and presented at the facility's next Medical Executive Committee (MEC) meeting. At the meeting, it was learned that various other anesthesiologists have faced similar events in which patients develop profound postinduction hypotension. Two areas of improvement were identified.

First, it was determined that, in general, there was no mechanism in place to provide good communication among the patient, surgicenter, surgeon, and anesthesiologist. Therefore, a new policy was written so that the surgicenter staff notifies the surgeon's office and the anesthesiologist whenever there is difficulty contacting a patient the day before surgery. The anesthesiologist may then attempt to contact the patient the night before surgery, after the surgicenter's staff has gone home for the day.

Second, there was a generalized lack of written policies and procedures at this facility, as well as at the surgeon's office. Demographically, this particular facility

serves a large population of orthopedic senior patients. Many of these patients suffer from hypertension which is often treated with multiple agents in combination, including diuretics, ACEIs, and ARBs. This is arguably the patient population most susceptible to postinduction hypotension caused by these agents. Therefore, a new policy was written and distributed to all the surgeons credentialed at this facility, creating antihypertensive guidelines prior to general anesthesia, to be provided to the patient at the time of the surgical pre-op visit. The policy clearly lists all the ACEIs and ARBs by both generic and trade name and instructs the patients scheduled for a general anesthetic to skip taking these medications the night before surgery and the morning of surgery. In general, such policies will vary from facility to facility, by taking into consideration the type of procedures routinely being done, patient demographics, and other factors.

L-5: **The use of opiates: sometimes less is better**

Large doses of narcotics are an excellent choice for complex, lengthy procedures on fragile patients and surgical procedures that require tight hemodynamic stability such as those in cardiac surgery and neurosurgeries. However, postoperatively, the patients in both of these kinds of cases usually go to the intensive care unit (ICU) sedated and sometimes intubated. Therefore, whereas 10–15 μg/kg of fentanyl may be appropriate to blunt the response to a sternotomy or a craniotomy, large narcotic doses may not constitute the best choice of anesthetic for an outpatient shoulder arthroscopy.

In most of these cases, the most stimulating part of the case may be the laryngoscopy and insertion of an endotracheal tube. Therefore, in most cases requiring an endotracheal intubation, 1–2 μg/kg fentanyl IV, plus 1–1.5 mg/kg lidocaine IV, infused 2–3 min prior to laryngoscopy and an LTA may be all that is necessary to blunt the hemodynamic response elicited by laryngoscopy and intubation. A small bolus of esmolol (20–30 mg) may also work well to help prevent tachycardia during this period. In most cases under 2 h, the residual effect of the LTA will also aid in preventing the patient from bucking and coughing upon emergence.

Intraoperatively, if the patient's blood pressure, heart rate, and respiratory rate are trending upward during a stimulating portion of the procedure, transiently deepening the level of anesthesia by increasing the concentration of the volatile agent and supplementing with propofol IV are alternatives to adding more narcotic. The volatile agent is rapidly eliminated once titrated down, and propofol, with the exception of large, prolonged doses, is redistributed rapidly. In this fashion, one can manage the transient episode without affecting the post-op management and prolonging PACU stay.

An alternative to adding more narcotics at the end of the procedure in order to promote a smooth extubation is to give a bolus of lidocaine 1–1.5 mg/kg IV, 2–3 min before anticipated emergence, and relying on the residual anesthetic effect of the LTA during short cases. This is a good choice for avoiding excessive bucking, especially in patients with reactive airway, such as smokers, asthmatics, and patients with GERD. However, the timing of administration must be precise. Given too early, the effect of lidocaine wears off before emergence; given too late, it only serves to

make the patient sleepy after extubation. If it is anticipated that the patient may require additional narcotics, consider using Demerol towards the end of the case. Twelve to 25 mg IV will provide some analgesia while reducing shivering during emergence, a major complaint of numerous patients.

Combined anesthetic techniques, featuring general and regional anesthesia, will also reduce narcotic requirements, both intraoperatively and postoperatively. Injecting bupivacaine at the end of the procedure, and supplementing with an NSAID such as ketorolac 30 mg IV, usually provides excellent post-op analgesia. With such a technique, along with minimal amounts of intraoperative narcotics (fentanyl 1–2 μg/kg), most of these patients should meet discharge criteria within about 45 min, after requiring only some oral analgesic such as hydrocodone/APAP (Vicodin, Norco, Lorcet) or oxycodone/APAP (Percocet, Endocet, Roxicet) tablets in the PACU.

It is also important to also clearly communicate to the patient that there is no expectation to be pain-free. After being discharged home, all that will be available to the patient, short of a visit to the ER, is an oral analgesic such as Vicodin or Percocet. Therefore, the goal should not be to have the patient pain-free at the end of surgery, only to escalate once he gets home. Mild-to-moderate postoperative pain and discomfort should be expected. This should be discussed with the patient during the pre-op interview.

As evidenced by the case being discussed, giving large doses of opiates or beta-blockers during intubation as a means to provide hemodynamic control, or later on to blunt the effects of transient episodes of intense stimulation, may end up resulting in an unarousable, hypotensive patient at the end of the procedure, especially if the surgeon injects local anesthetic at the end. Again, the key to narcotics is slow titration with small doses.

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Chapter 62
Eye Surgery at an Outpatient Surgicenter

Luis M. Rivera

The patient is an 89-year-old male scheduled for a repeat right eye trabeculectomy at a self-standing outpatient surgicenter. He currently resides at a local skilled nursing facility (SNF), ambulates with a walker, and requires assistance with his activities of daily living (ADLs). He was transported via ambulance to the surgicenter, as this is the method of transportation routinely used by this SNF.

His past medical history (PMH) is significant for glaucoma, osteoarthritis, hypertension (HTN), inoperable coronary artery disease (CAD) with stents, status-post multiple myocardial infarctions (MIs) with an ejection fraction (EF) of 36 %, chronic obstructive pulmonary disease (COPD) with numerous exacerbations, mild dementia, and valvular heart disease including calcific (degenerative) aortic stenosis (AS) with an aortic valve area (AVA) of 1.2 cm^2. His medicines include several inhalers and eye drops, donepezil (Aricept), lisinopril, hydrochlorothiazide (HCTZ), digitalis (Digoxin), sublingual nitroglycerin (NTG), clopidogrel (Plavix), and aspirin, the latter two held for 5 days under the direction of his cardiologist, who cleared the patient for the procedure under sedation as planned. His level of exercise tolerance is not well established, as he is sedentary and does not engage in physical activity requiring more than two to three metabolic equivalents (METs). He is scheduled for a peribulbar block by the surgeon, supplemented with minimal to moderate IV sedation. In the preop area, his vital signs are blood pressure (BP) of 128/56 mmHg, heart rate (HR) of 76 beats per minute (bpm), respiratory rate of 18 breaths/min, and O_2 saturation of 92 % on room air. He appears to have recovered from a pneumonia developed about 1 month ago, and his COPD is at his baseline.

In view of the patient's fragile medical condition, a discussion is held with the surgeon and the facility's nurse manager regarding the suitability of this case to be done at a self-standing outpatient surgicenter. The surgeon states that the patient had

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a similar procedure without any complications at another facility about 6 months ago, easily managed with a peribulbar block and minimal sedation. Stating that he would require nothing more than mild anxiolysis, if any, to supplement his block, the surgeon insists on proceeding with the case under these conditions. He also states that the patient has been in urgent need of this procedure for over 6 months, twice cancelled in the past due to pneumonias and COPD exacerbations. The surgery is scheduled for 45 min, and rescheduling it would pose many logistic challenges, including having to hold anticoagulation again, exposing the patient to further cardiovascular events. In view of the patient's ability to tolerate a similar recent procedure under the same circumstances, the decision is made to proceed with the case as planned, with the understanding that the patient's medical condition makes him an unsuitable candidate for a general anesthetic at this facility (**L-1**).

In the preop area, as routinely done, the patient is fitted with a nasal cannula with integrated capnography sampling tubing. Two minutes before entering the OR, the patient is sedated with midazolam (Versed) 0.5 mg IV and fentanyl 25 mcg IV and transported to the operating room (OR). Once in the OR, lidocaine 80 mg IV is administered, standard monitors are applied and, in order to preoxygenate the patient while attempting to maintain his hypoxic respiratory drive, he is instructed to take long, slow deep breaths of O_2 provided at 3 L/min via nasal cannula for the next minute or so, while the effect of the three aforementioned drugs peaks concurrently. Meanwhile, an anesthesia circuit fitted with a face mask is on standby, in case the patient requires positive pressure ventilation with 100 % O_2. A set of vital signs is obtained simultaneously showing HR of 72 bpm, BP of 120/53 mmHg, RR of 18 breaths/min, and O_2 saturation of 98 %. At this time, propofol 25 mg IV is infused. About 1 min later, the patient becomes sleepy, the respiratory rate becomes slower and somewhat irregular, and he is unable to produce coherent verbal responses. At this point, the surgeon performs an uneventful single-injection infraorbital peribulbar block. The patient continues to breathe spontaneously with the aid of gentle jaw thrust, and the O_2 saturation remains above 90 % throughout. He is then allowed to fully wake up after the propofol is redistributed, while the face is prepped and draped. A few minutes later, the surgeon makes the incision and the surgery proceeds as planned.

Approximately 5 min after the incision had been made and the surgery is in progress, the patient complains of severe eye pain, with no relief from additional local anesthetic drops instilled by the surgeon. The surgeon requests a deeper level of sedation. This is provided with additional midazolam 1.5 mg IV and fentanyl 75 μg IV in small increments. Unfortunately, this deeper level results in no pain relief. Instead, in response to further surgical stimulus, the patient becomes more restless, uncooperative, and attempts to reach with his hands towards the surgical field. Now struggling with hemostasis, the surgeon states that he cannot continue with the planned surgery under these conditions and requests even deeper sedation. However, by now the patient is obstructing his airway with intermittent episodes of oxygen desaturation below 85 %. The patient is not a suitable candidate for a general anesthetic at an outpatient facility, the provided amount of IV sedation is inadequate, further increasing the amount of sedation would risk airway and cardiovascular

complications, and stopping the surgery at this point in time may result in permanent loss of vision.

The anesthesiologist increases the oxygen flow to 6 L/min, proceeds to bolus the patient with 30 mg propofol and 20 mg ketamine, along with 25 μg phenylephrine IV. Meanwhile, the circulating nurse is instructed to apply soft restraints to both upper extremities, as the anesthesiologist inserts an oral airway and a nasal trumpet lubricated with 2 % lidocaine jelly by reaching under the drapes. Following this, the anesthesiologist quickly prepares a syringe containing 600 mg of propofol (60 mL) plus 50 mg of ketamine and begins an infusion of this mixture using a Bard InfusOR® propofol syringe infusion pump set for a propofol infusion rate of 50 μg/kg/min. In order to shorten the detection time for hemodynamic abnormalities, the blood pressure cuff is cycled every 1 min for the duration of the remainder of the case (approximately 35 min). Small boluses of phenylephrine 10–20 μg IV are infused periodically in order to maintain a BP in the range of 120–130/50–60 mmHg (**L-2**, **L-3**, **L-4**).

The surgery proceeds as planned with the BP and HR in the target range and an O_2 saturation greater than 90 %. Towards the end of the procedure, the infusions are terminated and the patient is taken to the postanesthesia care unit (PACU) in stable condition. He spends 1 h in the PACU and is transported back to the SNF without complications.

Lessons Learned

L-1: **Different levels of sedation and the slippery slope**

Sedation and analgesia include a continuum of states of consciousness ranging from minimal sedation (anxiolysis) to general anesthesia (GA). Monitored anesthesia care may include varying levels of sedation, analgesia, and anxiolysis as necessary. The American Society of Anesthesiologists (ASA) has defined these different levels of sedation and their effect on vital physiological functions (See Table 62.1) [1].

As it is often the case at outpatient facilities performing ophthalmologic surgeries, the patient population consists primarily of senior patients. Oftentimes, some of these patients are very fragile, have a complex medical history, and an advanced level of disease, rendering them unsuitable candidates for general anesthesia. Therefore, at this facility, complex ASA three patients are only scheduled for cases that can be done with minimal to moderate sedation. Deep sedation and general anesthesia are reserved for healthier patients. Complex patients that require a general anesthetic are usually scheduled to have their procedure done at a nearby hospital, where resources are available to better manage any potential complications.

The ASA has also defined a physical status classification system for assessing the fitness of patients before surgery (See Table 62.2).

In the case being discussed, the patient is a very complex ASA physical status 3, and it is questionable if he should have been a candidate for a self-standing outpatient facility. His multiorgan system dysfunction presented a high risk for potential

Table 62.1 Continuum of depth of sedation: definition of general anesthesia and levels of sedation/analgesia

Critical parameter	Minimal sedation (anxiolysis)	Moderate sedation/ analgesia (conscious sedation)	Deep sedation/ analgesia	General anesthesia
Responsiveness	Normal response to verbal stimulation	Purposeful response to verbal or tactile stimulation	Purposeful response following repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular function	Unaffected	Usually maintained	Usually maintained	May be impaired

Defined and adopted by the American Society of Anesthesiologists [1]

Table 62.2 ASA physical status patient classification

ASA physical status	Characteristics
1	A normal healthy patient
2	A patient with mild systemic disease
3	A patient with severe systemic disease
4	A patient with severe systemic disease that is a constant threat to life
5	A moribund patient who is not expected to survive without the operation
6	A declared brain-dead patient whose organs are being removed for donor purposes

Data from American Society of Anesthesiologists [1]

cardiovascular and pulmonary complications. Conversely, the difficulty anticipated in overcoming the logistic obstacles and the risk of exposing the patient to another anticoagulation hiatus introduced by postponing and rescheduling the procedure were taken into consideration by the surgeon and the anesthesiologist. The information presented by the surgeon regarding his recent uneventful procedure emboldened the team to proceed with the case as planned, not expecting the difficulties encountered intraoperatively. Even though only minimal sedation was requested by the surgeon, this proved to be insufficient and rapidly escalated into the deep sedation, if not the general anesthesia category. Whether or not this decision was justified is difficult to assess.

As evidenced by the events of this case, it is not always possible to predict an individual patient's response, and there is always a possibility of a need to escalate the level of anesthesia or even convert to general anesthesia when necessary. Therefore, the decision to accept a patient for any planned level of sedation must

take into consideration that events out of the anesthesiologist's control may force escalation to a higher level of sedation (See **L-2**). Conversely, an anesthetic that has migrated into a deeper level than intended requires prompt action and possibly some rescue cardiovascular and airway interventions.

L-2: **Importance of having an alternate anesthetic plan**

It is accepted that the efficacy of a single inferior injection peribulbar block is affected by numerous factors, including the local anesthetic agents used, the location of the block, the use of a single versus double injections, and, most importantly, the experience of the individual performing the block. Further, it is also commonly accepted that prior eye surgeries may increase the failure rate. At this facility, all peribulbar blocks are done by an experienced surgeon, not by the anesthesiologist.

Whereas most cases of inadequate peribulbar blocks involve incomplete globe akinesia (usually managed by directing the patient to keep looking in a specified direction and avoid looking around), or incomplete anesthesia/analgesia with the patient reporting mild pain and discomfort (usually managed with additional anesthetic drops instilled by the surgeon plus a modest increase in the level of IV sedation), the patient in this case complained of severe pain, not relieved by either local anesthetic drops or by an increase in the level of IV sedatives and analgesics. Because there is an intrinsic peribulbar block failure rate, a good anesthetic plan must anticipate this possibility and consider alternate techniques, should the block fail to provide adequate surgical conditions.

The anesthesiologist in this case was suddenly faced with the dilemma of canceling the procedure that had already been started, therefore exposing the patient to the risk of permanent vision loss or proceeding with a deeper level of anesthesia, risking airway and cardiovascular complications in a fragile patient with significant AS and numerous other medical issues. Therefore, the anesthesiologist had to devise an alternate plan and intervene immediately with rescue maneuvers (see Table 62.3).

First, the O_2 flow was increased to 6 L/min as the patient's level of sedation was increased. The strict goal of maintaining hemodynamic stability while the level of sedation was increased was accomplished by using a combination of small doses of propofol (30 mg IV), ketamine (20 mg IV), and phenylephrine (25 mcg IV) (See **L-4**). Second, maneuvers to rescue the airway were put in place immediately after the patient showed signs of an increased level of sedation by inserting an oral airway and a nasal trumpet. Lubricating the above with 2 % lidocaine jelly prior to insertion minimized stimulation. Third, the airway maneuvers were done underneath the sterile drapes, without the need to disrupt the surgical field, other than by having the surgeon pause for a few minutes. Fourth, the anesthesiologist delegated tasks by instructing the circulating nurse to apply soft restraints to the now sedated patient's upper extremities, thus preventing any further reaching into the surgical field. Fifth, in order to shorten the reaction time to hemodynamic changes in the absence of an arterial line, the anesthesiologist changed the noninvasive blood pressure (NIBP) cuff cycle interval to 1 min. Lastly, in order to provide continuous sedation and analgesia for the rest of the procedure while maintaining an adequate coronary perfusion pressure, the anesthesiologist prepared a syringe containing a

Table 62.3 Summary of quick rescue interventions and their advantages

Intervention	Advantage
Increasing O_2 flow	Minimizes periods of O_2 desaturation
Combination of small doses of propofol, ketamine, and phenylephrine	In drips and small amounts, ketamine provides both sedation and analgesia with hemodynamic stability, counteracts hypotension caused by propofol, and minimizes side effects
Insertion of oral and nasal airways	Easily tolerated in patient under moderate sedation without the need for airway instrumentation to induce GA
Lubrication of airway with 2 % lidocaine jelly	Causes minimal stimulation by the airway
Patient manipulation done under sterile drapes	No need for lengthy surgical interruptions and disruption of sterile field
Use of upper extremity soft restraints	Removes possibility of disrupting sterile field by patient's involuntary movements
Increase NIBP cycle to 1 min intervals	Shortens hemodynamic detection time in patient without arterial line
Use of Bard InfusOR® syringe pump	Readily available and quickly programmed

mixture of 60 mL of propofol (600 mg) and 50 mg of ketamine. He then started a continuous infusion of the above mixture using a Bard InfusOR® propofol syringe pump set to a rate of 50 μg/kg/min. This setting, along with periodic small boluses of phenylephrine, provided the sedation, analgesia, and hemodynamic stability necessary in this case.

All these complex maneuvers were well orchestrated by members of the OR team and resulted in restoring the surgical procedure within 2 min without the need for any further interventions.

L-3: **Special considerations for the patient with aortic stenosis**

While the range of values for a normal AVA in an adult is anywhere from 2–4 cm^2, most patients will not become symptomatic until their AVA becomes less than 0.9 cm^2. Hemodynamically significant stenosis occurs when AVA is less than 1.2 cm^2, but these patients are not necessarily symptomatic. Any procedures in which hemodynamic changes are anticipated require the use of an arterial catheter to shorten the detection time for hemodynamic abnormalities. Induction of general anesthesia usually mandates the guidance provided by an arterial catheter placed prior to induction. Our patient's last measured AVA was 1.2 cm^2. Therefore, it was expected that he would not tolerate hemodynamic instability well.

In this case, the noxious surgical stimulus resulting from a patchy peribulbar block was leading to hypertension and tachycardia, usually not well tolerated by patients with concurrent AS and CAD, as both of these hemodynamic changes further increase myocardial oxygen demand. Conversely, any increase in the level of sedation may risk causing significant hypotension, via a combination of diminished sympathetic tone, myocardial depression, and direct vasodilation. Potentially, the results of hypotension could be even more devastating.

This case posed numerous challenges for the anesthesiologist. Not only did maintaining a patent airway had become an issue, but the advanced degree of AS mandated avoidance of hemodynamic swings, including avoidance of arrhythmias (especially bradycardia), maintaining ventricular filling pressure, and maintaining coronary perfusion pressure by avoiding hypotension at all cost. As is usually the case, the outpatient surgicenter where the procedure was being conducted did not have any provisions for emergency invasive blood pressure measurements.

Since moderate sedation with midazolam and fentanyl was inadequate to supplement a failed peribulbar block, the next step in the continuum was deep sedation with propofol. However, it is well accepted that propofol, the injectable agent used by anesthesiologists in most cases requiring deep sedation, invariably leads to a significant reduction in blood pressure in a dose-dependent fashion. Ketamine, on the other hand, maintains an adequate afterload due to its sympathomimetic effects, although at the expense of increasing myocardial oxygen demands, in part by increasing the heart rate in a dose-dependent fashion. However, when used in combination with propofol in low doses, the resulting small hemodynamic changes are usually well tolerated (See **L-4**). Concurrently, periodic small doses of phenylephrine (10–25 μg IV) can be titrated in cases such as this one, where maintaining afterload is the main consideration.

The combination of the three drugs as described above was used successfully in order to provide the deep level of sedation required to complete the surgical procedure, while at the same time maintain tight hemodynamic control in the absence of an arterial cannula. The patient's blood pressure remained in the 110–130/50–60 mmHg range, his heart rate in the 70–80 bpm range, and his O_2 saturation above 90 % for the rest of the procedure. The NIBP was cycled every 1 min in order to finely titrate small phenylephrine boluses needed to maintain the desired blood pressure.

L-4: **Ketamine may be beneficial when used as an adjuvant to propofol in moderate/deep sedation**

Ketamine has been used for decades in the medical community. Its use by emergency room (ER) physicians in pediatric patients undergoing ER procedures that require sedation has been well described. Outside of the pain management community, perhaps due to the introduction of newer drugs, its history of veterinary use, its perception as a substance of abuse, and its side effect profile, ketamine's use for induction or maintenance of anesthesia has substantially diminished, and it is oftentimes not readily considered by general anesthesiologists as an integral part of their arsenal. Moreover, the option to use ketamine in subhypnotic doses as an adjuvant to propofol sedation is often completely ignored. Instead, anesthesiologists tend to routinely resort to supplemental opioids to enhance anesthesia and improve patient comfort. This combination may result in clinically significant respiratory depression, at times necessitating prompt airway rescue maneuvers (Table 62.4).

Ketamine, however, possesses qualities not shared by most other anesthetic agents. When used for sedation in small amounts, ketamine maintains cardiac output and vascular tone in cardiac and hypovolemic patients, maintains

Table 62.4 Advantages and disadvantages of using ketamine as an adjuvant in propofol sedation

Advantages	Disadvantages
Provides deep analgesia at subhypnotic doses	May cause psychotomimetic side effects[a]
Counteracts hypotensive effects of propofol	May cause visual disturbances[a]
Reduces opiate and propofol requirements	May increase salivation[a]
Causes less hypopnea/apnea than propofol alone	May increase intraocular and intracranial pressure[a]
Well established pharmacological profile	May cause hypertension and tachycardia[a]
May reduce patient involuntary movement (controversial)	Reputation as a veterinary drug
	Reputation as a drug of abuse

[a]Rarely documented when used at subhypnotic doses in conjunction with propofol

ventilatory drive, and causes some bronchodilatation. Ketamine also provides deep analgesia at plasma concentrations significantly lower than those producing hypnosis. It has also been suggested that ketamine produces a dose-dependent reduction in the incidence of patient responsiveness to local anesthetic infiltration and may help to minimize the involuntary movements often seen during sedation with propofol alone.

The adjunctive use of ketamine, when used in subhypnotic doses during continuous propofol sedation, provides significant analgesia and minimizes the need for supplemental rescue opioids, resulting in less respiratory depression. It also counteracts the hemodynamic depression of propofol. Subhypnotic doses, when used in combination with propofol are usually devoid of psychotomimetic side effects (e.g., dreams, hallucinations) and visual disturbances (e.g., double vision, nystagmus) [2].

The case being discussed required prompt escalation of the level of sedation in a patient with moderate/severe aortic stenosis. The patient was not a candidate for a general anesthesia at an outpatient surgicenter that does not even have the capabilities for emergency invasive monitoring. In this case, a small bolus loading dose of propofol 30 mg plus ketamine 20 mg was given. A mixture of ketamine and propofol (50 mg ketamine into 60 mL propofol, yielding a ketamine concentration of 0.833 mg/mL) was then provided as an IV continuous infusion using a syringe pump set for a propofol rate of 50 μg/kg/min. This combination proved very effective in providing the desired level of sedation and analgesia, while maintaining adequate coronary artery perfusion in a patient with AS. No additional opiates, which may have resulted in further compromising the already tenuous the patient's airway, were required. Small amounts of phenylephrine were used to maintain the BP in the desired range without causing the potential tachycardia that may have resulted by increasing the dose of ketamine any further. The hemodynamic stability of the propofol/ketamine combination and the analgesic effect of ketamine made it suitable for providing deep sedation in an urgent situation to this fragile patient.

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Chapter 63
Endoscopic Sinus Surgery at an Outpatient Surgicenter

Luis M. Rivera

The patient is an otherwise healthy 24-year-old, 86-kg male with a history of chronic sinusitis, scheduled for endoscopic sinus surgery (ESS) at an outpatient surgicenter. The procedure includes septoplasty, maxillary anthrotomy, ethmoidectomy, turbinate reduction, and submucosal resection. This is the patient's third nasal surgery with no history of prior complications. He has been smoking one pack of cigarettes per day for 6 years. Preoperative vital signs upon entering the operating room (OR) are blood pressure (BP) 128/84 mmHg, heart rate (HR) 86 beats per minute (bpm), respiratory rate (RR) 18 breaths per minute, and room air oxygen saturation (O_2 Sat) 97 %. The surgeon requests controlled hypotension in order to minimize bleeding and improve surgical field visual conditions throughout the procedure, with a target systolic blood pressure (SBP) in the 80–90 mmHg range.

General anesthesia is induced with midazolam 2 mg IV, lidocaine 100 mg IV, fentanyl 150 μg IV, propofol 200 mg IV and rocuronium 50 mg IV. He is easy to ventilate by mask. After loss of twitches on train-of-four (TOF) stimulation, the trachea is intubated easily and the table is turned 180°, with the head of the bed elevated 15°. A few minutes before the surgeon injects local anesthetic (lidocaine 1 % with epinephrine 1:200 K) into both nostrils of the patient's nasal mucosa, the anesthetic is transiently deepened with sevoflurane, and an additional 150 μg fentanyl and 100 mg propofol are administered in order to keep the patient's SBP from spiking due to absorption of the epinephrine contained in the local anesthetic injection. Additional boluses of propofol are given during the injection period with a resultant SBP varying between 70 and 120 mmHg, finally settling into the desired range. He is kept paralyzed and is mechanically ventilated throughout the rest of the case **(L-1)**.

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The surgery proceeds as planned with an SBP in the 70–80 mmHg target range and a clear surgical field. Anesthesia is maintained with 3–4 % sevoflurane and the head of the bed elevated 15° in order to maintain the SBP in the desired range **(L-2)**. Periodic rocuronium boluses are administered throughout the case to maintain muscle relaxation **(L-1)**. Approximately 1.5 h into the surgery, the surgeon moves from the right to the left nostril. This results in the patient's BP increasing to the 140–150/80–90 mmHg range and the HR to the 100–110 bpm range. What was a clear surgical field has suddenly become bloody. The SBP is brought back to the desired range with an additional fentanyl 150 μg IV bolus and two boluses of propofol 100 mg IV each. The surgeon injects additional local anesthetic into the left nostril mucosa, the field becomes less bloody, and the surgery continues as planned, although with a moderate degree of persistent oozing requiring frequent suctioning by the surgeon, lengthening the duration of the procedure to over 4 h.

At the end of the surgery, muscle relaxation is reversed with neostigmine 5 mg and glycopyrrolate 0.9 mg IV, the airway is suctioned meticulously, and the patient is allowed to emerge for an awake extubation. As the patient emerges, he begins to buck and cough, shakes his head vigorously, and attempts to pull on the endotracheal tube, as his heart rate increases to the 130s bpm and his BP to the 140s/90s mm Hg **(L-3)**. The patient is extubated while awake but copious amounts of blood are dripping from his nostrils. The surgeon is called back into the operating room, where he proceeds to repack the patient's nose while the patient is awake. In the recovery room, the patient is retching and blood is still coming out of his nose. The nausea is treated first with ondansetron 4 mg IV, followed by phenergan 12.5 mg IV with eventual resolution. However, the bleeding persists, and the surgeon decides to have the patient admitted to a nearby hospital for overnight observation due to persistent bleeding **(L-4)**.

Lessons Learned

L-1: **Advantages of succinylcholine over non-depolarizing neuromuscular blockers**

There are few outpatient procedures that truly require absolute intraoperative muscle relaxation. In most cases, muscle relaxation is only needed in order to facilitate intubation. Succinylcholine is an excellent choice in these cases. The main drawback is a high incidence of postoperative myalgias. Whereas a small defasciculating dose of a non-depolarizing agent (e.g., rocuronium 3–5 mg IV) given immediately prior to induction will not prevent hyperkalemia, elevated intracranial pressure (ICP), or intraocular pressure (IOP), it does prevent fasciculations and will usually decrease the incidence of myalgias. In general, in order to attain ideal intubating conditions, the use of a defasciculating agent will require that the dose of succinylcholine be increased to approximately 2 mg/kg (Table 63.1).

Succinylcholine has the advantage of not requiring reversal with neostigmine (a known pro-emetic drug) and glycopyrrolate, often causing tachycardia. It also has

Table 63.1 Advantages and disadvantages of succinylcholine over non-depolarizing neuromuscular blockers

Advantages	Disadvantages
Rapid onset and short duration	Increases ICP and IOP
Allows spontaneous breathing and the use of RR and $etCO_2$ as monitors	May produce hyperkalemia
No need for pro-emetic reversal agent and anticholinergic	May trigger malignant hyperthermia
Minimizes likelihood of residual paralysis at the end of the case	High incidence of fasciculation myalgias
Low cost and readily available	May cause extended paralysis in patients with undiagnosed pseudocholinesterase deficiency

the shortest onset and duration of all available neuromuscular blockers, and it is inexpensive and readily available at all surgical facilities.

Perhaps one of the most significant advantages of using succinylcholine is the ability of allowing the patient to breathe spontaneously during the surgical procedure. Spontaneous breathing allows the use of the variations of the RR and end-tidal carbon dioxide concentration ($etCO_2$) as monitors in assessing the need for further analgesia. Oftentimes, these two parameters will change in response to surgical stimulation well before the automated blood pressure cuff has had time to cycle in the next 3–5 min.

In general, muscle relaxants beyond those needed for intubation are usually unnecessary during ESS and other ear, nose, and throat (ENT) cases. In the rare occasion when muscle relaxation is required during some intraoperative periods, the shorter acting agents are preferred. Mivacurium, possibly the most used agent in outpatient surgery, has unfortunately been taken off the market. Its closest replacement (Nimbex, cisatracurium) has a much slower onset and longer duration of action. Alternatively, a short-acting agent such as rocuronium may be used in small doses to facilitate intubation (0.3–0.4 mg/kg) and, in most cases, its effect will dissipate within 30 min or less. This small dose will take longer to achieve adequate (although perhaps not ideal) intubating conditions, where the vocal cords are abducted, but the patient may exhibit some movement in response to the laryngoscopy. A 2- to 3-min onset should be expected, as opposed to about a 90-s onset when using 0.6 mg/kg. It remains controversial whether the wait time may be diminished somewhat by using a priming dose and potentiating its effect with a volatile agent. In cases that necessitate muscle relaxation, maintaining at least one twitch on TOF ensures that the agent is readily reversible when the surgeon suddenly announces that he is done. Frequent titration rather than large boluses is the key to timely reversal.

As demonstrated by the events of this case, it is imperative to stay vigilant throughout the case and inform the surgeon if he is operating outside the territory covered by the local anesthetic. A patient who is kept breathing spontaneously will typically increase his respiratory rate and decrease his $etCO_2$ well before a noninvasive BP cuff set to 3- or 5-min intervals has had a chance to cycle and display an updated value. Precious time can be wasted as the cuff recycles repeatedly due to it

Table 63.2 Surgical scoring of quality of field designed for use with ESS

Grade	Assessment
0	No bleeding (cadaveric)
1	Slight bleeding – no suctioning required
2	Slight bleeding – occasional suctioning required
3	Slight bleeding – frequent suctioning required; surgical field threatened a few seconds after suctioning removed
4	Moderate bleeding – frequent suctioning required; surgical field threatened directly after suctioning removed
5	Severe bleeding – constant suctioning required; bleeding appears faster than can be suctioned. Surgical field severely threatened and surgery usually not possible

Data from Refs. [1, 2]

being unable to measure an SBP that has suddenly increased beyond the expected range of the next automated measurement. Timely recognition of this scenario, followed by a quick bolus of propofol, and cessation of noxious stimulus by the surgeon will provide the surgeon adequate time to inject additional local anesthetic while the blood pressure is kept from escalating before resuming the surgery.

In some cases, as demonstrated herein, even a brief period of hypertension may cause dilatation of previously vasoconstricted arterioles, unnecessary and persistent rebleeding, and a compromised visual field. By limiting the use of a neuromuscular blocker to a single dose of succinylcholine in order to facilitate laryngoscopy and intubation, and allowing the patient to breathe spontaneously for the rest of the case, a rapidly escalating level of surgical stimulation may have been detected earlier resulting in a less dramatic SBP spike and ensuing rebleeding.

L-2: **The problem of nasal mucosal bleeding during nasal surgery and common methods available to provide controlled hypotension**

Intraoperative bleeding is one of the major problems encountered in ESS. The nasal mucosa is extremely vascular, and control of bleeding is one of the most important issues in this type of surgery. Many ENT cases, and certainly most if not all of the outpatient ESS and nasal cases, include the injection of local anesthetic by the surgeon at the beginning of the procedure. Most surgeons use lidocaine, bupivacaine, or a combination of both, with 1:100–1:400 K epinephrine to promote vasoconstriction. Most surgeons also routinely request a controlled hypotensive technique to minimize bleeding and optimize visibility. It has been demonstrated that controlled hypotension (CH) can improve the dryness of the surgical field in ESS. Specifically, surgical conditions have been reported to be influenced by the type of anesthetics, BP, and HR in a statistically significant manner. Consequently, the way the anesthetic is conducted has a significant impact on how successful the surgeon will be at controlling bleeding and therefore what kind of visibility and surgical conditions he will have.

Surgical field conditions in ESS are scored by many surgeons on a scale that was originally described in the anesthesia literature dating back to the 1980s (Table 63.2) [1, 2].

As the surgeon injects local anesthetic with epinephrine, inevitably, some of it will be rapidly absorbed by the vascular mucosa. Untreated, this will likely cause hypertension, tachycardia, and suboptimal vasoconstriction. Unless the patient has

Table 63.3 Common methods used to provide controlled hypertension

Method	Advantages	Disadvantages
Increased volatile agent concentration	Effective, titratable, wears off at the end of the case	Slow to become effective, delayed emergence, PONV[a], myocardial depression
Beta Blockers	Fast and effective	Bronchospasm in high doses, long acting except esmolol
Opiates	Fast and effective	PONV, hypopnea, delayed emergence, accumulate at high doses except remifentanil (costly)
Direct vasodilators	Fast, effective, and titratable	NTG[a] and SNP[a] typically require arterial line guidance, reflex tachycardia, hydralazine slow to effect
Propofol	Fast, effective, highly titratable, wears off quickly, no PONV	Cost, apnea at high doses
Calcium channel blockers	Effective	Long lasting except nicardipine IV
ACE Inhibitors	Effective	Slow to effect, long lasting, IV enalaprilat not readily available at most facilities, reflex tachycardia
Elevated head of the bed	Effective as adjuvant, promotes venous drainage	Potential for brain ischemia, usually not effective by itself

[a]*PONV* stands for post operative nausea and vomiting, *NTG* for nitroglycerin, *SNP* for sodium nitroprusside

significant risk factors that may lead to stroke or myocardial infarction, the transient tachycardia is usually well tolerated. However, in order to ensure high-quality surgical field conditions, the hypertensive spike should be minimized. Pharmacologically, deepening of the anesthetic with an increase in the volatile agent concentration for a few minutes before injection, plus a bolus of propofol given about 1 min before the surgeon infiltrates the nasal mucosa with local anesthetic, may be all that is required during this period. Procedurally, setting the BP cuff to 1-min intervals and vigilant monitoring of the vital signs during this critical period is an equally important requirement. Additional propofol boluses may be required to keep the SBP from escalating. While this technique is sufficient to momentarily prevent the hypertensive spike generated by the epinephrine absorbed by the nasal mucosa during the injection period, oftentimes it needs to be supplemented by another method in order to provide CH throughout the rest of the surgery.

There are various methods commonly available to the anesthesiologist to provide CH. Selection of a specific method will vary according to patient characteristics, anesthesiologist preference, and availability of pharmacological agents (Table 63.3).

For ESS, with the liberal use of infiltrated local anesthetic by the surgeon, the patient should wake up almost pain-free at the end of the surgery. Therefore, the use of narcotics beyond the amount needed for intubation is usually unnecessary. Additionally, due to accumulation, postoperative nausea and vomiting (PONV), and hypopnea, perhaps narcotics do not constitute the best method of providing CH in

this kind of surgery. Methods requiring arterial line guidance are also logistically impractical at outpatient surgicenters.

In this case, a seemingly innocuous event, namely, an intraoperative transient increase in the patient's BP resulted in turning the quality of the operative field from a Grade 2–3, to a Grade 3–4 (see Table 63.2), with deep repercussions. It is unclear whether the effect of the local anesthetic had partially worn off by the time the surgeon began surgery on the second nostril. It is also possible that the left nostril had been inadequately topicalized, or that the initial plan was to inject local anesthetic when ready to begin surgery in the left side, but this was overlooked or forgotten by the surgical team. Regardless of the reason, vigilance and astute actions by the anesthesiologist may have prevented the cascade of events leading to an unplanned hospital admission.

The anesthesiologist in this case chose to provide CH with a combination of high dose narcotics and high concentration volatile agent. Whereas this is certainly an acceptable method, there are others that, in retrospect, may have proved more effective (See Table 63.3). Once again, it is likely that the most stimulating portion of this surgery is the laryngoscopy and insertion of the endotracheal tube. With the liberal use of infiltrated local anesthetic by the surgeon, the patient should wake up almost pain-free, and the use of narcotics beyond the amount needed for intubation is usually unnecessary. Spraying the trachea with a laryngotracheal topical anesthesia (LTA) kit and coating the endotracheal tube with 4 % lidocaine paste or 2 % lidocaine jelly could have made it possible to avoid narcotics altogether during intubation. Further, the high dose of narcotics and volatile agent, combined with the likely significant amount of blood in the stomach due to persistent bleeding and the surgeon opting to not use a throat pack, may have contributed to PONV.

An alternate method to provide CH in this young, healthy patient is a combination of several agents devoid of these side effects. A propofol drip is usually highly effective and titratable. General anesthesia based on propofol infusion may have the advantage of decreased bleeding compared with conventional inhalation agents, making ESS technically easier and safer by improving endoscopic visualization of the surgical field [3]. The cumulative propofol dose may be reduced by using a weak intravenous direct vasodilator such as enalaprilat or hydralazine, given early in the procedure. A young, healthy patient is likely to compensate for the vasodilation by increasing the HR. Therefore, a beta-blocker such as labetalol may be used to keep the HR low. It is important to note that the half lives of both the vasodilators and the beta blockers are long compared to the half life propofol. Thus, they can be used to provide a baseline degree of hypotension, with any further deepening and titration done with propofol as required to meet the target SBP goal. The end result of this technique is typically a return of BP close to the preop value at the end of the case.

L-3: **Comparison of awake versus deep extubation**

There are numerous advantages and disadvantaged encountered with deep extubations. Perhaps one of the largest obstacles is the lack of experience resulting from discouragement in academia during training of residents. While the decision to perform a deep extubation is not trivial and requires careful thought and preparation, oftentimes it can yield superior results.

Table 63.4 Comparison of deep versus awake extubation

	Deep extubation	Awake extubation
Advantages	No bucking, coughing, choking, gagging	Decreased risk of compromised airway
	Avoidance of patient recall of ETT	Decreased risk of potential aspiration
	Saves time at end of case	
Disadvantages	Not encouraged in academia	Must wait until patient fully awake
	Contraindicated in patients with aspiration risks	Bucking, coughing, may cause rebleeding, wound dehiscence, increased ICP/IOP
	Relatively contraindicated in difficult airways	Choking, gagging may cause vomiting
	Relatively contraindicated in difficult to ventilate patients	

The most important factor in making this decision is the patient's characteristics. A patient that has a difficult airway, is difficult to ventilate by mask, or has significant tracheal aspiration risk factors is not a good candidate. Conversely, a patient in whom coughing, bucking, chocking, and gagging pose risk factors for complications is a likely candidate (Table 63.4).

The case being discussed has factors that merit consideration beyond the ones mentioned above. The patient is young and has a history of smoking and chronic mucosal inflammation due to chronic sinusitis. In general, these factors may result in a reactive airway, precipitating coughing, and bucking during emergence. On the other hand, he has active, although minimal, bleeding from his nasal mucosa. Although the pH of blood is not low enough to generally cause alveolar damage if he were to aspirate some blood, it may certainly precipitate laryngospasm in an unprotected airway.

To further complicate matters, prophylactic antiemetics were not used due the patient's lack of historical risk factors. However, the length of surgery, high dose of narcotics, high concentration of volatile agent, use of neostigmine, and large amount of swallowed blood resulting from not having a throat pack in place, may have turned this case from a low to a high risk of PONV, usually best managed by aggressive use of prophylactic antiemetics. The increased intra-abdominal and intrathoracic pressures produced during any coughing, bucking, gagging, retching, or vomiting may have contributed to nasal rebleeding and may have been avoided with a deep extubation and aggressive use of prophylactic antiemetics.

The decision to extubate deep is certainly a judgment call to be made by the anesthesiologist at the end of the procedure. However, it must be kept in mind so that the intraoperative course is conducted in a manner that facilitates this goal, even if it is abandoned later on. Meticulous suctioning of the airway and the stomach, along with insertion of an oral airway coated with lidocaine jelly, may facilitate a smooth deep extubation. If an awake extubation is necessary, lidocaine 1.5–2 mg/kg given IV approximately 1–2 min before anticipated emergence may serve to blunt airway reactivity without the need for opiates. Esmolol 30–50 mg IV may also be used to keep the HR from escalating during emergence.

L-4: **Repercussions of an unplanned hospital admission after an outpatient surgery**

An unplanned hospital admission after what was originally scheduled as an outpatient surgicenter procedure is not a trivial event. There are numerous ramifications including patient safety, allocation of resources, engagement of the emergency medical system (EMS), communication, and transfer of care issues. Although requirements vary from state to state, and state requirements may at times conflict with federal regulations and the different agencies that establish regulatory power over outpatient facilities, it is imperative that these requirements be met.

Presently, the State of California requires that, in the event that a patient needs continuance of specialized inpatient care, adequate arrangements must be made by the surgical facility with the receiving facility, including selecting the specific mode of transportation. All patient transfers must be made by ambulance. Without discussing the details in this document, let it suffice to say that a lengthy paper trail will then follow this event.

Unanticipated, but nonemergent transfers for conditions such as uncontrollable pain, nausea and vomiting, the need for additional diagnostic work-up, and monitoring and observation due to unexpected circumstances may be made by a contracted ambulance service. Emergency transfers usually require activation of the EMS ambulance via a call to 911 for transport to the nearest receiving hospital. Typically, an emergency transfer results in notification of the event to the surgical facility's administrative director, medical director, and department chiefs, and will be reviewed as part of the peer review program to assess whether there is something that could have been done or should be done differently to prevent the need for transfer.

The receiving hospital must also be notified of impending arrival, and all records must be completed and submitted with the patient at the time of transfer to the extent feasible. Oftentimes, the anesthesiologist must accompany an unstable patient to the receiving facility, with a significant impact on the surgical schedule for the remainder of the day. Upon discharge from the hospital, the surgicenter must obtain a discharge summary or observation stay report from the hospital.

As of the time of this writing, the State of California requires that an unplanned patient transfer from a surgicenter to a hospital be reported to the Office of Statewide Health Planning and Development, along with submission of the patient's chart, disposition, and the name of the physician requesting the transfer. It is unclear whether or not this agency is currently reporting the event to the state medical board. There is ongoing discussion at the federal Center for Medicare and Medicaid Services (CMS) regarding deducting a portion of the allocated payment to the surgical facility for any unplanned transfers.

In the case being discussed, the patient was transferred to a hospital for monitoring and observation due to unexpected circumstances. However, since no prior arrangements had been made with a contracted ambulance service, the patient was transferred by engaging the EMS system (calling 911 for an ambulance). In view of the need to engage EMS for an unplanned event where no prior arrangements had been made, the event was categorized as a surgical complication necessitating transfer, with the triggering event being excessive bleeding.

The event was discussed at the medical executive committee (MEC) meeting, reviewed by peers, and the lessons presented above discussed in an attempt to minimize the possibility of such events recurring in the future. While the unplanned admission resulted from a surgical complication (excessive bleeding), it is possible that timely and astute interventions by the anesthesiologist may have yielded a different outcome.

While the details of this process are best left to the administrators, it is imperative that physicians understand all the possible repercussions.

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