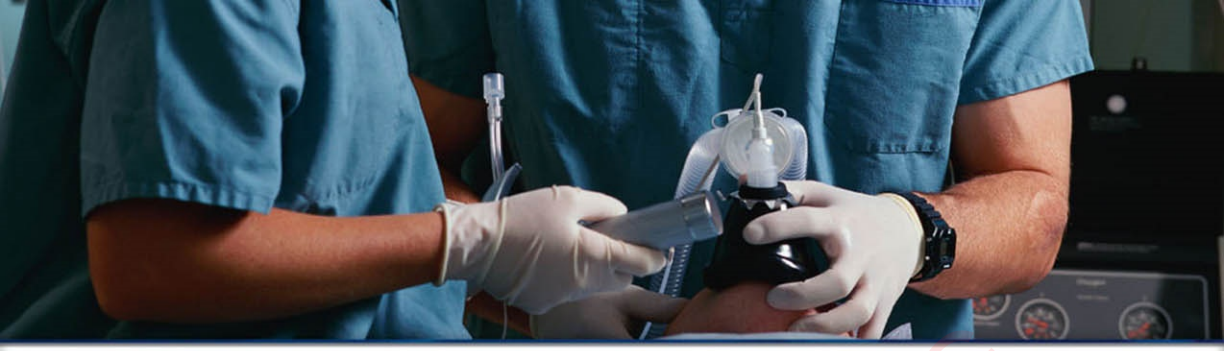

Anesthesiology Oral Board Flash Cards

150 two-sided cards help you develop the critical thinking skills necessary to ace the Anesthesiology Oral Boards

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McGraw Hill LANGE

Anesthesiology Oral Board Flash Cards

ABOUT THE CARDS

Congratulations! You have made a commitment to becoming an outstanding anesthesiologist. With these flash cards and some regular practice, you will easily gain the skills to become a decisive anesthesiologist who demonstrates wisdom, judgment, and skill in prioritizing, and who is well-respected by their peers, surgical colleagues, and patients.

There are many different ways that anesthesiology residents prepare for end-of-training examinations in addition to life as an attending or consultant. Examples include studying texts, reading journals, going over old questions, and undergoing mock oral examinations from local faculty. Others choose to attend "crash courses" of oral board preparation where they are advised to skirt tricky issues, hedge their bets, and "game the system." Wait a minute. What the…? "Game the system??!!!" Is that really the kind of anesthesiologist that you want to be? Someone that does not really know all the answers but can spread it on pretty thick? How is that going to save your patient's life at 3 a.m. when you are the only person managing their critical illness? It is especially important that at 3 a.m. you are able to fall back on a set of principles that guide you through information-gathering, synthesis, prioritizing, decision making, and reevaluation. In this collection of flash cards, we present a method of learning material and preparation that is tried and true, and will help you to both ace the oral examination and, more importantly, continue to think critically throughout your lifelong practice.

But wait, before we get ahead of ourselves, let us discuss what it means to think critically like an anesthesiologist. As with any professional, a competent anesthesiologist is defined by elements from many domains:

- Knowledge (and let us face it, we have to know a lot…)
- Academic qualifications
- Technical skills
- Ethics
- Ability to practice independently

Knowledge of the specialty is an essential component and the training to gain adequate knowledge takes many years. Numerous comprehensive textbooks and journals are dedicated to help fill this need. However, an anesthesiologist requires more than knowledge to be successful!

Critical thinking is the *application* of knowledge and requires you to show judgment, adaptability, and the ability to prioritize. An anesthesiologist must be able to create a sound anesthetic plan based on the information they acquire before and during a procedure. Given the constantly changing operating room environment, you must not only be able to anticipate problems but also rapidly diagnose and treat changes in your patient's status.

Why is critical thinking so important? There are a couple of reasons:

1. *Oral examinations*. As one of the last steps of anesthesiology education, trainees are required to pass oral examinations to prove this ability. Oral examinations do *not* test knowledge in and of themselves (although you will not pass if you do not know anything). That is what written examinations are for. The oral examination, in contrast, is designed to test your ability to *quickly* synthesize a plan from a great deal of information, deal with *conflicting priorities*, *communicate* it, be *adaptable*, and *manage* unexpected complications in an *organized* fashion. Try as you might, it is difficult to learn these skills from books. It takes practice of a different sort to master these abilities, and that is what these flash cards will help you with.
2. *The real world*. More important than examinations, thinking critically helps you earn confidence and respect of colleagues, surgeons, and patients. As a leader in perioperative medicine you must be able to convincingly advise and defend your opinions to surgical colleagues by synthesizing data from the preoperative evaluation and anticipated procedure, recognizing potential problems and conflicts in management goals, and communicating your plan logically and quickly. Likewise, we typically have a very short time to gain the trust of our patients before they entrust their lives with us. Credibly communicating an organized plan can go a long way to allaying patients' fears.

So now that we have discussed what critical thinking means—how do we learn it?

The key to thinking critically—especially under pressure—is in organizing your thoughts. Every day we are presented with a ton of information and are required to synthesize an anesthetic plan on the spot. A logical and consistent approach that you can reliably fall back on again and again is essential to avoid missing elements of the preoperative evaluation, and aid in the planning and execution of the anesthetic. This is where the flash cards come in. Let us go through one now.

HOW TO USE THE CARDS

The box of cards in front of you is organized into **three sections**. Section 1 is a collection of common **DISEASE STATES** and important clinical conditions that you *will* encounter in clinical practice (or on the oral examination!). Also included are some uncommon, but important disease states that you must know about as an anesthesiologist. Section 2 contains cards for a number of **PROCEDURES** that have special anesthetic considerations. In other words, giving anesthesia for an elective inguinal hernia does not have many special considerations, so it is not included; you will, on the other hand, find carotid endarterectomy here. Section 3, **EVENTS**, is a series of crises or things that can go wrong during your case. All the cards in each section are alphabetized, so that they are easy to find. Let us go through some examples in more detail to illustrate how the cards are constructed.

From section 1, **DISEASE STATES** (the blue cards), pull out the card marked…oh, let us say… "rheumatoid arthritis." The first thing you will notice is that it is divided into four panels; so is every other card in the "disease states" section and the "procedures" section. The "events" section is a little different, and we will get to that later. The card is a framework for how you organize your information and thinking, and follow the timeline of the anesthetic decision-making process, going left to right and top to bottom (Fig.1).

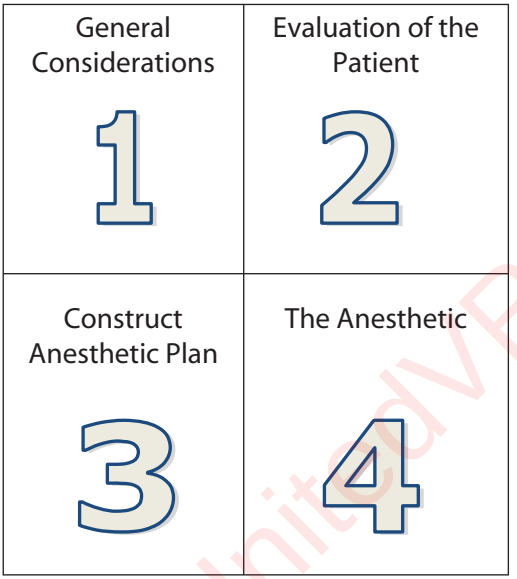

Figure 1. The flash card and its four quadrants. Numbers depict the order in which to read.

For example, the top left panel is a list of **general considerations** for each medical condition or surgical procedure that you will commonly encounter. For rheumatoid arthritis, note that we have listed six considerations—the number of considerations will vary but as a rule will be between 3 and 6. These are the main points that you will have in the back of your mind for each specific medical condition. Over time, you will become so familiar with this list that it just rolls off your tongue when someone mentions "rheumatoid arthritis." For each consideration, there may be several more specific items. For example, one major consideration is respiratory disease, examples of which are listed as bullet points: pleural effusions, rheumatoid nodules, pulmonary fibrosis, and costochondral involvement.

The second panel represents **your evaluation of the patient**, in the order that you would do it in real life: history, physical examination, investigations, and preoperative consults. Remember, you are a perioperative physician who is, in effect, being consulted by the surgeon and the primary care doctor to address any relevant anesthetic concerns, and formulate an integrated and comprehensive plan. Note that the items listed are *specific* to rheumatoid arthritis. Yes, you will order an ECG on many patients, but *in this case*, you are checking rhythm (conducting system nodules!) and ischemia (arteritis!). Surgeons and oral board examiners do not have all day for you to conduct every single physical examination maneuver and laboratory test; you have to be choosy and justify your choices.

The third panel is an important panel. It is here that you will **identify any potentially conflicting priorities**, **state your goals for the anesthetic, and then outline your plan to optimize the patient**

as best as you can prior to proceeding. At the end of the panel the anesthetic options are outlined.

The final panel is **the anesthetic** itself (notice that 75% of the thinking is done before you even hit the operating room). Here you will outline what is needed preoperatively (eg, antibiotics, steroids, blood in the OR, booking of an ICU bed, etc), the room setup (specifically for this patient), and then the induction, maintenance, and emergence/disposition. Again, note that the card is specific. ICU bed is not checked off, but preoperative steroid coverage is a possible consideration, and you may want a five-lead ECG, difficult airway cart, and an extra pair of hands for the awake fiberoptic intubation.

Next let us take a card from section 2, the **PROCEDURES** section (and color-coded green). Take "pulmonary resection" for example. You see immediately that the same structure exists, and for good reason. This procedure has its own major considerations, and requires certain elements of the history and physical that are specific to it. Perhaps more importantly, in panels 3 and 4, the goals, options and *how* the anesthetic can be performed are highlighted.

Finally, take a card from the third section, the **EVENTS** cards (and color-coded red). Let us pick… ummmmm…ok: "hypoxemia." The event cards are different than the disease states or the procedure cards. First off, there is no four-box structure. They are simply quick, bulleted lists consisting of a **definition, a differential diagnosis, and a step-by-step management plan**. These are designed to give you a structured framework for developing (1) a sensible but complete range of possibilities for the problem you are facing and (2) a logical way to quickly solve the crisis. There is no way to get around this—on the oral examination *and* in real life you *will*, we repeat *will*, get a child who goes profoundly hypoxemic and bradycardic on induction, a woman who suffers a massive postpartum hemorrhage, and a patient who unexpectedly has cardiovascular collapse due to anaphylaxis. Fine, you may not ever see malignant hyperthermia in real life (knock wood), but you sure as heck better be prepared for it just in case.

THE SYSTEM

Maybe you could research the information in these cards by yourself (taking *thousands* of back-breaking hours looking through textbooks and journal articles…) but you still need a system to put it all together! The trick to thinking critically—the trick to being able to synthesize information quickly, and communicate it effectively in the heat of the moment—**lies in organization**. And these cards provide you with a system that is tried and true. Here is how it works.

Select one card from each of the first two sections. Let us assume the cards selected are the ones outlined above. You will have a stem that looks like "A patient with a history of rheumatoid arthritis presents for a pulmonary resection" Hmmm…that could be an interesting case! Wait! **Do not read the details of the cards yet, just the titles.**

Now comes the fun…divide a blank sheet of paper into four quadrants. Collect your thoughts and list **your** considerations for this case in the upper left quadrant (Fig. 2). Once you are done,

compare with the considerations on quadrant 1 for both cards. You should have listed both rheumatoid arthritis *and* pulmonary resection in the first panel with the appropriate bullet points under each one. The first time you do this, you may miss many of the pertinent considerations. That is okay…as you review the card at the end of the case and continue to practice, you will go over each card many times, and the details will become second nature.

Considerations	History Physical Exam Lab tests/Imaging Consults
Conflicts Optimize/Goals Options	Preop Room Setup Induction Maintenance Emergence Disposition/Pain

Figure 2. Sheet of paper divided into quadrants and with corresponding subheads.

Continue to the next quadrant and list what you want to know on a focused history, physical examination, relevant labs and/or imaging, and consults (Fig. 2). Compare your thoughts to the information supplied on the cards. Miss anything?

Proceed to the third quadrant on your page and list any/all conflicts. These can become clear when you review quadrants 1 and 2. For example, the patient may require lung isolation with a double lumen ETT and lateral positioning. But the severity of RA may mean the patient is difficult to intubate with cervical spine instability and at risk of nerve/joint injury with positioning! What takes precedence in these situations? Ultimately, there may not be a right or wrong answer, and many conflicts can be defended either way, as long as you have considered the risks/benefits of each and provided rational reasons for your choice. The bottom line with conflicts is that their management will be *your* decision; so you want to make sure you have considered every angle (refer to quadrants 1 and 2!). In quadrant 3 you will also list your goals for the procedure and settle on an anesthetic plan (and a back-up plan!). Remember that options usually include general, regional, local, and canceling/postponing the case.

Finish by completing the fourth quadrant of your page: detail your plans for preoperative preparation, induction, maintenance, extubation, and disposition. Compare your reasoning with the cards. The more you practice the easier it will become!

At the end of the hypothetical case, take the card from the **EVENTS** section—hypoxemia! How will you deal with hypoxemia in the setting of a patient with RA undergoing a pulmonary resection? Again, review your thoughts with the information on the card.

How is this better than memorizing facts about a disease process from a review book? The key lies in restructuring the information that you already know in an organized **framework**. The oral examination is not about spitting out as many facts as you can about a disease or a surgical procedure. That is what the written examination covers. Applying this **framework** to everyday anesthetic scenarios will allow you to hone your skills at prioritizing, exercising judgment, and communication. Once you get good at this, cases like the one above, or any random combination of these cards, begin to roll off your tongue like a senior oral examiner.

Here is another way to use the cards, that is fun and extremely effective; imagine you are studying with your friend, and you have got your new box of cards. You know the general framework of the four-panel system. Your friend selects one card from each of the three sections, and with a little embellishment regarding the inconsequential details, begins to "play" oral board examiner by creating a scenario. Let us assume the cards selected are the ones outlined above. He or she may start by saying, "You have a 58-year-old woman with a history of rheumatoid arthritis who presents for a right upper lobectomy via thoracotomy." Notice that nothing else was stated—essentially just the titles of the first two cards and some made-up stuff regarding age and sex.

Then you do the most important thing in the whole process. You state to the examiner, "I am going to take a minute here to collect my thoughts." More mistakes have been made in the examination by rushing in headfirst without thinking first, so it is critical that you take some time *now* and organize, rather than waste precious time later floundering around and backtracking on your answers. The next thing you will do is to quickly list your considerations for this case in the upper left quadrant. Continue and fill in all four quadrants as best as you can with the information presented. Now your friend can prompt you and you carry on with the examination. At the end, you debrief—did you forget any major considerations? Forget to get flexion-extension views of the C-spine? Neglect to check the position of the double-lumen tube as a source of hypoxemia? Ah, there is always next time. In this way, you can practice speaking aloud and put yourself to the test. Are you organized? Can you think like an anesthesiologist? When you are done, switch around and put your friend in the hot seat. Good luck!

HINTS AND KEY PHRASES

We have referred to communication in the previous sections for good reason. You are sadly disadvantaged as an anesthesiologist (or oral examinee) because you have precious few minutes to convince the surgeon, patient, family, or examiners that you have a sound, well thought-out

plan and that you are a safe doctor. Hence the prime importance placed on communication. Nothing deflates a surgeon or examiner's confidence in you faster than someone who bumbles along with no clear message.

Of course, there are foundations of good communication that must be followed—obviously you always want to speak calmly but with conviction, maintain good eye contact, refrain from using slang, and avoid pitfalls like acting defensive. However, particularly for the oral board examinations, there are some key techniques that can help you avoid getting bogged down in areas where you will not score any points, and move along quickly to the high-yield parts of the examination. Remember, the examiners are waiting there with a checklist…if you do not cover the points they need to hear you say, you do not get the points.

1. *Recognition of the urgency of the case*. We know, this sounds silly to say. However, many examinees fail to do this, and it is a critical step in letting the examiner know that *you* know what is at stake here. Examples of ways to begin your dialogue with the examiners include: "This is an emergency situation and I would immediately proceed with…," or "This is an elective case and as such I have time to…." The only other category is semiurgent/semielective. The urgency will dictate how much workup you can do prior to the scheduled case. Keep in mind the urgency can change throughout the case; for example, the elective carotid you dropped off in the PACU is now stridorous. You should respond by acknowledging the new degree of urgency: "This is now an emergency and I would take action by doing the following…."
2. *Make your evaluation quick and focused*. The patient will be presented to you with an assortment of medical and surgical problems. As you outline your preoperative evaluation, one of the worst things you can do is to delve into every possible review of system's point that could come up. Show the examiners that you know what is important and what not to focus on. For example, let us take diabetes as an example. A good blurb to say is this:
   - I would proceed with my *usual* history including past medical history, past anesthetic history, meds, allergies, and last meal. Then I would do a *focused* history relating to diabetes including any history of coronary disease, cerebrovascular disease, renal disease, ophthalmologic complications, and peripheral and autonomic neuropathy such as gastroparesis and orthostatic symptoms.
   - I would then do my *usual* anesthetic physical including an airway and cardiorespiratory examination, and a *focused* physical looking for orthostatic changes, small joint disease in the airway, and potential hypovolemia due to polyuria.

   Wow! You have just spent 20 seconds impressing the heck out of the examiners. Usual history and physical—bang! Get it out of the way. Then focus on the relevant considerations.

3. *Do not complicate things*. This is a related point. At some point you may be asked, "what other information would you like to obtain in the preoperative evaluation?" This is a chance to quickly rhyme off anything else (eg, investigations, consults) from quadrant 2 (and look really slick doing it!). Then let them give you feedback. *Do not* continue to ask questions such as, "what is his ejection fraction?" or "is her FEV_1 normal?" etc. This is a low-yield fishing expedition and besides making you look disorganized, just annoys examiners. Annoyed examiner = not good. Gently put the ball back in their court by saying, "assuming the remainder of the

history and physical was unremarkable, I would proceed with…blah, blah, blah." That way, if there *is* something that they want you to elicit, they will be forced to tell you rather than you playing a guessing game.

4. *Prioritize.* The problems the examiners will throw at you may have many possible etiologies. We are not internists pondering the vagaries of serum rhubarb (no offense to internists… or rhubarb)—our patients' conditions change quickly and things go bad fast. That is why you have to prioritize. Hypoxemia during laparoscopy? Yes, it could be thyrotoxicosis causing a decreased venous admixture—but it is more likely to be a pneumothorax. Think of the clinical situation, and put the high-yield items at the top of your list. Hypoxemia during steep head-down position? I would put atelectasis or endobronchial intubation ahead of pulmonary edema or low cardiac output. This is a key aspect of critical thinking, and examiners are obliged to test you on it…simply repeating lists by rote is unacceptable in an oral examination, just as managing complications without any prioritization in real life is dangerous practice.
5. *Diagnose and manage.* Again, unlike some of our friends in other fields of medicine, we just do not have time to pontificate and order a bunch of tests. Let us say a patient has life-threatening hypotension. Now, picture what you would do in real life. Would you sit down and make a list of possibilities and then rule them out one by one, eventually deciding on one and initiating treatment? Of course not. At the same time, you would not just throw the kitchen sink at the patient and hope something worked. In real life you diagnose and resuscitate simultaneously. Again, this sounds simplistic, but you need to say it out loud. Practice; "I would diagnose and resuscitate simultaneously." There, didn't that feel good? For your hypotensive patient, a good blurb to say might be something like this:
   - This is an emergency and I will begin by telling you my differential, but please recognize that I would begin to immediately diagnose and resuscitate simultaneously (at this point the examiners look at each other with a "*this one is good*" look). In this particular case, (blank) is high on my list because of (blank), but I would also consider (blank), (blank), and (blank). I would begin by repeating the noninvasive blood pressure measurement. I would then alert the surgeon and check for surgical causes. At the same time, I would open up both IVs wide, and administer a bolus of 100 µg of phenylephrine. Next I would check the ECG for signs of ischemia…blah, blah, blah (the content will of course depend on what the clinical scenario is—see the hypotension card for complete differential and management).

6. *Be a quarterback*. Emergencies do not happen in a vacuum. You have helpers around, especially when the oral board scenarios take place in the PACU, the ER, or the ICU. These may start with you receiving a phone call alerting you to a change in patient status. Rather than running off half-cocked, show that you are in command of the situation. For example, imagine you are called by a nurse about a patient in the PACU who had a total knee replacement under epidural. A resident bolused the epidural a few minutes ago with bupivacaine and now the patient is bradycardic, hypotensive, and restless. A bad response might be, "um, how much did the resident give? What is the respiratory rate? Uh, I guess I would put some oxygen on the patient and…oh, I would give some ephedrine too." Try this instead:

- "This is a very urgent situation. The differential for this includes several things, but I am most concerned about acute systemic toxicity from the local anesthetic. I would come immediately ***but*** I would tell the nurse over the phone to:
    a. Administer oxygen by facemask.
    b. Open the IV wide open.
    c. Call for the code-cart with a defibrillator.
    d. Have another nurse get midazolam and lipid emulsion.
- When I get to the PACU, I will need to assess and resuscitate simultaneously. Another nurse can give 100% O2 by facemask. I will recheck the vital signs including ECG morphology. Assuming the patient is protecting his airway, I would…blah, blah, blah.

See how you delegated there? Especially over the phone—that way, when you showed up, the resuscitation was already in motion.

7. *Do not be a "sally show-off"*. This is a tough issue. You may be up to date on the latest advancements in a certain aspect of anesthetic care. Or you come from a place that does that new 5-D ultrasound-guided combined lumbar plexus and sciatic block for knee arthroscopy. That is great. Just keep things in perspective. Examiners are real people too, and many may not be as up to date on *that* particular thing. Insisting on using a new technique that is not widespread clinical practice shows lack of flexibility, even if you believe it is the best course of action. Instead, say something like, "There is some recent literature suggesting that using 'x' can lead to improved outcomes, and where I trained we had been doing that, but if 'x' was not available, I would proceed with technique 'y'". Keep in mind that the examiner might actually be an expert themselves in the same thing, and invite you to defend your knowledge-base!

INVESTIGATIONS

Now, a special word on "investigations," otherwise known as "What labs and other tests would you like to order, doctor?" The various investigations you order for a given case (labs, imaging, cardiac evaluation, consults, etc) should all be based on *risk assessment*. Assess the patient's risk for the procedure at hand; identify modifiable risk factors; and hopefully shift the balance of risk/reward to favor your patient! A thorough preoperative assessment should reduce delays and complications. The most important aspects of any assessment are the **history and physical examination**. Further tests should be ordered based on your findings and **only if the results will influence your anesthetic plan**. Random testing can actually lead to harm (either from needless delays, complications of the test, or complications from follow-up tests!)

For a healthy ASA I patient—no tests are necessary.

Exceptions:

- Use of contrast dye or >55 years of age—order serum creatinine (renal disease is silent and risk increases with age)
- If you think the patient is pregnant—order pregnancy test
- If the planned surgery is "high risk," that is, the expected blood loss is substantial—order a complete blood count to assess hemoglobin/hematocrit

What about the electrocardiogram (ECG)? Shouldn't we get a "baseline"? Many institutions have age cut-offs for ordering ECG's. Bottom-line: an ECG is not sensitive or specific enough to assess for CAD (use symptoms and risk factors—didn't you do a history and physical examination?) There are still many circumstances when ordering an ECG is appropriate:

- Alcohol abuse
- History of cardiovascular, pulmonary, or renal disease
- Diabetes, malnutrition, malabsorption
- Morbid obesity or OSA
- Poor exercise tolerance
- Rheumatoid arthritis, systemic lupus erythematosus
- Cigarette smoking
- Radiation therapy

What about assessing cardiac risk for patients undergoing noncardiac surgery? This is a very important area to understand! The most current guidelines were published by the ACC/AHA in 2007.

A noninvasive cardiac test is supposed to help evaluate and stratify risk for coronary artery disease. Noninvasive cardiac testing (vs invasive testing or cardiac catheterization) has a couple of components:

- "Stress"—exercise or pharmacologic (dipyridamole, adenosine, or dobutamine)
- Cardiac response—interpretation of ECG and/or myocardial perfusion and/or echocardiography

Unless it is an emergency, before proceeding with any surgery further evaluation and treatment is recommended for patients with "active conditions":

- Unstable coronary syndromes (CCS III or IV, or MI <30 days)
- Decompensated heart failure (NYHA IV)
- Significant arrhythmias
- Severe valvular disease (*as* with valve area <1.0 cm^2, or symptomatic MS)

If a patient does not have an active condition you need to ask the following questions:

(1) Is it an emergency? Yes—proceed to OR

(2) Is it low-risk surgery (endoscopy, superficial, cataract)? Yes—proceed to OR

(3) What is the patient's exercise tolerance? Is it greater than 4 METs (climb a flight of stairs?)—proceed to OR

(4) Poor or unknown exercise tolerance? Assess the number of risk factors for CAD. (There are five factors you need to remember! History of CAD, history of CHF, history of stroke, insulin therapy for DM, and preoperative serum creatinine >2.0 mg/dL.)

- Zero risk factors—proceed to OR
- One or two risk factors—proceed to OR **(consider noninvasive testing if it will change management)**

- Three or more risk factors and intermediate surgery (carotid endarterectomy, head/neck surgery, orthopedic surgery, abdominal/thoracic surgery)? Proceed to OR **(consider noninvasive testing if it will change management)**
- Three or more risk factors and vascular surgery? **Consider noninvasive testing if it will change management**

When should you order a hemoglobin/hematocrit? For surgery associated with significant blood loss or for patients with an iron deficient diet (malnutrition, alcohol abuse, etc), poor or abnormal hemoglobin production (thalassemia, sickle cell, etc), or ongoing blood loss (malignancy, bleeding disorder, hepatic disease, etc).

Order coagulation studies only for patients with coagulation disorders (including alcohol abuse, hepatic disease, malnutrition) or if they are taking anticoagulants.

Order a serum creatinine for patients with risk factors for renal disease (diabetes, hypertension, age >55 years, vascular disease, SLE, etc); the use of diuretics or planned use of contrast dyes.

Order serum electrolytes for patients with risk factors for renal disease, malnutrition, gastric bypass procedures, or patients who take digoxin or diuretics.

Order a serum glucose for patients with a history (or family history) of diabetes, or a history of obesity, stroke, use of steroids, or poor exercise tolerance.

Order liver function tests for patients with risk factors for alcohol abuse, bleeding disorders, hepatic disease, or hepatitis exposure.

Order ABGs for patients who have hypoxia (SpO_2 <90%), severe lung disease, decompensated CHF, or severe musculoskeletal disorders that affect ventilation. ABGs are also used to predict pulmonary function after lung resection.

Order a CXR for patients with *significant* cardiac or pulmonary symptoms/signs of undetermined cause. CXRs do not predict postop pulmonary complications.

Pulmonary function tests (PFTs) do not typically add value to risk assessment. Traditionally they are ordered for lung resection surgery to predict mortality after pneumonectomy.

Overall a number of studies have demonstrated that "routine" testing does not change perioperative patient outcome. **So remember—unless the test you order is going to make a difference with patient management—do not order it!**

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Abbreviation Key

ABG	arterial blood gas
ACE	angiotensin-converting enzyme
ACEI	angiotensin converting enzyme inhibitor
ACLS	advanced cardiac life support
ACT	activated clotting time
ACTH	adrenocorticotropic hormone
AFIB	atrial fibrillation
A flutter	atrial flutter
AFOI	awake fiberoptic intubation
AI/AR	aortic insufficiency/aortic regurgitation
ALA	6-Aminolevulinic acid
APL	adjustable pressure-limiting (valve)
aPTT	activated partial thromboplastin time
AR	aortic regurgitation
ARB	angiotensin receptor blocker
ARDS	acute respiratory distress syndrome
ARF	acute renal failure
AS	aortic stenosis
ASA	American Society of Anesthesiology
ASD	atrial septal defect
ATLS	advanced trauma life support
AV	aortic valve
AVN	avascular necrosis
AVR	aortic valve replacement
BiPAP	bilevel positive airway pressure
BMI	body mass index
BMP	basic metabolic profile
BP	blood pressure
BVM	bag-valve-mask
Ca	cancer or calcium (context dependent)
CABG	coronary artery bypass graft
CAD	coronary artery disease
CBC	complete blood count
CBF	cerebral blood flow
CF	cystic fibrosis
CHF	congestive heart failure
CO	cardiac output
Coags	coagulation panel (INR, PT, PTT)
CPAP	continuous positive airway pressure
CPB	cardiopulmonary bypass
Cr	creatinine
CRP	C-reactive protein
CSA/OSA	central sleep apnea/obstructive sleep apnea
CSE	combined spinal-epidural
CSF	cerebrospinal fluid
CT	computed tomography
CVA	cerebrovacular accident
CVP	central venous pressure
CV(S)	cardiovascular (system)
CXR	chest x-ray
DDAVP	1-desamino-8-D-arginine vasopressin
DI	diabetes insipidus
DIC	disseminated intravascular coagulation
DLT	double lumen tube
DM	diabetes mellitus
DVT	deep vein thrombosis
Dz	disease
ECG/EKG	electrocardiogram
ECMO	extracorporeal membrane oxygenation
EDC	estimated date of confinement
EEG	electroencephalogram
EF	ejection fraction
Epi	epinephrine
EPO	erythropoietin
ERCP	endoscopic retrograde cholangiopancreatography
Esp	especially
ETOH	ethanol
ETT	endotracheal tube
FB	foreign body
FEV1	forced expiratory volume (1st second)
FFP	fresh frozen plasma
FH	family history
FOB	fiberoptic bronchoscopy
FOI	fiberoptic intubation
FRC	functional residual capacity
FVC	forced vital capacity
GA	general anesthetic/anesthesia
GBS	Guillain Barre syndrome

GCS	Glasgow coma score
GERD	gastroesophageal reflux disease
GETA	general endotracheal tube anesthesia
GFR	glomerular filtration rate
GH	growth hormone
GI	gastrointestinal
HD	heart disease
Hem	hematologic
H/O	history of
HOCM	hypertrophic obstructive cardiomyopathy
HR	heart rate
HTN	hypertension
Hx	history
IABP	intra-aortic balloon pump
ICH	intracranial hemorrhage
ICP	intracranial pressure
ICS	intercostal space
ICU	intensive care unit
IE	infective endocarditis
ILMA	intubating laryngeal mask airway
Incl	including
INR	international normalized ratio
Intub	intubation
IOP	intraocular pressure
IVIG	intravenous immunoglobulin
LA	local anesthetic or left atrium (context dependent)
LAH	left atrial hypertrophy
LBBB	left bundle branch block
LFTs	liver function tests
LGL	Lown-Ganong-Levine syndrome
LLSB	left lower sternal border
LMA	laryngeal mask airway
LOC	level of consciousness
LR	lactated ringers
LV	left ventricle
LVAD	left ventricular assist device
LVH	left ventricular hypertrophy
JVD	jugular venous distension
JVP	jugular venous pressure
MAC	monitored anesthetic care
MAP	mean arterial pressure
Meds	medications
MEP	motor evoked potentials
METS	metabolic equivalent
MI	myocardial infarction
MR	mitral regurgitation
MRI	magnetic resonance imaging
MvO_2	mixed venous oxygen saturation
MVP	mitral valve prolapse
Mx	management
NDMR	non-depolarizing muscle relaxants
NE	Norepinephrine
NG	nasogastric
NHL	non-Hodgkin lymphoma
NIBP	non-invasive blood pressure
NICU	neurologic intensive care unit or neonatal intensive care unit (context dependent)
NIRS	near infrared spectroscopy
NK	natural killer (cells)
NMB	neuromuscular blocker
NMJ	neuromuscular junction
NO	nitric oxide
NPPV	non-invasive positive pressure ventilation
NSAIDs	nonsteroidal anti-inflammatory drugs
NTG	nitroglycerin
OSA	obstructive sleep apnea
PA	pulmonary artery
PAC	pulmonary artery catheter
PACU	post-anesthesia care unit
PAP	pulmonary artery pressure
PASP	pulmonary artery systolic pressure
PAWP	pulmonary artery wedge pressure
PDA	patent ductus arteriosus
PE	physical exam or pulmonary embolism (context dependent)
Peds	pediatric
PEEP	positive end-expiratory pressure
PET	positron emission tomography
PFO	patent foramen ovale
PFR	peak flow rate
PFTs	pulmonary functions tests
PHTN	pulmonary hypertension
Physio	physiotherapy
PIP	peak inspiratory pressure

PNB	peripheral nerve block
PND	paroxysmal nocturnal dyspnea
PONV	post-operative nausea and vomiting
PPV	positive pressure ventilation
PT	prothrombin time
PVD	peripheral vascular disease
PVR	pulmonary vascular resistance
RA	right atrium or regional anesthesia/ anesthetic or rheumatoid arthritis (context dependent)
RAD	right axis deviation
RBBB	right bundle branch block
RBF	renal blood flow
RDS	respiratory distress syndrome
Re:	in regards to
ROM	range of motion
ROP	retinopathy of prematurity
RSI	rapid sequence induction
RV	residual volume or right ventricle (context dependent)
RVH	right ventricular hypertrophy
RVOT	right ventricular outflow tract
Rx	treatment
SAH	subarachnoid hemorrhage
SBE	subacute bacterial endocarditis
SBP	systolic blood pressure
SC	subcutaneous
SEM	systolic ejection murmur
SIADH	Syndrome of inappropriate antidiuretic hormone
SLE	systemic lupus erythematosus
SNP	sodium nitroprusside
SpO_2	pulse oximetry
SSEP	somatosensory evoked potentials
Std	standard
Sux	succinylcholine
SVC	superior vena cava
SVR	systemic vascular resistance
SVT	supraventricular tachycardia
TB	tuberculosis
TBI	traumatic brain injury
TCD	transcranial Doppler
TEE	transesophageal echocardiography
TEF	tracheoesophageal fistula
TEG	thromboelastography
THA	total hip arthroplasty
TIA	transient ischemic attack
TIVA	total intravenous anesthesia
TKA	total knee arthroplasty
TMJ	temporomandibular joint
TPN	total parenteral nutrition
TSH	thyroid stimulating hormone
URI	upper respiratory infection
UTI	urinary tract infection
VATS	video-assisted thoracoscopic surgery
VC	vital capacity
Vent	ventilation
VF	ventricular fibrillation
VSD	ventricular septal defect
VT	tidal volume or ventricular tachycardia
vWD	von Willebrand disease
vWF	von Willebrand factor
WBC	white blood cells
WPW	Wolff-Parkinson-White syndrome

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Anesthesiology Oral Board Flash Cards

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Anesthesiology Oral Board Flash Cards

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in connection with new or infrequently used drugs

Contents

Procedures

Events

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Disease States

1 Achondroplasia
2 Acromegaly
3 Acute Porphyrias
4 Adrenal Insufficiency
5 Adrenocortical Excess (Cushing Syndrome)
6 Alcoholism
7 Amyotrophic Lateral Sclerosis (ALS)
8 Ankylosing Spondylitis
9 Anterior Mediastinal Mass
10 Aortic Dissection
11 Aortic Regurgitation
12 Aortic Stenosis
13 ASD/VSD
14 Asthma
15 Burns
16 Cancer
17 Carcinoid
18 Cardiac Contusion
19 Cardiac Tamponade
20 Cerebral Aneurysm
21 Chronic Obstructive Pulmonary Disease
22 Chronic Renal Failure
23 Congestive Heart Failure
24 Coronary Artery Disease
25 Cystic Fibrosis
26 Diabetes Mellitus
27 Down Syndrome (Trisomy 21)
28 Duchenne Muscular Dystrophy
29 Eisenmenger Syndrome
30 Epiglottitis and Croup
31 Epilepsy
32 Foreign Body Airway Obstruction
33 Geriatric Patient
34 Guillain Barré Syndrome
35 Hemophilia
36 HIV
37 Hypertension
38 Hyperthyroidism
39 Hypothyroidism
40 Liver Cirrhosis
41 Marfan Syndrome
42 Mitral Regurgitation
43 Mitral Stenosis
44 Mitral Valve Prolapse
45 Multiple Sclerosis
46 Myasthenia Gravis
47 Myasthenic (Eaton-Lambert) Syndrome
48 Myotonic Dystrophy
49 Neurofibromatosis Type 1
50 Noncardiac Surgery for Heart Transplant Patient
51 Obesity
52 Obstructive Sleep Apnea
53 Ocular Injury
54 Omphalocele and Gastroschisis
55 Patient with Pacemaker or AICD
56 Pediatric Patient
57 Pheochromocytoma
58 Placental Abruption
59 Placenta Previa
60 Preeclampsia
61 Pregnancy
62 Premature (Low Birth Weight) Infant
63 Progressive Systemic Sclerosis (Scleroderma)
64 Pulmonary Hypertension
65 Pulmonic Stenosis
66 Pyloric Stenosis
67 Rheumatoid Arthritis
68 Sarcoidosis
69 Scoliosis
70 Sickle Cell Disease
71 Spinal Cord Injury
72 Systemic Inflammatory Response Syndrome
73 Systemic Lupus Erythematosus
74 Tetralogy of Fallot
75 Tracheoesophageal Fistula
76 Traumatic Brain Injury
77 Von Willebrand Disease
78 Wolff-Parkinson-White & Lown-Ganong-Levine Syndromes

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Achondroplasia

Considerations

1. Most common form of dwarfism (more than 100 other types). Autosomal dominant inheritance

   Appearance: large head-to-body size difference, prominent forehead, shortened arms and legs, decreased muscle tone
2. Airway management difficulties
   Craniofacial and spinal abnormalities:
   - limited neck extension
   - foramen magnum stenosis
   - large tongue
   - large mandible
   - atlanto-axial instability
3. Kyphosis, scoliosis, and spinal stenosis : difficult and unpredictable spread of local anesthetics in epidural and subarachnoid spaces
4. Comorbidities: central and obstructive sleep apnea, otitis media (childhood), obesity

History

- Pain/ataxia/incontinence/apnea—due to cervicomedullary/spinal cord compression

Physical Exam

- Neurologic—hypotonia in infancy
- Craniofacial features—large head, midface hypoplasia, dental crowding
- Short stature (normal trunk length)
- Bow legs (genu varum)

Lab Tests/Imaging

Polysomnography (assess CSA/OSA)
Head/neck CT/MRI (assess craniocervical junction)

Consults

- Neurology as indicated from history and exam

Conflict(s)

- Endotracheal intubation and cervical instability
- CSA/OSA and use of sedatives/analgesics
- Regional anesthesia and spinal/neurologic abnormalities

Optimize/Goals

- Minimize movement of cervical spine during ETT placement (consider referral to neurology if new onset/worsening symptoms)
- Consider adjuvants to opioids for pain management regarding OSA
- Consider imaging techniques before regional anesthesia to assess anatomy of vertebrae and spinal cord; also consider epidural versus spinal (titratable)

Options

- General anesthesia, regional anesthesia, or sedation

Preop: Premed
Blood: as indicated by surgical procedure
ICU/stepdown bed: consider severity of OSA

Room Setup (special drugs/monitors)

- Difficult airway cart available
- Body size appropriate airways/laryngoscopes
- Consider use of alternative analgesics than opioids (dexmedetomidine, low-dose ketamine, regional)

Induction

- If GETA—consider AFOI as determined from airway/C-spine assessment
- If regional—use smaller doses of LA; beware high block

Maintenance

- Maintain neck in neutral position
- Positioning—consider patient's body habitus

Emergence

- If difficult intubation—consider leaving ETT in place or extubating fully awake, use of tube exchanger

Disposition/Pain

- Recovery/stepdown/ICU as required

Clinical Pearls

For the achondroplastic obstetric patient—consider imaging early in pregnancy to assess lumbar anatomy during labor—early placement of epidural catheter to allow for slow titration.

Reference

Shirley ED, Ain MC. Achondroplasia: manifestations and treatment. *J Am Acad Orthop Surg*. April 2009;17(4):231-241.

Acromegaly

Considerations

1. GH hypersecretion.
   Usually from GH secreting pituitary adenoma—may be complicated by headaches, visual field defects, elevated ICP, hypopituitarism (requires replacement with hydrocortisone/thyroxine)
2. Potentially difficult airway/difficult ventilation: gigantism, facial changes, large tongue, hypertrophy of pharyngeal mucosa, small glottic opening, prominent jaw (prognathism), obstructive sleep apnea (OSA)
3. Comorbidities: OSA, hypertension, cardiac arrhythmias, diastolic dysfunction (heart failure), coronary artery disease, glucose intolerance, renal failure, arthritis, kyphoscoliosis
4. Treatments: pituitary surgery (transsphenoidal), radiotherapy, medical (octreotide—suppresses GH secretion)

History

- Comorbidities: duration, severity, and functional capacity/limitations
- Respiratory—apnea, snoring, somnolence, PH OSA, use of CPAP, PH of difficult intubation?
- Cardiac disease—HTN? Angina? Exercise capacity?
- Therapies: medical, radiotherapy, surgical?

Physical Exam

- Body habitus/BMI
- Airway—hypertrophy of facial bones, mandible, tongue
- Vital signs—hypertension?
- Heart failure—tachycardia, elevated JVP, S3/S4, hepatomegaly, peripheral/pulmonary edema

Lab Tests/Imaging

- CBC—anemia? Electrolytes— hyponatremia, ↓K, hyperglycemia?
- TSH—thyroid function?
- EKG/echocardiography—LV hypertrophy? Systolic/diastolic dysfunction?

Conflict(s)

- Potential difficult airway/difficult mask ventilation (requiring AFOI) and potential cardiac disease (avoid further "stress"—hypertension, tachycardia)
- OSA and use of opioids for analgesia

Optimize/Goals

- Hypopituitarism—ensure hydrocortisone/thyroxine therapy as required (consider preop. endocrine consult)
- If difficult airway—prepare for AFOI
- If OSA—minimize use of opioids, constant positive airway pressure (CPAP) postoperative
- If heart failure—minimize further depression of cardiac function, optimize fluid therapy

Options

- General anesthesia, regional anesthesia, peripheral nerve blocks, or sedation

Preop: Premed: ±steroids
Blood: surgery dependent
ICU/stepdown bed: possibly given comorbidities

Room Setup (special drugs/monitors)

- Depending on patient size—may require larger-sized equipment (OR table, NIBP cuff)
- Consider invasive BP monitor
- Difficult airway cart—fiberoptic video laryngoscope

Induction

- Consider AFOI

Maintenance

- Optimize based on comorbidities

Emergence

- Fully awake for extubation; if OSA, consider early use of CPAP

Disposition/Pain

- PACU/ICU/stepdown as required
- Minimize opioids, consider adjuvants/nerve blocks

Clinical Pearls

For transsphenoidal pituitary surgery: the acromegalic patient will require oral intubation (regular or RAE tube) and the procedure is generally done in the sitting position. Complications include cranial nerve injury (II-VI), bleeding, CSF leak, DI (treat with DDAVP), or SIADH (fluid restrict).

References

Melmed S. Acromegaly pathogenesis and treatment. *J Clin Invest*. 2009;119:3189-3202.

Nemergut EC, Dumont AS, Barry UT, et al. Perioperative management of patients undergoing transsphenoidal pituitary surgery. *Anesth Analg*. 2005;101:1170-1181.

Considerations

1. Group of inherited enzymatic defects of heme synthesis, characterized by overproduction of heme precursors and intermittent symptomatic attacks
2. Multisystem manifestations including neurological, renal, cardiovascular
3. Drugs triggering attacks (wide range, but including):
   - barbiturates
   - ergots
   - metoclopramide
   - steroids
4. Nondrug triggers: dehydration, fasting, stress, infection

History

- History of attacks: abdominal pain, vomiting, fever, hallucinations, mental status changes, seizures (acute intermittent porphyria), blistering skin lesions (variegate porphyria)
- Prior anesthetic and specific drugs used

Physical Exam

- Signs of autonomic dysfunction (HTN, ↑ HR)
- Assess intravascular volume status
- Neuro exam (weakness, bulbar involvement, respiratory failure)
- Abdominal exam often normal

Lab Tests/Imaging

- Electrolytes (↓Na, ↓K, ↓Ca)
- Urine porphyrin & porphyrinogen precursors
- Red/purple urine

Consults

Conflict(s)

Optimize

- Carbohydrate loading suppresses the synthesis of porphyrins. Start IV with D10W (or D5LR if hyponatremic)

Options

- Regional anesthesia probably safe but take care with preexisting neurologic deficits

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

Induction

Maintenance

- Expect labile BP; volatiles safe

Emergence

Disposition/Pain

- Observe 24 hours for attack

Clinical Pearls

- The enzyme ALA synthetase is inducible by a wide variety of drugs, or by events that lead to an increased requirement for heme. The deficiency of various enzymes (depending on the type of porphyria) further along the biochemical pathway causes accumulation of precursors.
- The acute porphyrias are:
  - acute intermittent porphyria (most severe, mortality up to 10% with attacks)
  - variegate porphyria
  - hereditary coproporphyria
- All acute porphyrias share neurologic symptoms as a common feature. These are numerous and include autonomic and peripheral neuropathy, bulbar involvement, hypothalamic dysfunction, mental status changes, seizures, and coma. Many of the neurologic deficits can be permanent.
- If crisis:
  - remove trigger/end surgery
  - IV hydration with dextrose
  - **hematin** 3 to 4 mg/kg IV (inhibits ALA synthetase)
  - treat nausea and vomiting with antiemetics
  - treat pain with opioids
  - beta blockers for hypertension/tachycardia
  - if seizures, midazolam, NOT phenytoin
  - monitor electrolytes and treat accordingly
- "Safe drugs" for anesthesia:
  - lidocaine, bupivacaine
  - alfentanil, fentanyl, morphine, codeine, meperidine, sufentanil, naloxone
  - ASA, ibuprofen, ketoprofen, acetaminophen
  - epinephrine, atropine, phenylephrine, beta blockers, beta agonists
  - propofol, desflurane, isoflurane, nitrous oxide, NDMRs, neostigmine, succinylcholine
  - ondansetron, granisetron, prochlorperazine, droperidol
  - midazolam
  - gabapentin, pregabalin
- "Unsafe drugs" for anesthesia *or* drugs with unproven safety:
  - barbituates, etomidate, ropivacaine, hydralazine, nifedipine, phenyoxybenzamine, pentazocine

Reference

James MFM, Hift RJ. Porphyrias. *Br J Anaesth.* 2000;85:143-153.

Adrenal Insufficiency

Considerations

1. Establish cause: primary (failure to produce cortisol/aldosterone) versus secondary (failure to produce adrenocorticotropic hormone [ACTH])
2. DDx: primary—autoimmune destruction of adrenal glands, autoimmune polyendocrine deficiency syndromes, other infections (TB, AIDS), surgical removal; secondary causes—lack of ACTH from abrupt discontinuation of chronic steroids, hypopituitarism (tumors, radiation, surgery, drugs—etomidate, ketoconazole)
3. Addisonian crisis: back/leg/abdominal pain, vomiting, diarrhea, dehydration, hypotension, and loss of consciousness
4. Surgery—may require "stress" dose glucocorticoids

History

- Fatigue/headache
- Muscle weakness
- Loss of appetite/weight loss/nausea/vomiting

Physical Exam

- Altered mental status
- Orthostatic hypotension
- Hyperpigmentation (Addison's Disease)

Lab Tests/Imaging

- Electrolytes (hyponatremia, ↓ K, hypoglycemia)
- Serum cortisol (low), ACTH (high)
- BUN/ Creatinine —prerenal failure

Consults

- Endocrine

Conflict(s)

- Avoid elective/urgent procedures if Addisonian crisis

Optimize/Goals

- Addisonian crisis—treat with hydrocortisone 100 mg IV q6h, fludrocortisone, fluid resuscitation (NS), dextrose, and vasopressors as required
- May require supplemental "stress" dose hydrocortisone in perioperative period
- Adequate fluid resuscitation, supplement, and/or maintenance of electrolytes

Options

- General, regional, local, peripheral nerve block, or sedation
- Altered mental status, vomiting—will require GETA/RSI

Preop: Premed: steroids
Blood: surgery dependent
ICU/stepdown bed: possibly

Room Setup (special drugs/monitors)

- Standard monitors ± Foley catheter
- Consider A-line (BP, electrolyte monitoring) or central line, if requires large volume fluid resuscitation and/or vasopressors
- Treatments for hyperkalemia (insulin, dextrose, sodium bicarbonate)

Induction

- Often requires GETA/RSI (avoid etomidate)

Maintenance

- Balance technique

Emergence

- Routine

Disposition/Pain

- PACU, stepdown, ICU as required

Clinical Pearls

"Stress dose" steroids are given to prevent adrenal crises in the perioperative period for patients with adrenal insufficiency. Keep in the mind the concept of "stress dose" steroids for the perioperative period is controversial. The correct dose and frequency of corticosteroid administration are not standardized. Some believe that it is sufficient to simply continue the patient's current dose of corticosteroid throughout the perioperative period without an additional "stress" dose.

References

National Endocrine and Metabolic Diseases Information Service – Adrenal Insufficiency and Addison's Disease. http://endocrine.niddk.nih.gov/pubs/addison/addison.htm. Accessed May 12, 2010.

Husebye E, Lovas K. Pathogenesis of primary adrenal insufficiency. *Best Pract Res Clin Endocrinol Metab*. 2009;23:147-157.

Adrenocortical Excess (Cushing Syndrome)

Considerations

1. Differentiation of source of corticosteroid excess: pituitary (Cushing disease), adrenal, paraneoplastic, or exogenous (oral steroids)
2. Potential for ↑ ICP
3. Left ventricular hypertrophy (esp. asymmetric septal hypertrophy) with systolic and diastolic dysfunction
4. ↑ Incidence of OSA
5. ↑ Sensitivity to catecholamines
6. Increased infection and poor wound healing

History

- Symptoms of pituitary tumor: headache, bitemporal hemianopsia, ↓ libido (hyperprolactinemia), thirst (DI)
- Weight gain, weakness, lethargy, easy bruising, baldness, hirsutism, acne, hyperhidrosis
- Psychosis, depression
- Snoring, somnolence

Physical Exam

- "Moon" facies, striae, supraclavicular and cervical fat pads
- Careful volume assessment. HTN?
- Papilledema if ↑ ICP
- Signs of pulmonary HTN, cor pulmonale if OSA

Lab Tests/Imaging

- CBC (↑ Hb), electrolytes (↑ K, ↓ Ca^{++}), glucose
- EKG (LVH, strain), ± echo if indicated

Consults

- Endocrinology

Conflict(s)

Optimize

- Treat hypertension if severe
- Treat OSA preoperatively for 6 to 8 weeks if signs of pulmonary HTN/cor pulmonale/erythrocytosis

Goals

- Careful management of perioperative glucose → stress response of surgery/trauma leads to ↑ cortisol

Options

- May be difficult brachial plexus block → obesity/ supraclavicular fat pad

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Mannitol if ↑ ICP
- ± Difficult airway cart if predicted

Induction

Maintenance

Emergence

Disposition/Pain

- If resection of ACTH/cortisol secreting tumor, follow serum cortisol levels q6h and treat if laboratory evidence of adrenal insufficiency (ie, no empiric corticosteroid Rx)

Reference

Nieman LK, Ilias I. Evaluation and treatment of Cushing's syndrome. *Am J Med*. 2005;118:1340-1346.

Alcoholism

Considerations

1. Multisystem disease:

   Neuro—peripheral neuropathy, Wernicke-Korsakoff syndrome (ocular signs, ataxia, confusion)

   CVS—acute: tachycardia, hypertension; cardiomyopathy, palpitations, arrhythmias.

   Respiratory—comorbid: smoking, pneumonia, abscesses
   GI—reflux, gastritis, peptic ulcers
   Liver—fatty liver, hepatitis, cirrhosis
   Pancreas—pancreatitis

   Heme—pancytopenia
2. Chronic consumption: induction of liver enzymes and increased metabolism of other drugs (cross-tolerance)
3. Risk of alcohol withdrawal syndrome: tremors, sweating, nausea, vomiting, seizures (DTs within 48-72 hours post-alcohol cessation)
4. Avoid acetaminophen : higher risk of liver damage

History

- Screen for alcohol consumption/withdrawal symptoms
- Screen for polysubstance abuse, smoking
- Capacity for consent? Alternate decision maker?

Physical Exam

- Neuro—mental status, encephalopathy, tremors
- Stigmata of cirrhosis—jaundice, scleral icterus, ascites, spider angioma

Lab Tests/Imaging

- CBC, electrolytes, BUN/Cr, coags, LFTs
- Electrocardiography (EKG)

Consults

- Addiction/psychiatry

Conflict(s)

- Poorly optimized (inadequate preoperative care, noncompliant) but often present for emergency surgery

Optimize/Goals

- Prevent withdrawal, supplement nutrition—long-lasting benzodiazepines (diazepam, chlordiazepoxide) and thiamine supplementation (po/IV)
- General anesthesia—acute intoxication—more sensitive to anesthetics; chronic intake—tolerant to anesthetics
- May require FFP/platelet transfusion if coagulopathy

Options

- General, regional, local, peripheral nerve block, or sedation
- Often full stomach requiring GETA
- Cooperation may preclude regional/PNB techniques

Preop: Premed: benzodiazepine, GI prophylaxis
Blood: surgery dependent
ICU/stepdown bed: possibly

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter

Induction

- RSI for intoxicated patients, cirrhosis/ascites
- Chronic—require larger doses of induction agents (mindful of cardiomyopathy)

Maintenance

- Balanced technique

Emergence

- Beware of postoperative agitation/confusion

Disposition/Pain

- PACU, stepdown, ICU as required

References

Holder KJ, Weller RM. Alcohol consumption and anaesthesia. *Curr Anaesth Crit Care*. 1997;8:231-236.
Spies CD, Rommelspacher H. Alcohol withdrawal in the surgical patient: prevention and treatment. *Anesth Analg*. 1999;88:946-954.

Amyotrophic Lateral Sclerosis (ALS)

Considerations

1. Aspiration risk due to bulbar palsy
2. Altered responses to neuromuscular blockers
   Succinylcholine—hyperkalemia
   NDMR—prolonged response
3. ↑ Risk for requiring postoperative PPV
4. Potential autonomic dysfunction
5. Ethical issues surrounding preservation of life versus minimizing suffering

History

- H/o dysarthria, dysphagia
- H/o ventilatory support (eg, BiPAP)

Physical Exam

- Skeletal muscle atrophy, weakness, fasciculations
- Hyperreflexia
- Orthostatic hypotension
- Resting tachycardia

Lab Tests/Imaging

- CXR—pneumonia; association with lung ca
- Electrolytes, glucose (↓ K, hyperglycemia)
- PFTs: ↓ VC, PFR
- ABG

Consults

Conflict(s)

- Full stomach versus hyperkalemia with succinylcholine

Optimize

- Treat pneumonia if present

Goals

- Avoid worsening respiratory depression

Options

- GA but care with respiratory depressants
- Epidural, spinal, and CSE have all been used without neurologic exacerbation, and may be excellent choice for avoiding GA

Preop: Premed: care with respiratory depressants
Blood
ICU/stepdown bed: as needed

Room Setup (special drugs/monitors)

Induction

- Modified RSI with rocuronium

Maintenance

- ↓ Doses of NDMRs
- Careful neuromuscular monitoring

Emergence

- Fully awake

Disposition/Pain

- Avoid opioids if possible—multimodal analgesia/RA
- Postoperative chest physio, incentive spirometry

Reference

Hara K, Sakura S, Saito Y, et al. Epidural anesthesia and pulmonary function in a patient with amyotrophic lateral sclerosis. *Anesth Analg*. 1996;83:297-299.

Ankylosing Spondylitis

Considerations

1. AS—chronic inflammatory arthritis (HLA B-27 association)
   Affects mainly spine and sacroiliac joints ("bamboo spine") and insertion sites of ligaments/tendons
   Spine—fractures/collapse and nerve root/cord compression, cauda equina syndrome
   Comorbidity—ulcerative colitis/Crohn, psoriasis, uveitis
   Airway—potentially difficult (C-spine, TMJ involvement)
   Often present for hip/shoulder/spine surgery
   Extra-articular—CV (aortic insufficiency, conduction defects, MI) resp (restrictive defect), eye (uveitis)
2. Therapy—physiotherapy, NSAIDs, methotrexate, bisphosphonates, anti-TNF-alpha, intra-articular steroids, joint replacements/spinal surgery
3. Anti-TNF-alpha agents—increased risk of infection (TB), may worsen CHF—consider holding perioperative

History

- Pain assessment; morning stiffness (improves with exercise)
- Evaluate articular and extra-articular symptoms
- General—weight loss, fatigue

Physical Exam

- Neuro—ROM of joints, muscle strength, sensation (paresthesias)
- Rigid hyperkyphotic deformity of spine "hump," limited ROM in lumbar spine, limited chest expansion
- Unsteady gait
- CV—large volume pulse (water-hammer), diastolic murmur (AI)

Lab Tests/Imaging

- Pending severity: ECG, echocardiography, PFTs, C-spine imaging, ABG

Consults

- Rheumatology

Conflict(s)

- GETA and involvement of cervical spine/TMJ—difficult intubation and potentially impossible tracheostomy (if fixed flexion defect)

Optimize/Goals

- Consider preoperative imaging or indirect laryngoscopy to assess airway
- AFOI is primary option; alternatives include ILMA, video laryngoscope
- Spinal/epidural anesthesia—technically difficult; consider use of ultrasound to assess feasibility
- Beware high block (smaller epidural space)

Options

- General, regional, local, peripheral nerve block, or sedation often require AFOI

Preop:

Premed: ±steroids
Blood: surgery dependent
ICU/stepdown bed: possibly

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter
- Difficult airway cart/video laryngoscope
- Procedures requiring neuro monitoring (MEPs)—avoid muscle relaxants

Induction

- Optimize positioning
- AFOI; backup equipment available

Maintenance

- Balanced technique (mindful of SSEP/MEPs)

Emergence

- Minimize neck movement, care when transferring patient, early institution of chest physiotherapy

Disposition/Pain

- PACU, stepdown, ICU as required

Reference

Woodward LJ, Kam PCA. Ankylosing spondylitis: recent developments and anaesthetic implications. *Anaesthesia*. 2009;64:540-548.

Anterior Mediastinal Mass

Considerations

1. Risk of death from airway obstruction and/or cardiovascular collapse
2. Anatomy—location, size, degree of airway/CV compromise? (Children: tracheobronchial compression >50% precludes safe GETA)
3. Pathology—benign or malignant tumors, cysts, or aneurysms. Most common—Hodgkin's lymphoma or NHL
4. Proposed surgical procedure—usually for diagnosis: sternotomy, thoracotomy, mediastinoscopy (cervical or anterior parasternal), VATS, or extrathoracic mass biopsy. May also present for emergency surgery of other nature
5. If unsafe for GA—1) other options to get tissue for diagnosis: extrathoracic mass/lymph node for biopsy or CT-guided needle biopsy or awake anterior mediastinoscopy with LA, or 2) consider empiric chemo/radiotherapy to decrease size of mass
6. May require ECMO or cardiopulmonary bypass

History

- Chest pain/fullness
- Dyspnea/cough/orthopnea
- Night sweats/fever/fatigue
- Hoarseness/dysphagia
- Syncope
- Asymptomatic (noted on imaging study)

Physical Exam

- Acute distress?
- Lymphadenopathy, fever
- Pericardial rub
- Stridor, cyanosis, venous engorgement of neck, edema of head and neck (SVC syndrome)

Lab Tests/Imaging

- CBC, CXR, chest CT, ECG, echocardiogram, ± flow-vol loop (intra-thoracic obstruction)

Consults

- Thoracic, cardiac

Conflict(s)

- Need for GA versus risk of cardiorespiratory collapse

Optimize/Goals

- Maintain spontaneous ventilation until airway secured or procedure is complete
- If airway and/or vascular compression:
  1) if possible—awake patient
  2) reposition (determine preoperatively which position causes less compression—left/right/prone)
  3) rigid bronch and ventilation distal to obstruction
  4) sternotomy and surgical elevation of mass off compressed vessels
- If requires cardiopulmonary bypass (consider for patients with severe positional symptoms)—establish before induction; will take too long if airway/CVS deteriorates

Options

- General, local, may require AFOI

Preop: Premed:
Blood: surgery dependent
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Arterial line ± Foley catheter
- Difficult airway cart/video laryngoscope

Induction

- Slow, titrated induction maintaining spontaneous ventilation: sevoflurane, propofol, ketamine
- Rigid bronch equipment available (and someone capable of using rigid bronch)

Maintenance

- Muscle relaxants—manually ventilate to ensure positive pressure ventilation is possible before using

Emergence

- Ensure ventilation maintained; beware of postanesthetic obstruction

Disposition/Pain

- PACU, stepdown, ICU as required

Clinical Pearl

Anterior mediastinal mass and flow-volume loops: risk of airway collapse during induction supposed to correlate with increase in mid-expiratory plateau when going from upright to supine position. Not borne out with studies! History, physical exam, and chest imaging provide most valuable information.

Reference

Slinger P, Karsli C. Management of the patient with a large anterior mediastinal mass: recurring myths. *Curr Opin Anaesthesiol*. 2007; 20:1-3.

Aortic Dissection

Considerations

1. Potentially life-threatening (aortic rupture >50% mortality) need to assess urgency of procedure
2. Pathology—tear in intima of aorta:
   Stanford Type A—begins in ascending aorta
   Type B—begins in descending aorta
   DeBakey Type I—involves whole aorta
   Type II—ascending aorta
   Type III—descending aorta
3. Type A—requires surgery to repair aorta ± aortic valve ± coronaries (require CPB)
   Type B—may be treated medically, endovascular
4. Cardiac involvement? AI/tamponade/MI?
5. Risks—atherosclerosis, hypertension, trauma (blunt), bicuspid aortic valve, coarctation of aorta, Ehlers-Danlos syndrome, Marfan syndrome, pregnancy
6. Complications: hemorrhage/shock, emboli, stroke, renal failure
7. May require help: lines/blood products/resuscitation

History

- Chest pain: sudden, severe, sharp, stabbing, tearing; radiating to scapula/back
- Chest trauma? Confusion, anxiety, thirst, dyspnea

Physical Exam

- Acute distress/loss of consciousness/neurologic deficit?
- Difference in BP—R versus L (arms/legs)
- Pallor, dry mouth/skin, diaphoresis
- Rapid, weak pulse; hypotension; loss of distal pulse
- Diastolic murmur (AI), distant heart sounds (pericardial effusion)
- Crackles/rales (CHF?)

Lab Tests/Imaging

- Aortic angiography, chest MRI/CT, echocardiogram, TEE; CXR—wide mediastinum/pleural effusion/R shift trachea; EKG, CBC, electrolytes, coags, BUN/Cr, type/screen

Consults

- Cardiothoracic surgery

Conflict(s)

- Emergency surgery requiring controlled induction versus "full stomach" requiring RSI

Optimize/Goals

- Strict control of BP required (SBP<115 mm Hg)
- Avoid HTN (increased ejection velocity [dP/dt])—start beta blocker before vasodilator
- Neuroprotection? Consider EEG, SSEP, MEP, TCD, NIRS, CSF drain
  type A—proximal aorta or aortic arch—risk of stroke
  type B—spinal cord ischemia is a concern—SSEP, MEP, lumbar drain
- Large bore IV access
- Lung isolation?
- Blood transfusion requirements

Options

- GETA/CPB ± deep hypothermic circulatory arrest (type A)
- GETA/MAC/local (type B—endovascular repair)

Preop: Premed
Blood: surgery dependent
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Std monitors, Foley catheter, arterial monitor, ± CVP monitor ± PAC, ± TEE
- Vasopressors/inotropes; heparin

Induction

- Slow, titrated induction

Maintenance

- Balanced technique; effect on neuro monitoring?

Emergence

- Ensure hemodynamic goals maintained

Disposition/Pain

- PACU, stepdown, ICU as required

References

Khalil A, Tarik T, Porembka DT. Aortic pathology: aortic trauma, debris, dissection, and aneurysm. *Crit Care Med.* 2007;35:S392-S400.
Kohl BA, McGarvey ML. Anesthesia and neurocerebral monitoring for aortic dissection. *Semin Thorac Cardiovasc Surg*. 2005;17:236-246.

Aortic Regurgitation

Considerations

1. Valvular/cardiac surgery or noncardiac surgery?
2. Emergency?
3. Infective endocarditis antibiotic prophylaxis: prosthetic valve, PH endocarditis, congenital HD, cardiac transplant?
4. Mechanical valve—anticoagulants?
5. Etiology: congenital (bicuspid) or acquired (type A dissection, Marfan syndrome, rheumatic disease)
6. Pathophysiology:
   Acute AR—sudden increase in LV tension/work, decreased myocardial perfusion, decreased CO
   Chronic AR—LV volume overload; eccentric hypertrophy and dilatation
7. Severe AR—regurgitant fraction >0.6
8. Medical therapy—continue antihypertensive therapy; treat CHF
9. Procedures—AV replacement for severe AR; often present for noncardiac surgery!

History

- Acute AR—chest pain, dyspnea
- Chronic AR—asymptomatic for years; then dyspnea

Physical Exam

- Acute AR—high-pitched diastolic murmur, bounding pulses, wide pulse pressure, cardiogenic shock
- Chronic AR tachycardia
- Crackles/rales—pulmonary edema

Lab Tests/Imaging

- EKG—signs of LVH; CXR—LV enlargement; echocardiography—severity, coexisting AS; exercise stress test—functional capacity?

Consults

- Cardiology, cardiothoracic surgery as required

Conflict(s)

Optimize/Goals

- AR and noncardiac surgery:
  - maintain high HR
  - low afterload
  - increased preload
  - normal/increased contractility

Options

- General, regional, neuraxial, local, MAC as required

Preop: Premed: IE prophylaxis?
Blood: surgery dependent
ICU/stepdown bed: surgery dependent

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter, arterial monitor, CVP monitor, PAC, TEE

Induction

- Maintain cardiovascular goals; consider arterial line placement preinduction

Maintenance

- Balanced technique

Emergence

- Ensure hemodynamic goals maintained; assess for CHF

Disposition/Pain

- PACU, stepdown, ICU as required; monitor for symptoms of CHF

Reference

Mittnacht, AJC, Fanshawe M, Konstadt S. Anesthetic considerations in the patient with valvular heart disease undergoing noncardiac surgery. *Semin Cardiothorac Vasc Anesth*. 2008;12:33-59.

Aortic Stenosis

Considerations

1. Valvular/cardiac surgery or noncardiac surgery?
2. Elective, urgent, or emergency?
3. Infective endocarditis antibiotic prophylaxis: prosthetic valve, PH endocarditis, congenital HD, cardiac transplant?
4. Mechanical valve—anticoagulants?
5. Etiology: senile calcification, rheumatic heart disease, congenital abnormalities, endocarditis
6. Normal AV area is 2 to 4 cm^2

   "Severe" AS—velocity >4 m/s, mean gradient >50 mm Hg, AV area <1 cm^2

   As valve area decreases, chronic pressure gradient leads to concentric LVH and diastolic dysfunction

   LVH—increased O_2 demand—high risk of coronary ischemia
7. Procedures—AV replacement for severe AS (transcatheter approach for AVR—investigational stages for patients considered high risk for open heart surgery); often present for noncardiac surgery

History

- Mild/mod AS—asymptomatic
- Severe AS—angina, syncope, dyspnea, fatigue, palpitations

Physical Exam

- Pulsus "parvus et tardus" —small and slow pulse
- Crescendo-decrescendo systolic murmur, radiates to carotids
- S4
- Crackles/rales—pulmonary edema

Lab Tests/Imaging

- EKG—signs of LVH, ischemia? CXR—pulmonary edema; echocardiography—severity; exercise or pharmacologic stress test—ischemic disease; cardiac catheterization

Consults

- Cardiology, cardiothoracic surgery as required

Conflict(s)

- Symptomatic severe AS—cancel elective surgery
- Asymptomatic severe AS—reevaluate need for AVR in light of risk of planned noncardiac surgery

Optimize/Goals

- AS and noncardiac surgery:
  - maintenance of sinus rhythm—helps maintain SV
  - normal/decreased HR (avoid tachycardia)
  - increased afterload
  - increased preload
  - normal/increased contractility
- Avoid hypotension (decreased coronary perfusion)
- Balloon valvuloplasty—may temporize and allow for urgent noncardiac surgery

Options

- General, regional, local, MAC as required
- Spinal anesthesia relatively contraindicated

Preop: Premed: IE prophylaxis?
Blood: surgery dependent
ICU/stepdown bed: surgery dependent

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter, arterial monitor, CVP monitor, PAC (beware arrhythmias), TEE
- Defibrillator in OR, defibrillator pads on patient

Induction

- Maintain cardiovascular goals; consider arterial line placement preinduction

Maintenance

- Balanced technique; alpha agonists (phenylephrine) first choice to maintain BP

Emergence

- Ensure hemodynamic goals maintained

Disposition/Pain

- PACU, stepdown, ICU as required

Clinical Pearls

- As CO decreases, pressure gradients may not accurately portray the severity of AS.
- Gorlin formula: valve area is proportional to flow across valve divided by square root of mean pressure gradient. Therefore, CO and pressure gradient required to reliably estimate severity of AS.
- Avoid cardiac stress testing in symptomatic patients—proceed to cardiac catheterization.

Reference

Mittnacht, AJC, Fanshawe M, Konstadt S. Anesthetic considerations in the patient with valvular heart disease undergoing noncardiac surgery. *Semin Cardiothorac Vasc Anesth*. 2008;12:33-59.

ASD/VSD

Considerations

1. Left-to-right shunt at level of either atria (ASD) or ventricles (VSD) (Other left-to-right shunts—PDA, endocardial cushion defect, partial anomalous pulmonary venous return)
2. Acyanotic condition: leads to increased pulmonary flow
3. Increased flow can cause RV overload
4. Poor tolerance of negative inotropes
5. Increase in PVR can lead to reversal of shunt from left to right, to right to left (Eisenmenger syndrome)
6. IV induction agents—slower onset
7. Repair: either percutaneous techniques or open surgical repair
8. Other congenital associations: VSD and trisomy 21
9. Endocarditis prophylaxis? *Not for unrepaired ASD/VSD* —only for first 6 months after repair

History

- ASD: often asymptomatic
- Large VSD: delayed growth, poor exercise tolerance, pulmonary infections

Physical Exam

- Physical appearance—failure to thrive, diaphoresis?
- ASD: split S2, systolic ejection murmur
- VSD: split S2, holosystolic murmur
- CHF: tachycardia, tachypnea, wheezing, hepatomegaly
- Eisenmenger: cyanosis and clubbing

Lab Tests/Imaging

- CBC
- ASD—RAD, RVH, RBBB on ECG
- VSD—LVH, LAH on ECG
- Echo—position/size of defect, CXR—cardiomegaly?
- Electrolytes

Consults

- Cardiology, cardiac surgery

Conflict(s)

- Maintain oxygenation—realizing that 100% O_2 will decrease PVR and increase L-to-R shunt

Optimize/Goals

- Decrease shunt (propofol—greater decrease in SVR than PVR—decreased L-to-R shunt)
- Maintain perfusion and oxygenation; avoid bradycardia
- CHF management—digoxin, diuretics, afterload reduction (ACEI)
- Avoid air bubbles (paradoxical air embolus)

Options

- General, regional, local, MAC as required—usually will require GETA

Preop: Premed: midazolam (0.5 mg/kg po)
Blood: surgery dependent
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter
- Medications: inotropes (dobutamine, milrinone) as required

Induction

- Children: IV induction preferred over inhalation if CHF
- Nasal intubation may be preferred postoperatively

Maintenance

- Balanced technique

Emergence

- Controlled emergence; deep extubation as appropriate

Disposition/Pain

- ICU as required
- Beware heart block; postoperative pulmonary hypertension (if late repair)

Reference

Mann D, Qu JZ, Mehta V. Congenital heart diseases with left-to-right shunts. *International Anesthesiology Clinics.* 2004;42(4):45-58.

Asthma

Considerations

1. Reduction in airway diameter due to contraction of smooth muscle/chronic inflammatory processes. Leads to air-trapping, hyperinflation of lungs, increased work of breathing, V/Q mismatching
2. PFTs: reduced FEV_1 (n ≥80% predicted); reduced FEV_1/FVC ratio (n ≥70% predicted); increased RV, FRC; >12% improvement in FEV_1 after bronchodilator
3. Potential exacerbation with NSAIDs—triad of asthma, nasal polyposis, and aspirin insensitivity
4. High-dose β_2 agonists: hypokalemia, hyperglycemia, hypomagnesemia
5. May cause cor pulmonale (right-sided heart failure): cyanosis, dyspnea, clubbing, elevated JVP, P2, S3, S4, right axis deviation on ECG
6. Chronic steroids—HTN, weight gain, osteoporosis, infections, hyperglycemia

History

- Symptom exacerbation? Dyspnea?
- Recent URI?
- Recent visit to hospital for asthma?
- Use of bronchodilator medications?
- Allergies? Precipitating factors?
- PH of GETA?
- Medications? Steroids?

Physical Exam

- Respiratory distress: tachypnea, accessory muscle use, cyanosis?
- Chest—wheezing, prolonged expiratory phase
- Pulsus paradoxus
- Cor pulmonale?

Lab Tests/Imaging

- PFTs severity/reversibility
- ABG severity
- Peak flow meter (15-20% reduction—exacerbation)

Consults

- Pulmonary as required

Conflict(s)

- Risk of bronchospasm in patient requiring GETA
- Airway instrumentation—parasympathetic reflex bronchoconstriction

Optimize/Goals

- Avoid triggering stimuli—consider topical lidocaine, regional anesthesia, LMA versus ETT, use of volatile anesthetics, propofol, opioids, ketamine as appropriate
- Treatments—β_2 agonists, corticosteroids (consider preoperative oral prednisone (40-60 mg/d) for patients. with severe asthma)
- Pulmonary infections—treat with antibiotics
- Correct fluid/electrolytes imbalances
- Chest physio—for sputum clearance
- Smoking cessation as required

Options

- General, regional, local, MAC as required

Preop: Premed: $\beta_2$agonist
Blood: surgery dependent
ICU/stepdown bed: possibly

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter
- Medications: β_2 agonist, low-dose epinephrine

Induction

- Propofol or ketamine (consider anticholinergic for secretions); fentanyl (reduced-risk histamine release); avoid atracurium, mivacurium (histamine release)
- Intraoperative bronchospasm: 100% oxygen, deepen anesthesia (volatile/IV), β_2 agonists, epinephrine (SC/IV), corticosteroids (onset 4-8 hours)

Maintenance

- Inhalational anesthetics

Emergence

- Controlled emergence; deep extubation as appropriate

Disposition/Pain

- PACU, stepdown, ICU as required

References

Burburan SM, Xisto DG, Rocco PR. Anaesthetic management in asthma. *Minerva Anestesiol*. 2007;73:357-365.

Burns

Considerations

1. Body surface area—"Rule of 9's." Head 9%, arms 9% each, legs 18% each, chest 18%, back 18% (head is 18% for children)—count second- and third-degree burns
2. Inhalation injury? Risk airway obstruction
3. Shock—hypovolemic and distributive (for >72 hours)
4. Early effects (24-48 hours)—*decreased CO*, decreased response to catecholamines, *increased SVR*, metabolic acidosis
5. Late effects (>48 hours)—*increased CO* and *decreased SVR*
6. Parkland formula: Four cc LR/kg/% body surface area over 24 hours (half given in first 8 hours, other half given over 16 hours)
7. Succinylcholine—avoid 24 hours after burn (unclear how long receptor proliferation lasts for)
8. Carbon monoxide poisoning? Hyperbaric therapy?

History

- Inhalation injury? Closed-space fire (house/car)
- Type of burn—electrical, chemical, thermal

Physical Exam

- ABCs as per ACLS/ATLS
- Appearance—facial burns/edema, stridor, respiratory distress?
- Extent of burn
- Sites for IV access, invasive monitors

Lab Tests/Imaging

- ABG, carboxyhemoglobin, CBC, electrolytes, creatinine
- CXR
- Cyanide level (exposure from burning plastics)

Consults

- Referral to burn unit, plastic surgery

Conflict(s)

Optimize/Goals

- Secure airway early before edema
- Maintain U/O >0.5 to 1.0 cc/kg/h; IV hydration
- Avoid hypothermia
- Avoid infection—strict aseptic techniques
- Avoid compartment syndrome—may require fasciotomies/escharotomies
- Blood products as required (risk of significant blood loss)

Options

- Usually will require GETA; consider regional, PNB if appropriate for analgesia

Preop: Premed: GI prophylaxis
Blood: surgery dependent
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Std monitors, Foley catheter, arterial line, central line
- Difficult airway equipment as required (may require surgical airway depending on location of burn)

Induction

- Ketamine or etomidate or propofol as required
- Succinylcholine in first 24 hours; otherwise avoid
- Uncut ETT (beware postoperative edema)

Maintenance

- Balanced technique; resistance to NDMRs possible

Emergence

- Keep intubated (depending on size of burn)

Disposition/Pain

- ICU as required
- Nutritional support, pain control, DVT prophylaxis

Reference

MacLennan N, Heimbach DM, Cullen BF. Anesthesia for major thermal injury. *Anesthesiology*. 1998;89(3):749-770.

Considerations

1. Primary disease? Location, extent
2. Secondary (metastatic) disease? Location, extent
3. Paraneoplastic syndrome?
   - SIADH (hyponatremia)—carcinoma of lung, pancreas, bladder, prostate, breast, colon
   - Hypercalcemia
   - Cushing syndrome (secrete ACTH)
4. Chemotherapy? Radiotherapy?
5. Multisystem effects:
   Cardiac effects—arrhythmias, CHF (anthracycline)
   Pulmonary—pneumonitis
   Renal—prerenal failure or nephrotoxic therapy
   Pancytopenia—myelosuppression
   Infection
   Tumor lysis syndrome
6. General: depression, pain, cachexia, nausea, vomiting, mucositis

History

- Diagnosis
- Symptoms
- Nutrition, comorbid conditions, current/past therapies

Physical Exam

- As guided from history
- Sites for IV access, ± invasive monitors

Lab Tests/Imaging

- CBC, electrolytes, creatinine, coagulation
- CXR, echo as required

Consults

- Oncologist as required

Conflict(s)

- May have difficult vascular access
- May have thrombocytopenia or coagulopathy that precludes regional/PNB
- May have neuro, cardiac, pulm involvement that precludes sedation

Optimize/Goals

- Optimize preoperative status: hydration, electrolytes, anemia
- Blood products as required (risk of significant blood loss)
- Consider regional/PNB as appropriate: retrospective studies suggest that epidural analgesia may be superior to IV opiates in preventing tumor recurrence (opioids suppress NK cell activity)

Options

- General, regional, PNB, MAC, local as appropriate

Preop: Premed
Blood: surgery dependent
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter, arterial line, central line
- Difficult airway equipment as required

Induction

- Depending on preoperative status—may require RSI, placement of invasive hemodynamic monitors preoperatively, or advanced airway equipment

Maintenance

- Balanced technique; consider regional to reduce opioids

Emergence

- Routine

Disposition/Pain

- Recovery room, stepdown, ICU as required
- Nutritional support, pain control, DVT prophylaxis

Reference

Arain MR, Buggy DJ. Anaesthesia for cancer patients. *Curr Opin Anaesthesiol*. 2007;20:247-253.

Carcinoid

Considerations

1. Tumors of GI tract (also bronchi, lungs)—contain variety of hormones—serotonin, histamine, substance P, prostaglandins, kallikrein, etc
2. Tumor location? Imaging or PET scan
3. Symptomatic—obstruction, bleeding, diarrhea, weight loss, pain? Surgical and medical therapy (octreotide, antihistamines, 5-HT antagonists)
4. Carcinoid syndrome—systemic release of serotonin, histamine, etc (usually from liver mets or non-GI tumor). Results in skin flushing, edema, diarrhea, bronchoconstriction; progress to RV failure, tricuspid and pulmonary regurgitation/stenosis
5. Carcinoid crisis: due to handling of tumor during surgery—bronchoconstriction, hypotension (sometimes HTN), hyperglycemia—treat with octreotide
6. Present for GI/hepatic/cardiac/thoracic surgery
7. Associated with multiple endocrine neoplasia

History

- Symptoms? Dyspnea, bronchospasm, diarrhea?
- Triggers?
- Treatments

Physical Exam

- Routine and as directed from history
- Volume status: orthostatic changes, mucous membranes
- Cardiac: elevated JVP, murmur?
- Respiratory: wheezing?
- Skin: flushing?

Lab Tests/Imaging

- CBC, electrolytes, LFTs, glucose
- CXR, ECG, echo

Consults

- Endocrinologist as required

Conflict(s)

- Serotonin release can cause hyper-hypotension—catecholamines can potentially cause more release

Optimize/Goals

- Prevent release from tumor with octreotide (somatostatin analogue)—50 to 500 µg subcutaneously q8h; if crisis, can use IV slowly)
- Maintain adequate volume, electrolytes
- Maintain cardiovascular stability (avoid hyper- and hypotension—can provoke release of carcinoid mediators)
- Prevent bronchospasm (caution with beta agonists)
- Caution: anything known to release histamine, serotonin, or catecholamines (cause serotonin secretion). Use of octreotide may reduce this risk (epinephrine given successfully)
- Avoid anxiety, hypercapnia, hypothermia

Options

- Usually GETA; regional may provoke hypotension

Preop: Premed: octreotide, anti-histamine, GI prophylaxis
Blood: surgery dependent
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Std monitors, Foley catheter, arterial line, central line
- Octreotide; antihistamines; antihypertensives; vasopressin, phenylephrine to maintain BP

Induction

- Caution: ketamine, morphine, catecholamines (epinephrine), mivacurium, atracurium
- Prefer: propofol, etomidate, fentanyl
- Succinylcholine used successfully; caution: risk of mediator release (contractions/histamine)

Maintenance

- Balanced technique

Emergence

- Routine

Disposition/Pain

- Stepdown, ICU as required
- Nutritional support, pain control, DVT prophylaxis

Reference

Vaughan DJA, Brunner MD. Anesthesia for patients with carcinoid syndrome. *Int Anesthesiol Clin*. 1997;35(4):129-142.

Cardiac Contusion

Considerations

1. Usually due to blunt chest trauma (car accidents, falls, sports injury)—injury usually to right ventricle
2. Trauma patient? Other severe injuries possible
3. Spectrum of injury from mild to life-threatening
4. Potential emergency: life-threatening arrhythmias and cardiogenic shock
5. EKG—variety of changes: nonspecific ST-T wave elevation, Q-waves, conduction disorders, bradycardia, tachycardia, AFIB, VT, VF
6. Physical exam often not helpful:

   Cardiac troponins used to diagnose (may persist for days)—similar to infarction!

   Echo—wall motion abnormalities, pericardial effusion, thrombus, rupture?
7. Complications: early—arrhythmias, myocardial rupture, valvular damage, thrombosis; late—ventricular aneurysm, dilated cardiomyopathy, pericarditis, ventricular arrhythmias

History

- Nature of injury?
- Palpitations, chest pain?

Physical Exam

- As per ACLS/ATLS
- Evidence of chest trauma? Contusion/fracture?
- HR and rhythm?

Lab Tests/Imaging

- ECG, echo—transthoracic/esophageal (wall motion, EF, other injury?)
- Troponin
- CXR (other injuries?)

Consults

- Trauma team as required; cardiology

Conflict(s)

- Cardiac contusion often occurs in setting of emergency trauma—may require operative treatment of other injuries (and RSI) despite unstable cardiovascular status

Optimize/Goals

- Treat heart failure—may require invasive monitors of BP and CVP; fluid resuscitation; inotropes; intra-aortic balloon pump; LVAD
- Treat arrhythmias as per ACLS
- Treat associated injuries
- Avoid hypoxia/acidosis (aggravate arrhythmias)
- Avoid further depression of cardiac function

Options

- Usually GETA as required

Preop: Premed
Blood: surgery dependent
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Std monitors, Foley catheter, arterial line, central line
- Inotropes, vasopressors, antiarrhythmics

Induction

- Often require GETA/RSI for other injuries—beware induction hypotension
- Consider etomidate or ketamine; placement of invasive monitors before induction; support with inotropes and vasopressors as required

Maintenance

- Balanced technique

Emergence

- Routine

Disposition/Pain

- Stepdown, ICU (monitor ECG)

Reference

Sybrandy KC, Cramer MJM, Burgersdijk C. Diagnosing cardiac contusion: old wisdom and new insights. *Heart*. 2003;89:485-489.

Cardiac Tamponade

Considerations

1. Potential emergency
2. Obstructive shock—blocks cardiac filling: increased intrapericardial pressure leads to cardiac compression—decreased CO/BP
3. Chronic situation: the pericardium can stretch and accommodate more fluid; acute situation: small amount of fluid can lead to hemodynamic compromise. Cardiac inflow is limited as pericardial and cardiac diastolic pressures equalize
4. Causes—acute—usually trauma (penetrating/blunt), myocardial rupture (MI/aneurysm), iatrogenic during cardiac cath, etc; chronic—idiopathic, viral, neoplastic, uremia, SLE, hypothyroid.
5. Beck triad—elevated CVP, decreased blood pressure, quiet heart sounds
6. Compensation—tachycardia, increased SVR, increased contractility (sympathetic activation)

History

- Acute—often without symptoms
- History of trauma?
- Chronic—dyspnea, chest pain
- Associated comorbidities

Physical Exam

- As per ACLS/ATLS
- Elevated JVP, tachycardia, hypotension, muffled heart sounds
- Pulsus paradoxus—inspiratory fall in SBP >10 mm Hg
- Kussmaul sign—JVP distends with inspiration

Lab Tests/Imaging

- ECG—normal; tachycardia; electrical alternans
- Echo—essential—fluid and compressed heart chambers, diastolic collapse of RA/RV
- CXR—cardiomegaly, other trauma?

Consults

- Trauma team; cardiothoracic surgery; cardiology as required

Conflict(s)

- May require GETA but want to avoid decreases in sympathetic tone and positive pressure ventilation

Optimize/Goals

- Treatment—drain pericardial fluid—needle paracentesis or surgical drainage
- Medical therapy—inotropes (dobutamine)
- Avoid positive pressure ventilation. Decreased venous return and increased intrathoracic pressure—decreased CO
- If cardiac arrest—chest compressions not effective—immediate drainage preferred
- Avoid hypovolemia; maintain filling pressures
- Avoid bradycardia
- Avoid decreased SVR
- Maintain contractility

Options

- Evacuation of fluid emergently (subxiphoid needle)
- If GETA—beware cardiac arrest

Preop: Premed:
Blood: surgery dependent
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Std monitors, Foley catheter, arterial line, central line
- Large IV, inotropes, vasopressors

Induction

- Maintain sympathetic tone: consider ketamine or etomidate; placement of invasive monitors before induction; support with inotropes and vasopressors as required

Maintenance

- Balanced technique; spontaneous ventilation until tamponade relieved (or gentle positive pressure ventilation if tolerated)

Emergence

- Routine

Disposition/Pain

- Stepdown, ICU

Reference

Spodick DH. Acute cardiac tamponade. *N Engl J Med*. 2003;349:684-690.

Cerebral Aneurysm

Considerations

1. Potential emergency—subarachnoid hemorrhage (SAH). Mortality ~ 50%.
2. Elective treatment—endovascular or surgical repair
3. Risks: hypertension, smoking, alcohol abuse, cocaine use, FH, polycystic kidney disease, Ehlers-Danlos (lesser infection, trauma)
4. As ICP increases to systemic pressure—severe headache and loss of consciousness
5. SAH complications: rebleeding, mass effect, brain edema, seizures, hydrocephalus, vasospasm, cardiac arrhythmias, stunned myocardium, neurogenic pulmonary edema, increased serum troponin (due to release of high levels of catecholamines), hypovolemia, hyponatremia, hypomagnesemia
6. Treatment for vasospasm: triple H—hypertension, hypervolemia, hemodilution; also early angioplasty ± vasodilator infusion; nimodipine/nicardipine

History

- Age (elderly—poorer prognosis)
- Headache ("worst of my life"), seizure, neuro deficit?
- Nausea, stiff neck
- Risk factor assessment. Cocaine use?

Physical Exam

- ABCs
- Level of consciousness?
- Vomiting?
- Pupils (nonreactive, small)
- Focal neuro deficits

Lab Tests/Imaging

- CBC, coags, electrolytes, glucose, CXR, ECG, head CT (noncontrast), angiography (CT, MRA, selective cerebral)
- Toxicology as per history

Consults

- Neurosurgery, neuroradiology as required

Conflict(s)

- Secure airway in obtunded patient without changing blood pressure—higher blood pressure associated with rebleeding while lower blood pressure associated with neurologic deficit
- Maintain cerebral perfusion pressure (CPP) without further exacerbating cardiac dysfunction

Optimize/Goals

- Avoid changes in BP—nicardipine, labetalol, esmolol
- Brain protection: maintain CPP >60 mm Hg; normocapnia; mild hypothermia (32-34 C); avoid hyperglycemia, acidosis, and hypoxia; seizure prophylaxis
- Prevent/treat seizures, vasospasm
- Maintain volume and electrolytes (re: hyponatremia)

Options

- Typically GETA; MAC/local for diagnostic angiography
- Giant aneurysm: may require deep hypothermia with circulatory arrest and extracorporeal circulation

Preop: Premed
Blood: surgery dependent
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Std monitors, Foley catheter, arterial line placed preinduction, large bore IV, antihypertensives available

Induction

- Deep, controlled induction; avoid hypertension; avoid coughing/straining

Maintenance

- Balanced technique; TIVA technique superior for elevated ICP (volatile anesthetics will increase CBF >1.0 MAC)

Emergence

- Controlled, avoid hemodynamic changes; depending on neuro status may require postoperative intub/vent

Disposition/Pain

- Stepdown, ICU, neuro-ICU as available

Reference

Bederson JB, Connolly ES, Batjer HH, et al. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a statement for healthcare professionals from a Special Writing Group of the Stroke Council. American Heart Association. *Stroke*. 2009;40:994-1025.

Chronic Obstructive Pulmonary Disease

Considerations

1. Presence of V/Q mismatch with resulting hypoxemia and hypercarbia
2. Potential for chronic hypoxemia, pulmonary hypertension, cor pulmonale, polycythemia
3. Potential for bullous disease and risk of pneumothorax
4. Potential for postoperative mechanical ventilation
5. Coexisting diseases: CAD, PVD, CVA
6. Presence of carboxyhemoglobinemia

History

- Duration, severity, smoking history, therapy, exacerbating factors
- Current status and exercise tolerance
- Coexisting disease: CAD, PVD, CVA?
- Use of home oxygen or CPAP?

Physical Exam

- Body habitus: barrel-chested, plethoric, asthenic?
- Pursed lips?
- Able to talk in full sentences?
- RV failure: JVD, RV heave, pedal edema
- Cyanosis, clubbing

Lab Tests/Imaging

- CBC, ABG ± PFTs
- CXR if suspect bullous disease
- EKG (R heart strain, low voltage)

Consults

- Pulmonology

Conflict(s)

Optimize

- Preoperative chest physiotherapy
- Antibiotics for infection
- Beta agonists (albuterol) and anticholinergics (Atrovent)
- IV hydration
- Smoking cessation (will aid in CO levels)

Options

- Epidural anesthesia/postoperative analgesia reduces postoperative pulmonary complications

Preop: Premed: antibiotics
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Arterial line for ABG sampling

Induction

- Care with opioids

Maintenance

- I:E ratio >1:2 to prevent gas trapping

Emergence

Disposition/Pain

- Postoperative epidural
- Chest physio/incentive spirometry
- Early ambulation

Reference

Licker M, Schweizer A, Ellenberger C, Tschopp JM, Diaper J, Clergue F. Perioperative medical management of patients with COPD. *Int J Chron Obstruct Pulmon Dis.* 2007;2:493-515.

Chronic Renal Failure

Considerations

1. Presence of common coexisting diseases such as CAD, HTN, DM, PVD
2. Issues with volume overload
3. Issues with electrolytes ($\uparrow K^+$, $\uparrow Mg^{++}$, $\downarrow Ca^{++}$)
4. Potential presence of uremia (encephalopathy, autonomic/peripheral neuropathy, pleural/pericardial effusions, anemia, platelet dysfunction)
5. Pharmacokinetic changes due to $\downarrow$ elimination, acidosis and hypoalbuminemia
6. Potential difficult IV access
7. Cr >2.0 independent risk factor for cardiac complications

History

- Etiology: DM, HTN, cystic dz, glomerulonephritis, etc
- Mental status changes, glove and stocking neuropathy
- Easy mucosal bleeding, GERD
- Dialysis regimen

Physical Exam

- Careful volume status evaluation
- Autonomic evaluation: orthostatic vitals

Lab Tests/Imaging

- CBC, electrolytes, BUN, Cr, ±ABG if suspect acidosis
- EKG ($\uparrow$ K, ischemia, LVH)
- ±CXR

Consults

- Nephrology

Conflict(s)

- Hyperkalemia and need for emergency surgery

Optimize

- Dialyze up to 1 day prior (5-10% in chronic CHF)
- Treat hyperkalemia (see earlier) or other electrolyte disturbances
- If on peritoneal dialysis, drain dialysate to optimize respiratory function
- Consider transfusion if Hb<6 (rare now with EPO)

Goals

- Avoid nephrotoxic drugs (NSAIDs, aminoglycosides)
- Avoid renally excreted drugs (esp. morphine, meperidine, pancuronium)
- Maintain euvolemia

Options

- Consider local, regional, or general anesthesia as required

Preop: Premed: GI prophylaxis
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Avoid NIBP cuff over AV fistulae
- Careful positioning due to renal osteodystrophy
- Low threshold for CVP for volume status monitoring

Induction

- Consider RSI (care with sux if $\uparrow K^+$)
- Care with local anesthetic doses ($\uparrow$ systemic toxicity)

Maintenance

Emergence

Disposition/Pain

Clinical Pearls

- If hyperkalemic acutely:
  - 10 mL calcium gluconate 10% to restore difference between myocardial resting and threshold potentials
  - glucose + insulin (10 units IV)
  - β_2 agonists (albuterol)
  - ion exchange resins (eg, Kayexalate)
  - bicarbonate decreases K^+ slowly and minimally: use only if concomitant acidosis or if alkalinization of urine desired
  - hemodialysis is the fastest and most effective means of removing potassium from the body

Reference

Craig RG, Hunter JM. Recent developments in the perioperative management of adult patients with chronic kidney disease. *Br J Anaesth*. 2008;
101:296-310.

Considerations

1. Identification of the degree of ventricular dysfunction, ischemic burden, and adequacy of therapy
2. Assessment of the likelihood of fluid shifts/bleeding and implications of fluid loading
3. Prediction of need for special cardiovascular monitoring and inotropic agents
4. Potential for electrolyte abnormalities due to medications (especially hyperkalemia)

History

- Fatigue, dyspnea, exercise intolerance, ankle swelling
- Orthopnea, paroxysmal nocturnal dyspnea
- H/o CAD, HTN, valvular dz, DM, EtOH
- Current baseline? Progession?
- Medical rx: ACEI, ARBs, beta blockers, diuretics, digoxin, aldosterone antagonists, statins

Physical Exam

- JVD, S3, displaced cardiac apical impulse?
- Rales, dependent edema

Lab Tests/Imaging

- New or suspected CHF? → CBC, electrolytes, BUN, Cr, glucose, natriuretic peptides. If chronic, labs as indicated, eg, electrolytes if on ARBs/diuretics
- EKG, echo (± stress testing if new or suspect CAD)
- CXR if new or change in symptoms

Consults

- Cardiology

Conflict(s)

- Emergency surgery likely requiring ++ fluid versus failing heart

Optimize

- Consider preoperative coronary revascularization only if patient would benefit independent of surgery
- Coordinate with cardiology and ensure correct heart failure regimen (see earlier)
- Consider holding ACEI/ARB day of surgery if hypotension anticipated

Goals

- Prevent worsening ventricular function perioperatively

Options

- GA/regional/sedation. May not tolerate lying flat

Preop: Premed
Blood
ICU/stepdown bed: possibly

Room Setup (special drugs/monitors)

- Arterial line, ± PA catheter or TEE
- Vasopressors to rx hypotension (esp. if on ACEI/ARB)

Induction

- Slow: allow for ↑ arm-to-brain circulation time

Maintenance

- Volatile + opioid

Emergence

Disposition/Pain

- ± Stepdown unit for EKG monitoring

Clinical Pearls

Current practice in the medical management of congestive heart failure includes the following:

1. ACEI/ARBs (eg, ramipril, enalapril, candasartan)
   - vasodilation → ↓ afterload
   - ↓ salt/H2O reabsorption
   - prevent myocardial remodeling
   - mortality benefit in patients with symptomatic heart failure
2. Aldosterone antagonists (eg, aldosterone, eplerenone)
   - ↑ diuresis/natriuresis
   - ↓ myocardial collagen formation, NE levels
   - mortality benefit
3. Beta blockers (metoprolol, bisoprolol, carvedilol)
   - reversal of remodeling, blunting of neurohumoral response
   - improved myocardial O_2 supply/demand ratio
   - not used in acute CHF or cardiogenic shock
4. Others
   - diuretics for symptomatic treatment (usually in combo with above therapies)
   - digoxin → second- or third-line therapy in combo with above. Narrow therapeutic window and risk of toxicity

Reference

Wojciechowski P. Perioperative optimization of the heart failure patient. *Int Anesth Clin*. 2009;47:121-135.

Considerations

1. Cardiac or noncardiac surgery?
2. Elective, urgent, or emergency procedure?
3. Unstable—angina, CHF, severe arrhythmias/valvular disease/conduction disorders? Unless emergency these patients require further evaluation
4. Exercise tolerance? >4 METS?
5. Risk factors—PH CAD, PH CHF, stroke, insulin-requiring diabetes, creatinine >2.0 mg/dL
6. Noninvasive stress testing only if it will change Mx!
7. Therapy: medical, percutaneous, surgical?
8. Cardioprotection—statins, beta blockers, $\alpha_2$agonists, antiplatelet agents (aspirin)
9. Coronary stents? Continue clopidogrel/ASA for 12 months—avoid elective surgery
10. Perioperative beta blockade controversial. May reduce ischemia but higher mortality and stroke risk
11. Complications: MI (peak postoperative days 1-3), cardiac arrest, CHF, unstable angina, unstable arrhythmias

History

- Risk factors—PH CHF/CAD, diabetes, renal failure, stroke
- Presence/severity of angina, dyspnea, orthopnea, PND
- Exercise tolerance
- Medical/surgical management to date

Physical Exam

- Vital signs—compliant with medical therapy?
- Signs of atherosclerosis—xanthomas, diminished peripheral pulses, carotid bruit
- S3/S4 due to LV dysfunction/diastolic dysfunction
- CHF—elevated JVP, crackles/wheezing, hepatomegaly, peripheral edema

Lab Tests/Imaging

- CBC, electrolytes
- EKG—signs ischemia/MI? CXR, echocardiogram, exercise or pharmacologic stress test, cardiac catheterization as required

Consults

- Cardiology, cardiothoracic surgery as required

Conflict(s)

- No "best" anesthetic technique for myocardial protection. High-dose opioids popular due to cardiovascular stability. May not be practical for ambulatory surgery in patients with CAD

Optimize/Goals

- Myocardial ischemia—shift balance of supply/demand in favor of oxygen supply to myocardium: maintenance of sinus rhythm, decreased/normal HR (avoid tachycardia), maintenance of preload, afterload, and contractility (may require fluids, blood transfusion, diuretics, vasopressors, inotropes, or IABP depending on clinical situation)
- Consider if/when to abort procedure and urgent cardiac catheterization if not responsive to medical therapy
- Medical management—oxygen, analgesia, nitroglycerin, beta blockers, statins, ACEI, antiplatelet agents, and/or anticoagulants if not contraindicated

Options

- General, regional, local, MAC as required

Preop: Premed
Blood: surgery dependent
ICU/stepdown bed: surgery dependent

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter ± invasive monitors: arterial monitor, CVP monitor, PAC, TEE
- Monitor ECG leads V and II for ST segment changes

Induction

- Maintain cardiovascular goals; consider arterial line placement preinduction

Maintenance

- Balanced technique; volatile anesthetics may protect against ischemia-reperfusion injury.
- If ischemia—support hemodynamics, optimize myocardial oxygen supply/demand; abort procedure?

Emergence

- Ensure hemodynamic goals maintained

Disposition/Pain

- PACU/stepdown/ICU as required, follow troponin postoperatively in high-risk patients

Clinical Pearl

There is no single "best" anesthetic technique for patients with CAD. The technique, choice of monitors, and medications need to take into consideration the underlying goals for the patient and the type of procedure they are having. Local anesthesia or peripheral nerve blockade may be preferred depending on the clinical situation. Volatile anesthetics, opioids, α_2 agonists (clonidine, dexmedetomidine), and epidural or spinal anesthesia may be advantageous for certain patient populations.

References

Fleisher LA, Beckman JA, Brown KA, et al. ACC/AHA 2006 guideline update on perioperative cardiovascular evaluation for noncardiac surgery: focused update on perioperative beta-blocker therapy: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2002 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery): developed in collaboration with the American Society of Echocardiography, American Society of Nuclear Cardiology, Heart Rhythm Society, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society for Vascular Medicine and Biology. *Circulation*. 2006;113:2662-2674.

London M. Cardiovascular problems in noncardiac surgery. *Current Opinion in Critical Care*. 2009;15:333-341.

Cystic Fibrosis

Considerations

1. Genetic mutation for protein (CF transmembrane regulator) found on most exocrine glands. Causes changes in airway, GI tract, sweat glands, and GU system. Median age of survival is ~38 years
2. Airway: lungs—obstructive disease. Viscous mucous secretions with reduced mucociliary clearance leading to airway inflammation and chronic infection *(Pseudomonas aeruginosa,* etc) and chronic hypoxia/hypercarbia *(pulmonary hypertension/ cor pulmonale)*. Upper airway—nasal polyps, sinusitis
3. Pancreas: retention of digestive enzymes, autodigestion, fibrosis, pancreatitis. Can cause protein and fat malabsorption (vitamin K deficiency), diabetes. Require dietary supplements
4. Hepatobiliary: fatty liver, cirrhosis, portal HTN
5. GI: meconium ileus (neonate), intestinal obstruction
6. Bones: fractures, scoliosis/kyphosis
7. GU: late onset of puberty, decreased fertility

History

- Dyspnea, sputum production, respiratory infections?
- Home O_2, BiPAP, exercise tolerance
- Nutrition, comorbid conditions
- Antibiotic therapy, bronchodilators, steroids
- Previous surgeries—ERCP, liver transplant, lung transplant?

Physical Exam

- Body habitus. Vitals (SpO_2?)
- Pulmonary exam: cough, wheeze, crackles
- Cor pulmonale—split S2, S3, S4; distended neck veins, cyanosis, peripheral edema
- Stigmata of liver disease

Lab Tests/Imaging

- CBC, electrolytes, LFTs, coagulation, glucose
- CXR—hyperinflation, kyphoscoliosis?
- echo as required—RVH, cor pulmonale
- PFTs—obstructive pattern

Consults

- Primary care, pulmonary, endocrinology

Conflict(s)

- May have cardiac or pulmonary or hepatic involvement that precludes sedation yet GETA may lead to prolonged intubation/ ICU course.

Optimize/Goals

- Optimize preoperative status: nutrition, hydration, electrolytes, coagulation, glucose
- Bronchodilators for patients with reactive airways
- Minimize respiratory depression; full recovery of airway reflexes by end of surgery
- Muscle relaxation—caution in patients who rely on muscle tone for airway patency (worsen obstruction)

Options

- General, regional, PNB, MAC, local as appropriate (re: regional/ PNB—beware altered coagulation due to malabsorption)

Preop: Premed: beware multidrug resistant bugs with antibiotics
Blood: surgery dependent
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Std monitors + invasive monitors as required

Induction

- Minimize respiratory depression (short-acting agents)
- Avoid nasal intubation/nasal airways (re: polyps/sinusitis)

Maintenance

- Balanced technique; consider regional to reduce opioids
- May require frequent suctioning of ETT

Emergence

- Airway reflexes fully recovered, consider postoperative intubation/ventilation as required

Disposition/Pain

- Chest PT, BiPAP
- Recovery room, stepdown, ICU as required

Reference

Huffmyer JL, Littlewood KE, Nemergut EC. Perioperative management of the adult with cystic fibrosis. *Anesth Analg*. 2009;109:1949-1961.

Considerations

1. Metabolic disorder associated with microvascular (retinopathy, nephropathy) and macrovascular complications (cardio- and cerebrovascular)
2. Cause:
   DM1—insulin deficiency (autoimmune)
   DM2—insulin resistance, altered insulin secretion
3. DM1—risk of diabetes ketoacidosis (DKA) (dehydration, acidosis, vomiting, fatigue, confusion, unconsciousness)
   DM2—risk of hyperosmolar hyperglycemia syndrome
4. Metabolic syndrome—DM2, obesity, dyslipidemia, hypertension, procoagulant state, obstructive sleep apnea (OSA)
5. Perioperative hyperglycemia associated with wound infection. DM may increase morbidity (MI, renal failure, stroke) in certain surgical populations (CABG)
6. Medications?—insulin, sulfonylureas, biguanides, thiazolidinediones, DPP-4 inhibitors, incretin mimetics
7. Hypoglycemia—weakness, fatigue, confusion, seizures; sweating, tachycardia, hunger

History

- Type of diabetes (home monitor? Typical control?)
- Complications—CV, renal, neuropathy, retinopathy?
- Medications
- Exercise tolerance? Other CV risk factors?
- Gastroparesis? (heartburn, nausea, reflux)

Physical Exam

- CNS—peripheral neuropathies, autonomic neuropathy (orthostatic hypotension, decreased HR variability)
- Airway (stiff joints due to glycosylation)—limited cervical spine mobility?

Lab Tests/Imaging

- Fasting blood glucose, HbA_{1C}, urinalysis (ketones, protein), creatinine, electrolyes, CBC (Hct/Hgb)
- ECG

Consults

- Primary care, endocrinology, cardiology, nephrology as required

Conflict(s)

- What is optimal glucose level perioperatively?
- "Tight" glycemic control (<108 mg/dL or <6 mmol/L]) is controversial—may increase mortality
- "NICE-SUGAR" study (2009)—suggests target of <180 mg/dL or 10 mmol/L as more appropriate

Optimize/Goals

- Avoid hypo- or hyperglycemia, maintain electrolytes (potassium, magnesium, phosphate)
- Generally increased insulin requirements perioperatively:
  DM1—requires IV insulin (and IV glucose) to avoid acidosis and hypoglycemia
  DM2—holds hypoglycemia agents (may require IV insulin and glucose for larger procedures)
- Monitor glucose perioperatively at least every 1 to 2 hours (frequency depends on preop glycemic control and surgical procedure)

Options

- General, regional, local, MAC as required

Preop: Premed
Blood: surgery dependent
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Std monitors + glucose monitor
- Additional airway equipment pending airway examination

Induction

- Routine (consider RSI if gastroparesis)

Maintenance

- Balanced technique

Emergence

- Routine

Disposition/Pain

- Monitor glucose postoperatively. Restart regular therapy pending return of oral intake.

Clinical Pearl

Loss of normal heart rate variability with breathing suggests autonomic neuropathy (gastroparesis).

References

Robertshaw HJ, Hall GM. Diabetes mellitus: anaesthetic management. *Anaesthesia*. 2006;61:1187-1190.
Vann MA. Perioperative management of ambulatory surgical patients with diabetes mellitus. *Curr Opin Anaesthesiol*. 2009;22:718-724.

Down Syndrome (Trisomy 21)

Considerations

Multisystem disease:

1. Airway/Pulmonary—atlantoaxial (C1-C2) instability (AAI), mid-face hypoplasia, macroglossia, subglottic stenosis, OSA, pulmonary hypertension, chronic sinusitis
2. AAI—greater subluxation when the neck is flexed (although neck extension and rotation can also result in subluxation). Overall, ~2% of patients at risk for cord compression
3. If Hx/PE suggest cord compression—cancel elective surgery; otherwise treat with C-spine precautions
4. CNS—mental retardation, hearing loss
5. Cardiac—higher incidence of ASD, VSD, endocardial cushion defects, tetralogy of Fallot
6. GI—higher incidence of GERD, TEF, duodenal atresia, celiac disease
7. Other—hypothyroidism, Alzheimer disease

History

- Failure to thrive, developmental delay? Comorbidities?
- AAI—changes in gait, motor function, bowel or bladder function, syncope, behavior change?

Physical Exam

- Flattened face, small head, dysplastic ears, protruding tongue, narrow palate, short neck, epicanthal folds (skin of upper eyelid that covers inner corner of eye), Brushfield spots (small spots on periphery of iris), palmar crease
- Head/neck range of motion; tenderness?
- AAI—abnormal gait, weakness, spasticity, increased deep tendon reflexes (DTRs), positive Babinski, clonus of lower extremities
- Neuro—hypotonia; resp—stridor, wheezing
- Cardiac—see underlying cardiac condition

Lab Tests/Imaging

- Cervical x-rays

Consults

- Primary care, pediatrics, neurosurgery as required

Conflict(s)

- Cervical x-rays—in US screening with lateral x-rays for all patients once between age 3-5 years recommended. Not recommended in United Kingdom
- Perioperatively—consider cervical x-rays if new neurologic signs/symptoms, previous abnormal x-ray; or concern of difficult intubation or non-neutral neck position required for procedure

Optimize/Goals

- AAI—consider cervical spine x-rays
- Maintain neck in neutral position
- Avoid postintubation croup—often require smaller ETT than predicted by age; test for air leak
- Understand/optimize cardiopulmonary comorbidities: cardiac lesions, pulmonary hypertension, OSA

Options

- General, regional, local, MAC as required
- Patient's age and mental status may make general anesthesia preferable to sedation

Preop: Premed
Blood: surgery dependent
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Additional airway equipment pending airway examination

Induction

- Routine (consider ETT/RSI if GERD); minimize neck movement (consider LMA or FOI)
- Prepare smaller ETT re: tracheal stenosis

Maintenance

- Balanced technique; maintain neck in neutral position

Emergence

- Routine

Disposition/Pain

- May require inpatient setting
- Monitor postintubation croup

References

Hata T, Todd MM. Cervical spine considerations when anesthetizing patients with Down syndrome. *Anesthesiology*. 2005;102:680-685.
Meitzner MC, Skurnowicz JA. Anesthetic considerations for patients with Down syndrome. *AANA Journal.* 2005;73(2):103-107.

Duchenne Muscular Dystrophy

Considerations

1. DMD—most common childhood muscular dystrophy (~1 in 3500 male births). Onset ~3 to 5 years of age. (Becker muscular dystrophy is a milder dystrophinopathy)
2. Affects skeletal, cardiac, and smooth muscle (caused by lack of dystrophin)
3. Death due to respiratory failure
4. Cardiac—cardiomyopathy (often without signs/symptoms); smooth muscle—gastroparesis
5. Anesthetic risks: rhabdomyolysis and acute hyperkalemia
6. "Anesthesia-induced rhabdomyolysis"—inhalation agents and succinylcholine can cause rhabdomyolysis
7. MH—*not* at elevated risk of MH *but* rhabdomyolysis can make differential diagnosis challenging
8. Anesthetic: "trigger-free" anesthetic recommended to avoid inhalation agents and succinylcholine

History

- Progressive, worsening muscle weakness: frequent falls, fatigue, difficulty running, walking, dysphagia
- Mental retardation—(not present in all patients)

Physical Exam

- Body habitus: hypotonia, pseudohypertrophy of calf muscles, scoliosis; Gower sign: due to impaired leg muscles—characteristic way child uses arms to help stand upright
- Cardiac: arrhythmia (sinus tachycardia most common)
- Respiratory: pneumonia, aspiration? (Late in disease)

Lab Tests/Imaging

- Electrolytes (↓ K), CK; CXR—kyphoscoliosis, cardiomegaly; EKG; echo

Consults

- Primary care, pediatrics, neurology, cardiology as required

Conflict(s)

- Usually pediatric patients and want to avoid triggers of rhabdomyolysis—not candidates for inhalation induction

Optimize/Goals

- Optimize nutrition, cardiac function preoperatively
- Avoid aspiration; have noninvasive positive pressure ventilation (NPPV) prepared for high-risk patients postoperatively
- Avoid inhalation agents. Avoid succinylcholine
- Nondepolarizing muscle relaxants—more sensitive and longer duration
- Rhabdo/hyperkalemia treatment: calcium chloride, sodium bicarbonate, insulin/dextrose, hyperventilation, IV hydration, and mannitol to promote diuresis
- Dantrolene available if MH suspected

Options

- General, regional, PNB, MAC, local as appropriate (usually will require GA given age of patient)

Preop: Premed: GI prophylaxis
Blood: surgery dependent
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Flush anesthetic machine with 100% oxygen (consider booking as first case of day)
- Std monitors ± invasive monitors as required

Induction

- Avoid inhalation agents and succinylcholine

Maintenance

- TIVA—propofol, remifentanil; nondepolarizing muscle relaxant as required
- Monitor closely EKG signs of hyperkalemia; temperature, $ETCO_2$, rigidity, urine output.

Emergence

- Airway reflexes fully recovered, consider postoperative intubation/ventilation as required

Disposition/Pain

- Recovery room, stepdown, ICU as required; NPPV as required
- Chest PT as required

Clinical Pearl

Anesthetic risks of DMD include rhabdomyolysis and hyperkalemia. This is thought to be related to the underlying myopathy and not malignant hyperthermia (MH). Clinically to discriminate between rhabdomyolysis and MH: MH associated with hypermetabolism—rapidly rising end-tidal CO_2, unexplained metabolic acidosis, muscle rigidity, and temperature >38.8°C.

Reference

Hayes J, Veyckemans F, Bissonnette B. Duchenne muscular dystrophy: an old anesthesia problem revisited. *Pediatric Anesthesia.* 2008;18:100-106.

Eisenmenger Syndrome

Considerations

1. Usually unrepaired congenital cardiac shunt (left to right); eventually pulmonary hypertension leads to reversal of shunt (right to left) and cyanosis.
2. Cause: ASD, VSD, PDA? Any other noncardiac congenital abnormalities?
3. Complications: heart failure, pulmonary hypertension (mean PAP >25 mm Hg), chronic hypoxemia
4. Survival depends on RV function: avoid increased PVR, decreased SVR, hypoxemia, arrhythmia
5. Endocarditis prophylaxis? Required for unrepaired cyanotic heart disease undergoing certain dental, respiratory tract, and other procedures
6. Polycythemia/thrombotic events; reduced platelets and reduced platelet function
7. Paradoxical embolus (no air in IVs)
8. Higher risk of cholelithiasis/nephrolithiasis
9. May be candidates for heart-lung transplant or lung transplant with repair of cardiac lesion

History

- Underlying cardiac condition; other abnormalities?
- Dyspnea, orthopnea, fatigue, syncope, reduced exercise tolerance
- Myalgias, weakness, paresthesias, stroke (due to polycythemia)

Physical Exam

- Physical appearance—central cyanosis, clubbing, tachypnea
- RV heave, loud S2, S4, holosystolic murmur (tricuspid regurgitation), diastolic murmur (pulmonary insufficiency)

Lab Tests/Imaging

- CBC (increased Hct/Hgb), ABG (reduced $PaCO_2$ from tachypnea, reduced PaO_2 due to R-to-L shunt)
- CXR—RV and RA enlargement
- ECG—RVH—RAD, tall R wave V1
- Echo—position/size of defect, LV and RV size and function?

Consults

- Cardiology, cardiac surgery

Conflict(s)

- Goal is to maintain SVR—most anesthetics will decrease systemic BP and aggravate R-to-L shunt!

Optimize/Goals

- Maintain SVR and avoid decreases in SVR (increases R-to-L shunt)
- Avoid increases in PVR—avoid dehydration, pain, acidosis, hypoxia, hypercarbia, hypothermia, high PEEP, increased intrathoracic pressure
- CHF management—digoxin, diuretics
- Avoid air bubbles (paradoxical air embolus)
- Pulmonary vasodilator—consider prostacyclin, epoprostenol, bosentan, or sildenafil. Inhaled NO may be useful intraop

Options

- General, regional, local, MAC as required—regional may be preferred to avoid GETA

Preop: Premed: midazolam (0.5 mg/kg po)
Blood: surgery dependent
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter ± central line (monitor volume status!)
- Medications: inotropes (dobutamine, milrinone) as required

Induction

- Children: IV induction preferred over inhalation if CHF
- Nasal intubation may be preferred for postop

Maintenance

- Balanced technique

Emergence

- Controlled emergence; deep extubation as appropriate

Disposition/Pain

- ICU/CCU as required

Clinical Pearls

- It is critical to avoid dehydration in this patient population (increases blood viscosity and thrombotic complications).
- Pregnancy is contraindicated. Their high mortality is due to increased blood volume precipitating right heart failure, and pregnancy associated decreases in SVR that exacerbate the R-to-L shunt and increase hypoxemia.

Reference

Trojnarska O, Plaskota K. Therapeutic methods used in patients with Eisenmenger syndrome. *Cardiol J*. 2009;16 (6):500-506.

Epiglottitis and Croup

Considerations

1. Upper airway obstruction—potential airway emergency
2. Pediatric patients; although may occur in adults
3. Epiglottitis—minimal cough, sudden stridor, "toxic" appearance, dysphagia and drooling
4. Epiglottitis associated with *Haemophilus influenzae infections (or H. parainfluenzae,* Group A *Streptococcus,* pneumococci, staphylococci*)*—usually 3 months to 5 years of age
5. Croup—barky, nonproductive cough, hoarseness, gradual stridor, mild fever, minimal dysphagia, no drooling. Often improves with nebulized epinephrine and steroids
6. Croup associated with parainfluenza viruses—usually 6 months to 3 years of age
7. DDx—FB aspiration, epiglottitis, croup, tracheitis, tonsillitis, retropharyngeal abscesses, vascular rings, allergic reaction, laryngeal diphtheria

History

- Epiglottitis—fever, drooling, stridor (adults—slower onset, dysphagia and sore throat usually first)
- Croup—distinctive barky cough. Often mild and self-limiting; often preceded 24 to 48 hours by nonspecific URI symptoms

Physical Exam

- Physical appearance?
- Epiglottitis—dysphagia, drooling, high fever, anxious—sitting forward in "sniffing" position, stridor, "toxic"
- Croup—mild fever, hoarseness, stridor, not "toxic"

Lab Tests/Imaging

- Tests are not needed to confirm diagnosis in a patient with airway distress. Delays and manipulation may make situation worse
- Epiglottitis—CT or lateral neck x-rays— "thumb sign"
- Croup—AP x-ray—"steeple sign"

Consults

- ENT/surgeon experienced performing tracheostomy

Conflict(s)

- Pediatric patients will not tolerate stress of AFOI. Requires induction with spontaneous ventilation

Optimize/Goals

- Secure airway if respiratory distress—use most experienced personnel available
- Maintain spontaneous ventilation
- Croup:
  Steroids (dexamethasone 150-600 µg/kg po/IM/IV may reduce need for intubation); nebulized epinephrine (0.5 mL, 2.25% racemic epinephrine in 3 mL NS, or 5 mL of 1:1000 L-epinephrine); heliox (helium, oxygen mixture); if measures fail—secure airway
- Antipyretics for fever
- Epiglottitis—broad-spectrum antibiotics (beta-lactamase resistant)

Options

- Endotracheal intubation with inhalation induction
- Surgical backup for emergency tracheostomy

Preop: Premed
Blood
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Std monitors
- Difficult airway equipment; rigid bronchoscope (age appropriate); tracheostomy tray open

Induction

- Inhalation induction (sevoflurane)
- ETT—smaller sizes available

Maintenance

Emergence

- Transport to ICU intubated

Disposition/Pain

- Pediatric ICU

References

Bjornson CL, Johnson DW. Croup. *Lancet*. 2008;371:329-339.
Jenkins IA, Saunders M. Infections of the airway. *Pediatric Anesthesia*. 2009;19(S1):118-130.

Considerations

1. Emergency, urgent, or elective situation
2. Epilepsy—paroxysmal, recurring seizures
   Type: partial, generalized, pseudoseizures, or nonepileptic (alcohol/drug withdrawal, hypoglycemia, hyponatremia, stroke, hypoxia, eclampsia)
3. Present for nonepilepsy surgery, epilepsy surgery, or treatment of status epilepticus
4. Antiepileptic drugs—alter protein binding and/or enzyme induction which cause drug interactions
5. Anesthetics: may be pro- and/or anticonvulsant (generally proconvulsant at lower doses):
   Anticonvulsant—benzodiazepines
   Proconvultsant—opioids, sevoflurane
   Pro- and anticonvulsant: propofol, thiopental, isoflurane, etomidate, ketamine
6. Antiepileptic drugs (phenytoin, etc)—resistance to neuromuscular-blocking agents and opioids

History

- Symptoms, frequency, triggers
- Length of seizure (status epilepticus—>30 minutes continuous seizure activity)
- Comorbid psychiatric disorders, congenital syndromes
- Medications and compliance: phenytoin, benzodiazepines, phenobarbital , carbamazepine, lamotrigine, gabapentin, etc

Physical Exam

- Appearance? ABCs pending situation
- Neuro exam—mental status, gait, reflexes, sensory, motor
- Other trauma?

Lab Tests/Imaging

- Glucose
- LFTs, creatinine
- EEG, brain CT/MRI

Consults

- Primary care, neurology as required

Conflict(s)

- Anesthetics may cause epileptiform activity but rarely seizures. May have pro- and anticonvulsant effects.

Optimize/Goals

- Status epilepticus—emergency (time is brain)—ABCs and stop seizure
- Ensure antiepileptic medications continued periop
- Avoid drug interactions
- Consider EEG monitoring to detect seizures

Options

- Status epilepticus—may require GETA (both IV and inhalation agents successful)
- Functional epilepsy surgery (awake craniotomy)—local and sedation
- Other—general, regional, local, MAC as required

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Std monitors
- Antiepileptic drugs: midazolam, fosphenytoin/phenytoin

Induction

- Avoid etomidate, sevoflurane (>1.5 MAC)
- Status epilepticus:
  1) ABCs—manage airway (vomiting/secretions—RSI), support ventilation and hemodynamics (hypertension, hypotension, tachycardia, arrhythmias)
  2) Stop seizure as soon as possible: benzodiazepine, fosphenytoin or phenytoin, propofol, high MAC volatile anesthetic (isoflurane, desflurane), ketamine, or N_2O
  3) Treat acidosis, hyperkalemia, hyper/hypoglycemia

Maintenance

- Balanced technique or TIVA

Emergence

- Routine

Disposition/Pain

- Outpatient, inpatient, ICU as required

References

Kofke WA. Anesthetic management of the patient with epilepsy and prior seizures. *Curr Opin Anaesthesiol*. 2010;23:391-399.
Voss LJ, Sleigh JW, Barnard JPM, et al. The howling cortex: seizures and general anesthetic drugs. *Anesth Analg*. 2008;107(5)1689-1703.

Foreign Body Airway Obstruction

Considerations

1. Pediatric patient: tendency toward rapid desaturation, bradycardia, heat loss
2. Potential for sudden decline in respiratory status
3. Full stomach
4. Shared airway
5. High rate of morbidity and mortality

History

- Witnessed choking/gagging by parent
- Respiratory distress, dyspnea, recurrent pneumonia
- H/o "funny voice"
- ↓ LOC

Physical Exam

- Stridor, wheezing, coughing, ↓ SpO_2
- Fever, rales, ↓ breath sounds unilaterally
- Unilateral hyperinflation with tracheal deviation if check-valve effect in bronchi with mediastinal shift

Lab Tests/Imaging

- CXR (NB: most FBs are organic → radiolucent)
- L&R lateral decub CXRs to look for absence of gravitational mediastinal shift → suggests bronchial obstruction with hyperinflation
- Spiral CT if diagnosis in doubt and stable

Consults

Conflict(s)

- Goal of spontaneous ventilation versus 1) full stomach and 2) requirement for profound depth of anesthesia

Optimize

- If in distress, initiate first aid for choking (see Clinical Pearls section)
- Base urgency of procedure on degree of respiratory distress; if stable and FB chronically, consider waiting several hours to ↓ risk of gastric aspiration

Goals

- If in distress, spontaneous respiration with avoidance of paralysis
- Avoid pushing FB deeper with PPV (except if complete obstruction at carina → can be resolved by pushing FB into mainstem bronchus)
- Calm, smooth induction → may require sitting position

Options

- General anesthesia unless moribund (no anesthesia)
- If stable and no distress, consider IV induction (with paralysis) for optimal endoscopic conditions

Preop: Premed: atropine (↓ secretions & bradycardia)
Blood
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Jet ventilator, peds cricothyrotomy & trach set, rigid bronchoscope, skilled endoscopist gowned/gloved

Induction

- If time, topicalization of airway (lidocaine 4% by nebs)
- Sevoflurane in 100% O_2
- May be slow due to alveolar hypoventilation
- If full stomach and resp distress → RSI with ETT

Maintenance

- No muscle relaxants
- Periodic ventilation by anesthesiologist
- TIVA acceptable but watch apnea

Emergence

Disposition/Pain

- Depending on degree of edema and respiratory insult, may require period of mechanical ventilation

Clinical Pearls

First aid for foreign body in airway (FBAO):

- If **mild** (child coughing and making sounds), do not interfere. Allow victim to clear airway by coughing while observing for signs of severe FBAO
- If **severe** (victim unable to make sound):
  - For a child, perform subdiaphragmatic abdominal thrusts (Heimlich maneuver) until object expelled or victim becomes unresponsive
  - For infant, deliver 5 back blows (slaps) followed by 5 chest thrusts repeatedly until object expelled or victim becomes unresponsive
  - If victim becomes unresponsive, look in mouth for foreign body and remove **if visible**. Do not perform a blind finger sweep (may push object further in pharynx). Initiate ventilation and follow with chest compressions.

References

American Heart Association 2005. American Heart Association (AHA) guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiovascular care (ECC) of pediatric and neonatal patients: pediatric basic life support. *Pediatrics.* 2006;117:e989-e1004.

Zur KB, Litman RS. Pediatric airway foreign body retrieval: surgical and anesthetic perspectives. *Pediatr Anesth.* 2009;19:109-117.

Considerations

1. Elective surgery—usually positive outcomes
2. Emergency surgery—higher risk of M&M
3. Advanced age (>65-85) may be an independent risk factor for poor outcome (controversial as age may serve as marker of comorbid conditions)
4. Age-related changes: neuro—grey/white matter loss, dementia, deafness/blindness, impaired balance
   CV–LVH, diastolic dysfunction, blunted HR response
   Resp—muscle atrophy, chest wall stiffness, reduced response to hypoxia/hypercapnia
   Renal—reduced RBF/GFR, asymptomatic UTI
   GI—decreased GI motility, GERD, constipation
   MSK—reduced muscle, osteoporosis, fracture risk
   Skin—atrophy, bruising, pressure sores
5. Postoperative cognitive dysfunction (POCD)—risks: preop cognitive impairment, blood loss, sleep deprivation, infection, hyponatremia, drug withdrawal, pain, opioids/benzodiazepines

History

- Routine—focus on functional status, comorbidities
- Previous surgery/anesthetics—complications, POCD?
- Medications—compliance? Polypharmacy?

Physical Exam

- Appearance, vital signs
- Mental status
- Orthostatic vitals

Lab Tests/Imaging

- Determined by disease and procedure—not age
- Exception—age >55—creatinine

Consults

- Primary care, geriatrics, subspecialty as required

Conflict(s)

- Anesthesia/surgery may result in POCD—weigh risk/benefit of procedure

Optimize/Goals

- Maintain body temperature
- Avoid MSK injury; proper positioning
- Fluid therapy—maintain preload (invasive monitors?)
- Avoid hyponatremia and electrolyte alterations
- POCD—treat reversible causes, optimize environment, and adequate analgesia
  Treatment—antipsychotics (haloperidol 0.5-1.0 mg IV), physical restraints as temporary measure for severe cases

Options

- GA, regional, local, PNB, MAC as required
- Consider PNB as appropriate; regional anesthesia may or may not be protective re: POCD
- In certain situations dexmedetomidine or low-dose ketamine may reduce incidence of POCD

Preop: Premed
Blood: surgery dependent
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Std monitors ± additional as required

Induction

- Avoid opioids/benzodiazepines as appropriate

Maintenance

- Balanced

Emergence

- Routine

Disposition/Pain

- PACU, stepdown, ICU as required. Monitor/treat postoperative cognitive dysfunction

References

Deiner S, Silverstein JH. Postoperative delirium and cognitive dysfunction. *Br J Anaesth*. 2009;103:i41-i46.
Preston SD, Southall ARD, Nel M, et al. Geriatric surgery is about disease, not age. *J R Soc Med*. 2008;101:409-415.

Guillain Barré Syndrome

Considerations

1. May present for management of respiratory failure or for emergency/urgent procedures
2. Acute flaccid ascending paralysis, often after URI or gastroenteritis (or vaccination?)
3. Usually resolved weeks/months
4. Respiratory failure may require intubation/ventilation
5. Autonomic disturbance—arrhythmias, hypertension, ileus, urinary retention
6. DDx—Peripheral neuropathy (critical illness/diabetic neuropathy), NMJ disorder (myasthenia gravis), muscle disorder (periodic paralysis, infections), CNS disorder (stroke, encephalitis)
7. Fatigue is common during recovery. May require physical therapy
8. Treatment—plasmapheresis/IVIG (oral corticosteroids generally not effective)
9. Avoid succinylcholine re: hyperkalemia!

History

- Routine
- Focused neurologic history—symptoms, severity, onset, duration
- Dyspnea, fatigue, respiratory distress?

Physical Exam

- Appearance? Respiratory distress? ACLS as required
- Complete neurologic exam—cranial nerves, motor, sensory, autonomic, reflexes
- Generalized muscle weakness, ascending from lower to upper limbs
- Respiratory weakness
- Other—cardiac rate/rhythm, hypertension

Lab Tests/Imaging

- Nerve conduction studies/EMG, CSF analysis

Consults

- Neurology, ICU as required

Conflict(s)

- Regional anesthesia may aggravate symptoms of GBS (pro and con case reports). Risk/benefit must be evaluated and discussed for given patient

Optimize/Goals

- Supportive care:
  Monitor/control HR/BP/cardiac rhythm
  Mechanical ventilation—typically VC <15 to 20 cc/kg
- DVT prophylaxis as required
- Foley catheter for urinary retention
- Nutritional support

Options

- Risks with regional or GA; avoid PNB (multihit hypothesis of nerve injury)

Preop: Premed
Blood: surgery dependent
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Std monitors, Foley catheter ± invasive monitors as required

Induction

- May require intubation/ventilation for respiratory failure
- Avoid succinylcholine re: hyperkalemia

Maintenance

- Nondepolarizing muscle relaxants—may have prolonged effect

Emergence

- May require prolonged postop intub/vent (tracheostomy?)

Disposition/Pain

- ICU, neuro-ICU as required

References

Kocabas S, Karaman S, Firat V, et al. Anesthetic management of Guillain-Barre syndrome in pregnancy. *J Clin Anesth*. 2007;19:299-302.
Vucic S, Kiernan MC, Cornblath DR. Guillain-Barre syndrome: an update. *J Clin Neurosci*. 2009;16:733-741.

Hemophilia

Considerations

1. Hemopilia A and B—X-linked recessive disorders caused by deficiencies of factor VIII and factor IX
2. Spontaneous hematomas—usually joints but may also affect airway or CNS requiring urgent care
3. Many hemophiliacs develop inhibitors (IgG Abs) that neutralize infused clotting factors. These patients require more complex treatment and are more likely to have perioperative bleeds
4. Comorbidity—HIV, hepatitis C (from contaminated pooled factor concentrates) chronic pain, arthropathy, osteoporosis, hypertension, reduced quality of life
5. DDx for prolonged aPTT—factor deficiencies or antibodies (VIII, IX, XI, XII, or fibrinogen), vWF, lupus anticoagulants, or medications (heparin, activated protein C)
6. Do not perform elective surgical procedures if abnormal coagulation studies

History

- Bleeding: hematomas, hemarthroses, major bleeding with surgery
- Family history
- Comorbidities (HIV, hepatitis C, chronic pain, arthropathy, osteoporosis, hypertension, psychosocial)
- Treatments, presence of inhibitors?

Physical Exam

- Routine and as guided by history

Lab Tests/Imaging

- Hemophilia A—normal PT, normal bleeding time, prolonged aPTT; assay for factor VIII coagulant activity
- Hemophilia B—normal PT, normal bleeding time, prolonged aPTT
- CBC

Consults

- Hematology

Conflict(s)

- Hemophilia A and B require factor replacement before surgery but for mild, undiagnosed cases the first sign of hemophilia may be excessive bleeding perioperatively

Optimize/Goals

- Hemophilia A—recombinant factor VIII concentrates (reduce risk of viral transmission). Older treatments: factor VIII replacement with concentrates, FFP (large volume required), or cryoprecipitate. Other: DDAVP 0.3 μg/kg IV, aminocaproic acid. Hemophilia B—recombinant factor IX concentrates (reduce risk of viral transmission). Older treatment: factor IX replacement with concentrates.
- Avoid aspirin and NSAIDs; avoid IM injections

Options

- General anesthesia, MAC, local as required
- Regional or PNB—usually avoided—require factor replacement and close monitoring of factor levels

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter ± invasive monitors as required
- Hematology—consult re: factor replacement preoperatively (can give as IV bolus and IV infusion)
- If regional or PNB—confirm factor levels preprocedure

Induction

- Care with intubation, avoid nasal intubation as permitted re: bleeding

Maintenance

- Careful positioning re: hematomas/hemarthroses

Emergence

- Routine

Disposition/Pain

- Ambulatory, floor, stepdown, ICU as required

References

Lee JW. von Willebrand disease. Hemophilia A and B, and other factor deficiencies. *Int Anesthesiol Clin*. 2004;42(3):59-76.

Mauser-Bunschoten EP, Fransen Van de Putte DE, Schutgens REG. Co-morbidity in the ageing haemophilia patients: the down side of increased life expectancy. *Haemophilia*. 2009;15:853-863.

Considerations

1. HIV virus infects helper T cells and their loss results in opportunistic infections and neoplasms
2. Multisystem disease ranging from *asymptomatic* to *critically ill*
3. Complications as a result of:
   Direct effect of HIV infection
   Opportunistic infection/neoplasm
   Side effects of medications
4. Airway—dysphagia, infections (bronchitis, sinusitis, pneumonia), or obstruction (Kaposi sarcoma)
   Neuro—encephalopathy, dementia, neuropathy
   CVS—acute coronary syndromes, dilated cardiomyopathy, pericardial effusion, endocarditis
   GI—diarrhea, fatty liver, pancreatitis
   Renal—acute or chronic kidney disease
   Hem—anemia, neutropenia, thrombocytopenia
   Endocrine—lipodystrophy, DM, SIADH, hyper- or hypothyroidism

History

- Focus on complications related to disease
- Other infections—hepatitis C, TB?
- IV drug use?
- Medications—antiretroviral therapy: reverse transcriptase inhibitors, protease inhibitors, integrase inhibitors, and entry inhibitors

Physical Exam

- Vital signs, mental status
- Neurologic deficits?
- Targeted exam based on history

Lab Tests/Imaging

- CD4 count, viral load
- CBC, coagulation, electrolytes, creatinine, LFTs
- CXR, ECG (echocardiogram as indicated)

Consults

- Primary care, infectious disease, subspecialty as required

Conflict(s)

- Varied medical optimization—may present for urgent or emergent surgery without adequate preoperative management

Optimize/Goals

- Universal precautions (if occupational exposure, start postexposure prophylaxis)
- Avoid further infection in immunocompromised patient with strict aseptic technique as indicated
- Avoid drug interactions—antiretrovirals may prolong effect of fentanyl and midazolam (cytochrome P450 inhibition)
- Avoid acute renal/liver injury—adequate hydration
- Continue antiretroviral therapy through perioperative period (may require G-tube)

Options

- GA, regional, local, PNB, MAC as required
- Regional techniques: concern re: CNS infection or neoplasm, and coagulopathy

Preop: Premed
Blood: surgery dependent
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Std monitors ± additional as required

Induction

- Routine
- Regional: consider alternative techniques if advanced disease, neurologic symptoms present, or coagulopathy

Maintenance

- Balanced
- Consider possible prolonged effect of fentanyl

Emergence

- Routine

Disposition/Pain

- PACU, stepdown, ICU as required

Reference

Leelanukrom R. Anaesthetic considerations of the HIV-infected patients. *Curr Opin Anaesthesiol*. 2009;22:412-418.

Hypertension

Considerations

1. Presence of end-organ damage
   - CAD, CHF, CVA, renal doze, PVD
2. ↑ Lability of intraop BP (hypotension or hypertension)
3. Potential for secondary causes: renovascular, Cushing syndrome, hyperaldosteronism, pheochromocytoma, ↑ ICP
4. Altered cerebral autoregulation curve
5. Drug effects:
   - hypokalemia with diuretics
   - platelet inhibition with calcium channel blockers

History

- H/o complications: stroke/TIA, MI, renal failure, PVD
- Headache

Physical Exam

- LV heave, S4 (LVH)
- S3 (CHF)
- AV nicking on fundoscopy

Lab Tests/Imaging

- Electrolytes, BUN, Cr
- EKG (ST changes, left axis deviation, LVH/strain)

Consults

Conflict(s)

Optimize

- If on antihypertensives, continue until time of surgery, especially beta blockers. ACE inhibitors and ARBs may predispose to hypotension if BP already low (consider holding on morning of surgery)
- Patients with mild-mod HTN can proceed to surgery without delay or escalation in therapy; in *elective* surgery in patients with preop diastolic BP > 110 mm Hg, consider delay with 6 to 8 weeks of slow BP lowering

Goals

- Preop BP <170/100 mm Hg
- Maintain BP within 20% of preop range

Options

- Regional anesthesia avoids hypertensive response to laryngoscopy/extubation, *but* careful if volume contracted

Preop: Premed: anxiolytic
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Esmolol to blunt hypertensive response to laryngoscopy
- ± Arterial line if very labile

Induction

- Slow, titrated. Ensure deep level before laryngoscopy

Maintenance

Emergence

- Deep extubation often useful if no contraindication to avoid hypertensive response to extubation

Disposition/Pain

- Treat postop hypertension with same class of agent that patient is already on (eg, IV enalaprilat if on ACE inhibitor or SL nifedipine if on amlodipine). This is especially vital for beta blockers and clonidine → rebound tachycardia and hypertension

Reference

Fleisher LA. Peroperative evaluation of the patient with hypertension. *JAMA*. 2002;287:2043-2046.

Hyperthyroidism

Considerations

1. Excess thyroid hormone caused by Graves disease (autoimmune—also causes ophthalmopathy and dermopathy), toxic multinodular goiter, thyroiditis, pituitary adenoma, or excess thyroid hormone intake
2. Graves disease treatments—antithyroid drugs, radioiodine, or surgery
3. Effects: fatigue, anxiety, weight loss, heat insensitivity, palpitations, myopathy, osteoporosis, women—irregular menses
4. Thyroid storm (tachycardia, fever, altered mental status)—metabolic crisis that can be triggered by trauma, infection, or surgery (DDx includes MH)
5. May present for thyroid or nonthyroid surgery
6. Controlled hyperthyroidism—minimal anesthetic risk
7. Uncontrolled hyperthyroidism—increased anesthetic risk—avoid nonemergent surgery
8. Thyroid surgery—bleeding, airway obstruction, nerve injury

History

- Symptoms: hyperactivity, weight loss, tremor, dyspnea, palpitations—any changes recently?
- Associated endocrine disease? (Pheochromocytoma?)
- Medications—antithyroid: methimazole, carbimazole or propylthiouracil; beta blockers (propranolol)

Physical Exam

- Tachycardia, warm skin, tremors, exophthalmos (Graves)
- Enlarged thyroid/goiter—stridor, airway obstruction?
- CVS—irregular heart rate (AFIB), CHF—elevated JVP, edema, S3, murmur?

Lab Tests/Imaging

- Free thyroxine (T4), triiodothyroxine (T3), TSH
- CBC (anemia, thrombocytopenia)
- CXR (tracheal compression, retrosternal goiter?), CT/MRI as required
- EKG

Consults

- Endocrine

Conflict(s)

- Goiter—potential for difficult airway but also potential for difficult tracheostomy

Optimize/Goals

- Continue medical therapy for hyperthyroidism
- Airway—prepare for potentially difficult airway (AFOI)
- Avoid corneal abrasion in patients with Graves and exophthalmos eye protection
- Avoid sympathetic stimulation—beta blockers, maintain deep anesthetic
- Thyroid storm—fluid resuscitation, cooling, support hemodynamics, electrolyte replacement, steroids (if adrenal insufficiency), antithyroid medications, beta blockers (propranolol)

Options

- General, regional, local, peripheral nerve block, or sedation as required
- Thyroid surgery—general or PNB (cervical plexus). May require sternotomy for retrosternal goiter

Preop: Premed
Blood: surgery dependent
ICU/stepdown bed: possibly

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter ± invasive monitors
- Difficult airway equipment as required; beta blockers

Induction

- AFOI as indicated

Maintenance

- Balanced technique (may require deep anesthetic), monitor temperature

Emergence

- Beware postop hematoma, nerve injury (stridor), tracheomalacia—controlled, awake extubation

Disposition/Pain

- PACU, stepdown, ICU as required
- Post-thyroidectomy risks—hypoparathyroidism

Clinical Pearl

Maintain a high degree of suspicion when assessing the airway in a patient with a goiter—may have difficult intubation even if otherwise normal airway exam. Often the surgeon will document preoperative vocal cord function with the use of indirect laryngoscopy. The results of this exam can be very useful to assess the need for AFOI.

References

Cooper DS. Hyperthyroidism. *Lancet.* 2003;362:459-468.
Farling PA. Thyroid disease. *Br J Anaesth.* 2000;85(1):15-28.

Hypothyroidism

Considerations

1. Deficiency of thyroid hormone caused by iodine deficiency, congenital defect, autoimmune thyroiditis (Hashimoto), acquired deficiency due to thyroid surgery/radiation, drugs (amiodarone, lithium), or CNS tumors
2. Effects: altered mental state, cold intolerance, weight gain, dry skin, bradycardia, constipation, anemia
3. Controlled hypothyroidism—minimal anesthetic risk
4. Severe hypothyroidism (or myxedema coma)—avoid anesthesia unless emergency—complicated by coma, seizure, CHF, respiratory failure, hypothermia, hyponatremia, hypoglycemia, ileus, adrenal insufficiency, and coagulopathy
5. Management—thyroid hormone replacement (treatment may exacerbate myocardial ischemia in at-risk patients)
6. May be sensitive to sedatives and analgesics
7. May present for nonthyroid or thyroid surgery

History

- Symptoms: cold intolerance, fatigue, weakness, weight gain—any changes recently?
- Obstructive sleep apnea?
- Medications—levothyroxine, T3?

Physical Exam

- Airway—goiter? (usually improves with thyroxine replacement)
- Hypotension, bradycardia; dry, cool skin; coarse hair
- Muscle weakness, decreased tendon reflexes

Lab Tests/Imaging

- Free thyroxine (T4), triiodothyroxine (T3), TSH (elevated in primary hypothyroidism)
- CBC, electrolytes, PT/PTT/INR, glucose
- EKG (low voltage, prolonged P-R, QRS, Q-T intervals) echocardiogram (pericardial effusion, decreased systolic/diastolic function)

Consults

- Endocrine

Conflict(s)

- Myxedema coma can be precipitated by cold environment and medications including opioids and sedatives

Optimize/Goals

- Avoid elective procedures in symptomatic hypothyroid patients. Controversy whether delay for treatment improves outcome for patients with "mild" hypothyroidism ("mild" based on clinical or lab evidence)
- Continue medical therapy for hypothyroidism
- Potentially difficult airway? Consider AFOI
- Avoid hypothermia
- Avoid excess fluid (prone to CHF)—invasive monitors as required
- Myxedema coma—levothyroxine 500 μg IV, hydrocortisone 100 mg IV

Options

- General, regional, local, peripheral nerve block, or sedation as required
- Thyroid surgery—general or PNB (cervical plexus)

Preop: Premed: GI prophylaxis. **Avoid preop sedation**
Blood: surgery dependent
ICU/stepdown bed: possibly

Room Setup (special drugs/monitors)

Std monitors ± Foley catheter ± invasive monitors
Difficult airway equipment as required

Induction

May require AFOI, consider lower doses of anesthetic agents (postinduction hypotension)

Maintenance

Balanced technique, controlled ventilation, normothermia

Emergence

Controlled, awake extubation or keep intubated as required

Disposition/Pain

- Outpatient, inpatient, ICU as required

Clinical Pearl

In critically ill patients, the use of glucocorticoids, dopamine, or dobutamine can suppress TSH levels leading to false-positive results.

References

Farling PA. Thyroid disease. *Br J Anaesth*. 2000;85(1):15-28.
Roberts CGP, Ladenson PW. Hypothyroidism. *Lancet*. 2004;363:793-803.

Liver Cirrhosis

Considerations

1. Significant mortality for nontransplant procedures
2. Causes: autoimmune, alcohol, hepatitis B/C, obesity, cryptogenic, primary biliary cirrhosis, inherited (Wilson disease), drugs (acetaminophen)
3. Risk stratification: Child-Pugh classification. Considers albumin, PT, bilirubin, ascites, encephalopathy. Avoid elective surgery in class C

   MELD (model for end-stage liver disease) also used to stratify. Considers bilirubin, creatinine, INR
4. Highest-risk surgery: cardiac and open abdominal surgeries (laparotomy reduces liver blood flow)
5. Multisystem disease: encephalopathy, cardiac: increased CO, decreased SVR; pulmonary: hepatopulmonary syndrome (shunting), portopulmonary hypertension; ascites, renal failure (prerenal, hepatorenal), malnutrition, coagulopathy, anemia, metabolic—hyponatremia

History

- Risk factors: blood transfusions, tattoos, illicit drugs use, sexual promiscuity, FH, alcoholism, travel history, medications
- PH—ascites, edema, encephalopathy, variceal bleeding (varices treated with propranolol therapy or TIPS)
- Encephalopathy—who is responsible for consent?

Physical Exam

- Condition—cachexia, ascites, encephalopathy—LOC?
- Stigmata of liver disease: jaundice, palmar erythema, spider nevi, gynecomastia, testicular atrophy; portal hypertension—splenomegaly, ascites, asterixis
- Hepatopulmonary syndrome—orthodeoxia, platypnea (desaturation and dyspnea when *upright* vs supine)

Lab Tests/Imaging

- CBC, coags, electrolytes, glucose, LFTs, CXR, ECG, echo

Consults

- Hepatology as required

Conflict(s)

- Anesthetics/surgery further reduce hepatic blood flow and risk exacerbating underlying condition

Optimize/Goals

- Avoid further decrease in hepatic blood flow; avoid hypotension, hypoxemia, hemorrhage.
- Maintain volume (albumin) and electrolytes (hyponatremia—avoid rapid reversal re: CPM!)
- Reverse coagulopathy (FFP, platelets, cryo) as required
- Manage ascites—diuretic therapy, paracentesis as required
- Avoid renal failure—avoid NSAIDs, maintain preload
- Encephalopathy—caution with sedatives/analgesics, lactulose as required (reduces ammonia absorption)

Options

- Typically GETA; sedation may be contraindicated if altered mental status and/or massive ascites; regional/PNB often contraindicated due to coagulopathy

Preop: Premed: GI prophylaxis
Blood: surgery dependent
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Std monitors, Foley catheter, large bore IV; arterial line, central line as required; may require norepinephrine, vasopressin to maintain MAP

Induction

- GETA; RSI if ascites; propofol and succinylcholine

Maintenance

- Balanced technique (isoflurane for preserving hepatic blood flow), cisatracurium avoids hepatic metabolism
- Fentanyl better than morphine (liver metabolism)

Emergence

- May require postop intub/vent; fully awake for extub

Disposition/Pain

- PACU, stepdown, ICU—beware worsening ascites, encephalopathy, renal failure, liver failure, coagulopathy, wound infection, pneumonia, bleeding, sepsis!

Reference

Millwala F, Nguyen GC, Thuluvath PJ. Outcomes of patients with cirrhosis undergoing non-hepatic surgery: risk assessment and management. *World J Gastroenterol*. 2007;13(30):4056-4063.

Marfan Syndrome

Considerations

1. Connective tissue disorder (fibrillin-1 deficiency):
   Cardiovascular—MVP or mitral regurgitation, aortic regurgitation, dilated ascending aorta, aortic dissection, dilated pulmonary artery
   Skeletal—tall stature (long bone growth), pectus carinatum/excavatum, scoliosis, joint hypermobility
   Ocular—lens dislocation, retinal detachment, glaucoma
   Pulmonary—spontaneous pneumothorax
   Dural ectasia—widening of the dural sac—asymptomatic or may present with lumbar back pain. May result in failed spinal anesthesia due to dilution of anesthetic.
2. Medical management—beta blockers to delay aortic aneurysm/dissection
3. Surgery for aortic aneurysm—aortic root repair (± aortic valve)
4. May present emergently with aortic dissection

History

- FH—inherited as dominant trait (many de-novo cases due to mutations), comorbidities, functional status, medications—beta blocker?
- Aortic dissection—acute chest pain (tearing, stabbing) radiates to back, dyspnea, anxiety

Physical Exam

- Airway—high-arched palate, retrognathia, crowded teeth
- CVS—MVP/MR/AR—heart murmur
- Skeletal—long limbs, anterior chest deformity, scoliosis
- Aortic dissection—tachycardia, hypertension or hypotension, interarm variation in BP, altered level of consciousness, weakness/paralysis

Lab Tests/Imaging

- EKG—SVT, prolonged QT, echocardiogram (measurements of proximal aorta), CXR, PFTs

Consults

- Medical genetics, ophthalmology, orthopedic surgery, cardiology, cardiac surgery as required

Conflict(s)

Optimize/Goals

- Minimize aortic root shear forces—control both HR and BP—adequate analgesia/anesthesia, beta blockers

Options

- General, regional, local, peripheral nerve block, or sedation as required
- Regional—may be difficult in patient with scoliosis, and reports of failed spinal anesthesia due to dural ectasia

Preop: Premed
Blood: surgery dependent
ICU/stepdown bed: possibly

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter ± invasive monitors
- TEE as required
- Beta blockers, vasodilators, vasopressors

Induction

- Controlled, avoid hypertension/tachycardia with intubation

Maintenance

- Balanced technique, close hemodynamic monitoring
- TEE as required

Emergence

- Controlled, avoid hypertension/tachycardia with intubation

Disposition/Pain

- PACU, stepdown, ICU as required

References

Judge DP, Dietz HC. Marfan's syndrome. *Lancet*. 2005;366:1965-1976.

Singh SI, Brooks C, Dobkowski W. General anesthesia using remifentanil for cesarean delivery in a parturient with Marfan's syndrome. *Can J Anesth.* 2008;55(8):526-531.

Mitral Regurgitation

Considerations

1. Valvular/cardiac surgery or noncardiac surgery?
2. Etiology:

   Organic—permanent changes to MV leaflets—endocarditis, mitral valve prolapse (myxomatous degeneration), rheumatic disease, Marfan syndrome
   Functional—change in interaction of structures of MV—ischemic heart disease, cardiomyopathy
3. Pathophysiology:

   Acute MR—regurgitant volume—rise in left atrial pressure, increased pulmonary venous pressure, acute pulmonary edema
   Chronic MR—LV volume overload—eccentric hypertrophy, dilated LA, LV systolic dysfunction (underestimated by EF due to compliant LA)
4. Treatment—"organic" causes ultimately require surgery, "functional" causes may improve with treatment of underlying process
5. Severe MR—regurgitant fraction >0.6

History

- Acute MR—chest pain, dyspnea, orthopnea
- Chronic MR—asymptomatic for years; then fatigue, dyspnea
- Atrial fibrillation—anticoagulants?

Physical Exam

- Chronic MR—holosystolic murmur, irregular pulse—atrial fibrillation
- Acute MR—systolic soft, decrescendo murmur, S3, cardiogenic shock
- Crackles/rales—pulmonary edema

Lab Tests/Imaging

- EKG—atrial fibrillation, signs of LVH; CXR—cardiomegaly; echocardiography—severity

Consults

- Cardiology, cardiothoracic surgery as required

Conflict(s)

- Acute MR—emergency. Avoid nonemergency procedures

Optimize/Goals

- Medical therapy—continue antihypertensive therapy, vasodilators, treat CHF
- MR and noncardiac surgery:
  Maintain high HR
  Low afterload
  Increased preload
  Normal/increased contractility
- Acute MR—afterload reduction, diuretics, inotropic support, cardiac surgery
- Atrial fibrillation—beta blockers, calcium channel blockers, digoxin, amiodarone
- Infective endocarditis antibiotic prophylaxis: prosthetic valve, PH endocarditis, congenital HD, cardiac transplant

Options

- General, regional, neuraxial, local, MAC

Preop: Premed: IE prophylaxis?
Blood: surgery dependent
ICU/stepdown bed: surgery dependent

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter, arterial monitor, CVP monitor, PAC, TEE as required
- For hypotension—inotropes: ephedrine, epinephrine

Induction

- Maintain cardiovascular goals

Maintenance

- Balanced technique

Emergence

- Ensure hemodynamic goals maintained; assess for CHF

Disposition/Pain

- PACU, stepdown, ICU as required; monitor for symptoms of CHF

References

Mittnacht, AJC, Fanshawe M, Konstadt S. Anesthetic considerations in the patient with valvular heart disease undergoing noncardiac surgery. *Semin Cardiothorac Vasc Anesth*. 2008;12:33-59.

Stout KK, Verrier ED. Acute valvular regurgitation. *Circulation.* 2009;119:3232-3241.

Mitral Stenosis

Considerations

1. Valvular/cardiac surgery or noncardiac surgery?
2. Elective, urgent, or emergency?
3. Infective endocarditis antibiotic prophylaxis: prosthetic valve, PH endocarditis, congenital HD, cardiac transplant?
4. Etiology: acquired: rheumatic heart disease, infective endocarditis, SLE, RA; or congenital abnormalities
5. Normal MV area is 4 to 5 cm^2, gradient 2 to 4 mm Hg
   "Severe" MS–MV area <1 cm^2, gradient >15 mm Hg
   As valve area decreases, chronic pressure gradient leads to elevated LA pressures, pulmonary edema, pulmonary hypertension, and right heart failure. Reduced flow limits LV output
6. Atrial fibrillation—rate control and anticoagulation
7. Procedures—MV repair/replacement or balloon valvotomy (consider intervention once patient is symptomatic); often present for noncardiac surgery

History

- Mild/mod MS—asymptomatic for years
- Palpitations (atrial fibrillation), exercise tolerance?
- Severe MS—angina, syncope, dyspnea, fatigue, palpitations
- Medications—HR control, anticoagulation

Physical Exam

- Facial (malar) flushing, irregular pulse (atrial fibrillation)
- Reduced pulse pressure, elevated JVP, increased S1, opening snap, diastolic murmur (holodiastolic, decrescendo)—best heard left lateral decubitus position
- RV heave, increased P2, hepatomegaly, ascites (PHTN)
- Crackles/rales—pulmonary edema

Lab Tests/Imaging

- EKG—large P wave (LA enlargement), RAD/RVH, AFIB? CXR—pulmonary edema; echocardiography—severity, LA thrombus? Exercise or pharmacologic stress test—ischemic disease; cardiac catheterization as required

Consults

- Cardiology, cardiothoracic surgery as required

Conflict(s)

Optimize/Goals

- MS and noncardiac surgery:
  Maintain sinus rhythm—atrial kick important to maintain left ventricular filling
  Normal/decreased HR (avoid tachycardia)
  Normal/increased preload (avoid pulmonary edema)
  Increased afterload
  Normal contractility
- Pulmonary hypertension—avoid hypercarbia, acidosis, hypothermia; may require pulmonary vasodilators
- Avoid hypotension (decreased coronary perfusion)
- Periop anticoagulation—hold Coumadin, initiate heparin

Options

- General, regional, local, MAC as required
- Neuraxial—avoid hypotension/sympathectomy, contraindicated if ongoing anticoagulation

Preop: Premed: IE prophylaxis?
Blood: surgery dependent
ICU/stepdown bed: surgery dependent

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter, arterial monitor, CVP monitor, PAC (beware arrhythmias!), TEE as required
- Pulmonary hypertension—pulmonary vasodilators (NO)

Induction

- Maintain cardiovascular goals; consider arterial line or PAC placement preinduction

Maintenance

- Balanced technique; alpha agonists (phenylephrine) first choice to maintain BP

Emergence

- Ensure hemodynamic goals maintained

Disposition/Pain

- PACU, stepdown, ICU as required

Clinical Pearl

MS is two to three times more common in women. Often becomes symptomatic during pregnancy (exacerbated by physiologic changes)—may require balloon mitral valvotomy.

References

Carabello BA. Modern management of mitral stenosis. *Circulation*. 2005;112:432-437.

Mittnacht, AJC, Fanshawe M, Konstadt S. Anesthetic considerations in the patient with valvular heart disease undergoing noncardiac surgery. *Semin Cardiothorac Vasc Anesth*. 2008;12:33-59.

Mitral Valve Prolapse

Considerations

1. Valvular/cardiac surgery or noncardiac surgery?
2. Etiology: myxomatous degeneration most common. Other cardiac—HOCM, WPW, rheumatic disease, myocarditis; connective tissue diseases—Marfan syndrome, Ehlers-Danlos syndrome, scleroderma Other—polycystic kidney disease, von Willebrand disease, SLE, Graves disease, acromegaly
3. Pathophysiology:
   Displacement of mitral leaflets into the left atrium during systole. May progress to mitral valve regurgitation.
   Chronic MR—LV volume overload—eccentric hypertrophy, dilated LA, LV systolic dysfunction (underestimated by EF due to compliant LA)
4. Symptoms—most asymptomatic; or angina, dyspnea (pulmonary edema), palpitations, fatigue
5. Complications: MR, arrhythmia (SVT, PVCs, VT), emboli (stroke), infective endocarditis, sudden death

History

- Asymptomatic or angina, palpitations, fatigue, dyspnea, presyncope/syncope
- Acute MR—chest pain, dyspnea, orthopnea
- Chronic MR—asymptomatic for years; then fatigue, dyspnea
- Medications—anticoagulants (atrial fibrillation), beta blocker (palpitations), diuretics (heart failure)

Physical Exam

- Mid to late systolic click, ± systolic murmur
- S3, crackles/rales (pulmonary edema)
- Other abnormalities of coexisting disorder

Lab Tests/Imaging

- EKG—normal or atrial fibrillation, PVCs, repolarization abnormalities; echocardiography—severity, MR?

Consults

- Cardiology, cardiothoracic surgery as required

Conflict(s)

Optimize/Goals

- Medical therapy—continue perioperatively
- MVP and noncardiac surgery:
  Avoid decreased LV volume (worsens prolapse)—avoid tachycardia, hypovolemia, increased airway pressure, vasodilation.
  Maintain LV size—adequate preload, afterload, and contractility
- If mitral regurgitation—same hemodynamic goals as for MR
- Atrial fibrillation—beta blockers, calcium channel blockers, digoxin, amiodarone
- Infective endocarditis antibiotic prophylaxis: prosthetic valve, PH endocarditis, congenital HD, cardiac transplant

Options

- General, regional, neuraxial, local, MAC

Preop: Premed: IE prophylaxis?
Blood: surgery dependent
ICU/stepdown bed: surgery dependent

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter, arterial monitor, CVP monitor, PAC, TEE as required
- For hypotension—phenylephrine

Induction

- Maintain cardiovascular goals

Maintenance

- Balanced technique

Emergence

- Ensure hemodynamic goals maintained

Disposition/Pain

- PACU, stepdown, ICU as required

References

Hanson EW, Neerhut RK, Lynch C. Mitral valve prolapse. *Anesthesiology*. 1996;85(1)178-195.
Verma S, Mesana TG. Mitral-valve repair for mitral-valve prolapse. *N Engl J Med*. 2009;361:2261-2269.

Considerations

1. Disease of axonal inflammation and demyelination in the central nervous system (not peripheral)
2. Common triggers: infection, stress, trauma, hyperthermia
3. Occasional respiratory impairment due to ↓ inspiratory and expiratory strength
4. Neuraxial anesthesia implicated in postoperative relapse (spinal >> epidural)
5. ↓ Rate of relapse during pregnancy, but 3 × ↑ rate of relapse in first 3 months postpartum

History

- H/o weakness, visual disturbances, numbness/tingling, gait ataxia, neurogenic bladder (spastic or flaccid)
- Ability to cough, clear secretions
- Relevant meds: steroids, mitoxantrone (cardiotoxic), baclofen (potentiates neuromuscular blockade), cyclophosphamide (pancytopenia, pulmonary fibrosis, myocarditis)

Physical Exam

- Full neurologic exam
- Orthostasis and signs of autonomic neuropathy

Lab Tests/Imaging

- Echo if on mitoxantrone
- T2-weighted MRI shows multifocal lesions in CNS

Consults

Conflict(s)

- Risk of relapse with neuraxial anesthesia versus desire to avoid GA in full stomach, pregnancy, etc or stress/pain of labor

Optimize

- Delay elective surgery if acute exacerbation or if fever

Goals

- Counsel patient that despite careful attention to avoiding triggers, surgery and anesthesia may precipitate an exacerbation postop
- Avoid hyperthermia

Options

- GA + ETT/LMA
- PNB is good option as peripheral nerves *not* affected
- Potential risk of relapse with neuraxial should be balanced against benefits of avoiding GA

Preop: Premed: ± steroid cover
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Careful temperature monitoring

Induction

- Avoid sux if evidence of motor nerve involvement (spasticity, flaccidity, hyperreflexia)

Maintenance

- Unpredictable effect of NDMR: ↑ resistance in some (upregulation of ACh receptor) versus ↑ sensitivity in others (wasting, weakness)
- If RA, consider ↓ local anesthetic dose as impaired blood–brain barrier may ↑ risk for systemic neurotoxicity

Emergence

- Extubate when full recovery of baseline motor strength

Disposition/Pain

- Aggressive temperature monitoring and treatment of hyperthermia postop

Reference

Dorotta IR, Schubert A. Multiple sclerosis and anesthetic implications. *Curr Opin Anaesthesiol.* 2002;15:365-370.

Myasthenia Gravis

Considerations

1. Autoimmune disease targeting postjunctional acetycholine receptors, causing weakness
2. Potential respiratory failure
3. Potential bulbar involvement and aspiration risk
4. Altered responses to neuromuscular blockers: sensitive to NDMRs, resistant to sux
5. Potential for thymoma and anterior mediastinal mass
6. Potential for both myasthenic crisis or cholinergic crisis, both of which can cause acute respiratory failure

History

- Duration of disease, severity (see grading on back)
- Stress fatigue (arm abduction, upward gaze)
- Treatment: cholinesterase inhibitors, steroids, immunosuppressants, plasmapheresis, h/o thymectomy
- Orthopnea (mediastinal mass)

Physical Exam

- Chest infection
- Degree of muscle weakness
- Signs of other autoimmune diseases: SLE, RA, hypothyroidism

Lab Tests/Imaging

- Electrolytes (↓ K can worsen weakness)
- PFTs (esp. FEV_1, FVC, PFR)
- Glucose if on steroids

Consults

Conflict(s)

- Need for muscle relaxation versus difficulty using neuromuscular blockers and cholinesterase inhibitors

Optimize

- Plasmapheresis preoperatively for those with severe weakness. Effects can last weeks

Goals

- Avoid giving more cholinesterase inhibitor than usual daily dose → may precipitate cholinergic crisis (1 mg IV neostigmine—120 mg po pyridostigmine)

Options

- Regional anesthesia where possible
- GA without neuromuscular blockers (± thoracic epidural)
- GA with neuromuscular blockers (least desirable)

Preop: Premed: ± steroid cover; caution with sedation
GI prophylaxis
Blood
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

Induction

- May need RSI if aspiration risk → sux 1.5 to 2 mg/kg

Maintenance

Emergence

- Extubate fully awake with full return of baseline strength versus stage weaning from ventilation (see risk factors on back)

Disposition/Pain

- Careful with opioids → respiratory reserve ↓
- Multimodal: NSAIDs, tramadol
- Restart cholinesterase inhibitor slowly at smaller dose
- Observe carefully for myasthenic/cholinergic crises

Clinical Pearls

Autoimmune disease with destruction of postsynaptic nicotine acetylcholine receptors in NMJ
On average ~70 to 80% receptors destroyed
Affects women more than men, in women of age 20 to 30, men >60
Grading of severity:

1. type I—involves primarily extraocular muscles
2. type IIA—mild and slowly progressive, spares respiratory muscles
3. type II B—more severe and rapid, involves respiratory muscles
4. type III—acute onset, rapid deterioration
5. type IV—severe generalized

Risk factors for requiring postoperative mechanical ventilation (~30% will require):

- pyridostigmine dose > 750 mg/d
- disease duration >6 years
- vital capacity <2.9 L
- coexisting COPD
- upper abdominal surgery

Reference

Dillon FX. Anesthesia issues in the perioperative management of myasthenia gravis. *Semin Neurol.* 2004;24:83-94.

Myasthenic (Eaton-Lambert) Syndrome

Considerations

1. Muscle weakness, predisposing to respiratory failure after anesthesia; potential requirement for postoperative PPV
2. Association with lung carcinoma
3. Extremely sensitive to both succinylcholine and nondepolarizing muscle relaxants
4. Autonomic dysfunction presents in ~ 30%

History

- Proximal lower limb weakness—muscle power ↑ with exercise
- Neostigmine produces little/no improvement
- Dry mouth, ptosis, urinary retention

Physical Exam

- ↓ or absent lower limb reflexes
- Orthostatic hypotension

Lab Tests/Imaging

- Serology for antibodies confirms diagnosis
- CXR/CT chest for lung cancer
- EMG: characteristic finding is ↓ amplitude for low-frequency (2 Hz) stimulation but ↑↑ amplitude for high-frequency (50 Hz) stimulation

Consults

Conflict(s)

Optimize

- Counselling & preparation for possible postop PPV
- IVIG 2 g/kg over 2 days has been used to ↑ muscle strength, peaking at 2 to 4 weeks and declining by 8 weeks; alternative regimen: 400 mg/kg/d × 5 days preoperatively

Goals

- Minimize use of neuromuscular blockers, prevent need for postoperative PPV

Options

- Regional may be best option

Preop: Premed
Blood
ICU/stepdown bed: may require

Room Setup (special drugs/monitors)

Induction

Maintenance

- Avoid neuromuscular blockers if possible; if used, small doses and titrate carefully to nerve stimulator
- Response best monitored by post-tetanic facilitation, not train-of-four
- Care with myocardial depressants/vasodilating drugs if evidence of autonomic neuropathy

Emergence

- Fully awake

Disposition/Pain

- Possible postop ventilation required

Clinical Pearl

Mechanism: autoantibodies target presynaptic voltage-gated calcium channels → ↓acetylcholine release.

References

Biarnes A, Rochera MI. Lambert-Eaton (myasthenic) syndrome: pre-anaesthetic treatment with intravenous immunoglobulins. *Anaesthesia.* 1996;51:797.

Itoh H, Shibata K, Nitta S. Neuromuscular monitoring in myasthenic syndrome. *Anaesthesia.* 2001;56:562-567.

Myotonic Dystrophy

Considerations

1. Myotonia (spasm) may occur with diathermy, cold/shivering, succinylcholine, potassium
2. Respiratory weakness and hypoventilation: very sensitive to respiratory depressants
3. Cardiovascular disease & arrhythmias incl. mitral regurgitation, atrial fibrillation, conduction defects, cardiomyopathy, and biventricular heart failure
4. Aspiration risk 2-degree disordered esophageal contraction and delayed gastric emptying
5. Pregnancy: ↑ risk weakness, CHF, uterine atony, retained placenta

History

- H/o weakness, spasm, usual triggers
- Developmental delay, feeding difficulties
- ↓ Exercise tolerance, h/o CHF, pneumonias
- Hospital admission for respiratory support
- Meds: often phenytoin, procainamide, quinine

Physical Exam

- Wasting, ptosis, dysarthrias, frontal baldness
- Murmur of mitral regurgitation, parasternal heave

Lab Tests/Imaging

- CBC, electrolytes, LFTs (albumin→nutrition)
- CXR (thinned ribs, prominent PA, RVH)
- EKG (conduction defects in 50%)
- ± Echo if mitral regurgitation or cardiomyopathy suspected

Consults

- Old chart

Conflict(s)

- Potentiation of NDMRs with procainamide
- Full stomach versus sensitivity to muscle relaxants
- LMA (avoid relaxants) versus ETT (fully protected airway)

Optimize

- Preoperative antibiotics if pneumonia
- β_2-agonists, anticholinergics, steroid rx if reversible airways disease

Goals

- Minimize residual neuromuscular blockers, respiratory depressants
- Avoid episodes of myotonia, which ↑ VO_2, CO, and precipitates respiratory insufficiency

Options

- GA + ETT
- Regional if cooperative (does *not* prevent spasm)

Preop: Premed: GI, no benzos
Blood
ICU/stepdown bed: yes × 24 hours

Room Setup (special drugs/monitors)

- Warm room
- Defibrillator in room

Induction

- Inhalational versus IV with propofol

Maintenance

- Avoid myocardial depressants: consider TIVA

Emergence

- Fully awake
- Consider avoiding reversal agents (myotonia) and ventilating until fully recovered

Disposition/Pain

- Minimal opioids (NSAIDs good)
- Chest physio, incentive spirometry
- Possible postop ventilation required
- Treat shivering aggressively

Clinical Pearls

- Slowly progressive, multisystem disease with muscle atrophy as prominent sign
- Autosomal dominant, onset age 10 to 40 years, death usually not before age 40 years
- Smooth and skeletal muscle involvement (including uterus)
- Respiratory problems are the cause of most long-term deaths; also cause of most postoperative morbidity from anesthesia

Reference

White RJ, Bass SP. Myotonic dystrophy and paediatric anaesthesia. *Paed Anaesth.* 2008;13:94-102.

Neurofibromatosis Type 1

Considerations

1. Potential difficult airway with neurofibromas (NFs) of tongue, pharynx or larynx (incl. vocal cord palsy)
2. Potential pulmonary involvement including intrapulmonary/mediastinal NFs, pulmonary fibrosis, pulmonary HTN, restrictive disease from scoliosis
3. Association with pheochromocytoma, intestinal carcinoid tumors
4. Vertebral deformities or spinal tumors may make spinal/epidural techniques difficult/contraindicated
5. ↑ Incidence of epilepsy, deafness

History

- H/o dysarthria, dysphagia, stridor, voice change
- Cough, dyspnea
- Abdominal pain, bronchospasm, flushing, headache

Physical Exam

- Cafe au lait spots, cutaneous neurofibromas
- Signs of systemic HTN, LVH, right heart failure and pulmonary HTN
- Scoliosis/kyphosis
- Neuro exam for sensory/motor deficit
- Fundoscopy for papilledema (↑ ICP)

Lab Tests/Imaging

- EKG
- CXR ± chest CT, PFTs
- MRI spine prior to planned neuraxial (eg, labor epidural)

Consults

Conflict(s)

Optimize

Goals

Options

- Neuraxial should be preceded by MRI of lumbar spine to rule out spinal neurofibromas

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- ± Difficult airway cart

Induction

- Careful positioning if kyphoscoliosis

Maintenance

Emergence

Disposition/Pain

Reference

Hirsch NP, Murphy A, Radcliffe JJ. Neurofibromatosis: clinical presentations and anaesthetic implications. *Br J Anaesth.* 2001;86:555-564.

Noncardiac Surgery for Heart Transplant Patient

Considerations

1. Cardiac transplant—for end-stage heart failure (ischemic, dilated, or congenital cardiomyopathy)
2. Physiology of transplanted heart:
   Denervated but intrinsic mechanisms preserved
   Sensitive to changes in preload
   High-resting HR (90-110) as vagal tone lost
   Higher risk of arrhythmias or conduction abnormalities
3. Atropine or glycopyrrolate do not have usual effect. However, with time *reinnervation* possible
4. Complications: rejection, infection, malignancy, immunosuppression complications (DM, hypertension, seizures, cardiac vasculopathy)
5. Immunosuppressants—steroids, calcineurin inhibitors, monoclonal antibodies, etc
6. Renal failure—often cyclosporine induced
7. If blood products required—CMV status?
8. Presence of pacemaker?

History

- Scheduled surgical procedure and current symptoms
- Cardiac transplant—underlying condition, follow-up, medications, complications, current functional status
- Pacemaker?

Physical Exam

- Vital signs, resting HR
- Cardiopulmonary exam
- Targeted exam based on surgical procedure

Lab Tests/Imaging

- EKG (paced rhythm, may see 2 P waves)
- Recent echocardiography, cardiac biopsy, or angiography results

Consults

- Transplant team—cardiology, cardiothoracic, electrophysiology surgery as required

Conflict(s)

Optimize/Goals

- Continue immunosuppressant therapy
- Immunosuppressed—meticulous aseptic technique
- Infective endocarditis antibiotic prophylaxis indicated for cardiac transplant patients with valvulopathy
- Careful positioning—may have fragile bones due to chronic steroids

Options

- General, regional, neuraxial, local, or MAC as indicated

Preop: Premed: IE prophylaxis?
Blood: surgery dependent
ICU/stepdown bed: surgery dependent

Room Setup (special drugs/monitors)

- Std monitors ± Foley catheter, arterial monitor, CVP monitor, PAC, TEE as required for procedure
- Vasopressors, inotropes

Induction

- Maintain preload

Maintenance

- Balanced technique

Emergence

- Routine

Disposition/Pain

- PACU, stepdown, ICU as required; monitor preload, renal function
- Continue immunosuppressants as required

Reference

Blasco LM, Parameshwar J, Vuylsteke A, Anaesthesia for noncardiac surgery in the heart transplant recipient, *Curr Opin Anaesthesiol,* 2009;22:109-13.

Considerations

1. Associated diseases: HTN, CAD, CVA, PVD, OA
2. ↓ FRC and tendency to desaturate
3. Association with obstructive sleep apnea (5%)
4. Potentially difficult airway management (13%)
5. ↑ Glucose intolerance and presence of DM
6. Practical difficulties such as venous access, regional anesthesia, NIBP cuff size, fit on bed

History

- Screening for diseases as above
- Sx of OSA: somnolence, snoring, morning headache, CPAP use, h/o sleep study
- Exercise tolerance

Physical Exam

- Signs of R or L heart failure, HTN

Lab Tests/Imaging

- CBC, electrolytes, BUN, Cr, glucose ±room air ABG (venous CO_2 can be used as marker for CO_2 retention)
- EKG
- ± Echo (may be difficult), stress test if suspicion of CHF, CAD

Consults

Conflict(s)

Optimize

- Preop weight loss if evidence of hypoxemia, hypercarbia
- Treatment of HTN, CHF

Goals

- Avoid factors that ↑ PVR if pulmonary HTN

Options

- Consider regional anesthesia (including thoracic epidural for thoracoabdominal cases) to reduce opioid/inhalational anesthetics and facilitate early extubation
- ↓ Volume of local anesthetic in spinal/epidural space

Preop: Premed: ranitidine & metoclopramide
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- SC heparin for DVT prophylaxis
- Five-lead EKG
- Arterial line if inadequate NIBP cuff fit
- ± Difficult airway cart

Induction

- Pre-oxygenate well
- RSI versus awake FOBI
- Dose based on lean body mass (fat only 5% of cardiac output in obese)

Maintenance

- Use PEEP to prevent atelectasis

Emergence

- Fully awake

Disposition/Pain

- Careful with postoperative opioids

Clinical Pearls

- Obesity defined as BMI >30 kg/m^2
- Morbid obesity defined as BMI >35 kg/m^2
- Super-morbid obesity defined as BMI >55 kg/m^2

Reference

Adams JP, Murphy PG. Obesity in anaesthesia and intensive care. *Br J Anaesth.* 2000;85:91-108.

Obstructive Sleep Apnea

Considerations

1. Physiologic changes:
   - arterial hypoxemia
   - polycythemia
   - hypercarbia
   - systemic & pulmonary hypertension with biventricular failure
2. ↑ Risk of difficult airway
3. Sensitivity to respiratory depressants
4. ↑ Risk for early (1st 24 hours) or late (2-5 days) desaturation following anesthesia
5. Association with arrhythmias and heart block

History

- Snoring, daytime somnolence, morning headache
- H/o sleep studies: apnea-hypopnea index (AHI) >70 or minimal SaO_2<80% associated with ↑ risk postop respiratory complications
- CPAP, BiPAP use
- EtOH, drug use

Physical Exam

- Signs of R or L heart failure, S4 heart sound
- Height, weight, BMI calculation
- Neck circumference

Lab Tests/Imaging

- CBC, electrolytes, BUN, Cr, ±LFTs, ±room air ABG
- EKG
- ± Echo

Consults

- Pulmonology

Conflict(s)

- Potential for desaturation versus ambulatory surgery
- Procedures that ASA guidelines suggest should not be done on outpatient basis in this population: airway surgery, laparoscopic upper abdominal surgery, tonsillectomy in children <3 years of age
- Other factors: comorbidities, type of anesthesia, need for postop opioids, capabilities of facility (incl. availability of difficult airway equipment)

Optimize

- Ensure CPAP has been used consistently (× 2 weeks) preoperatively, and machine brought to hospital
- Treat associated conditions such as respiratory disease, heart failure, and pulmonary hypertension
- Preoperative weight loss if time

Goals

- Minimize use of respiratory depressants
- Options
- Regional may be best option (avoid opioids in infusate)

Preop: Premed: minimize benzos, opioids
Blood
ICU/stepdown bed: may require

Room Setup (special drugs/monitors)

- ±Difficult airway cart

Induction

Maintenance

- Short-acting agents (remifentanil, dexmedetomidine)
- Consider TIVA with propofol versus inhalational agent

Emergence

- Fully awake

Disposition/Pain

- Consider recovering in lateral position
- Discharge to floor with oximetry if concerned; supplementary O_2 should be provided
- NSAIDs, other opioid-sparing meds if no contraindication

Clinical Pearls

- Screening tools are useful for identifying patients in preoperative clinics with OSA, so that therapy can be initiated prior to the date of surgery. One tool is called the STOP-BANG questionnaire, which has a high sensitivity and negative predictive value, and is quick and simple to use.
  - yes to ≥3 questions—high risk of OSA
  - yes to <3 questions—low risk of OSA

S (snore)	Do you *snore* loudly (louder than talking or loud enough to be heard through closed doors)?	Yes/No
T (tired)	Do you often feel *tired*, fatigued, or sleepy during daytime?	Yes/No
O (observed)	Has anyone *observed* you stop breathing during sleep?	Yes/No
P (blood pressure)	Do you have or are you being treated for high blood *pressure*?	Yes/No
B (BMI)	*BMI* >35 kg/m^2?	Yes/No
A (age)	*Age* >50 years?	Yes/No
N (neck)	*Neck* circumference >40 cm?	Yes/No
G (gender)	*Gender* male?	Yes/No

References

Chung F, Yegneswaran B, Liao P, et al. STOP questionnaire. A tool to screen patients for obstructive sleep apnea. *Anesthesiology*. 2008;108:812-821.

Chung SA, Yuan H, Chung F. A systematic review of obstructive sleep apnea and its implications for anesthesiologists. *Anesth Analg*. 2008;107:1543-1563.

Ocular Injury

Considerations

1. Typically emergency or urgent situation
2. Type—foreign body, strike by blunt/sharp object, fight or assault, post-vehicle accident
3. If trauma—injuries? (Head/neck/other)
4. Intraocular pressure (IOP) increases with coughing, vomiting, Trendelenburg position, Valsalva maneuver, forceful facemask placement, intubation, or after succinylcholine
5. General versus regional anesthesia:

   General—most trauma, uncooperative, intoxicated, or pediatric patients

   Regional/local—cooperative patient or minimal injury
6. Oculocardiac reflex (OCR)—traction on extraocular muscles or periorbital injections—bradycardia (or AV block, asystole). Pathway: afferent—ciliary ganglion to trigeminal nerve, efferent—vagus nerve.
7. Succinylcholine—will increase IOP. Whether to use? Consider risk of aspiration/airway versus risk of eye injury

History

- Precipitating event
- Preop visual acuity
- Other trauma

Physical Exam

- Appearance? ABCs pending situation
- Visual exam, mental status, other neuro as required
- Other trauma? Primary and secondary survey

Lab Tests/Imaging

Consults

- Ophthalmology, trauma team as required

Conflict(s)

- Succinylcholine will increase IOP by 10 to 20 mm Hg for ~6 minutes versus preferred muscle relaxant for airway management of trauma patient

Optimize/Goals

- Avoid elevated IOP—blunt response to laryngoscopy with IV lidocaine or opioids and reverse Trendelenburg
- Avoid external ocular pressure
- Avoid/blunt oculocardiac reflex—eliminate stimulation, atropine/glycopyrrolate, local anesthetic—eye muscle
- Avoid respiratory acidosis (increases IOP)
- Smooth induction and emergence—avoid bucking, coughing, straining. PONV prophylaxis
- Consider aspiration prophylaxis if full stomach

Options

- General, regional, local, MAC as required
- Trauma—typically requires GETA/RSI (no published reports of further damage in ruptured globes associated with use of succinylcholine)

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Std monitors

Induction

- Trauma—GETA/RSI
  Avoid pressure with facemask
  Benzodiazepines/opioids—decrease IOP
  Propofol will decrease IOP (ketamine will increase IOP)
  ± Succinylcholine or nondepolarizing muscle relaxant

Maintenance

- Balanced technique (volatile anesthetics decrease IOP)
- Nondepolarizing muscle relaxants will decrease IOP

Emergence

- Consider deep extubation (if not full stomach!)
- Blunt cough with IV lidocaine (1.5 mg/kg) or opioids

Disposition/Pain

- Outpatient, inpatient, ICU as required

Reference

Kohli R, Ramsingh H, Makkad B. The anesthetic management of ocular trauma. *Int Anesthesiol Clin*. 2007;45(3):83-98.

Omphalocele and Gastroschisis

Considerations

1. Metabolic derangements including:
   - severe dehydration, shock
   - hypoglycemia
   - acidosis
2. ↑ Risk of infection
3. Exaggerated temperature loss
4. Risk of impaired ventilation
5. Omphalocele associated with other congential conditions:
   - trisomy 13, 18, 21
   - Beckwith-Wiedemann syndrome (hypoglycemia, macroglossia)
   - VSD, genitourinary defects

History

- Antenatal and perinatal history
- Prenatal ultrasonography diagnoses majority
- Review NICU records, flow sheets, ventilator settings

Physical Exam

- Assess ventilation
- Careful volume status (capillary refill, fontanelles, mucous membranes)
- May be murmur of VSD

Lab Tests/Imaging

- CBC, electrolytes, BUN, Cr, ABG
- CXR, abdominal x-ray (AXR)

Consults

- Cardiology, pulmonology, GI prn

Conflict(s)

Optimize

- Volume resuscitate until urine output >1 mg/kg/h
- Normalize electrolytes, glucose
- TPN is often required if bowel dysfunction with gastroschisis (or if *ruptured* omphalocele)
- Prevent heat/moisture loss by wrapping omphalocele in clear plastic

Goals

Options

- Epidural anesthesia + GETA

Preop: Premed: atropine 0.02 mg/kg IV
Blood
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Warm room, warm fluids, radiant heater; infusion pumps for all fluids
- Arterial line, CVP for access and TPN

Induction

- RSI versus intubate awake

Maintenance

- Avoid N_2O
- Careful attention to I/O; Hb, glucose, electrolytes q60min

Emergence

- ABG pre and post-closure
- At closure, if CVP ↑ >4 mm Hg, or intra-abdominal pressure ↑ >20 mm Hg, reopen and stage repair

Disposition/Pain

- ICU with PPV
- Continuous epidural or IV opioid infusion

Omphalocele and Gastroschisis *(continued)*

Clinical Pearls

- Omphalocele is a herniation of bowel (± viscera such as liver) into an enlarged umbilical cord. The membranes of the cord protect the contents from exposure to amniotic fluid and the external environment post-birth.
- Gastroschisis is a <5 cm defect to the right of the umbilicus through which bowel (and other intra-abdominal contents) herniate. These are susceptible to exposure to both amniotic fluid (causing a sclerosing effect that leads to bowel wall thickening), and, postdelivery, air (causing dehydration).

Reference

Liu LM, Pang LM. Neonatal surgical emergencies in anesthesiology. *Anesthesiol Clin North Am.* 2001;19:265-286.

Patient with Pacemaker or AICD

Considerations

1. Presence of severe cardiovascular disease (eg, severe cardiomyopathy, conduction system disease, myocardial ischemia)
2. Possibility of electromagnetic interference from:
   - electrocautery
   - radiofrequency ablation
   - MRI/radiation therapy
   - lithotripsy
   - electroconvulsive therapy

History

- Indication (eg, heart block, tachyarrhythmias, low EF)
- History of syncope, palpitations, MI, arrest
- Last device interrogation, type of device (ID card)
- Pacemaker-dependent if bradycardia? (eg, syncope, post-AV node ablation, pacemaker report)

Physical Exam

- Scars, palpate for device

Lab Tests/Imaging

- BMP (electrolytes $\rightarrow$ pacing thresholds)
- EKG, $\pm$ CXR for identification of type of device

Consults

- Cardiology
- Device manufacturer representative (interrogation and preop reprogramming)

Conflict(s)

Optimize

- Determine if EMI likely during procedure. If not likely, do nothing with device. If yes, see below

Goals

- Avoid inappropriate shocks/pacing from EMI:
  - suspend antitachycardia function and rate responsiveness during case (application of magnet usually results in asynchronous mode at predetermined rate without rate responsiveness; if in doubt, contact manufacturer/cardiologist)
  - advise surgeon to use bipolar electrocautery or ultrasonic (harmonic) scalpel if possible and use short, intermittent bursts
  - place current return pad for cautery such that vector of current does *not* pass through/near device

Options

- Consider local, regional, or general anesthesia as required

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Magnet to be placed over device
- Defibrillator/pacer in room (and depending on indication, type of surgery, and access to chest, place pads preoperatively and connect to defibrillator)

Induction

Maintenance

Emergence

Disposition/Pain

- Continuously monitor EKG and have backup pacing/defibrillator equipment immediately available
- Postoperative interrogation and restoration of function by cardiology or device manufacturer representative

Clinical Pearls

The North American Society for Pacing and Electrophysiology/British Pacing and Electrophysiology Group (NASPE/BPEG) pacemaker code

I	II	III	IV	V
chamber paced	chamber sensed	response to sensed event	rate modulation	multisite pacing
A (atrium)	O (none)	O (none)	O (none)	O (none)
V (ventricle)	A (atrium)	I (inhibit)	R (adaptive rate)	A (atrium)
D (dual: A+V)	V (ventricle)	T (triggered)		V (ventricle)
	D (dual: A+V)	D (dual: I+T)		D (dual: A+V)

Common examples:
DDD: Every atrial event followed by a ventricular event. If no activity in atrium, it will be paced, and after any sensed or paced atrial event, a ventricular event will either be allowed to occur within the allowed timeframe, or, if it has not occurred, it will be paced.
VOO: Asynchronous ventricle-only pacing with no regard for underlying rhythm
VVI: Ventricle-only pacing. Ventricle is sensed and if no event within predetermined timeframe, the ventricle is paced. If ventricular activity is sensed, the pacemaker is inhibited.

Reference

Atlee JL, Bernstein AD. Cardiac rhythm management devices (Part II): perioperative management. *Anesthesiology.* 2001;95:1492-1506.

Considerations
1. Altered airway anatomy including large occiput and tongue, short neck, narrower and softer epiglottis, and presence of cricoid ring as narrowest part of airway
2. Reliance on heart rate for CO since fixed stroke volume
3. Tendency to become bradycardic with stress
4. Tendency to desaturate rapidly with apnea or breath holding
5. Tendency toward rapid heat loss

History
- Birth history: full term? Vaginal versus C-section?
- History of recent upper respiratory infection (URI)?
- Fasting status

Physical Exam
- Precordial exam for murmur

Lab Tests/Imaging

Consults

Conflict(s)
- URI vs. need for semielective surgery

Optimize

Goals

Options
- If URI:
  - delay 4 to 6 weeks if severe symptoms:
    - fever >38.4°C
    - malaise
    - productive cough
    - wheezing
  - proceed with GA w/o ETT, or use RA if mild sx:
    - nonproductive cough, sneezing, nasal congestion
  - if surgery requires ETT, delay 4 to 6 weeks

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)
- Warm room, fluids, forced air warmer
- Variety of tube sizes (4 + age/4 as starting size, plus half size above and below), LMAs, oral airways
- 22 ga needles on sux (4 mg/kg) and atropine (20 µg/kg) for IM injection in emergency

Induction
- Inhalational versus IV if older child (tolerates IV placement)
- Avoid sux if possible, especially with young males

Maintenance
- MAC highest at 6 months of age

Emergence
- Extubate awake

Disposition/Pain

Clinical Pearls

- Debate exists regarding the use of cuffed versus uncuffed endotracheal tubes in young children. Cuffed endotracheal tubes have replaced uncuffed tubes in many centers, in children of all ages, for the following reasons:
  - mechanical ventilation through an ETT has largely replaced spontaneous ventilation, and consequently work of breathing is not affected by the internal diameter of the tube, allowing for smaller tubes with cuffs.
  - ETTs are now manufactured with high-volume, low-pressure cuffs, rather than low-volume, high-pressure cuffs, which may lead to less mucosal injury than in the past.
  - cuffed tubes in children are associated with a *decreased*, not increased, incidence of postintubation croup.
  - the use of cuffed tubes is associated with fewer reintubation attempts than uncuffed tubes.
  - cuffed tubes may provide increased protection against pulmonary aspiration.

References

Brennan LJ. Modern day-case anaesthesia for children. *Br J Anaesth.* 1999;83:91-103.
Elwood T, Bailey K. The pediatric patient and upper respiratory infections. *Best Pract Res Clin Anaesth.* 2005;19:35-46.

Pheochromocytoma

Considerations

1. Problems with severe HTN and risk of:
   - cerebral hemorrhage
   - encephalopathy
   - pulmonary edema
   - MI
   - ventricular arrhythmias
   - renal failure
2. Potential for refractory hypotension following tumor excision
3. Association with multiple endocrine neoplasia (thyroid & parathyroid Ca, insulinomas, mucosal adenomas), neurofibromatosis, and von Hippel-Landau disease (CNS hemangiomas)

History

- Headache, palpitations, diaphoresis, weight loss (NOT flushing)

Physical Exam

- Tremor
- ± Orthostatic vitals

Lab Tests/Imaging

- Free NE in 24-hour urine (most sensitive) ± urine VMA
- CBC (↑Hb from volume contraction), electrolytes (↓ K in MEN2b)
- Glucose
- EKG, echo, CT abdomen/pelvis for tumor

Consults

Conflict(s)

Optimize

- α_1 Blockade (outpatient):
  - doxazosin 1 mg po × 10 to 14 days (↑ up to 16 mg)

Goals:

- no BP>165/95, no BP<80/45
- EKG: no sustained ST changes, <1 PVC/min

- May need beta blockade if ↑HR/dysrhythmias but only after alpha blockade

Goals

- Minimize stimulation of catecholamine release
- Smooth intubation, careful pneumoperitoneum & handling of tumor
- Avoid drugs with sympathetic stimulation/histamine release

Options

- GA (±epidural if open). Careful—neuraxial does not prevent activation of postsynaptic adrenoceptors and can exacerbate ↓BP once excised

Preop: Premed
Blood
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Arterial line, ±CVP, PAC/TEE as indicated
- Phentolamine, labetalol, phenylephrine prepared

Induction

- Quiet, unhurried induction
- Deep intubation (propofol/volatile/opioid/etc)

Maintenance

- Volatile, opioid
- ✓ Blood sugar periodically

Emergence

Disposition/Pain

- To ICU/stepdown for continuous monitoring
- Epidural for postop analgesia if open procedure

Clinical Pearls

- Catecholamine-secreting tumor, most secrete NE>epinephrine.
- 90% located in adrenal medulla (rest abdomen, bladder, thorax, neck).
- Phenoxybenzamine used to be drug of choice to treat hypertension preop problem=nonselective alpha blockade. α_2 Antagonism causes:
  - uninhibited release of NE at cardiac sympathetic nerve endings $\rightarrow$ unwanted $\uparrow$ chronotropy and inotropy
  - sedation and postoperative somnolence (24-hour half-life)
  - orthostatic hypotension
  - headache and stuffy nose
- Selective α_1 antagonists (eg, doxazosin) have been used very effectively without these unwanted side effects. The last dose should be administered at 10 pm the evening before surgery (phenoxybenzamine requires 24 to 48 hours of cessation prior to surgery).
- Intraoperative HTN due to tumor manipulation can be anticipated and prophylaxis given (phentolamine 2 mg IV). In the case of epinephrine secreting tumors, tachycardia may accompany HTN$\rightarrow$labetalol is drug of choice.

Reference

Prys-Roberts C. Pheochromocytoma—recent progress in its management. *Br J Anaesth.* 2000;85:44-57.

Placental Abruption

Considerations

1. Potential for massive maternal blood loss
2. Potential for fetal asphyxia due to loss of placental surface area for gas exchange
3. Complications:
   - hemorrhagic shock
   - ARF
   - DIC (most common cause in pregnancy)
   - fetal compromise/demise (12% fetal mortality)

History

- Gestational history, prior cesarean delivery, EDC
- Vaginal bleeding, abdominal tenderness
- Increased uterine activity
- Transfusion history

Physical Exam

- Careful volume status evaluation

Lab Tests/Imaging

- CBC, coags, electrolytes, BUN, Cr, ±fibrinogen/FDPs
- Cross-match × 2 to 4

Consults

- Pediatrics/neonatology

Conflict(s)

- Desire for neuraxial anesthetic versus hypovolemia
- Neuraxial versus coagulopathy (or *potential* for coagulopathy)

Optimize

- Proper volume resuscitation with isotonic crystalloid or colloid
- Treatment of coagulopathy prn with specific blood component therapy, factors

Goals

- If preterm and bleeding stable, admit and observe until fetus mature

Options

- Vaginal delivery ± labor epidural analgesia
- Cesarean delivery with neuraxial (if nonemergent)
- Cesarean delivery with GA + RSI (if emergent)

Preop: Premed: ± steroids for lung maturation, GI prophylaxis
Blood: cross-match × 2 to 4
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Large bore IV access × 2
- Oxytocin, prostaglandin $F_{2\alpha}$, methylergonovine, ±rFVIIa
- Fetal heart rate monitor
- ±Arterial line

Induction

- If hypovolemic:
  - ketamine 0.5 to 1 mg/kg or etomidate 0.3 mg/kg

Maintenance

- 50:50 O_2:N_2O, low concentrations of volatile
- ↓ Dose of volatile after delivery (↓ tone of uterus)

Emergence

Disposition/Pain

Clinical Pearls

- Risk factors:
  - HTN
  - preeclampsia
  - advanced maternal age and parity
  - maternal and paternal tobacco use
  - cocaine use
  - trauma
  - premature rupture of (fetal) membranes (PROM)
  - chorioamnionitis
  - history of previous abruption

Reference

Mercier FJ, Van de Velde M. Major obstetric hemorrhage. *Anesthesiol Clin.* 2008;26:53-66.

Placenta Previa

Considerations

1. Potential for massive maternal blood loss
2. Potential for life-threatening fetal blood loss
3. Potential for "cretas":
   - placenta accreta (adhered to myometrium)
   - placenta increta (invasion of myometrium)
   - placenta percreta (invasion of serosa or other pelvic structures)

History

- Gestational history, prior cesarean delivery, EDC
- Painless vaginal bleeding in 2nd/3rd trimester
- Presence of contractions
- Transfusion history, tocolytic history

Physical Exam

- Careful volume status evaluation

Lab Tests/Imaging

- CBC, cross-match × 2 to 4
- Ultrasound of uterus/placenta to determine location (total/partial/marginal) and rule out coexisting abruption (10% incidence)

Consults

- Pediatrics/neonatology

Conflict(s)

- Desire for neuraxial anesthetic versus hypovolemia

Optimize

- Proper volume resuscitation with isotonic crystalloid

Goals

- If preterm and bleeding stable, admit and observe until fetus mature

Options

- Double setup examination (see back of card)
- Cesarean delivery with neuraxial (if nonemergent)
- Cesarean delivery with GA + RSI (if emergent)

Preop: Premed: ± steroids for lung maturation, GI prophylaxis
Blood: cross-match × 2 to 4
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Large bore IV access × 2
- Oxytocin, prostaglandin $F_{2\alpha}$, methylergonovine
- Fetal heart rate monitor

Induction

- If hypovolemic:
  - ketamine 0.5 to 1 mg/kg or etomidate 0.3 mg/kg

Maintenance

- 50:50 O_2:N_2O, low concentrations of volatile
- ↓ Dose of volatile after delivery (↓ tone of uterus)

Emergence

Disposition/Pain

Clinical Pearls

- Ultrasonography has made the use of the "double setup" examination all but obsolete in modern obstetrical practice. In those scenarios, the patient underwent a vaginal exam in the operating room with all personnel ready to perform an immediate cesarean delivery if a significant acute hemorrhage occurred or if placenta previa was diagnosed. However, it is still used in patients who cannot be adequately imaged (eg, the morbidly obese). Briefly the components are:
  - obstetric, anesthesiology, and pediatric staff present and ready in *or*
  - two large bore IVs, full monitors applied
  - administration of a nonparticulate antacid
  - sterile preparation and draping of the abdomen
  - two units of cross-matched blood in the *or*

References

Hong JY, Jee YS, Yoon HJ, Kim SM. Comparison of general and epidural anesthesia in elective cesarean section for placenta previa totalis: maternal hemodynamics, blood loss and neonatal outcome. *Int J Obstet Anesth.* 2003;12:12-16.

Oyelese Y, Smulian JC. Placenta previa, placenta accreta and vasa previa. *Obstet Gynecol.* 2006;107: 927-941.

Preeclampsia

Considerations

Multisystem disease whose main factors include:

1. Cardiovascular
   - HTN
   - volume contraction
   - heart failure
   - intracranial hemorrhage
   - abnormal pressor response
2. CNS: cerebral edema, headache, seizures
3. Pulmonary: pulmonary edema
4. Genitourinary (GU): oliguria/anuria
5. Heme: thrombocytopenia, DIC, hemolysis
6. Airway: ↑ pharyngeal/laryngeal edema & friability of airway
7. Uteroplacental insufficiency

History

- Headache, edema, scotoma, dyspnea
- Nausea/vomiting
- Mucosal bleeding
- Anticonvulsant/antihypertensive use

Physical Exam

- Careful airway/cardiopulmonary exam
- Patellar reflexes if on magnesium therapy

Lab Tests/Imaging

- CBC, electrolytes
- PT/PTT, fibrinogen if DIC suspected
- Check ↓ K level if on infusion (normal 4-6 mg/L)
- Urine protein
- ±Echo if signs of heart failure/pulmonary edema

Consults

Conflict(s)

- Volume contraction versus desire for neuraxial anesthetic
- Thrombocytopenia versus desire for neuraxial anesthetic
- Careful titrated induction to avoid hypertensive response versus full stomach
- Difficult airway versus full stomach

Optimize

- Antihypertensives if severe (≥160/110 mm Hg) HTN (labetalol, nifedipine, hydralazine)
- Volume expansion *not* recommended as serious risk of volume overload and pulm/cerebral edema

Goals

- Minimize stimulation of catecholamine release (no epi in solutions, smooth IV induction)

Options

- Epidural analgesia recommended for labor (↓ catecholamines)
- Spinal generally safe and effective for C-section; CSE if longer procedure anticipated (ie, repeat)

Preop: Premed: ± steroids (may be on for HELLP), GI prophylaxis
Antibiotics
Blood: cross-match if C-section
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Arterial line if ++ hypertensive or labile
- Labetalol (esmolol causes fetal acidosis from ↓ cardiac output) to titrate at induction
- Avoid ergotamine for Rx of PPH (↑ BP)

Induction

- Labor epidural: no epi, fentanyl 100 μg for test dose (sedation = positive test)
- C-section: no dose adjustment with spinal c/w healthy
- GA: slow, quiet, unhurried induction

Maintenance

- Care with muscle relaxants if on Mg^{++} (potentiation)

Emergence

Disposition/Pain

- Consider monitored setting; avoid NSAIDs if renal dz

Clinical Pearls

- Diagnosis:
  - SBP ≥140 mm Hg or DBP ≥90 mm Hg on at least two occasions, 4 to 6 hours apart, at 20 weeks gestation or later (in women known to be normotensive beforehand)
  - 300 mg or more of protein in 24-hour urine sample (urine dipstick correlates poorly with 24-hour quantification)
- *Severe* preeclampsia:
  - BP≥160/110 *and/or*
  - 24 urine protein ≥5 g *and/or*
  - multiorgan involvement such as:
    - pulmonary edema
    - seizures
    - oliguria (<500 mL/d)
    - thrombocytopenia (<100,000/μL)
    - abnormal liver enzymes
    - epigastric/right upper quadrant pain
    - persistent and severe CNS symptoms (altered mental status, headaches, blurred vision, blindness)
  - Maternal and fetal complications:
    - abruption (1-4%)
    - DIC/HELLP syndrome (10-20%)
    - pulmonary edema/aspiration (2-5%)
    - ARF (1-5%)
    - eclampsia (<1%)
    - stroke (rare)
    - death (rare)
    - preterm delivery (15-67%)
    - fetal growth restriction (10-25%)
    - hypoxia-neurologic fetal injury (<1%)

Reference

Sibai B, Dekker G, Kupferminc M. Pre-eclampsia. *Lancet*. 2005;365:785-799.

Considerations
1. Physiologic changes of pregnancy including tendency to desaturate and increased risk of aspiration
2. Two patients: mother and fetus
3. Prone to aortocaval compression
4. Tendency for a friable, edematous airway
5. ↓ MAC
6. May present for non-OB surgery

History
- Pregnancy history including complications such as bleeding, hyperemesis

Physical Exam
- Wide, loud split S1, S3, systolic ejection murmur

Lab Tests/Imaging
- CBC, electrolytes, BUN/Cr

Consults
- OBGYN if non-OB surgery

Conflict(s)
- Straight epidural for labor (slower, but knowledge of working catheter) versus CSE (rapid onset, but untested catheter and possible ↑ PDPH)
- C-section: difficult airway and full stomach versus urgency d/t fetal distress

Optimize
- O_2 if fetal distress

Goals
- Preserve uteroplacental perfusion (MAP>70 mm Hg)
- Avoid suspected teratogenic agents: N_2O, benzodiazepines

Options
- Labor analgesia: epidural, CSE, single shot spinal (if maximally dilated), IV PCA remifentanil
- C-section: epidural/CSE/spinal preferred d/t ↓ mortality; GA if truly stat section

Preop: Premed: GI prophylaxis
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)
- Fetal heart monitor
- Vasopressors: phenylephrine and/or ephedrine

Induction
- C-section: wedge under right hip; pre-oxygenate for 3 minutes or 8 vital capacity breaths

Maintenance

Emergence

Disposition/Pain

Reference

Fanzago E. Anaesthesia for non obstetric surgery in pregnant patients. *Minerva Anestesiol.* 2003;69:416-427.

Premature (Low Birth Weight) Infant

Considerations

1. Impaired temperature regulation
2. Susceptibility to respiratory distress syndrome and bronchopulmonary dysplasia
3. ↑ Risk of postoperative apnea
4. Persistent fetal circulation (PDA, PFO) → L-to-R shunt, ↑ pulmonary flow, RVH, CHF, & ↓ systemic perfusion
5. Retinopathy of prematurity
6. Intraventricular hemorrhage associated with HTN, hypoxemia, hypercarbia, hypoglycemia, anemia, and hyperosmolar fluids
7. ↑ Risk of necrotizing enterocolitis, sepsis

History

- Perinatal, NICU history
- Apneic episodes, ventilatory/oxygenation requirements
- Meds: indomethacin for PDA
- Infections
- H/o TPN

Physical Exam

- Signs of respiratory distress
- Volume status
- Murmur of PDA; signs of CHF

Lab Tests/Imaging

- CBC, electrolytes, BUN/Cr, glucose, ABG
- CXR
- Echo

Consults

Conflict(s)

- ↑ O_2 requirements in RDS versus risk of ROP; however, "brain before eye"→use as much O_2 as needed to ensure vital organ oxygenation
- Surgery in OR (clean, familiar environment) versus in NICU (no need for potentially dangerous transport)

Optimize

Goals

- ↓ ROP: keep PaO_2 60 to 80 mm Hg
- ↓ IVH: maintain normocarbia, prevent HTN, hypoglycemia
- Avoid paradoxical embolism: keep PVR low to prevent R-to-L shunt through PFO

Options

- Consider local, regional, or general anesthesia as required

Preop: Premed: GI prophylaxis
Blood: yes (see note on reverse)
ICU/stepdown bed: yes (NICU)

Room Setup (special drugs/monitors)

- Warm room (27°C) + radiant/forced air warmers
- Glucose infusion setup; others prn (eg, inotropes)
- Pressure-controlled ventilator capable of delivering small tidal volumes + PEEP
- ± Arterial line

Induction

- IV or inhalational (RSI is controversial; apnea not tolerated well)

Maintenance

- Balanced (↓ MAC, longer elimination t½ for opioids)
- Fraction of inspired oxygen (FiO_2) to keep SaO_2 85 to 95% if risk of ROP

Emergence

Disposition/Pain

- NICU

Clinical Pearls

- Treat intraoperative hypoglycemia with a 2 mL/kg bolus of dextrose 10%, followed by an infusion of dextrose at 4 to 6 mg/kg/min. Check glucose levels frequently.
- Daily maintenance fluid requirements are approximately 100 mL/kg/d. Intraoperative maintenance fluids should be isotonic (LR or NS) with added dextrose if required.
- Cross-matched blood should be available when expected loss equals 10% of blood volume (ie, 9 mL for a 1000 g baby). Estimating blood loss is challenging, but can be guided by capillary Hb and cardiopulmonary status. The extremely premature and those infants with cyanotic heart disease require a hematocrit of 35 to 40% for adequate oxygenation.
- Risk of postoperative apnea is likely <1% for most premature infants after 60 weeks postconceptual age. Other risk factors are anemia, neurologic disease, and chronic lung disease.

References

Kinouchi K. Anaesthetic considerations for the management of very low and extremely low birth weight infants. *Best Pract Res Clin Anaesthesiol.* 2004;18:273-290.

Peiris K, Fell D. The prematurely born infant and anaesthesia. *Contin Educ Anaesth Crit Care Pain*. 2009;9:73-77.

Progressive Systemic Sclerosis (Scleroderma)

Considerations

1. Skin changes: joint contractures, ↓ mouth opening, difficult IV access, oral/nasal telangiectasias
2. Pulmonary: pulmonary HTN and hemorrhage, interstitial lung dz
3. Cardiac fibrosis (CHF), conduction system defects, sclerosis of coronary arteries, HTN, pericarditis/effusions/tamponade
4. Esophageal dysmotility with aspiration risk
5. Impaired nerve conduction with peripheral and/or cranial neuropathy
6. Renal artery obstruction from intimal proliferation ⟶ renal failure

History

- Neuropathy, trigeminal neuralgia, Raynaud
- Dyspnea, angina, syncope, peripheral edema
- Dysphagia, reflux

Physical Exam

- Skin of face, neck, mouth opening, neck ROM
- Signs of heart failure, pulmonary HTN

Lab Tests/Imaging

- CBC, electrolytes, coags (vitamin K malabsorption)
- EKG
- ±CXR, echo, stress-echo, CrCl, ABG
- PFTs rarely necessary

Consults

- Rheumatology ±cardiology/pulm/renal prn

Conflict(s)

- RSI for aspiration risk versus potentially difficult a/w (FOBI)
- Monitoring of poorly contractile heart: PA catheter ↑ risk with pulmonary HTN *but* TEE ↑ risk with esophageal mucosal thinning

Optimize

Goals

- Avoid further increases in pulmonary arterial pressure (avoid hypoxemia, hypercarbia, hypothermia, acidosis, N_2O, catecholamines, high a/w pressures, pain)

Options

- Regional often best option to avoid airway/PPV
- If GA, expect increased a/w pressures and FiO_2 to maintain ventilation and oxygenation respectively

Preop: Premed: GI prophylaxis
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Warm room & fluids (vasoconstriction)
- ±Arterial line, CVP, cardiac output monitoring
- Fiberoptic scope if difficult airway anticipated

Induction

- RSI versus FOBI

Maintenance

- Care with drugs that require renal clearance if renal function impaired

Emergence

Disposition/Pain

Reference

Roberts JG, Sabar R, Gianoli JA, Kaye AD. Progressive systemic sclerosis: clinical manifestations and anesthetic considerations. *J Clin Anesth*. 2002;14:
474-477.

Pulmonary Hypertension

Considerations

1. Establishment of cause (primary vs secondary)
2. Associated diseases such as collagen vascular disease, Raynaud disease, cirrhosis, sickle cell disease
3. Special medications: Coumadin, vasodilators
4. Possibility of biventricular failure

History

- Duration, severity
- Dyspnea, angina, syncope, hemoptysis
- Drug therapy (vasodilators, calcium channel blockers, PDE-5 inhibitors)

Physical Exam

- RV heave + S3, loud P2
- Signs of low cardiac output: mottling, hypotension, cyanosis
- Raynaud: blanched fingers

Lab Tests/Imaging

- CBC (↑Hb), electrolytes, coags, ABG, ± LFTs
- CXR (assess RV size, presence of large PA)
- EKG (RV strain, RVH, RBBB)
- Echo (RVH, paradoxical septal motion, PASP)
- ± Cardiac catheterization (gold std for assessment)

Consults

- Pulmonology and cardiology

Conflict(s)

Optimize/Goals

- *Hemodynamic*
  - avoid increased PVR (avoid N2O, hypoxemia, hypercarbia, acidosis, hypothermia, pain)
  - avoid high airway pressures, PEEP, alpha agonists
  - avoid decreased RV preload
  - maintain RV contractility (early inotropic support)
  - maintain LV afterload (care with neuraxial)
- *Preop hydration*
- *Chest physio*

Options

- Consider local, regional, or general anesthesia as required

Preop: Premed
Blood
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- A-line, TEE/PA catheter (careful.± balloon inflation)
- Warm room, Bair hugger
- Epoprostenol (continue same dose), SNP drip, NE drip, ± NO (20-40 ppm)

Induction

- Careful titrated induction (opioids have no effect on PVR)

Maintenance

- No N_2O, careful with PEEP
- If hypotension, NE (least increase in PVR)
- Minimize a/w pressures

Emergence

Disposition/Pain

- ICU/stepdown
- Aggressive pain regimen

Reference

Blaise G, Langleben D, Hubert B. Pulmonary arterial hypertension. Pathophysiology and anesthetic approach. *Anesthesiology*. 2003;99:1415-1432.

Pulmonic Stenosis

Considerations

1. Etiology: valvular (90%) versus sub/supravalvular (10%)
2. Coexisting cardiac abnormalities:
   - ASD
   - VSD
   - TOF
   - PDA

History

- Dyspnea, angina, syncope
- Duration & severity
- Treatment: percutaneous balloon valvuloplasty

Physical Exam

- Murmur: loud SEM second left ICS
- Right heart failure: edema, hepatosplenomegaly, RV heave, ascites

Lab Tests/Imaging

- CBC, LFTs (if right heart failure)
- ABG
- Echo

Consults

- Cardiology

Conflict(s)

- Very difficult to resuscitate if cardiac arrest

Optimize

Goals

- Avoid ↑ RV O_2 consumption (eg, ↑ HR, contractility)
- Maintain preload as right cardiac output fixed
- Maintain sinus rhythm if possible

Options

- GA with ETT
- Neuraxial (care with ↓ preload → ↓↓ cardiac output)

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Defibrillator in room
- Arterial line
- Phenylephrine

Induction

- Careful titrated induction

Maintenance

- Treat arrhythmias aggressively
- Treat systemic hypotension aggressively with vasopressors

Emergence

Disposition/Pain

References

Brickner ME, Hillis LD, Lange RA. Congenital heart disease in adults. *N Engl J Med.* 2000;342:256-263.

Ransom DM, Leicht CH. Continuous spinal analgesia with sufentanil for labor and delivery in a parturient with severe pulmonary stenosis. *Anesth Analg.* 1995;80:418-421.

Pyloric Stenosis

Considerations

1. Medical, not surgical, emergency
2. Metabolic derangements including:
   - ↓ Na^+
   - ↓ K^+
   - ↓ Cl^+
   - metabolic alkalosis → if severe enough, metabolic acidosis
3. Full stomach and aspiration risk
4. Hypovolemia
5. Tendency to postoperative depression of ventilation

History

- During of vomiting
- Last feed

Physical exam

- Careful volume assessment, skin turgor, mucous membranes, fontanelle, capillary refill

Lab Tests/Imaging

- CBC, electrolytes, BUN/Cr, glucose, ABG

Consults

- Pediatrics for preoperative hydration, correction of metabolic abnormalities

Conflict(s)

- Full stomach versus rapid desaturation as infant
  - *intubate* either *awake* (potentially suboptimal intubating conditions) or *modified RSI* (ie, ventilate prior to laryngoscopy). True RSI with period of apnea *not* tolerated in this infant population

Optimize

- Normal saline and electrolyte replacement. Endpoints:
  - vitals: HR<120 bpm, SBP 60 to 90 mm Hg
  - pH 7.3 to 7.5
  - K^+ >3 mmol/L
  - Cl^+ >90 mmol/L
  - HCO_3^- <30 mmol/L
  - U/O > 1 mL/kg/h

Goals

Options

- GA with ETT

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Warm room + radiant/forced air warmers
- ± Glucose infusion if hypoglycemic

Induction

- Preoperative orogastric suctioning
- IV or inhalational (RSI is controversial; apnea not tolerated well)

Maintenance

- Volatiles, no N_2O

Emergence

- Fully awake

Disposition/Pain

- Postanesthesia care unit (PACU)
- Watch carefully for hypoventilation
- ✓ Sugars

References

Eich C, Timmermann A Russo SG, et al. A controlled rapid-sequence induction technique for infants may reduce unsafe actions and stress. *Acta Anaesthesiol Scand.* 2009;53:1167-1172.

Fell D, S Chelliah. Infantile pyloric stenosis. *Contin Educ Anaesth Crit Care Pain.* 2001;1:85-89.

Considerations

1. **Airway:** TMJ, cricoarytenoid involvement; atlantoaxial subluxation
2. **CVS:** pericarditis, myocarditis, coronary arteritis, conduction system abnormalities, aortic insufficiency
3. **Resp:** pleural effusions, pulmonary fibrosis, pulmonary rheumatoid nodules, restrictive disease from costochondral involvement
4. **CNS:** cerebral vasculitis, peripheral neuropathy (especially ulnar nerve)
5. **GI:** Felty syndrome (RA, hepatosplenomegaly, leukopenia)
6. **Effects of drugs:** steroids, NSAIDs, DMARDs, and immunosuppressives

History

- Neck symptoms: pain, paresthesias
- Peripheral neuropathies
- Drug history
- Exercise tolerance

Physical Exam

- TMJ, careful neck ROM
- Precordial exam (pericarditis, tamponade)
- Peripheral neurological exam

Lab Tests/Imaging

- EKG (conduction system), CBC (↓Hb, Felty)
- Lateral flexion/extension views of C-spine (>4 mm—positive for subluxation)
- Echo if effusion or aortic insufficiency suspected
- CXR ± spirometry if pulmonary symptoms/signs

Consults

Conflict(s)

Optimize

Options

- Often for orthopedic procedures—RA appropriate for many
- Must consider plan for airway if cannot perform RA or if GA required
- Awake fiberoptic followed by positioning and brief neurological exam before induction may be the safest option if concerned about C-spine instability

Preop: Premed: ± steroid cover
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Fiberoptic equipment if necessary
- Five-lead EKG

Induction

- IV induction versus awake FOBI

Maintenance

- Care with positioning

Emergence

- Smooth, awake. Stabilize C-spine

Disposition/Pain

- PACU

Reference

Lisowska B, Rutkowska-Sak L, Maldyk P, Cwiek R. Anaesthesiological problems in patients with rheumatoid arthritis undergoing orthopaedic surgeries. *Clin Rheumatol.* 2008;27:553-556.

Sarcoidosis

Considerations

Multisystem granulomatous disease with:

1. Pulmonary involvement: airway granulomas, restrictive (±obstructive) disease, alveolar fibrosis, ↓diffusion capacity
2. Airway involvement: laryngeal, nasal mucosal sarcoidosis
3. CNS involvement: dementia, encephalopathy, seizures, headache, facial nerve neuropathy, uveitis
4. Myocardial involvement: heart block, pericardial disease, restrictive cardiomyopathy

History

- Dyspnea, cough, chest pain, stridor
- Seizures, headache
- Exercise tolerance, shortness of breath on exertion (SOBOE)

Physical Exam

- Signs of cor pulmonale, pericardial rub
- Hepatosplenomegaly
- Skin nodules

Lab Tests/Imaging

- Electrolytes, calcium, ABG, LFTs
- EKG
- CXR
- ±CT chest/PFTs (?anterior mediastinal mass), ±echo

Consults

- Pulmonology

Conflict(s)

Optimize

- Treat heart failure, arrhythmias (may require pacemaker/AICD)
- A delay to treat upper airway sarcoidosis with steroids may be prudent
- Other indications for urgent steroids: hypercalcemia, neurological involvement, cardiac or ocular involvement

Goals

Options

- Consider local, regional, or general anesthesia as required

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- ±Arterial line if significant pulmonary disease

Induction

- RSI versus FOBI

Maintenance

Emergence

Disposition/Pain

- May require postoperative PPV

References

Dempset OJ, Paterson EW, Kerr KM, Denison AR. Sarcoidosis. *BMJ.* 2009;339:b3206.
Polychronopoulos VS, Prakash UB. Airway involvement in sarcoidosis. *Chest.* 2009;136:1371-1380.

Scoliosis

Considerations

1. Potential respiratory impairment and need for postoperative PPV
2. Potential pulmonary HTN and cor pulmonale
3. Association with neuromuscular disease and congenital heart disease
4. Potential difficult airway
5. Possible difficult regional anesthesia

History

- Location of curve, age of onset, severity, etiology
- Most curves to right convex; if left convex, ↑ suspicion for underlying conditions

Physical Exam

- Cervical mobility
- RV failure: RV heave, loud P2, ↑ JVP, hepatic congestion
- Brief neuro assessment

Lab Tests/Imaging

- CBC, ABG
- CXR: ✓ Cobb angle: surgery indicated if 45 to 50 degrees; postop respiratory problems expected if >60 degrees
- PFTs
- EKG ±echo

Consults

Conflict(s)

- Difficult airway versus need for double lumen tube

Optimize

- Treat right heart failure
- VC <50% predicted *and/or* ↑PCO_2 suggests need for postop PPV

Goals

Options

- Consider local, regional, or general anesthesia as required

Preop: Premed
Blood
ICU/stepdown bed: **based on predicted need**

Room Setup (special drugs/monitors)

- For correction of scoliosis:
  - arterial line, CVP, Foley
  - ±PA catheter if pulmonary HTN
  - SSEPs/MEPs ± wake-up test
  - intraoperative blood salvage
  - fiberoptic scope for DLT confirmation if used

Induction

Maintenance

- No NMBs if MEPs are used

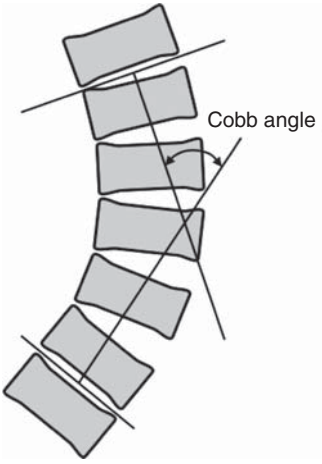
Emergence

Disposition/Pain

- Possible need for postop PPV

Clinical Pearls

- Etiology:
  - idiopathic (70%)
  - congenital
  - associated with neuromuscular/connective tissue disorders (CP, muscular dystrophy, Marfan, etc)



Scoliotic spine illustrating the method of calculation of the Cobb angle. This can be done using the chest or abdominal plain films.

Reference

Gibson PR. Anaesthesia for the correction of scoliosis in children. *Anaesth Intensive Care*. 2004;32:548-559.

Sickle Cell Disease

Considerations

1. Tendency to small vessel occlusion and infarction in organs including bone marrow, spleen, brain, lung
2. Tendency toward other crises including:
   - sequestration (pooling in spleen)
   - aplastic (marrow depression)
   - hemolytic
   - chest (fever, atelectasis, infiltrates)
3. Possible functional asplenia and risk of infection
4. Anemia and ↓ O_2-carrying capacity
5. Progressive ↓ in renal concentrating ability that exacerbates dehydration

History

- Crisis history, treatment, splenic function
- Hemoglobin subtype if known
- Vaso-occlusive complications: CVA, AVN, lung infarction, MI

Physical Exam

- Temperature, chest exam

Lab Tests/Imaging

- Hb electrophoresis to determine subtype
- CBC, electrolytes, urine dipstick
- CXR if chest crisis suspected

Consults

- Hematology

Conflict(s)

Optimize

- Preop admission and IV hydration, chest physio
- Delay if acute infection
- Transfuse to Hb>10 g/dL *if* crisis *or* emergency/major surgery. Otherwise, just pay meticulous attention to avoiding risk factors for sickling

Goals

- Avoid sickling by keeping:
  - warm
  - well-hydrated (po and IV)
  - well-oxygenated
  - normal acid-base status ⟶ normocapnia
- Avoid tourniquets (discuss with surgeon)

Options

- RA theoretically good option if possible: ↑ blood flow to extremities, superior pain control

Preop: Premed: antibiotics especially with splenectomized
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Warm room, fluids, forced air warmer

Induction

- RSI versus FOBI

Maintenance

- FiO_2—0.3 to 0.5 at minimum

Emergence

Disposition/Pain

- O_2 and IVF to prevent chest crisis (peak incidence at 72 hours)
- Aggressive pain control: opioid PCA, NSAIDs, acetaminophen, regional blocks

Clinical Pearls

- Pulmonary and neurologic disease are the leading causes of morbidity and mortality.
- Acute chest syndrome (ACS)—a finding of a new pulmonary infiltrate involving at least one complete lung segment on CXR *plus* one or more of these new symptoms:
  - chest pain
  - fever >38.5°C
  - tachypnea
  - wheezing
  - cough
- Preoperative blood transfusion tends to reduce complication rates only in moderate-to-high-risk cases, and should be balanced against the usual risks of transfusion.
- The goal for transfusion should be a total Hb>10 g/dL (not HbS<30%—this is associated with a higher incidence of complications).

Reference

Firth PG, Head CA. Sickle cell disease and anesthesia. *Anesthesiology*. 2004;101:766-785.

Considerations
1. Potential for spinal shock (hypotension ± bradycardia depending on level)
2. Respiratory compromise if >C4-5 level→hypoventilation, inability to clear secretions
3. Poikilothermia → risk for hypothermia
4. Risk for autonomic dysreflexia
5. Risk for succinylcholine-induced hyperkalemia
6. Immobilization problems: DVT, ulcers, infection

History
- Cause (trauma, tumor, vascular, MS), level, & timing of injury

Physical Exam
- Chest exam for pneumonia
- Orthostasis
- C-spine mobility

Lab Tests/Imaging
- CBC (↓Hb), coags if on warfarin (DVT)
- BUN + Cr to r/o renal impairment (chronic UTI/reflux)
- Spirometry in all patients with lesions >T7
- If in doubt re: resp status, CXR, ABG

Consults

Conflict(s)

Optimize
- ±Po nifedipine 10 mg 1 hour preop (AD prevention)

Goals
- Prevent AD if at risk
- Keep warm, euvolemic
- Minimize need for postop PPV

Options
- Standby: if below lesion, may not need anesthesia. Based on:
  - level/completeness of lesion (>T1=high risk)
  - risk of procedure (ie, urology=high risk)
  - likelihood of spasms in response to surgery
  - patient willingness
- General: deep GA often best option
- Regional: theoretically good but can be difficult

Preop: Premed
Blood
ICU/stepdown bed: **based on predicted need**

Room Setup (special drugs/monitors)
- Extra padding for limbs → osteoporosis/fractures
- Warm room, IVF, forced air warmer

Induction
- Sux OK in first 48 to 72 hours, and again after 6 to 12 months
- Atropine 0.01 mg/kg if HR<60 at induction
- Lower blood volume →↑ sensitivity to induction agents

Maintenance
- Deep anesthesia prevents AD

Emergence

Disposition/Pain
- Possible need for postop PPV
- Often in chronic pain →requires aggressive Rx

Clinical Pearls

- Spinal shock (vasodilation, bradycardia, myocardial dysfunction) lasts from 2 to 3 days to 6 to 8 weeks after injury.
- Gradual (but abnormal) neural connections develop distal to the site of injury and lead to return of sympathetic efferent discharge, muscle tone, and reflexes ("reflexic phase").
- Spasticity develops as final phase at 2 to 3 months
- **Autonomic dysreflexia**
  - 85% in lesions above T6; unlikely if below T10
  - paroxysmal HTN (often severe), headache, sweating + flushing/pallor above level of injury, reflex bradycardia
  - complications include stroke, seizures, coma, MI, pulmonary edema, and death
  - two mechanisms: loss of descending inhibitory impulses *and* disorganized connections between ascending afferent fibers and injured preganglionic sympathetic fibers. Result is widespread inappropriate sympathetic response
  - typical triggers: bladder distention, uterine contractions, defecation; cutaneous stimuli less common
- **Management**:
  - remove offending stimulus (eg,✓ Foley for blockage, remove tight clothing)
  - if no relief, treat HTN: labetalol 10 to 20 mg and phentolamine 2 to 10 mg IV are fast agents

References

Fox R, Watling G. Anaesthesia for patients with chronic spinal cord injury. *Curr Anaesth Crit Care.* 2001;12:154-158.
Hambly PR, Martin B. Anaesthesia for chronic spinal cord lesions. *Anaesthesia.* 1998;53 273-289.

Systemic Inflammatory Response Syndrome

Considerations

1. A nonspecific, multisystem state of systemic inflammation and organ dysfunction defined by the presence of two or more of the following:
   - body temperature <36°C or > 38°C
   - heart rate >90 bpm
   - respiratory rate >20 or $PaCO_2$ <32 mm Hg
   - WBC <4000 cells/mm^3 or >12,000 cells/mm^3; or the presence of >10% immature neutrophils
2. Often associated with infection (SIRS + infection = *sepsis*), but may be due to trauma, burns, pancreatitis, anaphylaxis, drugs, ischemia, hemorrhage
3. Common perioperative problems: anemia, hypotension, hypovolemia, coagulopathy, electrolyte disturbances, and acidosis

History

- Confusion, fever, chills
- Unexplained bleeding/bruising
- Dizziness, syncope, lethargy
- Antecedent event (trauma, burn, drug ingestion, infection, etc)

Physical Exam

- Confusion, tachypnea, hypotension, tachycardia
- Fever or hypothermia, oliguria, evidence of DIC
- Postoperative respiratory failure

Lab Tests/Imaging

- CBC, electrolytes, glucose, ABG
- ± blood & urine cultures (& others prn), cardiac enzymes, liver profile, amylase, lactate, CRP, procalcitonin
- Echo (often myocardial depression), CXR
- Other labs/imaging as indicated by history/symptoms

Consults

- Infectious diseases (especially in immunocompromised)

Conflict(s)

Optimize

- Two large-bore IVs, fluids and vasopressors if hypotensive
- Antibiotics if known infection, or if:
  - immunocompromised
  - hemodynamically unstable
- Consider activated protein C if gram-negative septic shock
- Maintain blood glucose at 80 to 110 mg/dL
- Consider low-dose steroid (hydrocortisone 50 mg q6h)
- Enteral feedings, DVT, and stress ulcer prophylaxis

Goals

- IV fluid, vasopressors, inotropes, and oxygen to maintain CVP of 8 mm Hg, MAP >65 mm Hg, U/O of 0.5 mL/kg/h, and MvO_2 >60 to 65%

Options

- Neuraxial should be undertaken with extreme caution (hypotension & spinal infection); PNBs can be excellent for avoiding GA and neuraxial

Preop: Premed:**review current antibiotics**
Blood
ICU/stepdown bed: **yes**

Room Setup (special drugs/monitors)

- Arterial line, CVP, Foley ± PA catheter, TEE
- Temperature monitoring
- Prepare vasoactive infusions (eg, epinephrine)

Induction

- Careful titrated induction with *minimal* agent (consider ketamine or etomidate)
- Anticipate postinduction hypotension and treat early

Maintenance

- Care with NMBs (critical illness myopathy)

Emergence

Disposition/Pain

- Transfer back to ICU/other monitoring setting

Reference

Schuerholz T, Marx G. Management of sepsis. *Minerva Anestesiol*. 2008;74:181-195.

Systemic Lupus Erythematosus

Considerations
1. Cardiac: conduction abnormalities, myocarditis, pericarditis/effusion, noninfective vegetations, coronary artery disease
2. Pulmonary: pleuritis/effusions, restrictive lung disease, lupus pneumonitis, pulmonary HTN
3. Renal: lupus nephritis
4. CNS: seizures, psychosis, peripheral neuropathy
5. Heme: ↓ Hb, ↓WBC, ↓ platelets; antiphospholipid antibody → arterial & venous thrombosis, MI, PE, fetal loss
6. Meds: often on steroids, NSAIDs, heparin, cyclophosphamide, antimalarials

History
- CNS: headache, neuropathy
- CVS: SOBOE, angina, peripheral edema
- Pulm: dyspnea
- Heme: h/o thrombosis

Physical Exam
- Pleural effusion
- Pericardial rub
- Signs of heart failure

Lab Tests/Imaging
- CBC, electrolytes, coags
- CXR, EKG, ±echo (if symptoms)
- Thrombin time, TEG, or ACT if neuraxial planned

Consults
- Rheumatology ±cardiology/pulm/renal prn

Conflict(s)
- Difficult to assess coagulation status as PTT can be ↑ due to lupus anticoagulant (despite patient being thrombophilic)

Optimize
- Treat exacerbation with course of corticosteroids
- If severe exacerbations (refractory renal dz, alveolar hemorrhage, TTP, catastrophic antiphospholipid antibody syndrome, neuropsychiatric symptoms), may try preoperative plasmapheresis

Goals

Options
- Consider local, regional, or general anesthesia as required

Preop: Premed: **consider steroid cover; SBE prophlax if vegetations**
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

Induction

Maintenance

Emergence

Disposition/Pain

References

Fessler BJ, Boumpas DT. Severe major organ involvement in systemic lupus erythematosus. Diagnosis and management. *Rheum Dis Clin North Am.* 1995;21:81-98.

Wetzl RG. Anaesthesiological aspects of pregnancy in patients with rheumatic diseases. *Lupus.* 2004;13:699-702.

Tetralogy of Fallot

Considerations

1. Main lesion—R→L shunt
2. Associated abnormalities: ASD, coronary arterial abnormalities, right-sided aortic arch, scoliosis
3. Potential for paradoxical emboli
4. Potential for SBE
5. Potential for erythrocytosis and thrombotic complications

History

- Severity, history of cyanotic or syncopal spells, surgical history of correction
- History of CVA
- Dyspnea, exercise tolerance

Physical Exam

- Pulmonic stenosis murmur → SEM at LLSB

Lab Tests/Imaging

- CBC (↑Hb)
- EKG (RVH ± RBBB)
- CXR (boot-shaped heart, ↓ pulmonary vascularity)
- Echo, ±catheterization

Consults

- Cardiology

Conflict(s)

Optimize

- IV fluids for dehydration, glucose

Goals

- Avoid dehydration
  - IV fluids, clears until 2 hours before surgery (first case of the day)
- Minimize R→L shunting
  - Maintain afterload
  - Avoid ↑ in PVR (avoid hypoxemia, hypercarbia, hypothermia, acidosis, N_2O, catecholamines, pain, ↑ a/w pressures)
  - Avoid ↑ contractility

Options

- GA, GA + epidural

Preop: Premed: **antibiotics for SBE prophylaxis**
Blood
ICU/stepdown bed: **yes**

Room Setup (special drugs/monitors)

- Warm room & fluids
- Bubble traps in lines
- Phenylephrine and beta blocker prepared

Induction

- Ketamine good *or*
- Careful inhalational induction watching ↓SVR

Maintenance

- Avoid muscle relaxants/opioids that release histamine

Emergence

Disposition/Pain

- ICU, careful rhythm monitoring (atrial fib/flutter common)

Clinical Pearls

- Most common congenital heart lesion associated with right-to-left shunt consists of:
  - VSD
  - RVH
  - pulmonic stenosis with RVOT obstruction
  - overriding aorta (receives flow from both ventricles)
- Corrective procedures open RVOT and close the VSD.
- Palliative procedures anastomose a systemic artery to the pulmonary artery to increase flow (eg, Blalock-Taussig shunt → subclavian artery to PA). However, complications include pulmonary hypertension and LV overload.
- The pathophysiology of TOF depends on:
  - the degree of RVOT obstruction (↑ obstruction—↑ shunting via VSD): examples of dynamic changes that can occur include RV infundibular spasm or closure of the ductus arteriosus
  - the degree of SVR relative to the RVOT obstruction (↓ SVR—↑ shunting via VSD)
- A "tet spell" consists of acute hypoxemia and acidemia and/or syncope brought about by one or both of the above factors characterized by hyperpnea, prolonged crying, intense cyanosis, ↓ intensity of murmur. Treatment should address:
  - ↓ SVR: phenylephrine, metaraminol, norepinephrine; knee-chest position
  - infundibular spasm: beta blockers (esmolol 0.5 mg/kg)
  - ↓ respiratory drive: morphine 0.1 to 0.2 mg/kg IM (care with ↓ SVR)
  - ↑ PVR: oxygen

References

Apitz C, Webb GD, Redington AN. Tetralogy of Fallot. *Lancet* .2009;374:1462-1471.
Qu, JZ. Congenital heart diseases with right-to-left shunts. *Int Anesthesiol Clin.* 2004;42:59-72.

Tracheoesophageal Fistula

Considerations

1. Considerations for pediatric patient (see peds card)
2. High risk for pulmonary aspiration
3. Metabolic derangements including dehydration, acidosis, and hypoglycemia
4. Association with VACTERL constellation of abnormalities:
   - *V*ertebral defects
   - *A*nal atresia
   - *C*ardiac anomalies
   - *T*racheo-*E*sophageal fistulae, esophageal atresia
   - *R*enal anomalies
   - *L*imb defects (radial aplasia, syndactyly)

History

- Respiratory distress with feeding
- Inability to pass NG tube

Physical Exam

- Dehydration/hypovolemia
- Hypoxemia (SpO_2)
- Evidence of VACTERL abnormalities (see list)

Lab Tests/Imaging

- ABG
- EKG
- CXR, renal U/S, ±echocardiogram

Consults

Conflict(s)

Optimize

- IV fluids for dehydration, glucose

Goals

- Prevent further aspiration
  - consider gastrostomy under local to decompress stomach
  - no PPV prior to endotracheal tube confirmation
  - keep spontaneously breathing if possible

Options

- GA with ETT

Preop: Premed
Blood
ICU/stepdown bed: **yes**

Room Setup (special drugs/monitors)

- Preductal (right arm) arterial line for ABGs

Induction

- Intubate awake with judicious topicalization
- Maintain spontaneous ventilation
- Advance cuffed tube until just endobronchial on right side, then pull back slightly so breath sounds heard on left

Maintenance

Emergence

- Extubate awake

Disposition/Pain

Clinical Pearls

- Possible pitfalls during/after repair of TOF:
  - PTX
  - air leak with anastomosis → pneumomediastinum
  - tracheal collapse postoperatively (±inability to pass tube → immediate rigid bronchoscopy)

Reference

Krosnar S, Baxter A. Thoracoscopic repair of esophageal atresia with tracheoesophageal fistula: anesthetic and intensive care management of a series of eight neonates. *Paediatr Anaesth.* 2005;15:541-546.

Traumatic Brain Injury

Considerations

1. Emergency? Follow ACLS/ATLS guidelines
2. C-spine injury? Other injury? Full stomach?
3. *Primary injury*—initial physical injury (not modifiable); avoid *secondary injury*! Avoid hypotension, hypoxemia, fever, hyperglycemia, and hypercapnia
4. Risk factors—penetrating injury (worse than blunt), pedestrian or cyclist (worse than vehicle occupants), ejection from vehicle, increased age
5. Ensure cerebral perfusion pressure (CPP) >60 mm Hg. (CPP = MAP – ICP)
6. Reduce elevations in ICP—4 components—*reduce brain tissue*—mannitol, hypertonic saline, surgery; *reduce blood volume*—hyperventilation, elevate head of bed 30 degrees, prevent seizures; *reduce CSF*—drain; *remove pathologic lesions*—hematoma, tumor

History

- Events—mechanism of injury
- Witnessed—LOC, seizure, neuro deficit?

Physical Exam

- ABCs—C-spine collar?
- Level of consciousness? GCS?
- Vomiting?
- Pupils, focal neuro deficits

Lab Tests/Imaging

- CBC, electrolytes, glucose, CXR, ECG, head CT (noncontrast)

Consults

- Neurosurgery, NICU, trauma team as required

Conflict(s)

- Secure airway in obtunded patient and maintain cerebral perfusion pressure (CPP); avoid secondary injury to brain

Optimize/Goals

- Brain protection: maintain CPP >60 mm Hg; normocapnia (hyperventilate if herniating); avoid hyperglycemia, acidosis, and hypoxia; seizure prophylaxis
- Maintain volume and electrolytes

Options

- Typically GETA
- Beware sedating patient with TBI for imaging (think full stomach, altered mental status, loss of airway reflexes, hypoxia, and hypercapnia!)

Preop: Premed
Blood: surgery dependent
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Std monitors, Foley catheter, arterial line, large bore IV, central line as required, ICP monitor, $ETCO_2$ monitor
- Drugs: mannitol, hypertonic saline

Induction

- Controlled induction; avoid hypotension; avoid coughing/straining

Maintenance

- Balanced technique; TIVA technique superior for elevated ICP (volatile anesthetics will increase CBF >1.0 MAC)

Emergence

- Controlled, avoid hemodynamic changes; depending on neuro status, may require postoperative intub/vent

Disposition/Pain

- ICU, neuro-ICU as available

Clinical Pearl

Hyperventilate or not? Hypercapnia increases cerebral blood volume (cerebral vasodilation). Hyperventilation previously used to manage TBI. However, studies show that cerebral blood flow is *reduced* by more than 50% (!) immediately after TBI and $PaCO_2$ <30 mm Hg correlates with poorer outcome. Therefore, avoid in the first 24 hours after injury and only consider hyperventilation as a temporizing measure for signs of brain herniation (abnormal posturing, altered LOC, dilated pupils, and vomiting)

Reference

Moppett IK. Traumatic brain injury: assessment, resuscitation and early management. *BJA.* 2007;99(1):18-31.

Von Willebrand Disease

Considerations

1. Potential for excessive bleeding
2. Potential side effects of desmopressin therapy (eg, hyponatremia)
3. Avoid IM/PR routes
4. Avoid antiplatelet drugs

History

- Onset, duration, severity
- Easy bruising, gum bleeding, heavy periods
- Subtype if known

Physical Exam

- Ecchymoses, petechiae

Lab Tests/Imaging

- CBC, electrolytes (Na^+), BUN, Cr, PT/PTT
- Type & screen
- vWF/FVIII, ristocetin cofactor assay if diagnosis unsure

Consults

- Hematology

Conflict(s)

Optimize

- Rx:
  - Less severe (type 1), DDAVP 0.3 µg/kg 20 to 30 minutes preop
  - Type 2 or 3, or current bleeding? vWF/FVIII concentrates. Goal to increase factor concentrations >50% normal level
  - Platelet transfusion does not correct bleeding time but if massive blood loss, may require

Options

- Regional anesthesia ok if mild and PTT/BT close to normal

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

Induction

Maintenance

Emergence

Disposition/Pain

- No NSAIDs for pain

Clinical Pearls

- vWF is synthesized by the endothelium and platelets, and serves two roles:
  - an adhesion protein for platelets to the injured vessel wall
  - forms a noncovalent complex with factor VIII in plasma, thereby protecting it from inactivation and clearance
- Therefore, a deficiency of vWF affects both platelet plug formation and fibrin formation, leading to oozing and prolonged bleeding after surgery (typical of coagulopathies) and mucosal hemorrhages (typical of platelet disorders).
- Classification:
  - type 1 (60-80%). vWF/factor VIII at about 5 to 30% of normal levels
  - type 2 (10-30%)—qualitative defect of vWF
  - type 3 (1-5%). Low or undetectable vWF. Symptoms more severe, like hemophilia (eg, spontaneous hemorrhage)
- Diagnosis not reliable with PT/PTT or bleeding time. Use assays for vWF, FVIII, and ristocetin cofactor instead.
- Desmopressin releases endogenous vWF and FVIII into plasma, increasing levels almost immediately by two to fourfold.
- Because type 2 disease has defective vWF, desmopressin has minimal effect; in the type 2b subtype, it causes platelet aggregation and thrombocytopenia and is contraindicated.
- vWF and factor VIII levels rise spontaneously through pregnancy in type 1 disease, often reaching normal at term.

References

Mannucci PM. Treatment of von Willebrand's disease. *N Engl J Med.* 2004;351:683-694.

Michiels JJ, van Vliet HHDM, Berneman Z, Schroyens W, Gadisseur A. Managing patients with von Willebrand Disease Type 1, 2 and 3 with desmopressin and von Willebrand Factor-Factor VIII concentrate in surgical settings. *Acta Haematol.* 2009;121:167-176.

Wolff-Parkinson-White & Lown-Ganong-Levine Syndromes

Considerations

1. Congenital pre-excitation syndromes with predisposition to tachyarrhythmias
2. Tendency to rapid ventricular rates and VF with atrial fibrillation due to conduction through accessory pathway
3. Avoidance of techniques and drugs that increase HR
4. Avoidance of drugs that increase AV nodal refractory period (beta blockers, calcium channel blockers, digoxin, adenosine) ***in the setting of atrial fibrillation***

History

- H/o palpitations (induced by exercise, stress, excitement), syncope, cardiac arrest
- H/o ablations, activation-induced cell death (AICD) insertion
- Drug therapy

Physical Exam

- Heart failure

Lab Tests/Imaging

- EKG
  - *May be normal!*
  - Shortened PR (<0.12 millisecond)—look at lead V1
  - Widened QRS with delta wave (WPW). No delta wave with LGL because accessory fibers connect directly to bundle of His

Consults

Conflict(s)

Optimize/Goals

- Goals
  - avoid sympathetic stimulation, drugs producing tachycardia
  - choose early defibrillation if tachyarrhythmias, especially if atrial fibrillation, which can easily progress to VF

Options

- Consider local, regional, or general anesthesia as required

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Defibrillator in the room
- Amiodarone, adenosine, esmolol, procainamide available

Induction

- Avoid ketamine

Maintenance

- Careful with desflurane (increased HR). Isoflurane depresses conduction in AV and accessory pathways. Sevoflurane has no effect on conduction and may be the best
- Maintain good anesthetic depth to avoid sympathetic response to surgical stimulation

Emergence

Disposition/Pain

- Aggressive pain control to control HR

Wolff-Parkinson-White & Lown-Ganong-Levine Syndromes *(continued)*

Clinical Pearls

- Incidence 0.3% of general population. Sudden death occurs in 0 to 4%.
- **If an acute attack occurs:**
  (1) *for atrial fibrillation:*
    i) goal is to slow transmission rate through accessory pathway, which is directly opposite to the goal of reentrant tachycardias (slowing of AV nodal transmission).
    ii) treatment with nodal blockers (beta blockers, calcium channel blockers, adenosine, and digoxin) can result in an increase in the rate of transmission through the accessory pathway, and a corresponding increase in the ventricular rate. This can lead to severe hemodynamic compromise, and lethal ventricular arrhythmias such as VF.
    iii) if any question of stability, immediate synchronized cardioversion is the treatment of choice (remember, electricity is cheap and fast). Start with 100 J and go up from there.
    iv) If hemodynamically stable, two medications can be recommended: Procainamide 17 mg/kg at 50 mg/min maximum infusion rate, and amiodarone 150 mg. Both can cause hypotension if given too fast.
  (2) *for supraventricular tachycardias (NOT atrial fibrillation)*
    i) two therapies are commonly recommended: cardioversion and adenosine (6 mg IV push). Bear in mind that if you have misdiagnosed the rhythm, you may accidentally convert atrial fibrillation into ventricular fibrillation. The regularity or irregularity of very fast atrial rhythms can be difficult to identify. For this reason, if using adenosine in WPW, you must be prepared to perform cardioversion.

Reference

Redfearn DP, Krahn AD, Skanes AC, Yee R, Klein GJ. Use of medications in Wolff-Parkinson-White syndrome. *Expert Opin Pharmacother.* 2005;6:955-963.

Procedures

1 Airway Surgery
2 Ambulatory Surgery
3 Carotid Endarterectomy
4 Cesarean Section
5 Craniotomy
6 ECT
7 Exploratory or Open Laparotomy
8 Hepatic Resection or Transplant
9 Kidney or Pancreas Transplant
10 Laparoscopy
11 Major Joint Replacement
12 Neck Surgery
13 Nonoperating Room Anesthesia
14 Off-Pump Coronary Artery Bypass Graft Surgery
15 Open Abdominal Aortic Aneurysm Repair
16 Open Cardiac Surgery
17 Ophthalmic Surgery
18 Oral and Maxillofacial Surgery
19 Percutaneous Tracheostomy
20 Prone Position of Surgical Patient
21 Pulmonary Resection
22 Spine Surgery
23 Tonsillectomy and/or Adenoidectomy
24 Tracheal Resection
25 Trauma Resuscitation
26 TURP and Hysteroscopy
27 Vascular Surgery—Open or Endovascular Bypass

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Airway Surgery

Considerations

1. Procedures—direct laryngoscopy, fiberoptic bronchoscopy, rigid bronchoscopy, foreign body removal, laser surgery, airway stents, or airway trauma
2. Requires close communication between surgeon and anesthetist
3. If respiratory distress—surgical airway
4. High prevalence of cigarette smoking (COPD, airway tumors)
5. Options for ventilation:
   Spontaneous ventilation + local anesthesia/sedation—limited procedures that patients can tolerate
   Spontaneous ventilation + GA—for upper airway endoscopy
   Positive pressure ventilation (with small ETT) + GA—obscures surgical view but allows for standard equipment
   Jet ventilation + GA—unobstructed view but risk barotrauma
6. Lasers—risk of airway fire. Use minimal inspired oxygen concentration to maintain oxygenation and use specialized laser airway endotracheal tubes
7. TIVA useful to maintain anesthesia and/or reduce pollution

History

- Location, size, and symptoms of airway abnormality (dyspnea, hoarseness, dysarthria, aspiration, coughing)
- Tumors—medications, radiation, surgery?
- Foreign body? Often pediatric patients
- Comorbid cardiopulmonary disease
- Other—cigarette smoking, alcohol use

Physical Exam

- Vital signs
- Airway exam—stridor? Neck ROM, MP score
- Features of difficult mask ventilation—obesity, beard, or no teeth
- Cardiopulmonary exam

Lab Tests/Imaging

- Airway—imaging, bronchoscopy results, fiberoptic assessment

Consults

Conflict(s)

- Weigh pros and cons of various ventilation modes for given patient and surgical requirements

Optimize

- Positioning—protect neck, teeth, eyes
- Anesthesia—often TIVA—ensure adequate IV access

Options

- MAC/local anesthesia
- General anesthesia—spontaneous ventilation, jet ventilation, or with ETT or LMA as required

Preop:

Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Consider arterial line for hemodynamic monitoring or blood gases as required

Induction

- Depending on assessment:
  surgical airway
  AFOI
  induce but maintain spontaneous ventilation
  induce, muscle relaxants, and PPV

Maintenance

- TIVA or balanced technique ± muscle relaxants
- Short-acting agents often preferred
- Dexamethasone 10 mg IV—to reduce airway edema
- Laser surgery—protective eye equipment for patient and staff

Emergence

- Assess airway—edema or bleeding may limit ability to extubate

Disposition/Pain

- Assess re: airway edema, obstruction
- Stridor: nebulized epinephrine, heliox
- Emergency airway equipment available

References

Moorthy SS, Gupta S, Laurent B. Management of airway in patients with laryngeal tumors. *J Clin Anesth*. 2005;17:604-609.

English J, Norris A, Bedforth N. Anaesthesia for airway surgery. Continuing education in Anaesthesia. *Critical Care and Pain*. 2006; 6(1):28-31.

Considerations

1. Anesthetics with adequate recovery profile
2. Major complications (MI, stroke, PE, respiratory failure) are rare
3. Minor complications (pain, PONV, sore throat, somnolence, hypotension, hypertension, and bleeding) are common
4. Ease of transfer in case of unplanned admission? Know your surroundings—office-based procedure, freestanding ambulatory surgery center, or hospital
5. Predictors of hospital admission after ambulatory surgery: >65 years, prior inpatient admission in last 6 months, invasiveness of surgery, surgery >2 hours, general anesthesia rather than regional anesthesia
6. Unanticipated admission causes:
   Medication—complications from preexisting disease
   Surgical—direct complication, pain, bleeding
   Anesthesia—aspiration, PONV, somnolence
   Social—no escort, long distance from home
7. Controversial patients for ambulatory surgery—elderly, morbid obesity, severe OSA, significant COPD or asthma, and infants (risk of apnea if postconceptual age <60 weeks)
8. ENT, urology, and generally surgery—higher rates of unanticipated admission

History

- Planned procedure and symptoms
- Active upper respiratory tract infection?
- Any changes from baseline function?
- Comorbid cardiopulmonary disease, cardiac stents in last 12 months?
- Predictors for unanticipated admission?
- Fasting status?

Physical Exam

- Vital signs, airway, BMI
- Cardiopulmonary exam

Lab Tests/Imaging

Consults

- Primary care physician as required

Conflict(s)

- Appropriate patient selection for ambulatory surgery is controversial. All members of team (anesthesia, surgery, and nursing) must be comfortable with role and level of support before undertaking procedure

Optimize

- Pain—orthopedics and plastic surgery highest risk
  Peripheral nerve blocks as appropriate
  Multimodal analgesia—acetaminophen, NSAIDs, opioids, adjuncts (gabapentin, dexmedetomidine, ketamine) as required
- Anesthesia: propofol, sevoflurane, remifentanil may have pharmacologic advantage
  LMA reduces incidence of sore throat versus ETT
- PONV—risks: female, nonsmoker, PH, intraoperative opioids. Rx: multimodal—5-HT_3 antagonists, TIVA, dexamethasone, avoid N_2O, minimize opioids, minimize neostigmine, adequate hydration

Goals

- Rapid recovery and discharge desirable
- Systems to treat common complications (pain, PONV)

Options

- General anesthesia—supraglottic airways (LMA) may have faster recovery versus ETT
- Neuraxial, PNB, or MAC/local as appropriate

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

Induction

- Propofol—fast recovery, reduce PONV

Maintenance

- Balanced technique. Short-acting agents or TIVA often preferred
- Fentanyl—reduced postdischarge nausea/vomiting versus morphine
- Avoid N_2O—PONV risk

Emergence

- Assess if able to bypass directly to phase II recovery
- Phase I recovery—"early"—until recovery of protective reflexes and motor function
- Phase II recovery—"intermediate"—patient ready for discharge home (assess vital signs, activity, PONV, pain, and bleeding)

Disposition/Pain

- Assess re: discharge—pain score, PONV, bleeding, vital signs, level of activity
- Appropriate follow-up instructions, ensure escort home

References

Fleisher LA, Pasternak LR, Lyles A. A novel index of elevated risk of inpatient hospital admission immediately following outpatient surgery. *Arch Surg*. 2007;142(3):263-268.

Gan TJ, Meyer TA, Apfel CC, et al. Society for Ambulatory Anesthesia guidelines for the management of postoperative nausea and vomiting. *Anesth Analg*. 2007;105(6):1615-1628.

Carotid Endarterectomy

Considerations

1. Risk of stroke from plaque at carotid bifurcation
2. Definite benefit for symptomatic patient (TIA or stroke) with >70% stenosis. Benefits less certain for asymptomatic patients
3. Open carotid endarterectomy—internal, external, and common carotid arteries are cross-clamped (heparinization)
4. Carotid artery stenting (CAS) alternative to CEA. Risk/benefit still emerging. May have higher early stroke risk
5. Cerebral monitoring—stump pressure (>40-50 mm Hg), EEG (raw or processed), SSEPs, transcranial doppler, or cerebral oximetry
6. Shunting—blood from common carotid to internal carotid across cross-clamp. Use varies from routine to selective if cerebral ischemia. Risks—air/plaque emboli, intimal tears, carotid dissection
7. Procedure risks—stroke, MI, cranial nerve injury, hematoma, airway edema
8. Anesthesia—regional or general anesthesia. No difference in rate of death or stroke. Surgeon and patient preference determine anesthetic technique
9. Increased cerebral blood flow postop can cause hyperperfusion syndrome of headache, hypertension, seizures, and neurologic deficits

History

- Neurologic history—stroke, TIA—residual symptoms
- Comorbid conditions: hypertension, atherosclerosis, CAD, cigarette smoking, diabetes
- Uncontrolled hypertension—higher risk of postoperative stroke
- If regional technique—assess for communication barrier or history of claustrophobia or anxiety

Physical Exam

- Blood pressure
- Neurologic deficits
- Cardiopulmonary exam
- If regional technique—assess ability to tolerate surgical position (supine, shoulder roll, head turned to side)

Lab Tests/Imaging

- Cardiac testing: ECG, echocardiogram, stress test as required

Consults

Conflict(s)

- GA provides controlled environment but local anesthesia or cervical plexus block allows direct neurologic monitoring of patient

Optimize

- Blood pressure
- Avoid cerebral ischemia—note change in mental status if awake or change in neurologic monitoring if GA
  Treatment—shunt, increase blood pressure, maintain oxygenation, and normocarbia

Goals

- Hemodynamic stability
- Monitor neurologic status

Options

- General anesthesia with endotracheal tube (LMA also described)
- Regional anesthesia—superficial cervical plexus block. Deep cervical plexus block—no added benefit. (Cervical epidural also described but higher complication rate)
- Local anesthesia

Preop: Premed: anxiolytic/sedative
Blood: cross-match
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Arterial line
- Medications—heparin, vasopressors, vasodilators
- Cerebral monitor

Induction

- Propofol may offer neuroprotection

Maintenance

- Balanced technique (volatile anesthetics may offer neuroprotection)
- N_2O—will preserve EEG signal compared to propofol or volatile anesthetic
- If GA—often require phenylephrine infusion to maintain MAP
- ECG—ST-segment analysis, maintain normocarbia
- Cross-clamp often associated with hypertension

Emergence

- Assess airway re: hematoma, airway edema

Disposition/Pain:

- Hypotension and hypertension common
- Monitor for wound hematoma
- Monitor for hyperperfusion syndrome—treat hypertension
- postoperative MI—monitor

Clinical Pearls

Regional anesthesia—cervical plexus formed from C1-C4. At transverse processes of C2-C4 divide and form deep and superficial branches. Deep
branches—motor. Superficial branches—sensory. Superficial block—needle inserted at the mid-point of the posterior border of the sternocleidomastoid muscle (Erb's point).

References

Howell SJ. Carotid endarterectomy. *Br J Anaesth*. 2007;99(1):119-131.
Guay J. Regional anesthesia for carotid surgery. *Curr Opin Anaesthesiol.* 2008;21:638-644.

Considerations

1. Either elective or emergency
2. Any immediate threat to life of mother or fetus is an emergency (eg, maternal hemorrhage with sustained fetal bradycardia). Aim for delivery <30 minutes
3. Two patients potentially at risk: mother and fetus
4. Labor pain: uterus/cervix T10 to L1, perineum (pudendal nerve) S2 to S4. Blockade to T4 for cesarean section
5. Options: regional (epidural, CSE, spinal) or general
6. Contraindications to regional include coagulopathy, uncorrected hypovolemia, infection at needle puncture site, and increased ICP
7. Side effects of regional include hypotension, high or total spinal, infection, bleeding, PDPH, and nerve injury
8. For failed labor epidural requiring C-section—either GA or CSE (with *low* dose given risks of high spinal)
9. If GA—neonatal respiratory depression will require management

History

- Complete prenatal history
- Comorbid conditions, preexisting neurologic symptoms, coagulation disorders
- Previous C-sections show any associated complications with regional anesthesia
- Last meal

Physical Exam

- Airway exam
- Assess re: hypovolemia—vital signs, urine output, mucous membranes, blood loss
- Lumbar spine
- Sites for IV access
- Fetus—fetal heart rate

Lab Tests/Imaging

- Hemoglobin; PT/PTT/INR as required

Consults

- Pediatrics or neonatology for neonatal resuscitation

Conflict(s)

- Regional anesthesia may avoid airway manipulation of mother but emergency scenario may require GA

Optimize

- Preload—1 L IV crystalloid as required
- Maintain MAP: phenylephrine infusion (± bolus)
- Prevention of aspiration pneumonitis—regional anesthesia, antacids, H_2 antagonists
- Fetal resuscitation:
  Stop oxytocin, left lateral position, oxygen, IV crystalloid, treat hypotension, tocolysis: terbutaline 250 µg IV/SC or nitroglycerin 400 µg SL as required
- Blood products: type and cross-match as required

Options

- GETA/RSI
- Spinal—hyperbaric bupivacaine 12.5 mg, preservative-free morphine 100 to 250 µg
- Top-up of existing epidural catheter

Preop: Premed: aspiration prophylaxis

Room Setup (special drugs/monitors)

- Pediatrician or neonatologist for neonatal resuscitation
- Emergency airway equipment

Induction

- RSI with cricoid pressure—propofol/succinylcholine commonly used

Maintenance

- If GA: balanced technique (volatile anesthetics will decrease uterine tone); maintain MAP
- Oxytocin immediately after delivery (5-10 units IV bolus then 10 units/h for at least 4 hours)
- Monitor re: postpartum hemorrhage

Emergence

- Aspirate stomach contents with OG tube before extubation

Disposition/Pain

- Postoperative analgesia—intrathecal/epidural opioids or PCA

Clinical Pearls

- Spinal-induced hypotension:
  - ephedrine associated with lower umbilical artery pH, more nausea, and higher incidence of hypotension compared to phenylephrine
  - start phenylephrine infusion prophylactically or as soon as systolic BP decreases 10% (suggested starting doses of phenylephrine range from 50 to 100 µg/min IV with titration to effect)

References

Hawkins JL. Epidural analgesia for labor and delivery. *NEJM.* 2010;362:1503-1510.
Levy DM. Emergency caesarean section: best practice. *Anaesthesia.* 2006;61:786-779

Craniotomy

Considerations

1. Brain accessed by removing bone flap from skull. For a variety of procedures including tumor resection, aneurysm clipping, vascular malformation repair, and trauma
2. Awake craniotomy—epilepsy, stereotactic biopsy or requiring functional or electrophysiology testing (deep brain stimulation for Parkinson disease or tumors near essential areas)
3. Risks: neurologic injury, seizures, infection nausea/vomiting, bleeding, increased ICP, air embolus
4. Volatile anesthetics—affect cerebral autoregulation and ICP
5. Head pinning—adequate depth of anesthesia, immobility
6. Brain relaxation (decrease ICP) for neurosurgery:
   Position—elevate head of bed 15 to 30 degrees
   TIVA—propofol/remifentanil versus volatile anesthetics
   Hyperventilation—$PaCO_2$ ~ 30 mm Hg
   Mannitol—0.25 to 0.5 mg/kg IV
   Dexamethasone—10 mg IV
   CSF drainage
   EEG—additional propofol until burst suppression
7. Rapid assessment postprocedure—anesthetics and analgesics with rapid recovery profile

History

- Neurologic history—symptoms and deficits
- Comorbid conditions
- If awake craniotomy planned—anxiety, abnormal movements, claustrophobia

Physical Exam

- Mental status
- Neurologic deficits
- Cardiopulmonary exam
- If awake technique—assess ability to tolerate surgical position

Lab Tests/Imaging

- CBC

Consults

- Depending on facility and procedure, may require specialized personnel for electrophysiology testing

Conflict(s)

- GA provides controlled environment but local anesthesia allows direct neurologic monitoring of patient

Optimize

- Maintain cerebral perfusion pressure:
  CPP—MAP–ICP (or jugular venous pressure if greater) normal CPP ~80 mm Hg
  May require fluids or phenylephrine infusion to maintain MAP
- Brain relaxation (decrease ICP) for neurosurgery
- Seizure prophylaxis as required—fosphenytoin 15 to 20 PE mg/kg
- Maintain normoglycemia, avoid hypercapnia
- Awake: avoid obstruction, hypoventilation, and loss of cooperation/disinhibition

Goals

- Hemodynamic stability, reduce cerebral metabolism, preserve cerebral autoregulation, avoid increases in ICP, and rapid recovery

Options

- General anesthesia with endotracheal tube (LMA also described for certain procedures—may allow for "wake-up test" without provoking airway reflexes)
- Local anesthesia and sedation for awake craniotomy

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Arterial line
- Adequate IV access for TIVA

Induction

- Propofol may offer neuroprotection
- Maintain CPP

Maintenance

- TIVA or balanced technique $\pm$ N_2O (0.5-1.0 MAC volatile anesthetic). TIVA superior if elevated ICP
- Patients on chronic anticonvulsant therapy may be resistant to nondepolarizing muscle relaxants—assess TOF
- If awake—avoid hypoventilation and apnea
- Cerebral aneurysm—may require mild hypothermia (benefit not proven), temporary clipping (and induced hypertension), or deep hypothermic circulatory arrest (for giant aneurysms)

Emergence

- Ensure ability to protect airway, limit coughing/straining
- If unable to extubate may perform wake-up test to assess neurologic status

Disposition/Pain

- PACU or ICU

Clinical Pearls

- Nitrous oxide and neuroprotection—N_2O is a NMDA antagonist (potentially neuroprotective) but in some animal studies N_2O is neurotoxic. To date, clinical research suggests that relevant concentrations of N_2O are not neurotoxic. N_2O may not be desirable as it increases cerebral blood flow, cerebral metabolic rate, and ICP.
- Hyperventilation ($PaCO_2$ ~30 mm Hg) can improve surgical operating conditions and decrease ICP. However, hyperventilation not recommended for initial treatment of acute subarachnoid hemorrhage or traumatic brain injury unless signs of transtentorial herniation (no benefit and excessive hyperventilation can cause cerebral ischemia).

References

Cole CD, Gottfried ON, Gupta DK, et al. Total intravenous anesthesia: advantages for intracranial surgery. *Neurosurgery*. 2007;61: S369-S378.

Bonhomme V, Franssen C, Hans P. Awake craniotomy. *European Journal of Anaesthesiology.* 2009;26:906-912.

Haelewyn B, David HN, Rouillon C, et al. Neuroprotection by nitrous oxide: facts and evidence. *Crit Care Med.* 2008;36(9):2651-2659.

Considerations

1. For schizophrenia, unipolar depression, bipolar depression, and acute mania
2. Polypharmacy: antidepressant, antipsychotic medications
3. Anesthetics and seizure threshold—lidocaine, benzodiazepines, and most induction agents inhibit seizure duration
4. No absolute contraindication to ECT but increased risk of ECT with:
   Uncontrolled hypertension
   Decompensated heart failure
   Recent MI/unstable angina
   Severe valvular disease
   Elevated ICP or intracranial aneurysms
   Pheochromocytoma
   Uncontrolled hyperthyroidism
   Pregnancy
5. Seizures: initial parasympathetic bradycardia followed by sympathetic tachycardia, hypertension, and increased myocardial oxygen demand
6. Contraindications to succinylcholine?
7. Risk of cognitive side effects (amnesia)—higher risk with dementia or preexisting brain injury
8. Usually multiple ECT treatments—note previous anesthetic and any complications

History

- Psychiatric history and treatments
- Previous ECT treatments, complications
- Comorbid conditions with particular attention to cardiac disease

Physical Exam

- Mental status
- Airway—difficult mask ventilation?
- Cardiopulmonary exam

Lab Tests/Imaging

- CBC, electrolytes, urea/creatinine, ECG
- Further cardiac testing depending on comorbidities

Consults

Conflict(s)

- Brief procedure (10 minutes) with significant cardiovascular changes. Need to weigh risk and benefit in high-risk patients

Optimize

- Avoid cardiac complications:
  - pretreatment with anticholinergic (atropine 0.4-0.8 mg IV or glycopyrrolate 0.2-0.4 mg IV) to avoid severe bradycardia
  - pretreat with esmolol 0.5 to 1.0 mg/kg or labetalol 20 to 30 mg IV to blunt sympathetic response (may decrease seizure duration)
- Use a bite block to avoid tongue injury during seizure
- If prolonged seizure—use midazolam to break

Goals

- Hemodynamic stability and rapid recovery

Options

- General anesthesia, muscle relaxant, and mask ventilation

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Anticholinergics, beta blockers

Induction

- Use pretreatments and titrate induction agent to unconsciousness (smaller dose—longer seizure)
  Methohexital 0.75 to 1.5 mg/kg
  Consider propofol, etomidate, ketamine as appropriate
- Propofol more stable hemodynamics than methohexital
- Muscle relaxant—succinylcholine 0.5 to 1.5 mg/kg unless contraindicated

Maintenance

- Mask ventilate with 100% oxygen, hyperventilate to increase seizure duration
- Document muscle relaxation
- Insert oral bite block
- Seizure initiated (usually 30-90 seconds)
- Maintain adequate ventilation (hypoxia and hypercarbia aggravate hypertension and tachycardia after seizure)
- Monitor hemodynamics, ECG

Emergence

- Ensure ability to protect airway, adequate strength

Disposition/Pain

- PACU

References

Pandya M, Pozuelo L, Malone D. Electroconvulsive therapy: what the internist needs to know. *Cleve Clin J Med*. 2007;74(9):679-685.
Saito S. Anesthesia management for electroconvulsive therapy: hemodynamic and respiratory management. *J Anesth.* 2005;19:142-149.

Exploratory or Open Laparotomy

Considerations

1. Wide range of potential indications (pain, bleeding, trauma, cancer, infection, vascular malformations, autoimmune, metabolic, toxic, or congenital conditions)
2. Wide range of procedures (bowel, hepatobiliary, spleen, pancreas, kidney, adrenals, ureters, bladder, vasculature, uterus, ovaries)
3. Range of acuity from elective, outpatient procedure to emergency procedure in critically ill
4. Position—supine, lateral, lithotomy?
5. Incision—subcostal, midline?
6. Aspiration risk—consider RSI
7. Regional anesthesia—may reduce postop CV and pulmonary complications and decrease postop ileus
8. Sepsis—acute organ dysfunction secondary to infection; septic shock—sepsis + hypotension
9. Abdominal compartment syndrome—increased intra-abdominal pressure (>25 mm Hg) and organ dysfunction (oliguria, hypoxia, acidosis). Consider trauma, bleeding, tense ascites, or intestinal obstruction
10. Organ injury—cardiac, lung, liver, or kidney failure
11. Hemorrhage—adequate IV access, blood products
12. Glycemic control—monitoring and insulin therapy

History

- HPI—acuity, pain score, symptoms, treatments to date
- Comorbid disease
- Previous abdominal surgery (scarring/adhesions can make dissection difficult and increase blood loss)

Physical Exam

- Vital signs, mental status—need for acute resuscitation?
- Cardiopulmonary exam
- Volume assessment—mucous membranes, skin turgor, tachycardia, postural hypotension, decreased urine output
- Sites for IV access, central access

Lab Tests/Imaging

- CBC, electrolytes, ECG, CXR, ABG, serum lactate (if sepsis), coagulation, blood cultures as required

Consults

- Critical care

Conflict(s)

- Critically ill patients may require resuscitation before safe induction of anesthesia

Optimize

- Resuscitation as required: MAP >65 mm Hg, CVP 8 to 12 mmHg, UO >0.5 cc/kg/h, SvO_2>65%
- Volume infusion, blood products, vasopressors, or inotropes as required to meet goals
- Antibiotic coverage—broad spectrum, follow susceptibilities
- Septic shock—hydrocortisone (100 mg IV) for adult if poor response to therapy
- Protective ventilation strategy—Tv 6 cc/kg, plateau pressure <30 cm H_2O, with PEEP to avoid lung collapse
- Maintain glucose <150 mg/dL (8.3 mmol/L)
- DVT prophylaxis—heparin or LMWH unless contraindicated

Goals

- Maintain resuscitation goals and allow for surgical management of underlying cause

Options

- General anesthesia with endotracheal intubation, often RSI
- Regional anesthesia (adjunct to GA or sole anesthetic) depending on clinical scenario and if no contraindications

Preop: Premed
Blood: cross-match
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Arterial line ± central line, PAC, TEE as required and Foley catheter
- Vasopressors, inotropes, insulin, blood products, steroids, antibiotics, ± CVVHD as required

Induction

- GA/ETT with RSI. Avoid induction hypotension

Maintenance

- Balanced technique
- Monitor and maintain resuscitation goals

Emergence

- Assess airway—keep intubated if required

Disposition/Pain

- ICU as required

References

Dellinger RP, Levy MM, Carlet JM, et al. Surviving Sepsis campaign: International guidelines for management of severe sepsis and septic shock: 2008. *Crit Care Med.* 2008;36(1):296-327.

Hanna MN, Murphy JD, Kumar K, et al. Regional techniques and outcome: what is the evidence? *Curr Opin Anaesthesiol*. 2009;22:672-677.

Hepatic Resection or Transplant

Considerations

1. Orthotopic liver transplant (OLT) for acute or chronic hepatic failure (often hepatitis B or C or alcoholic cirrhosis)
2. Liver resection for hepatic metastases, cholestatic diseases, or donation for hepatic transplantation
3. Liver failure is a multi-system disease:
   CV—hyperdynamic circulation, increased CI, LVH, PHTN
   Respiratory—restrictive defect (ascites), pleural effusion, shunting (hepatopulmonary syndrome)
   Renal—hepatorenal syndrome, ATN
   Metabolic—hyponatremia, hypomagnesemia, hyperkalemia, metabolic acidosis, hypoglycemia
   Hematology—reduced synthesis of vitamin K-dependent factors, DIC, anemia, thrombocytopenia
   CNS—encephalopathy, cerebral edema
4. Risk of massive bleeding and hemorrhage
5. Surgical maneuvers to reduce hepatic inflow (and reduce bleeding): *Pringle maneuver*—clamp portal vein and hepatic artery—decrease CO and increase afterload. *Total hepatic vascular occlusion*—clamp supra- and infra-hepatic IVC, portal vein and hepatic artery—hypotension and decrease CO (up to 60%)
6. Postoperative liver failure—jaundice, encephalopathy, coagulopathy ~72 hours postsurgery
7. Liver transplant—immunosuppressants (steroids)

History

- Assess presenting condition—hepatitis B or C, alcohol cirrhosis, acetaminophen overdose, hepatic tumor, or cholestatic diseases (primary biliary cirrhosis, etc)
- Nausea, fatigue, diarrhea, bleeding, pruritus
- Comorbid disease—cardiac, respiratory, renal failure
- Previous abdominal surgery (scarring/adhesions can make dissection difficult and increase blood loss)

Physical Exam

- Stigmata of liver failure: encephalopathy, ascites, jaundice, scleral icterus, spider angioma, palmar erythema, gynecomastia, and asterixis
- Cardiopulmonary exam
- Sites for IV access, central access

Lab Tests/Imaging

- CBC, electrolytes, albumin, bilirubin, PT, INR, ECG, CXR, echocardiogram, cardiac stress testing as required

Consults

- Cardiology, intensive care, or neurosurgery as required

Conflict(s)

- Secure airway quickly and safely (often require RSI) but also avoid induction hypotension (significant ascites)

Optimize

- CV—maintain low CVP (goal <5 mm Hg), support MAP with vasopressors, consider piggyback technique (avoids total hepatic vascular occlusion) or venovenous bypass if cardiac disease, avoid transplant if MPAP >35 mm Hg
- Respiratory—drain large pleural effusions, PEEP
- Renal—consider CVVHD for hyperkalemia, renal failure
- Metabolic—aggressively treat hyperkalemia, monitor and treat hypo-/hyperglycemia, hyponatremia
- Hematology—treat coagulopathy, maintain normothermia, consider tranexamic acid or aminocaproic acid or FVIIa as required
- CNS—consider ICP monitoring; maintain CPP >60 mm Hg (norepinephrine, mannitol, hypertonic saline)
- Reduce ischemia-reperfusion injury—consider *N*-acetylcysteine

Options

- General anesthesia with endotracheal intubation, often RSI
- Regional anesthesia combined with GA (single-shot spinal opioids or continuous epidural) depending on clinical scenario and if no contraindications (coagulopathy)

Preop: Premed: no
Blood: cross-match for PRBC, FFP, platelets as required
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Large bore IVs, arterial line ± central line, PAC, TEE as required, and Foley catheter
- Vasopressors, inotropes, insulin, blood products, antibiotics, ± immunosuppressants, and CVVHD as required
- Rapid transfusion device and cell salvage as indicated

Induction

- GA/ETT with RSI. Avoid induction hypotension

Maintenance

- Balanced technique. Avoid N_2O (gas embolus)
- Monitor and maintain anesthetic goals
- Reperfusion hypotension—DDx: acidosis, hyperkalemia, hypocalcemia, and hypovolemia
  Rx: hyperventilation, sodium bicarbonate, insulin, calcium chloride, blood products, epinephrine

Emergence

- Assess airway—keep intubated if required

Disposition/Pain

- ICU as required
- Monitor for bleeding, liver, renal, or metabolic dysfunction
- Avoid acetaminophen/paracetamol

Clinical Pearls

Hyperkalemia can cause fatal ventricular fibrillation. It requires aggressive treatment (see "Hyperkalemia") and close monitoring (ECG), particularly immediately after reperfusion of transplanted liver. If hyperkalemia is an issue but ongoing blood loss requires blood transfusion, the potassium load can be minimized by having the PRBCs washed.

References

Hartog A, Mills G. Anaesthesia for hepatic resection surgery. Continuing education in Anaesthesia. *Critical Care and Pain.* 2009;9(1):1-5.
Ozier Y, Klinck JR. Anesthetic management of hepatic transplantation. *Cur Opin Anaesthesiol.* 2008;21:391-400.

Kidney or Pancreas Transplant

Considerations

1. Procedure: kidney transplant, simultaneous kidney and pancreas (SPK), pancreas after kidney (PAK), or pancreas alone transplant (PAT)
2. Pancreas transplant—treatment of DM
3. DDx of renal failure—prerenal, renal, or postrenal causes
4. ESRD comorbidities—DM, CAD, CHF, HTN, anemia, dyslipidemia, infections (Hep B/C), hypercalcemia, hyperphosphatemia
5. Diabetic complications: infections, hypoglycemia, hyperglycemia, DKA, nonketotic hyperosmolar state, CAD, hypertension, stroke, nephropathy, retinopathy, neuropathy
6. Succinylcholine—avoid in ESRD with hyperkalemia (may increase K^+ 0.5 mEq/L and induce arrhythmias)
7. Altered pharmacokinetics/pharmacodynamics of medications due to ESRD
8. Immunosuppressed—meticulous aseptic technique
9. Volume assessment in patients with little or no urine output—may require invasive monitors

History

- Assess underlying condition—DM, etc
- Symptoms of ESRD: asymptomatic, fatigue, weakness, confusion, nausea, vomiting, paresthesias, dyspnea, pruritus, nocturia, reduced urine output
- Comorbid disease
- Previous abdominal surgery (scarring/adhesions can make dissection difficult and increase blood loss)

Physical Exam

- ESRD: altered mental status, neuropathies, hyperreflexia, seizures, weight loss, HTN, dependent edema, orthostatic hypotension
- Sites for IV access, central access

Lab Tests/Imaging

- CBC, urea/creatinine, electrolytes, glucose, CXR
- ECG, echocardiogram, cardiac stress testing

Consults

- Nephrology or cardiology as required

Conflict(s)

- Avoid transplant if active infection, recent MI, malignancy, or incomplete work-up of comorbid conditions

Optimize

- CV—maintain CVP (10-15 mm Hg) with crystalloids (LR). Balance graft perfusion with risk of pulmonary edema due to volume overload. Use invasive monitors as required
- Metabolic—monitor and treat hyperkalemia, hypoglycemia, or hyperglycemia
- Kidney transplant—mannitol and furosemide often used to promote diuresis
- Pharmacokinetics of drugs:
  Avoid medications with renally excreted active metabolites (morphine, meperidine)
  Vecuronium and rocuronium may have prolonged effect—monitor TOF or use cisatracurium

Options

- General anesthesia with endotracheal intubation, often RSI
- Regional anesthesia combined with GA (continuous epidural) depending on clinical scenario and if no contraindications (coagulopathy)

Preop: Premed: aspiration prophylaxis as required
Blood: cross-match for PRBC
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Large bore IVs, arterial line ± central line, and Foley catheter
- Antibiotics, immunosuppressants (steroids, monoclonal antibodies, calcineurin inhibitors, etc)
- Insulin, dextrose, vasopressors, and inotropes

Induction

- GA/ETT with RSI as required

Maintenance

- Balanced technique. Avoid N_2O (bowel distension, gas embolus)
- Monitor glucose (q 15 minutes at time of pancreatic anastomosis—hold insulin, may require dextrose infusion)
- Reperfusion of kidney—iliac vessels unclamped leading to decreased SVR and BP ± hyperkalemia

Emergence

- Assess airway—keep intubated if required

Disposition/Pain

- PACU or ICU as required
- Monitor for bleeding, metabolic dysfunction
- Avoid NSAIDs for kidney transplant patients
- TPN for pancreas-transplant patients

Clinical Pearls

Fluid replacement for renal transplant recipients is controversial. It is often suggested to avoid potassium-containing fluids (such as lactated Ringer) and use normal saline. However, normal saline is hypernatremic and hyperchloremic. Large volumes of NS (>20-30 cc/kg) can cause a hyperchloremic metabolic acidosis and worsen hyperkalemia! In renal transplant recipients, LR is associated with less hyperkalemia and acidosis compared to NS.

References

Sarin Kapoor H, Kaur R, Kaur H. Anaesthesia for renal transplant surgery. *Acta Anaesthesiol Scand.* 2007;51:1354-1367.

Pichel AC, Macnab WR. Anaesthesia for pancreas transplantation. Continuing education in Anaesthesia. *Critical Care and Pain.* 2005;5(5):149-152.

O'Malley CM, Frumento RJ, Hardy MA, et al. A randomized, double-blind comparison of lactated Ringer's solution and 0.9% NaCl during renal transplantation. *Anesth Analg.* 2005;100(5):1518-1524.

Laparoscopy

Considerations

1. Advantages—reduced pain, reduced hospital stay, improved postoperative respiratory function
2. CO_2 gas commonly used for pneumoperitoneum (not air or oxygen—fire). Gasless laparoscopy relies on abdominal wall lift to create space
3. CO_2 absorption across peritoneum causes hypercapnia. Venous gas embolus also possible
   Hypercapnia activates SNS—hypertension, tachycardia, arrhythmias, increased myocardial contractility
4. Pneumoperitoneum—increases intra-abdominal pressure (IAP):
   Cardiovascular—hemodynamic effects depend on patient position and IAP
   IAP <15 mm Hg—increased venous return, increased CO, hypertension
   IAP >15 mm Hg—decreased venous return, decreased CO, hypotension
   Bradycardia reported due to vagal stimulation (trocar placement, peritoneal stretch or CO_2 embolus)
   Respiratory—reduced lung volumes, increased PAP, V/Q mismatch, pneumothorax, pneumomediastinum
   Neurologic—increased ICP
   Renal/hepatic—decreased blood flow

History

- Comorbid cardiopulmonary disease
- Previous abdominal surgery (scarring may preclude laparoscopic technique)

Physical exam

- Vital signs
- Cardiopulmonary exam

Lab Tests/Imaging

- Cardiac and pulmonary testing as required (ECG, echocardiogram, stress test; CXR, ABG, PFTs)

Consults

Conflict(s)

- Laparoscopy may have distinct advantages over open procedure (particularly recovery) but patients with cardiopulmonary disease may not tolerate pneumoperitoneum

Optimize

- Optimize preload, afterload changes—volume infusion, positioning, and insufflation pressures
- Head-up position and laparoscopy—reduced venous return, CO, and hypotension. Head-down position and laparoscopy worsens respiratory function
- Release pneumoperitoneum if hypoxemia, hypercapnia, or elevated PAP—consider conversion to open surgery

Goals

- Frequently ambulatory surgery—rapid recovery and discharge desirable
- Compensate for changes induced by pneumoperitoneum

Options

- General anesthesia—usually endotracheal intubation but supraglottic airways (LMA) described for selected laparoscopic procedures (appropriate patient selection with low insufflation pressures and limited Trendelenburg position)
- Spinal/epidural anesthesia also an alternative (patients adjust minute ventilation)
- PNB as adjunct for analgesia

Preop: Premed
Blood: cross-match
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Consider arterial line for hemodynamic monitoring or blood gases and Foley catheter for urine output
- Atropine available if marked bradycardia

Induction

Maintenance

- Balanced technique. Short-acting agents often preferred
- Avoid N_2O—limits surgical view due to diffusion into bowel and diffusion may pose risk of fire
- May have significant gradient between $ETCO_2$ and $PaCO_2$ (consider ABG)
- Monitor PAP—beware pneumothorax

Emergence

- Assess airway—risk of extraperitoneal insufflation of CO_2 and subcutaneous emphysema (crepitus over chest wall/neck)

Disposition/Pain

- Early postoperative—higher $ETCO_2$ leads to increased RR. Patients with impaired baseline respiratory function may be at higher risk of respiratory failure
- Postoperative shoulder pain common (secondary to diaphragm irritation)

Clinical Pearls

Robotic surgery is a specialized form of laparoscopy with the same general considerations. The robot is under the control of the surgeon who sits at the operator console. There are a variety of potential robotic procedures including: general surgery, cardiothoracic surgery, urology (prostatectomy), and
gynecology (hysterectomy). Special considerations for robotic procedures include:

1. Depending on the surgeon's experience—anticipate a long-duration procedure.
2. Positioning:
   - Once the robot is in place the OR table cannot be changed and access to the patient is generally limited. Disengagement of the robot is required if rapid access to the patient is required.
   - Given length of procedure and fixed position, take care to avoid peripheral nerve injuries due to compression and position to avoid crush injuries by robotic arms.
3. Cardiothoracic procedures—one-lung ventilation mandatory.

Reference

Gerges FJ, Kanazi G, Jabbour-Khoury SI. Anesthesia for laparoscopy: a review. *J Clin Anesth.* 2006;18:67-78.

Considerations

1. Elderly patient (physiologic changes, concurrent disease, ↑ volume of distribution, ↑ risk hypothermia)
2. Potentially large blood loss (esp. hip)
3. Implications with arthritis (OA vs. RA, side effects from drugs, eg, NSAIDs, steroids)
4. Potentially difficult regional anesthetic
5. ↑ risk DVT/PE postoperatively
6. Use of methylmethacrylate cement → ↓ BP

History

- Effects of OA, RA
- Drug history and duration; side effects
- Exercise tolerance

Physical Exam

- Neck mobility, mouth opening (RA)
- Examine spine

Lab Tests/Imaging

- CBC, coags
- EKG if elderly
- ± Flexion/extension C-spine films if ++ RA disease

Consults

Conflict(s)

- Regional (↓ blood loss, DVT) versus GA (less hypotension, secured airway if long surgery in lateral position)

Optimize

- Consider preoperative multimodal po regimen to ↓ postop opioid use. For example:
  - oxyContin 20 mg po
  - acetaminophen 1000 mg po
  - celecoxib 200 mg po
  - gabapentin 600 mg po

Goals

- Provide quality pain control postoperatively that facilitates rehabilitation

Options

- GA
- Neuraxial (spinal, epidural, CSE)
- Peripheral nerve blocks/catheters for postop pain

Preop: Premed: ± steroid cover
Blood: 2 units
ICU/stepdown bed

Room Setup (special drugs/monitors)

- ± Difficult airway cart

Induction

- If severe RA with neck involvement, consider awake FOB intubation

Maintenance

Emergence

Disposition/Pain

- Perineural catheters (see Clinical Pearls Section)
- Continue multimodal regimen × 48 hours

Clinical Pearls

Postoperative regional anesthesia regimen for TKA

- Femoral nerve catheter. Initial bolus—20 mL of 0.2% ropivacaine, followed by patient-controlled perineural infusion. Initial settings: 0.2% ropivacaine, 5 mL/h, 5 mL bolus q 45 minutes (may be fine-tuned at later time)
- ± Sciatic nerve block with 15 to 20 mL of 0.2% ropivacaine

Postoperative regional anesthesia regimen for THA

- If primary THA, no nerve blocks required. Multimodal analgesia only
- If revision THA, consider single shot lumbar plexus block (20 mL 0.2% ropivacaine) or continuous catheter → initial bolus—20 mL of 0.2% ropivacaine, followed by patient-controlled perineural infusion. Initial settings: 0.2% ropivacaine, 5 mL/h, 5 mL bolus q 45 minutes (may be fine-tuned at later time). This decision may be influenced by the regimen of low- molecular weight heparin used, that is if twice-daily prophylaxis postop, many clinicians are uncomfortable placing/using a lumbar plexus catheter (vs. once-daily dosing)

Reference

Hebl JR, Dilger JA, Byer DE, et al. A pre-emptive multimodal pathway featuring peripheral nerve block improves perioperative outcomes after major orthopedic surgery. *Reg Anesth Pain Med.* 2008;33:510-517.

Neck Surgery

Considerations

1. Frequently shared airway with remote access to head of bed
2. Possible difficult airway (vocal cord palsy, h/o prior neck surgery, radiation rx, infection/abscess, tumor)
3. Potential need for postoperative PPV due for airway protection (edema, laryngeal nerve palsies)
4. Considerations for cancer if present (chemotherapeutic agents, local compressive effects, distant mets, endocrine effects)
5. Considerations for endocrine disease (thyroid/parathyroid) if present

History

- H/o disease process
- Stridor, hoarseness, dysphagia, dysphonia
- H/o OSA, COPD

Physical Exam

- Meticulous airway examination
- C-spine range of motion

Lab Tests/Imaging

- Review MRI/CT neck
- Review ENT report of office laryngoscopy (cord function) if available

Consults

Conflict(s)

Optimize

Goals

- Safe securing of airway
- Ablate reflexes to prevent hemodynamic response/movement → short-acting opioids such as remifentanil ideal
- If monitoring cranial nerve function (VII, XI) will need to avoid paralysis

Options

- GA (ETT vs. supraglottic device)
- Cervical plexus block for surgery or for postoperative pain control

Preop: Premed: ±antisialogogue (eg, glycopyrrolate)
Blood
ICU/stepdown bed: possibly

Room Setup (special drugs/monitors)

- ±Difficult airway cart, bougies, LMAs, smaller ETTs, video laryngoscope, tracheostomy set

Induction

- If doubt about airway, awake FOB intubation safest; if IV induction first, consider look without paralysis first or use succinylcholine

Maintenance

- Remifentanil 0.1 to 0.2 µg/kg/min + volatile, ±NDMR

Emergence

- Smooth emergence/extubation to ↓ bleeding
- Consider leak test to ensure patent natural airway

Disposition/Pain

- Ensure adequate antiemetic prophylaxis
- Careful postop airway evaluation before discharge home

Reference

Bonner S, Taylor M. Airway obstruction in head and neck surgery. *Anaesthesia.* 2000;55:290-291.

Considerations

1. Remote locations often considerable distance from other anesthesiologists if help is needed
2. Potentially limited working space
3. Potential lack of skilled nursing or technical personnel, drugs, or resuscitation equipment, especially in office setting
4. Unique hazards such as magnetic field in MRI suite, moving C-arms in cardiac catheterization lab
5. Often remote access to airway (eg, MRI/CT scanner, dental anesthesia)
6. Often behavioral issues (eg, dementia, claustrophobia, mentally challenged) leading to request for anesthesia services
7. Patients commonly children

History

- Careful history for airway obstruction, snoring
- Ferrous implants if MRI (ICDs, surgical clips)

Physical Exam

Lab Tests/Imaging

Consults

Conflict(s)

- Unfamiliar environment versus need to maintain (often very sick) patients in state of semiconsciousness for long periods of time (eg, ERCP for acute cholecystitis: often ill, obese, prone position, unsecured airway)

Optimize

- Decide if patient should have procedure in main OR, if possible (eg, endoscopy, ERCP, facelift)
- Ensure environment is safe for provision of anesthesia (see Clinical Pearls section)
- Patients (especially children) often require preop sedation—assess and provide prn

Goals

- Prevent the primary cause of remote location injuries: adverse respiratory events. Remain extremely vigilant

Options

- Sedation/GA/RA

Preop: Premed: anxiolysis
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- MRI compatible equipment if necessary (pumps, laryngoscopes, ventilators, etc)
- Lipid emulsion for resuscitation of local anesthetic systemic toxicity at any location regional anesthesia is performed (eg, labor and delivery)

Induction

Maintenance

- Often TIVA or intermittent boluses

Emergence

Disposition/Pain

- To monitor recovery area with same standards as main OR recovery area; transport with resuscitation drugs/equipment

Clinical Pearls

Ensure that the following recommendations are adhered to (from ASA guidelines for nonoperating room anesthetizing locations):

1. Reliable oxygen source including a backup supply
2. Adequate and reliable suction
3. Adequate and reliable scavenging system if anesthetic gases are to be used
4. Self-inflating resuscitation bag capable of delivering an inspired oxygen fraction (FiO_2) of 0.90
5. Adequate drugs, supplies, and equipment for the planned activity
6. Adequate monitoring equipment to adhere to standards for basic anesthetic monitoring
7. Sufficient electrical outlets, isolated electric power or electric circuits with ground fault interruption in "wet areas" like cystoscopy, arthroscopy, labor and delivery suites, with access to emergency power supply
8. Sufficient space for equipment and personnel and transportation
9. Immediate suitability of an emergency cart with defibrillator, emergency drugs, etc
10. Reliable two-way communication
11. Observation of all applicable building and safety codes and facility standards
12. Appropriate postanesthesia management

References

American Society of Anesthesiologists. "Statement on Nonoperating Room Anesthetizing Locations." (2008). https://www.asahq.org/For-Healthcare-Professionals/Standards-Guidelines-and-Statements.aspx. Accessed 3/31/2011.

Melloni C. Anesthesia and sedation outside the operating room: how to prevent risk and maintain good quality. *Curr Opin Anaesthesiol* . 2007;20:513-519.

Off-Pump Coronary Artery Bypass Graft Surgery

Considerations

1. CAD and considerations related to primary disease
2. Avoidance of potential adverse effects of CPB including SIRS, coagulopathy, ↑ stress hormone release, and cerebral, myocardial, pulmonary, and renal impairment
3. Avoidance of potential adverse effects related to aortic cross-clamp including atheroembolization
4. Occasional sudden and profound hypotension associated with surgical manipulation of the beating heart
5. Difficulty in monitoring for ischemia with traditional monitors such as SE-segment changes or TEE due to positional changes of the heart
6. Potential for intraoperative dysrhythmias

History

- Coexisting diseases: smoking, HTN, DM, COPD, obesity
- Duration/pattern of CAD, anginal class, dyspnea, exercise tolerance, MI, antianginal meds
- Previous PTCA/stents, CABG, valvular repair

Physical exam

- Evidence of left or right heart failure
- S3 or S4

Lab Tests/Imaging

- CBC, electrolytes, coags, glucose
- EKG, stress echo
- Review cath report

Consults

- Cardiology

Conflict(s)

Optimize

- To avoid severe hypotension from heart displacement:
  - volume load with crystalloid/blood
  - Trendelenburg position → ↑ preload
  - alpha agonists (eg, phenylephrine, NE)
- If ↓ LV function preop, consider placement of IABP after induction to ↑ cerebral/coronary perfusion and ↓ LV work

Goals

- Teamwork and communication with surgical team
- Stable hemodynamics, prevention of myocardial ischemia
- Early extubation and ambulation (analgesia)

Options

- GA alone or combined with RA (thoracic epidural, paravertebral blocks); RA alone has been described

Preop: Premed: anxiolysis
Blood: two units
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Arterial line, CVP, ± PAC, TEE
- SvO_2 may be most reliable monitor of CO
- Must have CPB available as backup

Induction

- Slow, smooth; treat ↑ HR with esmolol ± phenylephrine if ↓ BP

Maintenance

- Volatile or TIVA, opioids; epidural

Emergence

- Extubation in OR early in ICU stay (1-3 hours)

Disposition/Pain

- ICU
- IVPCA opioids ± NSAIDS; regional anesthesia (thoracic epidural, paravertebral blocks, intrapleural analgesia) is also an excellent option

Clinical Pearls

Benefits of OPCAB compared to CABG using CPB

- Shorter ICU and in-hospital stay
- Less bleeding and need for transfusion

Reference

Virmani S, Tempe DK. Anaesthesia for off-pump coronary artery surgery. *Ann Card Anaesth*. 2007;10:65-71.

Open Abdominal Aortic Aneurysm Repair

Considerations

1. Vasculopathy with multiple coexisting diseases (eg, HTN, DM, CAD, CVA, CHF, COPD, PVD)
2. Potential for aortic rupture at any time → severe hypovolemia requiring immediate resuscitation
3. Potential for massive blood loss, coagulopathy, and hypothermia
4. Complications related to aortic cross-clamping including renal and gut hypoperfusion, cardiac failure, and (rarely) spinal cord ischemia
5. High risk for postoperative complications, such as myocardial ischemia, acute kidney failure

History

- History of coexisting disease (see considerations)
- Smoking, complications from diabetes

Physical Exam

- Careful volume status exam
- Carotid bruits
- Signs of right or left heart failure
- Pneumonia, wheezing

Lab Tests/Imaging

- CBC, electrolytes, coags, glucose
- EKG (ischemia, LVH)
- Noninvasive testing for ischemia (no evidence of ↑ risk for rupture with dobutamine stress test)

Consults

- Cardiology ± pulmonology

Conflict(s)

- Full stomach versus need to perform smooth, careful induction

Optimize

- Smoking cessation, bronchodilators/antibiotics prn for COPD
- If preop evidence of myocardial ischemia and ↑ HR/BP, consider starting preoperative β blockers slowly (over weeks)

Goals

- Anticipate the effects of aortic clamping/unclamping
- Preserve myocardial, cerebrospinal, renal perfusion
- Maintain adequate cardiac output
- Prevent rupture of aorta

Options

- GA alone + IV PCA
- GA + thoracic epidural (↓ pain, duration of mechanical ventilation, MI, gastric and renal complications)

Preop:

Premed: anxiolysis
Blood: 4 units cross-matched
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Arterial line, CVP, ±TEE for cardiac output and monitoring for ischemia (PAC usually not indicated)
- Rapid infusor, cell salvage
- Vasodilators/pressors (eg, NTG, SNP, phenylephrine, NE)

Induction

- Smooth, careful titrated induction; if full stomach, *modified* RSI to prevent hypertensive response

Maintenance

- Volatile + O_2; epidural morphine 3 to 5 mg

Emergence

- Extubate in OR if hemodynamic/metabolic status ok
- Esmolol prn to ↓ HR, BP

Disposition/Pain

- ICU

Clinical Pearls

Physiologic effects of aortic clamping and unclamping

- Effects depend largely on level of clamp (suprarenal, infrarenal), baseline myocardial function, volume status, and effect of anesthetic drugs on myocardial and vascular tone

Aortic cross-clamping (generally) leads to:

- ↑ BP proximal to the clamp
- ↑ LV wall stress → LV failure if unable to meet O_2 demands
- Redistribution of blood from tissues distal to the clamp to tissues proximal to the clamp
  - if clamp supraceliac, blood from the splanchnic vasculature redistributes to heart → ↑ preload
  - if clamp sub-celiac, blood volume may shift to heart or to the splanchnic circulation, depending on the vascular tone of the splanchnic vasculature, therefore may see ↑ or ↓ in preload
- Little change in HR
- ↓ CO or no change in CO
- ↑ Coronary blood flow → ↑ contractility if myocardial O_2 supply adequate

Goals for management of aortic cross-clamping:

- ↓ Afterload (arteriolar vasodilators, eg, nitroprusside) prior to clamp (goal SBP~90 mm Hg)
- Normalize preload (may require venodilators), contractility, and coronary blood flow

Aortic unclamping leads to:

- ↓ BP, contractility (acidosis, cytokines), venous return, pH

Goals for management of aortic unclamping:

- Fluid load with crystalloid and turn off vasodilators
- Pretreat with small doses of vasopressors to raise SBP >140 mm Hg; gradual release of clamp. Communicate with surgeon re: need to reapply clamp if BP ↓↓

References

Wozniak MF, LaMuraglia GM, Musch G. Anesthesia for open abdominal aortic surgery. *Int Anesthesiol Clin.* 2005;43:61-78.

Nishimori M, Ballantyne JC, Low JH. Epidural pain relief versus systemic opioid-based pain relief for abdominal aortic surgery. *Cochrane Database Syst Rev.* 2006;19:CD005059.

Open Cardiac Surgery

Considerations

1. Procedure—coronary artery bypass graft, valve repair/replacement, ASD/VSD repair, heart/lung transplant, aortic root or arch replacement
2. "Open" procedures require CPB (off-pump and endovascular techniques also described for many procedures)
3. No "standard" anesthetic for cardiac operations—know considerations for underlying condition
4. One lung ventilation—if approach via thoracotomy
5. CPB—requires anticoagulation (heparin)
6. Risks: stroke, cognitive dysfunction, paralysis, peripheral nerve injury, coagulopathy, hemorrhage, air emboli, atheroemboli, organ failure—heart, lungs, liver, kidney
7. Temperature changes—deliberate hypothermia or deep hypothermia circulatory arrest. Avoid hyperthermia on rewarming
8. Electrolytes changes—sodium, potassium, calcium, magnesium, glucose
9. Cardioplegia—hyperkalemic solutions
10. Spinal drain—CSF drain for thoracoabdominal aortic surgery

History

- Cardiopulmonary disease: CAD, cardiomyopathy, left or right heart failure, valvular disease, cardiac tamponade, pulmonary hypertension
- Functional status
- Medications optimized? Beta blockers, alpha blockers, other antihypertensives, antiarrhythmics, anticoagulants, diuretics, inotropes, pulmonary vasodilators
- Reoperation? Increased bleeding risk

Physical Exam

- Vital signs, airway/dentition (re: TEE placement)
- Cardiopulmonary exam
- Access sites for peripheral and invasive monitors

Lab Tests/Imaging

- CBC, electrolytes, urea/creatinine, glucose, PT/PTT/INR
- Cardiac and pulmonary testing: ECG, echocardiogram, stress test; CXR, ABG, PFTs as required

Consults

- cardiology

Conflict(s)

- Depending on preoperative functional status: patient may be at high risk of perioperative complications. Informed patient consent required

Optimize

- Hemodynamics for underlying condition
- Avoid arrhythmias—optimize electrolytes, have pacing and defibrillator available (internal and external)
- Anticoagulation for CPB:
  Heparin 300 U/kg IV. Check ACT. Optimal ACT for CPB unknown but ACT 300 to 400 generally acceptable for initiation of CPB

Goals

- Hemodynamic stability; avoid hypertension, hypotension, arrhythmias, and cardiovascular depression
- Maintenance of temperature, electrolytes, and adequate systemic anticoagulation for CPB

Options

- General anesthesia with endotracheal intubation (DLT required for some procedures)
- Spinal/epidural anesthesia—hematoma risk—limited use

Preop: Premed: anxiolytic/sedative
Blood: cross-match: yes
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Arterial line, central line, PAC—place preinduction as appropriate, and TEE
- Medications—heparin, vasopressors, inotropes, vasodilators, antiarrhythmic agents
- Cardiac pacing, defibrillator
- Temperature: PAC—blood, nasopharyngeal or tympanic—brain, esophageal, rectal or bladder—core temperature

Induction

- Choice of agents depends on hemodynamic goals

Maintenance

- High-dose opioids to blunt sympathetic reflexes
- N_2O—generally avoid (increases size of gas emboli)
- Adjust anesthetic, hemodynamics, and ventilation for key events—sternotomy, chest wall retraction, cannulation of aorta, initiation of CPB, rewarming during CPB, separation from CPB, sternal wiring

Emergence:

- Postoperative intubation/ventilation common

Disposition/Pain

- Opioid-based anesthetic—prolonged recovery
- Transport to ICU with monitors

Clinical Pearls

Weaning from CPB:

- Stable rhythm (pacemaker available)
- Resuscitation medications available—inotropes, vasopressors, vasodilators
- Adequate anesthesia and paralysis
- Normothermia
- Normal electrolytes, glucose, ABG
- Acceptable hemoglobin
- Normal SVR
- Re-zero pressure transducers
- TEE—clearance of air
- Ventilation with 100% oxygen and re-expand lungs
- Protamine sulfate available to reverse heparin (1 mg protamine will neutralize approximately 100 U heparin)

Heparin resistance:

- Need for higher than normal doses of heparin to reach sufficient anticoagulation for CPB
- Differential diagnosis includes: ATIII deficiency, sepsis, thrombocytosis, and preoperative heparin therapy (heparin shortens ATIII half-life).
- Treatment: increase dose of heparin, ATIII replacement, FFP (contains ATIII), or consider alternatives to heparin such as direct thrombin inhibitors (argatroban, bivalirudin)
- Note—heparin alternatives required for patients with heparin-induced thrombocytopenia (HIT)

Reference

Savino J Si , Floyd T Fi , Cheung A Ti . Cardiac Anesthesia. In: Cohn LH, Edmunds LH Jr, eds. *Cardiac Surgery in the Adult*. New York: McGraw-Hill, 2003:249-281.

Considerations

1. Elderly patient (physiologic changes, concurrent disease, ↑ volume of distribution, ↑ risk hypothermia)
2. Complications related to peribulbar and retrobulbar blocks:
   - brainstem anesthesia
   - seizures
   - retrobulbar/peribulbar hemorrhage
   - globe penetration
   - retinal vascular occlusion
   - ocular myotoxicity → ptosis
3. Potential for oculocardiac reflex, especially in children (bradycardia/junctional rhythms)
4. If open eye injury, concern regarding anesthetic-related ↑ in IOP and extrusion of globe contents
5. ↑ Risk of postoperative nausea and vomiting with strabismus surgery

History

- Comorbidities: HTN, DM, COPD
- Ability to lie flat for duration of procedure
- ? Chronic cough

Physical Exam

Lab Tests/Imaging

- If cataract surgery → none

Consults

Conflict(s)

- Open eye and full stomach: succinylcholine facilitates RSI and prompt control of airway but ↑ IOP

Optimize

- Ensure blood pressure and glucose are within normal range for patient

Goals

- Prevent ↑ IOP and extrusion of globe contents if open eye: coughing/bucking, vomiting, hypercapnia, hypoxia, ± succinylcholine (controversial as to degree of increase)

Options

- GA with ETT (if full stomach, open eye injury, or unable to lie flat); LMA for pediatric strabismus surgery
- Regional/topical anesthesia (elective)

Preop: Premed: anxiolysis; GI prophylaxis if full stomach (care with metoclopramide → oculogyric crisis)
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Preparation for GA even if sedation planned
- Atropine prepared for oculocardiac reflex

Induction

- Pre-O_2 (care with face mask and eye)
- GA and open eye: smooth, modified RSI with rocuronium 1.2 mg/kg. Ensure deep plane before laryngoscopy (lidocaine/esmolol aid in blunting response)

Maintenance

- Avoid hypercapnia

Emergence

- Decompress stomach prior to awakening; smooth emergence and extubation

Disposition/Pain

- PACU

Reference

McGoldrick KE, Foldes PJ. General anesthesia for ophthalmic surgery. *Ophthalmol Clin North Am.* 2006;19:179-191.

Considerations

1. Potential difficult airway due to congential syndromes (eg, Treacher Collins, Pierre-Robin, Goldenhar), facial injury/burn, C-spine fusion, tumors, neck radiation, TMJ dysfunction
2. Shared airway with remote access to head
3. Presence of OSA due to chronic obstruction
4. Potential for large blood loss (ie, >1.5 L)
5. Need for nasal intubation and potential complications (epistaxis, turbinate fracture, submucosal dissection)
6. Hypotensive anesthetic technique and potential ischemic complications

History

- History of OSA?
- Stridor, hoarseness, dysphagia, dysphonia
- Trauma? associated injuries?

Physical Exam

- Meticulous airway examination
- Signs of RV failure, pulmonary HTN if OSA

Lab Tests/Imaging

- CBC

Consults

- May be team approach with plastics, neurosurgery, ENT

Conflict(s)

- Surgeon desires for hypotensive anesthesia versus potential for hypoperfusion of critical organs

Optimize

- If OMF trauma review trauma records and determine status of cervical spine
- If severely distorted airway, consider preoperative elective tracheostomy

Goals

- Safe securing of airway
- Prevent excessive bleeding: consider deliberate hypotension if no contraindication (mean BP of 50-65 mm Hg in healthy or 80 mm Hg in elderly)

Options

- GA (usually nasal ETT $\longrightarrow$ phenylephrine or oxymetazoline nasal drops help prevent bleeding)

Preop: Premed: ±antisialogogue (eg, glycopyrrolate)
Blood
ICU/stepdown bed: possibly

Room Setup (special drugs/monitors)

- ± Difficult airway cart
- ± A-line if controlled hypotension

Induction

- If doubt about airway, awake FOB intubation safest; if IV induction first, consider look without paralysis first or use succinylcholine

Maintenance

- Remifentanil 0.1 to 0.2 µg/kg/min + volatile, ±NDMR

Emergence

- Consider leak test to ensure patent natural airway
- Remove packing from throat

Disposition/Pain

- Frequently require PPV for swelling to subside
- If jaw wired shut, wire cutters MUST be present at bedside at all times

Reference

Krohner RG. Anesthetic considerations and techniques for oral and maxillofacial surgery. *Int Anesth Clin.* 2003; 41:67-89.

Percutaneous Tracheostomy

Considerations

1. Coexisting problems relating to critical illness: infection (pneumonia, sepsis), traumatic injuries, brain injury
2. Shared airway and potential for loss of airway during procedure
3. Unfamiliar environment (ICU)
4. Potential complications from procedure:
   - bleeding
   - creation of false passage
   - subcutaneous emphysema
   - pneumothorax
   - esophageal puncture
   - tracheal tube transfixion

History

- Indication for tracheostomy: reason for respiratory failure (emergent indications are usually treated with cricothyrotomy, ie, complete obstruction)
- Current treatment
- Ventilator settings (plateau pressure, PEEP, FiO_2)

Physical Exam

- Rales, wheezes, bilateral air entry
- Careful examination of neck tissues

Lab Tests/Imaging

- ABG
- CXR
- Coags

Consults

Conflict(s)

- Risk of airway fire (electrocautery) with 100% O_2 versus risk of hypoxia during procedure with FiO_2 <100%

Optimize

- Ensure no active respiratory issues; bronchodilators, anticholinergics, steroids, antibiotics prn
- Preprocedure suctioning

Goals

- Work together with surgeon to secure long-term airway in safe and expedient manner (see Clinical Pearls section)

Options

- GA

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Oxygen, suction, BVM, device for monitoring $ETCO_2$
- Cricothyrotomy kit available
- Supraglottic airway device (eg, LMA) bedside

Induction

- 100% oxygen for 2 to 3 minutes prior
- IV induction with propofol (watch BP), NDMR

Maintenance

- TIVA propofol, opioid, ventilation with BVM 100% O_2

Emergence

- Remains on ventilator

Disposition/Pain

- Maintain sedation until NMB worn off
- Continue suctioning

Clinical Pearls

Stepwise approach to anesthesia for percutaneous tracheostomy

- Position IV bag between scapulae, neck extended, head resting on head ring
- Aspirate NG tube and clear oropharynx
- Surgeon begins to scrub and prepare patient
- Ensure adequate sedation and paralysis
- When surgeon is ready to perform needle tracheostomy, switch to hand ventilation, deflate cuff, and pull ETT back slowly until cuff just below level of cords. Reinflate → surgeon should be able to confirm position by feeling for cuff through trachea
- After needle insertion, twist ETT clockwise and counterclockwise to rule out needle transfixion of ETT
- During dilation, ↑ PIP may be required to ventilate. Use capnography to verify ventilation
- After dilation, surgeon places tracheostomy tube. Detach BVM from ETT and ventilate tracheostomy tube, ensuring adequate chest rise, bag compliance, and $ETCO_2$ on capnography. Once confirmed, remove ETT
- If patient is prematurely extubated during procedure, and laryngoscopy is difficult/impossible, patient should be hand-ventilated with 100% O_2 while a supraglottic device (eg, LMA) is prepared and inserted. The procedure can be completed with this in place provided adequate oxygenation and ventilation. If not, fiberoptic bronchoscopy should be performed via the LMA and a #6.0 ETT inserted into the trachea
- Postoperative CXR to exclude pneumothorax and to check position

Reference

Groves DS, Durbin CG Jr. Tracheostomy in the critically ill: indications, timing and techniques. *Curr Opin Crit Care.* 2007;13: 90-97.

Prone Position of Surgical Patient

Considerations

1. Physiologic changes:
   Cardiovascular—decreased CO, increased SVR and PVR (increased intrathoracic pressure and abdominal compression leading to impaired venous return)
   Respiratory—increased FRC, may decrease V/Q mismatching!
2. Positioning injuries—pressure points (genitals, iliac crests, chest, forehead/chin), nerve injuries (direct pressure or stretch)
3. Visual loss—majority of cases *not* associated with direct pressure on globe. Possible causes: hypotension , elevated venous pressure, anemia, length of procedure (>6 hours), and direct compression on globe
4. Loss of airway prone—secure airway before turning and have stretcher immediately available if need to turn back supine emergently
5. Informed patient consent of risks of prone position

History

- Preexisting visual defect?
- Preexisting neurologic deficit? Paresthesias, numbness, weakness?
- Comorbid cardiopulmonary disease

Physical Exam

- Neurologic exam
- Assess ROM in limbs and neck

Lab Tests/Imaging

- Hemoglobin

Consults

- Consider ophthalmology consult if undocumented visual defect

Conflict(s)

- Turning patient prone is physically demanding. Requires appropriate personnel and technique, must avoid disruption of lines and monitors

Optimize

- Hemodynamics—ensure abdomen not compressed
- Avoid peripheral nerve injury with proper positioning—inspection, adequate padding, avoid abdominal compression, arms <90 degree abducted
- Avoid visual loss—no direct pressure on eyes, limit blood loss/anemia, maintain blood pressure, avoid induced hypotension, and keep surgery <6 hours
- Avoid neurologic injury—avoid excessive neck movement and keep neck in neutral position to avoid carotid/vertebral artery occlusion, cervical spine injury
- Avoid air embolus—more common in Neuro SX

Options

- Typically GETA but consider spinal or MAC/local depending on type of procedure

Preop: Premed
Blood: depending on type of surgery
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Specialized OR table (Jackson table) as required
- Adequate padding, bolsters, prone face padding

Induction

- Induce supine on stretcher then turn prone
- Ensure adequate vascular access before turning

Maintenance

- Balanced technique but alterations may be required for SSEP or MEP monitoring (spinal procedures)
- Ensure eyes are not directly compressed (document)

Emergence

- Prone position associated with facial/airway swelling due to dependent edema. May require period of postoperative ventilation

Disposition/Pain

- PACU or ICU as required

Clinical Pearls

Postoperative visual loss (POVL):

POVL is rare. Prone spinal surgery may have a higher risk of POVL. The two most common types of injuries are central retinal artery occlusion (CRAO) and ischemic optic neuropathy (ION). Direct pressure on the globe can lead to central retinal artery occlusion. Inadequate oxygen delivery to the optic nerve can lead to ION. This can occur even if there is no direct pressure on the globe (prone position and head pinned). Perfusion of the optic nerve—MAP—intraocular pressure or venous pressure (whichever is greater). Prone position can cause increased intraocular pressure and increased venous pressure. MAP may decrease during prone procedures due to hypotension or decreased CO from abdominal pressure. This explains the need to maintain perfusion pressure and to avoid anemia, hypotension, and increased venous pressure.

Reference

Edgcombe H, Carter K, Yarrow S. Anaesthesia in the prone position. *Br J Anaesth.* 2008;100 (2):165-183.

Considerations

1. Considerations relating to lung cancer: mass effects, airway narrowing, postobstructive pneumonia, metastases, chemo, Eaton-Lambert syndrome, hypercalcemia
2. Complications related to removal of bronchopulmonary segments and vasculature: pulmonary hypertension and right heart failure, dysrhythmias, pulmonary edema
3. Lateral position: potential nerve/eye/brachial plexus injuries
4. Anticipation of arterial hypoxemia with one-lung ventilation
5. Moderate-to-severe pain that interferes with pulmonary recovery

History

- Coexiting diseases: smoking, COPD, dyspnea, orthopnea, exercise tolerance
- Chemo: bleomycin (lung toxicity)

Physical Exam

- Respiratory rate, wheezing, rales
- Cyanosis, clubbing, evidence of SVC syndrome
- Room air SpO_2

Lab Tests/Imaging

- CBC, electrolytes, Ca^{++}, ±ABG if COPD
- Review CXR and CT scan (difficult placement of DLT due to tracheobronchial distortion?)
- PFTs, ±V/Q scan if ppoFEV_1<40%

Consults

- Pulmonology

Conflict(s)

- Difficult airway versus need for large DLT
- Hypoxemia and need to inflate operative lung versus interference with surgical exposure

Optimize

- Risk assessment (see Clinical Pearls section)
- Preop physio: cough/deep breathing, incentive spirometry
- Smoking cessation
- Prescribe/continue bronchodilator rx ± steroids

Goals

- Maintain lung separation and oxygenation
- Prevent postoperative pulmonary complications

Options

- GA + thoracic epidural (TEA) or paravertebral block (PVB)

Preop: Premed: antisialogogue for FOB
Blood: two units
ICU/stepdown bed: yes

Room Setup (special drugs/monitors)

- Arterial line, difficult airway cart with FOB

Induction

- Standard. DLT (37-39 Fr for males, 35-37 Fr for females)

Maintenance

- Volatile in 100% + epidural/PVB analgesia
- May require CPAP on operative lung ± minimal PEEP to nonoperative lung for hypoxemia
- Adjust TV to keep PIP <35 cm H_20

Emergence

- See algorithm in the Clinical Pearls section

Disposition/Pain

- Regional anesthesia (TEA or PVB) decreases postop respiratory complications
- No NSAIDs if on cis-platinum (renal toxicity)

Clinical Pearls

Risk assessment for pulmonary resection

It is useful to utilize the "three-legged stool" method of preop assessment for pulmonary resection after Slinger and Johnston.

1. *Respiratory mechanics*
   - most valid single test for postthoracotomy respiratory complications is the predicted postoperative FEV_1% ($ppoFEV_1$%). Calculated as: preop FEV_1% (predicted FEV_1%) x (1—the % functional lung tissue removed/100)
   - to estimate amount of tissue removed, remember: RUL = 6 segments, RML = 4 segments, RLL = 12 segments; LUL and LLL are 10 segments each. ($\therefore$ 42 segments total)
   - **$ppoFEV_1$ <30 to 40%** are associated with respiratory complications and need for mechanical ventilation postoperatively
2. *Lung parenchymal function*
   - **$ppoD_{LCO}$ <40%** predicted is associated with an increased risk of both cardiac and respiratory complications (and is independent of the FEV_1)
3. *Cardiopulmonary interaction (exercise testing)*
   - VO_2 max correlates well with risk of morbidity and mortality but may be impractical
   - stair-climbing is a useful marker: <2 flights is high risk
   - an objective test is the 6-minute-walk test: patients walk as far as they can (eg, back and forth down a hallway of known distance) for 6 minutes. **Distance <2000 ft** correlates with an increase in morbidity and mortality

If patient has $ppoFEV_1$ >40%, should be possible to extubate in the operating room provided the patient is awake, comfortable, and warm

If $ppoFEV_1$ is between 30% and 40%, and lung parenchymal function and exercise testing are above the increased risk thresholds, extubation should be possible provided minimal coexisting disease (eg, CAD, dysrhythmias, COPD)

If $ppoFEV_1$ is between 20% and 30% and parenchymal function/exercise testing is favorable, early extubation can be considered if thoracic epidural or paravertebral analgesia is used. Otherwise, patients should be transported to the ICU for mechanical ventilation and staged weaning

Reference

Slinger PD, Johnston MR. Preoperative assessment for pulmonary resection. *J Cardiothor Vasc Anesth.* 2000;14:202-211.

Considerations

1. Same considerations and planning as **"Prone Surgery"** (certain spinal procedures can have anterior approach)
2. Neurophysiologic monitoring may include: visual evoked potentials (VEPs), auditory evoked potentials (AEPs), somatosensory evoked potentials (SSEPs), and motor evoked potentials (MEPs)
3. Spinal cord function frequently assessed with combination of SSEPs and MEPs:

   SSEPs—dorsal columns (posterior spinal artery)
   MEPs—corticospinal tract (anterior spinal artery)
4. MEPs—risk tongue laceration with contraction of facial muscles—use a bite block
5. Potential for significant blood loss and venous air embolus
6. Cervical spine—instability may require AFOI and careful positioning
7. TIVA often required for optimal neuro monitoring

History

- Preexisting neurologic deficit? Paresthesias, numbness, weakness?
- Comorbid cardiopulmonary disease

Physical Exam

- Airway exam—neck ROM
- Neurologic exam
- Assess ROM in limbs and neck
- Sites for vascular access

Lab Tests/Imaging

- Hemoglobin

Consults

- Depending on monitoring, may require specialized personnel for neurophysiologic testing

Conflict(s)

- Optimal anesthetic for neurophysiologic monitoring may not be optimal anesthetic given patient's comorbidities

Optimize

- Anesthetic: TIVA (propofol and remifentanil) $\pm$ N_2O or dexmedetomidine or low-dose volatile anesthetic
- Neuromuscular blocking agent—MEPs may preclude use
- Blood loss—adequate IV access, type, and cross-match, invasive hemodynamic monitors
- Air embolus—vigilance in monitoring (hypotension, decreased $ETCO_2$), vasopressors, and inotropes available, consider placement of central line re: aspiration
- avoid hypothermia—suppresses MEP readings

Goals

- Maintain anesthetic goals while at the same time minimize interference with neurophysiologic testing

Options

- GETA

Preop: Premed
Blood: depending on type of surgery
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Arterial line $\pm$ central line and Foley catheter
- Fluid warmers, blood transfusion set
- Neurophysiologic monitors—SSEP/MEP as required
- Vasopressors

Induction

- AFOI—if unstable cervical spine

Maintenance

- Often TIVA (consider use of EEG monitor to assess depth)
- Avoid muscle relaxants (or minimize based on TOF)

Emergence

- May have significant facial edema—assess feasibility of extubation
- May require wake-up test if unable to extubate

Disposition/Pain

- PACU or ICU as required

Clinical Pearls

Anesthetic effects on SSEP and MEP monitoring:

Key—maintain stable anesthetic as a sudden change may make SSEP or MEP interpretation difficult
Most IV anesthetics—decrease amplitude and increase latency of SSEPs and MEPs (dose-dependent). Etomidate and ketamine increase amplitude of SSEPs and MEPs
Volatile anesthetics—decrease amplitude and increase latency of SSEPs (dose-dependent), low doses abolish transcranial MEPs
N_2O—decreases amplitude and increases latency of SSEPs (dose-dependent), dose-dependent decrease of transcranial MEPs
Opioids—minimal effects on SSEPs and MEPs
Muscle relaxants—little effect on SSEPs but prevent recording of MEPs
Optimal anesthetic for SSEPs and MEPs—TIVA (propofol, remifentanil) without N_2O, volatile anesthetics, or muscle relaxants (Propofol causes a dose-dependent decrease in MEP and SSEP amplitude, but is more stable than volatile anesthetics)

References

Sloan TB, Heyer EJ. Anesthesia for intraoperative neurophysiologic monitoring of the spinal cord. *J Clin Neurophysiol*. 2002;19(5):430-443.

Gonzalez AA, Jeyanandarajan D, Hansen C, et al. Intraoperative neurophysiological monitoring during spine surgery: a review. *Neurosurg Focus*. 2009;27(4):E6.

Wang AC, Khoi DT, Etame AB, et al. Impact of anesthesia on transcrnail electric motor evoked potential monitoring during spine surgery: a review of the literature. *Neurosurg Focus*. 2009;27(4):E7.

Tonsillectomy and/or Adenoidectomy

Considerations

1. Indication for surgery: presence of obstructive sleep apnea → risk of postop obstruction
2. Shared airway
3. ↑ Likelihood of concurrent upper respiratory tract infection (URTI)
4. Potential for tube kinks or disconnection with mouth gag and surgical manipulation of the head/neck
5. Potential for disguised bleeding (child swallows blood from pharynx) postoperatively

History

- Snoring, OSA, mouth breathing
- Current URTI? Fever? Nasal congestion? Rhinorrhea?
- Failure to thrive if right heart failure
- History of easy bruising or bleeding?

Physical Exam

- Evidence of pulmonary hypertension & cor pulmonale

Lab Tests/Imaging

- CBC
- ± CXR, EKG if severe OSA

Consults

- Pediatric cardiologist if severe OSA

Conflict(s)

- Current URTI versus desire to proceed

Optimize

- If OSA and evidence of pulmonary HTN or erythrocytosis, consider delaying to institute weight loss, CPAP; weigh against benefit of proceeding

Goals

- Smooth emergence with no coughing/straining
- Prevent nausea and vomiting:
  - two or three agent prophylaxis including dexamethasone and a 5-HT_3 antagonist is indicated
- Reduce risk of postop bleeding
  - control hypertension → good analgesia
- Avoid NSAIDs

Options

- GA (with ETT vs. flexible LMA) LMA reduces the incidence of postop stridor, laryngospasm, and desaturation due to protected glottis and trachea

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

- Oral RAE tubes (cuffed)

Induction

- Mask induction in child; IV in adult
- ± Muscle relaxation (not strictly necessary)
- Be prepared for airway obstruction → oral airway

Maintenance

- OSA patients sensitive to opioids (use small doses of fentanyl or codeine); acetaminophen IV/PR

Emergence

- Suction stomach before wakening; remove throat pack
- Ensure smooth emergence: lidocaine/esmolol IV

Disposition/Pain

- To PACU for observation for at least 6 hours (1-degree bleeding); children <2 years → ↑ risk of airway complications → consider overnight admission

Clinical Pearls

Post-tonsillectomy bleeding is the most-feared complication of this procedure. Classified as primary (<24 hours and due to inadequate hemostasis) or secondary (usually 5-10 days postop and likely due to sloughing of the eschar and reexposure of blood vessels).

Considerations

1. Hypovolemia (complicated by fact that IV access may have been removed or lost ⟶ IVs may now be difficult to reestablish with hypovolemia)
2. Full stomach ± aspiration if inhaled blood
3. Airway concerns: swollen tissue, obscured by bleeding
4. Patient who is in pain, bleeding, and agitated ⟶ uncooperative

Management

1. Recognize that this is an emergency situation and needs prompt intervention
2. Quickly scan vitals and assess patient:
   - BP (lying and sitting), HR, RR, SpO_2
   - assess volume depletion: skin temperature and color, capillary refill, mental status changes, pulse pressure
   - quickly review anesthetic and PACU records and quantify blood loss (if possible ⟶ much of the blood is usually swallowed in children)
3. Optimize:
   - two large bore IVs if possible: send blood for stat CBC, coags and cross-match 2 to 4 units
   - volume resuscitate with 20 mL/kg IV crystalloid bolus
   - talk to surgeon: can packs/ice help? Is an anesthetic necessary?
   - glycopyrrolate 10 µg/kg for secretions
   - ± DDAVP 0.3 mg/kg IV if on ASA/NSAIDs
   - transport to OR with 100% O_2, lateral position in Trendelenburg
4. In OR, need:
   - two suctions (blood clogs suction easily), Magill forceps
   - an array of ETTs with stylets
   - dedicated assistant (preferably another anesthesiologist)
5. Anesthetic:
   - preoxygenation, rapid sequence induction with cricoid pressure
   - ketamine 2 mg/kg, succinylcholine 2 mg/kg
   - after intubation, NG to suction stomach. Extubate awake

Reference

Johr M. Anaesthesia for tonsillectomy. *Curr Opin Anaesthesiol.* 2006;19:260-261.

Tracheal Resection

Considerations

1. Tracheal anatomy: size and location of lesion
2. Pathology: tumor, trauma, infectious, stricture
3. Shared airway
4. Difficult airway with potential for loss once induced

History

- Pathology and anatomy of lesion
- Severity of obstruction: exercise tolerance, stridor, positional respiratory difficulty, cough, wheezing
- Hemoptysis, smoking history

Physical Exam

- Careful a/w exam including palpation, C-spine ROM

Lab Tests/Imaging

- CBC
- Spirometry with flow-volume loop, CXR, CT chest

Consults

Conflict(s)

- Need for early extubation (prevent disruption of suture line) in presence of airway edema and cervical flexion posturing

Optimize

- Chest physio; smoking cessation

Goals

- Good surgical access
- Early extubation

Options

- If danger of tracheal collapse after induction:
  - awake FOBI with minimal sedation
  - *versus*
  - pre-O_2, slow inhalational induction
- Epidural good for postoperative pain if thoracotomy

Preop: Premed
Blood: cross-match 4 units
ICU/stepdown bed: **yes**

Room Setup (special drugs/monitors)

- Arterial line (left)
- Assortment of ETTs, difficult a/w cart
- Sterile circuit + armored tube to pass to surgeon
- ±Surgeon with rigid bronch ready if large tracheal mass/ tracheomalacia

Induction

- NMB only after secure airway placed

Maintenance

- Volatile versus TIVA (useful if jet ventilation used)

Emergence

- Raise head of bed; smooth extubation (±remifentanil) to avoid bucking

Disposition/Pain

- To ICU; if edema, racemic epinephrine, steroids, diuresis

Reference

Sandberg W. Anesthesia and airway management for tracheal resection and reconstruction. *Int Anesthesiol Clin.* 2000;38:55-75.

Considerations

1. Potential difficult airway
2. Full stomach
3. Multisystem injury including closed head injury and unstable cervical spine
4. Hypovolemia and potential for massive blood loss
5. Hypothermia
6. Coingestion of alcohol and drugs

Primary Survey

1. **Airway and cervical spine control**
   - assess for patency
   - chin-lift/jaw-thrust/oral or nasal airway
   - maintain C-spine in neutral position
2. **Breathing: ventilation and oxygenation**
   - assess RR, tracheal deviation, breath sounds
   - ventilate with high FiO_2 oxygen, monitor SpO_2
   - treat tension PTX, seal open PTX
3. **Circulation with hemorrhage control**
   - pulse quality, HR, skin color, BP, obvious bleeding
   - two large bore IVs, 1 to 2 L warm LR, send trauma labs*
   - direct pressure to external bleeding? Internal
4. **Disability: brief neurologic assessment**
   - Glasgow coma scale
   - pupil size, equality, reaction
5. **Exposure/Environment**
   - undress completely but prevent hypothermia
6. **Adjuncts to primary survey (see Clinical Pearls section)**

Conflict(s)

- Hypovolemia and full stomach
- Difficult airway (C-spine precautions, oral injury) and full stomach

Optimize

Goals

Options

- Usually GA is required due to anxiety, intoxication, and for control of airway, ICP
- Nerve blocks can be combined for postoperative pain but use low concentrations of local anesthetics to avoid masking development of compartment syndrome

Preop: Premed
Blood: as needed
ICU/stepdown bed: usually

Room Setup (special drugs/monitors)

- Keep room warm
- Rapid infusor for fluids/blood
- Arterial line ± CVP

Induction

- RSI with in-line stabilization versus awake fiberoptic if difficulty anticipated. Blind nasal okay if no suspicion of skull base fracture
- Always be prepared for surgical airway
- Ketamine 2 mg/kg or etomidate 0.3 mg/kg

Maintenance

Emergence

Disposition/Pain

Clinical Pearls

1. Adjuncts to primary survey:
   - send ABG
   - monitor exhaled CO_2
   - connect to EKG monitor
   - insert urinary and gastric catheters unless contraindicated and monitor hourly urine output
   - consider the need for trauma series x-rays:
     - AP chest
     - AP pelvis
     - lateral, cross-table cervical spine
   - consider need for FAST (focused abdominal sonography for trauma) scan or diagnostic peritoneal lavage (DPL)
2. Trauma labs at time of IV placement include:
   - CBC
   - PT, PTT, INR
   - Electrolytes panel, BUN, Cr, Ca^{++}, Mg^{++}, PO_4^{-}
   - serum pregnancy test
   - type and cross-match
3. Following the primary survey and resuscitation, proceed to the secondary survey which involves a focused history including mechanism of injury, and a head-to-toe physical exam looking for other injuries

Reference

American College of Surgeons. Advanced Trauma Life Support Program for Doctors. 7th Ed. Chicago, IL, 2004.

TURP and Hysteroscopy

Considerations

1. Complications related to surgical disease → menorrhagia (bleeding), BPH (nephropathy)
2. Ambulatory patients → need to minimize pain/N&V
3. Often elderly with coexisting disease (CAD, CHF, PVD, CVA, COPD)
4. Potential for ↑ blood loss
5. At risk for complications due to absorption of the irrigation fluid → TURP syndrome:
   - hyponatremia
   - hypo-osmolality → cerebral edema/seizures
   - fluid overload → hypertension or hypotension
   - shock and cardiovascular collapse
   - chest pain/dyspnea/pulmonary edema
   - hyperglycinemia → blindness
   - hyperammonemia → encephalopathy
   - dysrhythmias
   - hypothermia

History

- History of renal dz, CHF, obstructive nephropathy
- Menorrhagia, anemia, dizziness
- Preop neurologic function (eg, dementia)
- Exercise tolerance

Physical Exam

- Careful volume status exam

Lab Tests/Imaging

- CBC
- Electrolytes (Na^+, BUN, Cr)
- EKG

Consults

Conflict(s)

Optimize

Goals

- Prevent TURP syndrome:
  - limit duration of resection <90 to 120 minutes
  - limit intravesicular (<30 mm Hg) or intrauterine (<60 mm Hg) pressure
  - use isotonic irrigation fluid (1.5% glycine is hypotonic)

Options

- Neuraxial may allow for earlier and easier detection of TURP syndrome (hyponatremia and/or fluid overload)

Preop: Premed
Blood
ICU/stepdown bed

Room Setup (special drugs/monitors)

Induction

Maintenance

Emergence

Disposition/Pain

- PACU → home

Clinical Pearls

Management of suspected TURP syndrome

1. Ensure adequate oxygenation and ventilation $\rightarrow O_2$, talk to patient
2. Assess restlessness (confusion?)
3. Review history: CAD, CHF, low $Na^+ \rightarrow$ more likely to have TURP syndrome
4. Exam: CVP raised? CVS instability? Nausea, visual changes?
5. ✓ BP, HR, SaO_2, CO_2, EKG changes
6. ✓ Duration of procedure. Large prostate? Extensive resection?
7. Notify surgeon—cease surgery as soon as possible
8. How much irrigation fluid used? Decrease height of irrigation fluid
9. ✓ Suction bottles: clots? Blood stained?
10. Reposition patient to neutral
11. ✓ Temperature
12. Send blood for Na^+ and K^+ and osmolarity, coags, Hb

If patient is not too restless and has stable CVS and respiratory parameters, await results of BMP. If $Na^+ > 130$ mmol/L, give 0.9% normal saline and cease operation; if Na^+ 121 to 130 mmol/L, give 0.9% normal saline, cease operation, administer 10 to 20 mg furosemide, and observe; if Na^+ 110 to
120 mmol/L, give 3% normal saline (max 3 mL/kg). Give slowly as risk of central pontine myelinolysis (no more than 0.5 mEq/L per hour change). Use
furosemide, fluid restrict, $NaHCO_3$, O_2, and monitor. If profound confusion $\rightarrow$ secure airway with ETT and treat as above.

Blindness post-TURP/hysteroscopy likely due to glycine toxicity. Treatment consists of magnesium therapy to correct plasma-magnesium levels and reassurance that blindness is transient and will resolve over next 24 hours.

References

Gravenstein D. Transurethral resection of the prostate (TURP) syndrome: a review of the pathophysiology and management. *Anesth Analg.* 1997;84:438-446.

Mushambi MC, Williamson K. Anaesthetic considerations for hysteroscopic surgery. *Best Pract Res Clin Anaesthesiol.* 2002;16:35-52.

Considerations

1. Peripheral arterial disease (PAD): increased age, atherosclerosis, DM, hypertension, hypercholesterolemia, tobacco use, chronic kidney injury, hypercoagulable state
2. Indications: emergency limb salvage, nonhealing ulcers or gangrene, or improve functional capacity
3. Options: *endovascular*—focal stenosis or short-segment occlusion. *Surgical bypass*—long-segment occlusion, or multi-segment disease
4. Epidural versus general anesthesia. Epidural—improved vascular graft blood flow, decreased blood loss, decreased thromboembolic complications, improved postoperative pulmonary function, and improved postoperative analgesia. No change in CV complications
5. Anticoagulants and timing of regional anesthesia: frequent use of clopidogrel and perioperative heparin use
6. Complications: perioperative MI, cardiac death, stroke, wound or graft infection, peripheral nerve injury or vessel rupture and bleeding
7. DDx—venous claudication, compartment syndrome, hip or knee OA, peripheral neuropathy
8. Coronary revascularization—CARP trial suggests not necessary for *stable* CAD and elective vascular surgery
9. Periop beta blockers—POISE trial suggests reduced CV risk but at an increased risk of stroke, hypotension, bradycardia, and overall death

History

- Limb ischemia—asymptomatic or claudication with activity; location—aortoiliac, femoropopliteal, below the knee
- Comorbid diseases, exercise tolerance
- Cardiac evaluation based on patient history and clinical predictors: CAD, CHF, DM, renal insufficiency, stroke
- Treatments—smoking cessation, diabetes treatment, medications—aspirin, clopidogrel, statins, antihypertensive agents (often ACEI or ARB), and cilostazol or pentoxifylline (for intermittent claudication)

Physical Exam

- Vital signs, cardiopulmonary exam; limb—dependent rubor, pallor on elevation, weak or absent pulses, ischemic ulceration; *acute occlusion*—pulseless, pain, pallor, poikilothermia, paresthesia, paralysis

Lab Tests/Imaging

- Cardiac testing as required, ankle-brachial index (ABI <0.90 threshold for PAD), angiography

Consults

- Cardiology as required

Conflict(s)

- Cardiac optimization—including preoperative cardiac testing, perioperative beta blockade and perioperative antiplatelet medications requires close communication with surgeon and cardiologist. Risk/benefit ratio and cardiac treatment plans (surgical or medical) need to be tailored to patient. May require adjustment of anesthetic plan accordingly

Optimize

- Comorbid conditions—glycemic control, blood pressure, urine output (hydration), smoking cessation
- IV access—may be difficult. Consider central line or peripherally inserted central catheter as required
- If regional anesthetic planned—discontinue anticoagulants and antiplatelet agents for safe period of time. To reduce risk of spinal or epidural hematoma current guidelines suggest clopidogrel should be held for at least 7 days before a neuraxial technique. ASA may be continued
- Heparinization—before cross-clamping vessels. Follow ACT

Options

- Endovascular—usually local anesthesia with sedation
- Open vascular: general anesthesia with endotracheal intubation or epidural anesthesia (combined with general anesthesia or as sole anesthetic)

Preop: Premed
Blood: cross-match
ICU/stepdown bed: possibly

Room Setup (special drugs/monitors)

- Arterial line ± central line, PAC; and Foley catheter
- Heparin, beta blockers, vasopressors, inotropes
- Infusion pumps
- Blood transfusion set, fluid warmers

Induction

- Local anesthesia and sedation, or epidural—oxygen via nasal cannula or face mask
- GETA—avoid induction hypotension

Maintenance

- Epidural—load epidural and maintain level (0.5% bupivacaine or 0.5% ropivacaine)
- GETA—balanced technique
- Monitor—ECG ST segments, blood loss
- Labs for glucose, electrolytes, hemoglobin, ABG, ACT
- Heparinization before cross-clamping of vessels

Emergence

- If GETA—assess for safe extubation

Disposition/Pain

- PACU or ICU as required
- Monitor for postoperative cardiac complications

Clinical Pearls

Contrast-induced nephropathy (CIN) due to radiocontrast dyes is a serious complication of endovascular procedures. Risk factors for CIN include preexisting renal dysfunction and diabetes. Prevention of CIN centers on optimal peri-procedure hydration. Unfortunately, the ideal hydration protocol is unknown with many studies advocating use of normal saline, sodium bicarbonate, or medications such as *N*-acetylcysteine.

A suggested protocol:

Isotonic sodium bicarbonate (154 mEq/L in D5W): 3 to 5 cc/kg/hr X1 hour preprocedure, followed by 1 cc/kg/h X 12 hours.

Complications of sodium bicarbonate include volume overload, hypernatremia, and hyperosmolality.

References

Wesner L, Marone LK, Dennehy KC. Anesthesia for lower extremity bypass. *Int Anesthesiol Clin*. 2005;43(1):93-109.

Gornik HL, Creager MA. Contemporary management of peripheral arterial disease: cardiovascular risk-factor modification. *Cleve Clin J Med*. 2006;73:S30-S37.

Reddan D, Laville M, Garovic VD. Contrast-induced nephropathy and its prevention: what do we really know from evidence-based findings? *J Nephrol*. 2009;22:333-351.

Events

1 Acute Adrenal Crisis
2 Acute Pain
3 Amniotic Fluid Embolism
4 Anaphylaxis/Anaphylactoid Reactions
5 Anesthetic Awareness
6 Aspiration of Gastric Contents
7 Autonomic Hyperreflexia
8 Bradycardia
9 Breathing Circuit Leak
10 Bronchospasm
11 Chronic Pain
12 Delayed Awakening
13 Fat Embolism Syndrome
14 High Peak Inspiratory Pressure
15 Hypercalcemia
16 Hypercarbia
17 Hyperkalemia
18 Hypertension
19 Hyperthermia
20 Hypocalcemia
21 Hypoglycemia
22 Hypokalemia
23 Hyponatremia
24 Hypotension
25 Hypothermia
26 Hypoxemia
27 Laryngospasm
28 Local Anesthetic Systemic Toxicity
29 Malignant Hyperthermia
30 Management of the Difficult Airway
31 Massive Blood Transfusion
32 Metabolic Acidosis
33 Myocardial Ischemia/Infarction
34 Obstetrical Hemorrhage
35 Oliguria
36 Pneumothorax
37 Postoperative Stridor
38 Pulseless Cardiac Arrest
39 Respiratory Alkalosis
40 Serotonin Syndrome
41 Sinus Tachycardia
42 Supraventricular Tachycardia
43 Total Spinal
44 Transfusion Reaction
45 Venous Air Embolism
46 Wide-Complex Tachycardia

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Acute Adrenal Crisis

Definition: A life-threatening state of adrenocorticosteroid hormone deficiency leading to hypotension and hemodynamic shock.

Clinical features

1. Hypotension/shock, orthostatic hypotension
2. Anorexia, nausea/vomiting
3. Abdominal pain
4. Fever
5. Lethargy/weakness

Differential diagnosis

1. Shock: septic, cardiogenic, spinal
2. Anaphylaxis
3. Other causes of hypotension (see Hypotension card)

Management

1. Ensure adequate airway and gas exchange
2. Administer high-flow oxygen/100% oxygen
3. Expand the circulating volume rapidly with dextrose in normal saline (D5NS) $\longrightarrow$ up to 2 to 3 L may be required
4. Draw blood for random cortisol and ACTH levels
5. Immediately following this, administer IV glucocorticoid: hydrocortisone 100 mg (and continue q6h as needed)
6. Support systemic blood pressure with vasopressors
7. Correct any of the following common electrolyte disturbances:
   - hypoglycemia
   - hyponatremia
   - hyperkalemia
   - hypercalcemia
8. Identify precipitating cause (eg, iatrogenic adrenal insufficiency due to exogenous corticosteroid administration)
9. Rule out other causes on the differential diagnosis (especially those that are rapidly correctable):
   - anaphylaxis
   - tension pneumothorax
   - massive hemorrhage
   - syringe swap
   - myocardial ischemia/infarction

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Definition: Physiologic response to a noxious chemical, thermal or mechanical stimulus; associated with invasive procedures, trauma, and disease; generally time-limited until injury heals.

Clinical Features

1. Pain varies greatly—mild to severe (0-10 scale)
2. Onset—acute
3. Location—site of injury, radiation, or referred pain
4. Duration—continuous, intermittent, relapsing
5. Characteristics—sharp/stabbing, cramping
6. Associated features tachycardia, tachypnea, hypertension, diaphoresis

Differential Diagnosis

1. Acute pain:
   - surgical/procedural
   - trauma
   - disease process
2. Chronic pain:
   - nociceptive:
     - somatic—muscles, bones, skin, connective tissue, blood vessels
     - visceral—viscera (organs)
   - neuropathic:
     - peripheral nervous system
     - central nervous system
     - CRPS I/II
3. Acute on chronic pain
4. Anxiety, panic attack
5. Drug-seeking behavior

Management

1. Treat pain and determine underlying cause:
   - acute surgical pain typically requires intravenous opioids (consider bolus, infusion, or patient-controlled analgesia as appropriate)
   - pain out of proportion to surgery/procedure or new onset pain requires history, physical exam, and appropriate laboratory investigations, imaging, and surgical consultation
2. Adjust therapy if patient is tolerant to analgesic medications:
   - chronic opioid use for chronic pain/addiction
3. Consider multimodal analgesia as clinical situation permits:
   - nonopioid analgesics (acetaminophen/paracetamol, NSAIDs)
   - α_2 agonists (clonidine, dexmedetomidine)
   - gabapentin
   - NMDA antagonists (ketamine, methadone)
   - local anesthetics
   - peripheral nerve blockade (single shot or indwelling catheter)
   - neuraxial blockade

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Definition: Passage of amniotic fluid into the maternal circulation typically resulting in sudden, profound, and unexpected shock, and cardiopulmonary collapse.

Clinical features

1. Hypotension, cardiopulmonary arrest
2. Fetal distress
3. Pulmonary edema/ARDS
4. Cyanosis, dyspnea
5. Coagulopathy
6. Seizures

Differential diagnosis

1. Obstetric causes:
   - acute hemorrhage
   - placental abruption
   - uterine atony
   - peripartum cardiomyopathy
   - eclampsia
2. Anesthetic causes:
   - high/total spinal
   - aspiration
   - local anesthetic systemic toxicity
3. Other causes:
   - pulmonary thromboembolism
   - venous air embolism
   - anaphylaxis
   - sepsis
   - intracranial hemorrhage
   - transfusion reaction

Management

1. Call for assistance (another anesthesiologist) and inform obstetrician:
   - prompt delivery of the fetus improves maternal resuscitation outcomes
2. Ensure adequate oxygenation and ventilation:
   - intubate trachea and ventilate with 100% O2
3. If pulseless, commence chest compressions and ACLS protocol:
   - maintain left uterine displacement to ensure venous return
4. Establish large-bore IV access × 2
5. Expand circulating volume
   - IV bolus of normal saline × 2 to 3 L
6. Support circulation with vasopressors:
   - phenylephrine is a rational choice as early in the crisis, vasodilation is the dominant circulatory abnormality. However, epinephrine may be required
   - later in the course of the event, inotropic support may be required (eg, norepinephrine, epinephrine, dobutamine, or milrinone)
   - vasopressin is a good choice of a systemic vasopressor to avoid further increases in pulmonary vascular resistance
7. Start arterial line for pressure monitoring and frequent blood draws
8. Establish central venous access for infusion of vasopressors/inotropes:
   - consider drawing pulmonary capillary sample from PA catheter if in situ → presence of squamous cells strongly suggest diagnosis
9. Send stat bloods for:
   - ABG
   - CBC
   - PT, PTT, INR, fibrinogen, FDP
   - serum tryptase
   - cross-match for 4 to 6 units of packed cells
10. Treat coagulopathy:
    - maintain oxygen-carrying capacity by replacing blood loss with packed red cells (type O negative or group-specific if cross-match not available)
    - fresh frozen plasma and platelet concentrates as necessary
    - cryoprecipitate not first line, but useful if fibrinogen is low and volume overload or ARDS are concerns
    - consider use of recombinant factor VIIa for refractory bleeding (recognize possibility of intravascular thrombosis)
11. Guide therapy by using:
    - invasive hemodynamic data (blood pressure, CVP, PAP, CO, SVR)
    - urine output (>1 mg/kg/h)
    - echocardiography (TEE or TTE): Amniotic fluid has different appearance in heart than blood

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Anaphylaxis/Anaphylactoid Reactions

Definition: Anaphylaxis is a type I immediate hypersensitivity reaction involving IgE antibody-antigen interaction (and usually requires previous exposure); anaphylactoid reactions are direct, nonimmune-mediated release of vasoactive mediators from mast cells and basophils. The two are clinically indistinguishable.

Clinical features

1. Grade I: mucocutaneous signs only:
   - erythema, urticaria ± angioedema
2. Grade II: moderate multivisceral signs:
   - mucocutaneous signs PLUS
   - hypotension ± tachycardia ± dyspnea ± GI disturbances (vomiting/diarrhea/crampy abdominal pain)
3. Grade III: life-threatening mono- or multivisceral signs:
   - mucocutaneous signs PLUS
   - cardiovascular collapse, tachycardia or bradycardia
   - cardiac dysrhythmias
   - bronchospasm
   - GI disturbances
4. Grade IV: cardiac arrest

Differential diagnosis

1. Skin manifestations:
   - nonanaphylaxis-related rash
   - angioedema related to ACE inhibitors, trauma, C1-esterase deficiency
2. Pulmonary manifestations:
   - bronchospasm
   - pulmonary edema
   - pneumothorax
   - pulmonary embolism
   - increased airway pressure from another cause (see High Peak Inspiratory Pressure)
   - pulmonary aspiration
3. Cardiovascular manifestations:
   - pericardial tamponade
   - venous air embolism, fat embolism
   - shock (septic, cardiogenic, spinal)
   - other hypotensive causes (see Hypotension card)
   - transfusion reaction

Management

1. Call for assistance, especially with Grade III and IV anaphylaxis
2. Withdraw suspected culprit drug. If reaction occurs on induction, stop all anesthetic drugs
3. Check with surgeons to see if they have introduced any possible antigenic material
4. Maintain airway and support oxygenation and ventilation with 100% oxygen
5. Early tracheal intubation should be strongly considered → the laryngeal tissues can rapidly become edematous and make intubation difficult/impossible
6. Place patient in Trendelenburg position to maximize venous return
7. If surgery commenced, abbreviate ASAP
8. Epinephrine is the life-saving drug of choice:
   - 10 to 20 μg IV prn for hypotension, escalate dose as needed
   - 0.5 to 1 mg IV prn for cardiovascular collapse
   - infusion of 1 to 10 μg/min may be required
9. Begin rapid expansion of circulating fluid volume:
   - 5 to 10 mL/kg of crystalloid in first 5 minutes
   - 2 to 4 L in initial resuscitation is average (but 7 L not uncommon)
10. Secondary therapy:
    - administer an H_1 blocker → diphenhydramine 50 mg IV
    - steroid: hydrocortisone 100 to 200 mg IV (fastest corticosteroid)
    - H_2 blockers not indicated → may block helpful effects of H_2 agonism (eg, inotropy and coronary vasodilatation)
11. If refractory hypotension, consider vasopressin:
    - start with 2 units IV, and titrate up to max 15 units
12. Glucagon therapy:
    - ↑ inotropy/chronotropy independent of α or β-adrenergic mechanism. 1000 mg IV adults, 500 mg IV children, particularly effective if on beta blockers
13. Draw blood for serum tryptase to establish conclusive diagnosis of anaphylaxis/anaphylactoid reaction

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Definition: Recollection of experiences during a period of intended general anesthesia.

Clinical Features

1. During awareness:
   - hypertension
   - tachycardia
   - diaphoresis
   - pain
   - sensation of paralysis/weakness
   - hearing noises/voices
   - feelings of panic, helplessness, impending death
2. Postoperative:
   - nightmares
   - sleep disturbances
   - anxiety
   - fear of future anesthesia

Differential Diagnosis

1. True awareness:
   - equipment problems
   - drug errors
   - deliberate light anesthesia (crisis management)
   - prolonged intubation
2. Awareness under sedation/regional anesthesia:
   - communication breakdown
3. Secondary gain

Management

1. If suspected during general anesthesia:
   - stop surgery
   - verbally reassure
   - deepen anesthesia
   - amnestic medications (benzodiazepines)
2. Postoperative:
   - interview
   - reassure
   - disclose/explain circumstances
   - arrange follow-up/counseling

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Aspiration of Gastric Contents

Definition: The presence in the tracheobronchial tree of liquid or particulate matter that originates in the stomach.

Clinical features

1. Cough, laryngospasm
2. Bronchospasm
3. Hypoxemia
4. Tachypnea if breathing spontaneously
5. ↑ Airway pressures

Differential diagnosis

1. Hypoxemia from other causes
2. Asthma/bronchospasm
3. Pulmonary edema/ARDS
4. Pulmonary embolism
5. ↑ Airway pressures from other causes

Management

1. Immediately at regurgitation/vomiting:
   - place patient in Trendelenburg position to limit passage of aspirate into trachea
   - suction pharynx with rigid suction—if particulate matter, remove with Magill forceps
   - maintain cricoid pressure if possible (skilled assistant)
2. Intubate with cuffed ETT to prevent further aspiration
3. Ventilate with 100% oxygen initially; titrate down to maintain SpO_2 >95%
4. Pass soft suction catheter down ETT in attempt to suction any contents that passed into trachea. If SpO_2 ↓, do not persist
5. Assess for bronchospasm, cyanosis, difficulty ventilating, pulmonary edema and treat appropriately
6. Support circulation:
   - can be ↑↑ fluid losses through pulmonary edema
   - crystalloid boluses prn
7. If bronchospasm, albuterol inhaler into circuit or 5 µg/kg IV over 20 minutes, followed by infusion
8. Once initial resuscitation is done, consider bronchoscopy to assess soiling and remove any particulate matter
9. Postpone/abbreviate surgery if gross aspiration. If mild, discuss with surgeon and ± proceed with repeated assessment of respiratory and cardiovascular status
10. No evidence of supporting empirical antibiotics → manage based on results of the Gram stain of the pulmonary aspirate
11. Steroids not routinely recommended
12. CXR (usually in PACU as initially will be normal) to delineate extent of damage

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Autonomic Hyperreflexia

Definition: A syndrome of widespread reflex sympathetic discharge in patients with chronic spinal cord lesions (usually at or above the T7 level) in response to stimuli below the lesion.

Clinical features

Descending inhibitory signals from supraspinal centers are unable to modulate afferent input from below the level of the cord lesion. As a result, sympathetic stimulation below the level of the lesion causes a reflex arc leading to massive sympathetic efferent discharge below the level of the lesion. The ensuing hypertension initiates corrective reflexes such as bradycardia and vasodilation

1. Below the lesion:
   - pallor
   - pilomotor erection
   - intense somatic and visceral muscle contraction
   - ↑ spasticity
2. Above the lesion:
   - flushing of face and neck
   - mucous membrane and conjunctival congestion
   - intense sweating
   - mydriasis
3. Bradycardia, AV block, PACs, PVCs
4. Severe headache, seizures, SAH, unconsciousness
5. Dyspnea, LV failure/pulmonary edema
6. Blurred vision
7. Anxiety, agitation
8. Chest pain/myocardial ischemia
9. Nausea

Differential diagnosis

1. Chronic hypertension
2. Hypertensive crisis of other etiology
3. Pheochromocytoma
4. Drug toxicity (eg, cocaine)
5. Thyrotoxicosis
6. CNS herniation

Management

1. Communicate with surgeon and remove precipitating stimulus (eg, emptying the bladder)
2. Consider deepening level of anesthesia if under GA
3. If epidural anesthesia/analgesia being used, consider raising level of block
4. Treat severe hypertension (ideal agent is fast-acting and easily titratable):
   a. α-blocking agents (eg, phentolamine 5 mg IV prn)
   b. direct vasodilators (eg, hydralazine 10-20 mg IV)
   c. sodium nitroprusside: start 1 μg/kg/min, up to 3 to 4 μg/kg/min
   d. calcium channel blockers (eg, nicardipine 5 mg/h IV, titrating up to 15 mg/h max)
5. Look for evidence of end-organ involvement of hypertension and treat accordingly (eg, pulmonary edema, EKG changes, seizures)

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Bradycardia

Definition: A heart rate of <60 beats per minute (bpm) in adults.

Clinical features

1. Slow heart rate (<60 bpm)
2. Hypotension
3. Dizziness, mental status changes, nausea/vomiting

Differential diagnosis

1. CNS:
   - ↑ ICP
   - vagal stimulation
   - spinal shock
2. Cardiovascular:
   - myocardial ischemia/infarction
   - sick sinus syndrome
   - AV blocks
3. Pulmonary:
   - hypoxemia
4. Metabolic:
   - acidosis
   - hypothermia
5. Endocrine:
   - hypothyroidism
   - adrenal insufficiency
6. Drugs:
   - beta blockers
   - anesthetic drug
   - K^+
   - Ca^{++}
   - digoxin

Management

1. Verify bradycardia ⟶ heart rate tracing on SpO_2, EKG, manual pulse
2. Maintain patent airway, assist breathing as needed
3. Confirm rhythm on EKG (sinus vs. AV block, junctional, etc)
4. Check blood pressure
5. Quickly check with surgical team to see if surgical cause (eg, traction on bowel)
6. If symptoms of poor perfusion caused by bradycardia (eg, ↓ mental status, chest pain, profound hypotension):
   - atropine 0.5 mg IV
   - epinephrine IV 10 to 20 µg bolus, repeating prn with escalating doses to effect
   - may require infusion of epinephrine (2-10 µg/min)
   - if refractory to pharmacologic measures, prepare for transcutaneous pacing
7. If bradycardia associated with mild symptoms (mild-moderate hypotension, N&V):
   - atropine 0.4 mg IV
   - ephedrine IV 5 to 10 mg prn
   - glycopyrrolate IV 0.2 mg
8. If signs of adequate perfusion, observe and monitor
9. Identify and treat correctable cause

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Breathing Circuit Leak

Definition: An unexpected leak of gas from the breathing circuit. A leak becomes significant if unable to maintain positive pressure ventilation.

Clinical Features

1. If breathing spontaneously:
   - higher than normal fresh gas flow rates require to inflate reservoir bag between breaths
   - odor of volatile anesthetic
   - signs of light anesthesia

Management

1. Maintain adequate oxygenation and ventilation:
   - If necessary at any time, switch to a separate bag-valve mask connected to 100% oxygen, and initiate TIVA in order to keep patient anesthetized
2. If breathing spontaneously:
   - close the APL valve
   - ↑ the fresh gas flow into the circuit
   - if reservoir bag does not refill, press the O_2 flush button → listen for major leak and fix
   - check and tighten circuit connections, O_2 sensor, inspiratory/expiratory valve covers, $ETCO_2$ sampling line
3. If mechanically ventilated:
   - ↑ the fresh gas flow into the circuit
   - stop the ventilator, switch to spontaneous ventilation, and test ability of reservoir bag to deliver positive pressure breath
   - feel compliance of reservoir bag while assessing chest rise and breath sounds
   - if able to deliver positive pressure breath with manual ventilation, leak is in ventilator
4. Test integrity of circuit:
   - disconnect Y-piece from patient and occlude while holding O_2 flush. If positive pressure maintained, problem is with patient or ETT
   - If still leaking, and no disconnection found, use alternative mode of ventilation (and anesthesia) and replace machine at earliest convenience
5. Check for leak in machine:
   - check O_2 pipeline pressure and function of O_2 flowmeter
   - check for vaporizer leak → turn off and assess for continued leak
   - may be internal problem with machine
6. Check for ETT leak:
   - listen carefully over mouth while applying positive pressure in circuit
   - check inflation of pilot balloon → if deflated, reinflate and listen again
   - if cuff appears defective, consider feasibility and risk to replacing ETT (consider initial ease of intubation, risk of aspiration, positioning considerations, etc)
   - if not advisable to extubate, consider replacing with LMA or packing pharynx temporarily

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Bronchospasm

Definition: Reversible spasm of the bronchial smooth muscle resulting in narrowing of the airways.

Clinical Features

1. Wheezing
2. ↑ Airway pressures
3. ↓ Pulmonary compliance/↓ tidal volume
4. Hypoxemia
5. Hypercarbia

Differential Diagnosis

1. Pulmonary aspiration
2. Laryngospasm
3. Pulmonary edema
4. Pulmonary embolism/fat embolism/amniotic fluid embolism
5. Pneumothorax
6. Anaphylaxis/anaphylactoid reactions
7. ETT obstruction, endobronchial intubation

Management

1. Maintain oxygenation and ventilation:
   a. ensure adequate airway
   b. administer 100% O_2
   c. hand-ventilate if artificial airway in place to evaluate pulmonary compliance
2. Auscultate the chest for bilateral breath sounds, wheezing
3. Pass suction catheter down ETT to rule out kinking
4. If light anesthesia, deepen with propofol IV 20 to 50 mg, followed by an ↑ in the concentration of volatile agent (providing not hypotensive)
5. Administer inhaled $\beta_2$agonist:
   a. albuterol MDI via circuit: 4 to 20 puffs/h
6. If severe, consider IV bronchodilator therapy titrated to hemodynamic and bronchodilator response:
   a. albuterol 5 µg/kg over 20 minutes
   b. isoproterenol 1 to 3 µg/min
   c. epinephrine 2 to 8 µg/min
7. Anticholinergic therapy is useful in severe bronchospasm:
   a. ipratropium MDI 4 to 8 puffs q 15 minutes
   b. atropine 20 µg/kg IV (or nebulized 2 mg)
   c. glycopyrrolate 10 µg/kg IV (or nebulized 1 mg)
8. Steroids are indicated in acute asthma, but take time for onset:
   a. methylprednisolone 40 mg IV q6h
9. Alter ventilator settings to decrease risk of air trapping/barotrauma:
   a. adjust I:E ratio AND respiratory rate to maximize expiratory time (start with rate of 6-8/min)
   b. maintain PIP <50 cm H_2O
   c. pressure control ventilation will allow for greater inspiratory flow, which permits a longer expiratory time and ↓ dynamic hyperinflation
   d. allow mild-moderate hypercapnia in order to achieve above
10. For status asthmaticus consider the following:
   a. prolonged ventilation with isoflurane
   b. heliox (↓ airway resistance)
   c. ECMO if inability to oxygenate

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Definition: Pain lasting >3 to 6 months; may begin as acute pain but persists beyond typical time to resolve.

Clinical Features

1. Varies greatly—mild to severe (0-10 scale)
2. Location—site of injury, radiation, or referred pain
3. Duration—continuous, intermittent, relapsing
4. Characteristics—sharp/stabbing, cramping, burning
5. Associated features—depression, suicide, anxiety, loss of appetite, diminished libido, sleep disturbances, autonomic changes (diaphoresis, vasomotor changes), motor weakness, skin/bone atrophy, joint contractures

Differential Diagnosis

1. Chronic pain:
   - nociceptive:
     - somatic—muscles, bones, skin, connective tissue, blood vessels
     - visceral—viscera (organs)
   - neuropathic:
     - peripheral nervous system
     - central nervous system
     - CRPS I/II
2. Acute pain:
   - surgical/procedural
   - trauma
   - disease process
3. Acute on chronic pain
4. Anxiety, panic attack
5. Substance abuse/dependency

Management

1. Determine underlying cause—history, physical exam, laboratory investigations, imaging, and consultation
2. Consider multimodal therapy for chronic pain:
   - counseling
   - physical therapy
   - medications:
     - antidepressants
     - anticonvulsants
     - opioids
   - nerve blocks/ablation
   - surgery:
     - spinal cord stimulator
     - intrathecal drug delivery
   - transcutaneous electrical nerve stimulation (TENS)
   - acupuncture
   - biofeedback

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Delayed Awakening

Definition: Delayed return of consciousness despite which was expected for a given anesthetic.

Clinical Features

1. Absence of purposeful movements or response to stimuli

Differential Diagnosis

1. Pharmacologic:
   - residual drugs:
     - premedication (benzodiazepines, antihistamines)
     - induction agents/anesthetic agents
     - muscle relaxants
     - decreased metabolism, excretion, protein binding
   - increase sensitivity to drugs:
     - elderly
     - drug interactions
     - renal/hepatic disease
     - hypothermia
     - biologic variability
2. Metabolic causes:
   - renal/hepatic disease (encephalopathy)
   - hypothyroidism/myxedema coma
   - adrenal insufficiency
   - hypoxemia
   - hypercapnia
   - hypoglycemia
   - hypothermia
   - hyponatremia
   - sepsis
   - malignant hyperthermia
3. Neurologic causes:
   - hypoperfusion:
     - decrease CO
     - cerebrovascular disease
     - embolism, thrombus, retraction
   - hyperperfusion (eg, ICH)
   - increase ICP
   - subdural/epidural hemorrhage
   - cerebral edema
   - undetected head injury

Management

1. Scan monitors and ensure adequate O_2 and ventilation (check SpO_2 & $ETCO_2$)
2. Check that all anesthetics have been turned off (IV and inhaled)
3. Stimulate the patient using gentle airway suctioning
4. If combative, use physical restraints to protect patient and staff until etiology ruled out
5. R/O metabolic causes:
   - quick finger stick for glucose
   - check temperature
   - send stat electrolytes and ABG
6. Review doses of medications, check for syringe swap
7. Consider reversing:
   - naloxone 0.04 mg q 1 minute up to 0.4 mg
   - flumazenil 0.2 mg q 1 minute up to 1 mg
   - check train of four
8. Perform neurologic examination:
   - pupillary reaction
   - corneal/gag reflexes
   - limb reflexes
9. Consult neurology; ICU for continued care

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Fat Embolism Syndrome

Definition: Fat emboli damage pulmonary capillaries causing respiratory failure.

Clinical Features

1. Trauma to long bones or pelvis
2. Major criteria:
   - respiratory failure—tachypnea, dyspnea, hypoxia
   - CNS—agitation, seizures, coma
   - petechial rash—upper body, axillae
3. Minor criteria:
   - tachycardia
   - fever
   - jaundice
   - acute kidney injury
   - anemia, thrombocytopenia
   - retinal fat globules

Differential Diagnosis

1. Fat embolism—trauma, burns, acute pancreatitis, parenteral lipids
2. Pulmonary embolus
3. Pneumonia

Management

1. Supportive care:
   - assess need for intubation or cardiopulmonary resuscitation
   - maintain oxygenation/ventilation/hemodynamics
   - intravenous fluid resuscitation

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High Peak Inspiratory Pressure

Definition: Increase in peak inspiratory pressure > 40 cm H_2O or an elevation above an established baseline level.

Etiology

1. Circuit or machine problem:
   - ventilator/bag switch in wrong position
   - stuck valve (inspiratory/expiratory/APL)
   - O_2 flush valve stuck in "on" position
   - kinked/misconnected hose in circuit/scavenge limb
   - failure of check valves/regulators in machine, allowing high-pressure gas into low-pressure circuit
   - PEEP valve accidentally placed in inspiratory limb
2. ETT/supraglottic airway problem:
   - kinked tube
   - malpositioned supraglottic airway
   - endobronchial, esophageal, submucosal intubation
   - herniated cuff obstructing end of tube
   - foreign body/secretions plugging end of tube
   - dissection of interior surface of tube, leading to airway narrowing
3. Decreased pulmonary compliance:
   - increased intra-abdominal pressure
   - pulmonary aspiration
   - bronchospasm not related to aspiration
   - decreased chest wall compliance
   - pulmonary edema
   - pneumothorax
4. Drug-induced problem:
   - opioid-induced chest wall rigidity
   - inadequate muscle relaxation
   - malignant hyperthermia
5. Laryngospasm (if using supraglottic airway)

Management

1. Increase FiO_2 to 100%
2. Verify the peak inspiratory pressure (check manometer/gauge)
3. Switch to manually using reservoir bag; assess pulmonary and circuit compliance
4. Disconnect circuit from ETT and squeeze bag:
   - if PIP still high, obstruction in circuit; ventilate using BVM connected to 100% O_2
   - get help to replace/repair circuit
5. Auscultate chest and neck:
   - listen for symmetry (endobronchial, tension, or simple PTX) and for adventitious sounds (pulmonary edema, bronchospasm)
   - listen to stridorous sound of laryngospasm
6. Examine trachea for deviation, check HR, BP
7. Exclude ETT obstruction:
   - pass suction catheter down ETT and apply suction to clear secretions
   - if ETT obstructed, deflate cuff and repeat
   - consider FOB to elucidate problem
   - remove and reintubate if necessary
8. Check for other causes of decreased chest compliance:
   - MH
   - aspiration
   - inadequate muscle relaxation
   - opiates
   - excessive surgical retraction
   - abnormal anatomy (eg, scoliosis)

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Hypercalcemia

Definition: Elevated calcium level in the blood; normal ionized calcium is 1 to 1.25 mmol/L (reference ranges vary according to laboratory).

Clinical Features

1. Asymptomatic
2. Nausea, anorexia
3. Emotional lability, lethargy, stupor, coma
4. Polyuria, kidney stones, hypovolemia
5. Hypertension, heart block, arrhythmias
6. Osteoporosis, bone pain

Differential Diagnosis

1. Hyperparathyroidism—primary, secondary, tertiary
2. Vitamin D intoxication
3. Malignancy
4. Granulomatous disease—sarcoidosis, tuberculosis
5. Medications—thiazide diuretics, lithium, milk-alkali syndrome
6. Endocrine—hyperthyroidism, adrenal insufficiency, acromegaly, pheochromocytoma
7. Immobilization
8. Decreased calcium excretion—hypocalciuric hypercalcemia

Management

1. Assess and treat underlying cause
2. Intravenous hydration with normal saline
3. Once volume replete, induce diuresis with loop diuretic (furosemide)
4. Hemodialysis for life-threatening hypercalcemia
5. Consult endocrinology regarding other treatments: bisphosphonates, glucocorticoids, calcitonin

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Hypercarbia

Definition: The presence of abnormally high levels of carbon dioxide in the blood, usually defined as $PaCO_2$ >45 mm Hg on ABG.

Clinical Features

1. Early symptoms, especially if awake:
   - flushed skin, sweating
   - muscle tremor
   - headache
   - confusion, lethargy, CO_2 narcosis
2. Sympathetic stimulation:
   - tachycardia
   - hypertension
   - extrasystoles and other dysrhythmias
3. If spontaneously breathing:
   - dyspnea and tachypnea
4. If mechanically ventilated:
   - patient may attempt to overbreathing the ventilator

Differential Diagnosis

1. ↑ Production of CO_2:
   - fever, sepsis
   - malignant hyperthermia
   - thyrotoxicosis
   - TPN with high carbohydrate content
2. ↓ Elimination of CO_2:
   - ↓ cardiac output (shock, hypotension)
   - CNS depression → hypoventilation
   - ventilator malfunction
   - neuromuscular disease (eg, Guillan-Barre syndrome)
   - ↓ lung/chest wall compliance
   - splinting from pain of upper abdominal incision
3. Exogenous addition of CO_2:
   - CO_2 pneumoperitoneum during laparoscopy

Management

1. Establish/maintain a patent airway
2. Ensure adequate oxygenation (titrate FiO_2 as needed)
3. Ensure adequate ventilation:
   - if spontaneously breathing, consider assisting with CPAP:
     - if refractory to above measures, consider tracheal intubation
   - if mechanically ventilated:
     - increase minute ventilation (↑ tidal volume, respiratory rate, or both as appropriate)
     - test for leak in circuit (see Breathing Circuit Leak card)
4. Confirm diagnosis with ABG
5. Confirm and treat cause of hypercarbia:
   - evaluate for presence of residual anesthetics, opioids, neuromuscular blockers
   - check inspired CO_2 level:
     - potential causes: stuck valve in breathing circuit, exhausted soda lime, administration of exogenous CO_2
   - check temperature, presence of muscle rigidity, reddish-brown urine (MH)
   - review chart to look for presence of thyroid disease, TPN regimen
   - check for syringe swap (accidental opioid, neuromuscular blocker administration)

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Hyperkalemia

Definition: The presence of an abnormally high serum potassium level (usually >5.5 mEq/L)

Clinical Features

1. Symptoms:
   - fatigue
   - weakness
   - paresthesias
   - palpitations
2. EKG changes (with increasing severity):
   - peaked T waves
   - ↓ QT interval
   - ST-segment depression
   - ↑ PR interval
   - widened QRS complex
   - ↓ P-wave amplitude
   - sine wave
   - VF or asystole

Differential Diagnosis

1. Release of potassium into extracellular space:
   - potassium supplements
   - rhabdomyolysis
   - hemolysis
   - burns
2. ↓ Elimination of potassium:
   - acute/chronic renal failure
   - potassium-sparing diuretics
   - adrenal insufficiency
3. Transmembrane shifts:
   - acidosis
   - meds (digitalis toxicity, beta blockers, succinylcholine)
4. Pseudohyperkalemia:
   - lysis of red cells in phlebotomy specimen

Management

1. If no EKG signs, send stat VBG or ABG to confirm diagnosis before instituting treatment
2. If real, and K^+ >6.0 mmol/L or if EKG changes present, start emergency treatment:
   - calcium gluconate 10% 10 mL IV over 2 to 3 minutes (raises threshold potential and restores myocyte excitability)
   - insulin 10 units IV with 50 mL of 50% dextrose (shifts K^+ into cells. No effect on total body K^+)
   - albuterol 10 to 20 mg by neb over 10 minutes (shifts K^+ into cells)
   - ↑ blood pH (shifts K^+ into cells):
     - periodically check ABG
     - sodium bicarbonate 50 to 150 mEq
     - treat metabolic acidosis if present
     - mild hyperventilation
   - force diuresis:
     - increase fluid administration
     - furosemide 10 mg IV
3. Consider hemodialysis if life threatening or accompanied by volume overload
4. If mild, cation exchange resins (eg, Kayexalate) can be used to exchange K^+ for Na^+ in the gut

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Hypertension

Definition: A rise in blood pressure greater than 20% of the preoperative value.

Clinical Features

1. Elevated systolic, diastolic, or mean blood pressure
2. Headache
3. Chest pain
4. Dyspnea
5. Anxiety

Differential Diagnosis

1. CNS:
   - anxiety
   - pain
   - light anesthesia/laryngoscopy/surgical stimulation
   - autonomic hyperreflexia
   - ↑ ICP
2. Cardiovascular:
   - essential hypertension
   - ischemia
   - acute ↑ in afterload (eg, aortic cross-clamp)
   - pre-eclampsia
3. Pulmonary:
   - hypoxia
   - hypercarbia
4. Renal:
   - full bladder
   - renal failure/volume overload
   - renovascular disease
5. Endocrine:
   - Cushing syndrome
   - pheochromocytoma
   - thyrotoxicosis
   - hypoglycemia
6. Drugs:
   - catecholamines/vasopressors
   - anticholinergics
   - withdrawal of antihypertensives
   - withdrawal of alcohol/opioids
   - naloxone

Management

1. Verify that hypertension is real:
   - repeat measurement
   - adjust NIBP cuff, re-zero or flush arterial line
2. Assess depth of anesthesia:
   - check heart rate, respiratory rate, signs of light anesthesia (eg, sweating, tearing), MAC, BIS, inspired concentration of volatile agent
   - check function of TIVA pump if using
   - check for new surgical stimulus
   - deepen if necessary with IV propofol 20 to 50 mg, or volatile agent
3. Ensure adequate oxygenation and ventilation
4. Check for inadvertent vasopressor use
5. Review chart for trend of preoperative blood pressures
6. Rule out other cause as per patient's history (eg, hypoglycemia, ↑ ICP)
7. Assess the likelihood of distended bladder; insert Foley catheter if feasible
8. If no correctable cause, consider administering antihypertensive:
   - if possible, use from same class patient is taking
   - labetalol, 5 to 10 mg IV at a time
   - enalaprilat 1.25 to 2.5 mg IV at a time
   - verapamil 2.5 mg IV at a time
   - hydralazine 10 to 20 mg IV at a time
   - nifedipine 10 mg SL
   - if severe, consider nicardipine infusion (2-5 μg/kg/min) or phentolamine boluses (0.5-1 mg IV at a time)

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Hyperthermia

Definition: Normal body temperature is approximately 37.0°C (98.6°F) with normal variability of 0.5 to 1.0°C. Hyperthermia (fever) is a core body temperature >38.3°C (101°F). Heat stroke is a core body temperature >40°C (104°F).

Clinical Features

1. Hot, dry skin
2. Nausea, vomiting
3. Headache
4. Hypotension, tachycardia, tachypnea
5. Syncope
6. Mental status changes, seizures, coma

Differential Diagnosis

1. Infection—bacterial, viral, fungal
   (sites—indwelling catheters, surgical site, respiratory, cardiac—endocarditis, sinuses, urinary tract, stool, thrombophlebitis)
2. Malignancy (leukemia, lymphoma, metastatic, etc)
3. Autoimmune (collagen vascular disease, rheumatoid arthritis, SLE)
4. Noninfectious: malignant hyperthermia, neuroleptic malignant syndrome, thyroid storm, drug-induced, pulmonary embolism, hepatitis, withdrawal from alcohol, opiates, benzodiazepines
5. External heating

Management

1. Verify temperature is real:
   - repeat measurement
   - gold standard—pulmonary artery catheter (or esophageal probe, or rectal)
2. Assess vital signs and mental status to determine whether urgent or emergent management is required
3. Narrow differential diagnosis: history and physical exam including review of all medications and blood products
4. Supportive measures as required:
   - decrease temperature—acetaminophen, ice packs, cool IV fluids
   - intubation/ventilation for respiratory compromise
   - fluids, vasopressors/inotropes to maintain hemodynamic parameters
5. Noninfectious causes:
   - MH—use of halogenated volatile anesthetics or succinylcholine—muscle rigidity, hypercapnia, tachycardia, fever
     Management—see Malignant Hyperthermia
   - NMS—associated with antipsychotic neuroleptic medications—muscle rigidity, fever
     Management—stop offending agents. Muscle relaxation—dantrolene, nondepolarizing neuromuscular blocking agents, dopamine agonists
6. Infectious causes:
   - assess central venous catheters for inflammation/purulence at insertion site:
     - remove catheter if signs of infection
     - if required, insert new catheter at different site
   - surgical site infection—inspect site, open and culture if suspicion:
     - CBC, blood culture (3-4 separate sites)
     - diarrhea—culture for *Clostridium difficile*
     - urine culture
     - further studies: PPD, brain/sinus/chest/ab/pelvic imaging, bronchoscopy/BAL, TTE/TEE, venous Doppler study, lumbar puncture, bone marrow biopsy, as required
7. If infectious cause suspected—empiric antimicrobial therapy (choice depends on suspected etiology, community or hospital acquired, and whether the patient is immunocompromised)

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Hypocalcemia

Definition: Decreased calcium level in the blood. Normal ionized calcium is 1 to 1.25 mmol/L (reference ranges vary according to laboratory).

Clinical Features

1. Neuropsychiatric—anxiety, dementia, depression, psychosis, seizures, papilledema
2. Neuromuscular—tetany, muscle cramping/weakness:
   - Chvostek sign (tapping facial nerve by tragus of ear results in twitching)
   - Trousseau sign (inflating arm BP cuff results in carpal spasm)
3. Respiratory—apnea, laryngeal spasm, bronchospasm
4. Cardiovascular—arrhythmias, heart failure, hypotension, ECG—prolonged QT interval, wide QRS, flat T waves

Differential Diagnosis

1. Parathyroid hormone deficiency—primary, postparathyroid surgery
2. Vitamin D deficiency
3. Hyperphosphatemia
4. Hypomagnesemia
5. Citrate chelation—massive blood transfusion
6. Alkalemia
7. Renal failure
8. Liver failure
9. Medications—proton pump inhibitors (decreased calcium absorption)
10. Other—acute pancreatitis, rhabdomyolysis, sepsis

Management

1. Assess for underlying cause
2. If symptomatic:
   - assess need for intubation or cardiopulmonary resuscitation
   - maintain oxygenation/ventilation/hemodynamics
   - intravenous fluid resuscitation
   - seizures do not respond to typical antiseizure medications until calcium is restored
   - 10% calcium gluconate 10 mL IV over 10 minutes
3. Calcium replacement:
   - use extreme care with peripheral IV infusion as extravasation can cause tissue necrosis
   - central line preferred
4. Follow lab values

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Hypoglycemia

Definition: Reduced plasma glucose concentration; normal fasting glucose is usually 5.0 to 7.2 mmol/L (70-130 mg/dL). For healthy adults, symptomatic when plasma glucose approximately <3 mmol/L (55 mg/dL).

Clinical Features

1. SNS activation—diaphoresis, tremor, tachycardia, anxiety, hunger
2. Neuroglycopenic—weakness, fatigue, altered mental status, coma

Differential Diagnosis

1. Exogenous insulin
2. Critical illness—sepsis
3. Endocrine—Addison disease, adrenal crisis, hypopituitarism
4. Insulin-producing tumors
5. Fasting hypoglycemia—inherited liver/fatty acid oxidation enzyme deficiencies, drugs—ethanol, haloperidol
6. Reactive (postprandial) hypoglycemia—idiopathic or enzyme deficiencies

Management

1. Confirm glucose level
2. Supplement with oral sugars or intravenous dextrose
3. History and physical exam to assess for underlying cause
4. Continue to monitor glucose levels until stable

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Hypokalemia

Definition: Potassium concentration <3.5 mEq/liter; normal potassium concentration—3.5 to 5.0 mEq/liter.

Clinical Features

1. Asymptomatic
2. Musculoskeletal—muscle weakness, cramps, paralysis, decreased reflexes
3. CVS:
   - hyperpolarization of cells—delayed conduction, arrhythmias
   - ECG—flat, inverted T waves, prominent U waves, ST depression

Differential Diagnosis

1. Intracellular shift—medications ($\beta_2$agonists, phosphodiesterase inhibitors, calcium channel blockers, exogenous insulin), hyperthyroidism, delirium tremens, familial periodic paralysis
2. Renal loss—medications (diuretics, exogenous mineralocorticoids, penicillin, cisplatin), mineralocorticoid excess (primary hyperaldosteronism, Cushing disease, renin secreting tumors)
3. GI loss—diarrhea, NG losses

Management

1. Treat underlying cause
2. Replace potassium—oral or intravenous. Central line preferred for intravenous replacement (20 mEq KCl in 100 cc NS—infuse at 10-20 mEq/h)
3. Monitor ECG

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Definition: Sodium concentration <135 mEq/liter.

Clinical Features

1. Asymptomatic
2. Nausea, vomiting, tremors, cramps
3. Altered mental status—coma, seizures
4. Cerebral edema—papilledema
5. Decreased reflexes

Differential Diagnosis

1. Hypotonic hyponatremia (most common)—excess free water. Consider *volume status*:
   - hypovolemic—water/excess sodium losses from GI tract, skin, 3rd space, kidneys (diuretics), cerebral salt wasting
   - euvolemic—SIADH, hypothyroid, adrenal insufficiency, water intoxication, malnutrition
   - hypervolemic—heart failure, liver cirrhosis, nephrotic syndrome
2. Hypertonic hyponatremia—dilutional hyponatremia (non-sodium solute—glucose, mannitol, ethanol), TURP syndrome (plasma osmolality variable)
3. Normotonic hyponatremia—TURP syndrome (plasma osmolality variable)

Management

1. Assess duration of hyponatremia and severity
2. Measure osmolality—low, normal, or high, and assess volume status
3. Treat underlying cause
4. Do not correct hyponatremia too quickly! Risk—lcentral pontine myelinolysis. For chronic, severe hyponatremia, correct 0.5 to 1.0 mEq/L/h to a maximum of 12 mEq/L/d
5. Depending on diagnosis, treat with hypertonic saline, isotonic saline, or fluid restriction

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Hypotension

Definition: A decrease in blood pressure greater than 20% of the preoperative value.

Clinical Features

1. Reduced systolic, diastolic, or mean blood pressure
2. Orthostasis
3. Dizziness, syncope, ↓ LOC
4. Nausea
5. Pallor, cool moist skin
6. Tachypnea
7. Tachycardia

Differential Diagnosis

1. ↓ Preload:
   - hypovolemia
   - ↓ venous return from surgical retraction, positioning
   - ↑ intrathoracic pressure (tension pneumothorax, air trapping)
   - venous pooling from venodilation (eg, neuraxial block)
   - pericardial tamponade
   - pulmonary embolism
2. ↓ Afterload:
   - vasodilation from anesthetic agents, vasoactive drugs
   - sepsis
   - anaphylaxis
   - endocrine causes (adrenal insufficiency, myxedema coma)
   - sudden ↓ in afterload (eg, removal of aortic cross-clamp)
3. ↓ Contractility:
   - myocardial ischemia/infarction
   - cardiomyopathy
   - negative inotropic drugs
   - valvular disease
   - dysrhythmias
4. ↓ Heart rate

Management

1. Ensure adequate oxygenation and ventilation:
   - establish/maintain patent airway
   - ensure gas exchange with high-flow/100% oxygen
2. Verify hypotension
3. Be prepared to treat as cardiac arrest
4. Inform surgeon and ask about recent maneuvers (eg, pressure on IVC, etc)
5. Turn off vaporizers and other vasodilating drugs
6. Improve position:
   - temporarily place in Trendelenburg to ↑ venous return
   - raise legs
7. Expand circulating volume:
   - IV crystalloid bolus of 10 mL/kg, repeat as necessary
8. Administer vasopressor:
   - phenylephrine 100 µg IV bolus
   - metaraminol 0.25 to 0.5 mg IV bolus
   - if severe, epinephrine 1 µg/kg IV bolus, repeat as necessary
9. Review and treat probable causes:
   - hypovolemia (blood loss, dehydration, diuresis, sepsis)
   - check urine output, blood loss
   - consider infusions of inotropes and/or vasopressors
   - consider placing an arterial line
   - consider the use of TEE to evaluate cardiac function

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Definition: Core body temperature <35°C, mild—32 to 35°C, moderate—28 to 32°C, severe—<28°C.

Clinical Features

Mild hypothermia (32-35°C)

1. Shivering
2. CNS—confusion, slurred speech
3. Tachypnea
4. Activation of sympathetic nervous system—tachycardia
5. Diuresis

Moderate hypothermia (28-32°C)

1. Loss of shivering
2. CNS—stupor
3. Pupils may become dilated
4. Hypoventilation
5. Increased risk of arrhythmias—atrial fibrillation and other atrial and ventricular arrhythmias
6. Bradycardia, hypotension, decreased CO, peripheral vasoconstriction
7. ECG—J or Osborne waves
8. Coagulopathy—decreased platelet function, decreased coagulation cascade activation

Severe hypothermia (<28°C)

1. CNS—coma
2. Decreased EEG activity
3. Fixed pupils
4. Pulmonary edema
5. Arrhythmias—spontaneous asystole/VFIB
6. Decreased myocardial contractility, hypotension
7. Oliguria

Differential Diagnosis

1. Decreased heat production—hypopituitarism, adrenal insufficiency, hypothyroidism, malnutrition, hypoglycemia
2. Increased heat loss—accidental cold exposure (homeless person), iatrogenic (postoperative patient), burn patients, drug-induced (general anesthetics, ethanol, sedatives, clonidine, meperidine)
3. Impaired thermoregulation—CNS trauma, strokes, intracranial bleeding, Parkinson disease
4. Therapeutic hypothermia—postcardiac arrest

   (perioperative risks: very young or elderly, emergency surgery, prolonged surgery, low preoperative temperature)

Management

1. Verify temperature and determine degree of hypothermia
2. Assess need for intubation/ventilation and support of circulation
3. Rewarm—external rewarming (forced-air warming systems), warmed IV fluids, warmed, humidified oxygen
4. Correct electrolytes as required (hypo- or hyperkalemia; hypo- or hyperglycemia)
5. Correct fluid deficit
6. Treat coagulopathy as required
7. Consider need for invasive core rewarming: gastric/peritoneal/thoracic lavage, cardiopulmonary bypass, ECMO
8. Ventricular fibrillation—perform CPR and treat with defibrillation and antiarrhythmics. (Defibrillation may be ineffective at core temperatures <30°C—consider initiating cardiopulmonary bypass if available)
9. Postoperative shivering (causes SNS activation and increased oxygen consumption)—rewarm patient; treat shivering with opioids (meperidine) or $\alpha_2$agonists (clonidine, dexmedetomidine)

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Hypoxemia

Definition: SaO_2 <90% or PaO_2 <60 mm Hg.

Clinical Features

1. ↓ SpO_2
2. Cyanosis
3. Tachycardia
4. Dysrhythmias
5. Bradycardia ⟶ asystole
6. Hypotension ⟶ cardiovascular collapse

Etiology

1. ↓ FiO_2
2. Hypoventilation:
   - iatrogenic (inadequate ventilator settings/ventilator malfunction)
   - respiratory depressant drugs (eg, opioids, residual neuromuscular blockade)
   - bronchospasm
   - breath-holding/laryngospasm in infants
3. V/Q mismatch:
   - hypotension (ventilation to West zone I, perfusion to West zone III)
   - atelectasis
   - aspiration
   - PE
4. Anatomic shunt:
   - Eisenmenger sydrome
   - congenital heart disease (eg, tetralogy of Fallot)
5. Excessive metabolic demand or ↓ mixed venous oxygen content:
   - sepsis
   - fever
6. Diffusion problem:
   - pulmonary edema
   - COPD
7. Abnormal hemoglobin species (carboxyhemoglobin, methemoglobin)

Management

1. ↑ FiO_2 to 100%:
   - use high flow and verify FiO_2 from oxygen sensor approaches 1.0
2. Check Ventilation adequate:
   - check $ETCO_2$
   - switch to hand ventilation to assess compliance
   - give recruiting breaths to treat atelectasis
   - watch for symmetric rise of the chest
   - consider addition of PEEP and maintain large VT
   - check ABG. Ask lab to check for abnormal hemoglobin if clinically indicated
3. Check Position of ETT:
   - auscultate chest bilaterally (wheezing, rales, symmetry)
   - consider FOB to check interior of tube, trachea
4. Verify function of pulse oximeter (NB: hypoxemia should be assumed real until proven otherwise):
   - check probe position
   - correlate with EKG tracing
   - change site: ear, nose, lips, tongue, etc
5. Pass suction catheter down ETT and suction secretions/mucus
6. Consider addition of PEEP and maintain large VT
7. Restore adequate circulating volume with crystalloid and/or packed red cells to maintain CO- and oxygen-carrying capacity
8. Check for surgical cause of hypoxemia (eg, retractors-impeding ventilation)

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Laryngospasm

Definition: Form of airway obstruction, caused by involuntary contraction of the vocal folds.

Clinical Features

1. Inspiratory stridor
2. Desaturation
3. Bradycardia
4. Cyanosis
5. Increased inspiratory effort/tracheal tug
6. Paradoxical chest/abdominal movement

Differential Diagnosis

1. Laryngospasm:
   - airway irritation/obstruction: due to blood or secretions in airway
   - aspiration or regurgitation
   - excessive stimulation (suctioning)
   - light depth of anesthesia
2. Consider other causes of stridor:
   - anatomic/acquired malformations—foreign body, large tonsils or epiglottis, congenital abnormalities
   - infection—epiglottitis, acute tonsillitis, retropharyngeal abscess, croup
   - neoplasms—larynx, trachea, or esophagus
   - neurologic disorders—bulbar palsy, myasthenia gravis
   - trauma—to larynx or neck, airway, edema, or subglottic stenosis due to instrumentation or intubation
   - allergic reaction
   - vascular rings

Management

1. Stop offending stimulation/surgery; consider need for additional assistance
2. 100% oxygen
3. Open airway—chin lift/jaw thrust, assess re: aspiration
4. Deepen anesthesia (IV induction agent)
5. Positive pressure mask ventilation
6. If unsuccessful—treat with succinylcholine as appropriate
7. If bradycardia—treat with atropine
8. If ventilation does not improve—intubate/ventilate

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Local Anesthetic Systemic Toxicity

Definition: A constellation of clinical symptoms associated with high plasma levels of local anesthetic, and ranging from dizziness to complete cardiovascular collapse.

Clinical Features

NB: The "classic" description of clinical features involves worsening neurologic symptoms with increasing concentrations of local anesthetic, progressing to seizures, loss of consciousness, followed by cardiovascular manifestations. Examination of the literature has shown this to be oversimplified, many cases present initially with cardiovascular signs (11%) or a combination of neurologic symptoms and cardiovascular signs (44%)

1. Neurologic symptoms:
   - seizure (68%)
   - agitation (11%)
   - loss of consciousness (7%)
   - dysarthria, perioral numbness, tinnitus, dizziness, dysphoria (18%)
2. Cardiovascular signs:
   - bradycardia/asystole (27%)
   - hypotension (18%)
   - tachycardia (16%)
   - VF/VT (13%)
   - widening of QRS complex (12%)
   - ST changes, chest pain, dyspnea, hypertension (9%)
   - ventricular ectopy (5%)

Differential Diagnosis

1. Seizure from other causes
2. Bradycardia, tachycardia, or cardiac arrest from other causes
3. Pulmonary embolus
4. Anaphylaxis
5. Myocardial ischemia/infarction
6. Methemoglobinemia (esp. if using benzocaine or prilocaine)

Management

1. Stop injecting local anesthetic
2. Call for help
3. Ensure adequate oxygenation and ventilation:
   - mask ventilation is often sufficient, but if full stomach or if cardiovascular collapse, endotracheal intubation should be performed
   - this initial step is critical in preventing hypoxia and acidosis, both of which potentiate local anesthetic toxicity
4. Halt seizures:
   - benzodiazepines are first-line treatment, for example, midazolam 2 to 4 mg
   - small doses of propofol (20-40 mg) are acceptable, although cardiovascular depression is a concern
5. Circulatory support:
   - expand circulating volume with IV crystalloid
   - chest compression is severe hypotension or pulselessness
   - electrical therapy as indicated by ACLS protocols
   - vasopressor therapy is controversial based on a number of experimental studies. If cardiac arrest occurs, epinephrine should be used in small doses (10-100 µg)
   - do not use vasopressin, calcium channel blockers, beta blockers, or lidocaine in treatment of cardiac arrest/arrhythmias
   - amiodarone is antiarrhythmic of choice
6. Lipid emulsion therapy:
   - consider administering at the first sign of toxicity (even mild symptoms)
   - dosing regimen:
     - 1.5 mL/kg of 20% lipid emulsion bolus (approximately 100 mL for most adults)
     - infusion of 0.25 mL/kg/min, continued for at least 10 minutes after return of circulatory stability
     - if circulatory stability not attained, consider rebolusing and increasing infusion dose to 0.5 mL/kg/min
     - maximum dose is approximately 10 mL/kg over first 30 minutes
     - do not use propofol $\longrightarrow$ while it has a lipid emulsion as a constituent, the amount required to achieve an effect would cause profound circulatory depression
7. Consider institution of cardiopulmonary bypass if available and resuscitation not responsive to above measures

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Malignant Hyperthermia

Definition: Hypermetabolic state caused by succinylcholine or halogenated volatile anesthetics in genetically susceptible patients.

Clinical Features

1. Hyperthermia (rapid, sustained rise)
2. Rigidity
3. Activation of SNS—tachycardia, hypertension, tachypnea, arrhythmias
4. Acidosis (elevated $ETCO_2$), hyperkalemia
5. Rhabdomyolysis—elevated creatine kinase
6. Acute kidney injury—myoglobinuria

Differential Diagnosis

1. Malignant hyperthermia
2. Neuroleptic malignant syndrome—similar presentation to MH but associated with use of antipsychotic neuroleptic medications (also treated with dantrolene)
3. Thyroid storm—fever, tachycardia, altered mental status—assess for history of hyperthyroidism
4. Anaphylaxis—cardiovascular collapse without hypermetabolic features
5. Pheochromocytoma—significant hypertension
6. Drug toxicity—consider clinical context, screen urine/plasma
7. Consider other causes of fever—infection, malignancy, autoimmune

Management

1. Consider differential diagnosis and exclude other causes
2. Stop offending anesthetic, stop surgery
3. Hyperventilate with 100% oxygen
4. Call for assistance
5. Dantrolene 2.5 mg/kg IV, repeat prn
6. Cool patient: IV fluids, ice packs, gastric/peritoneal lavage
7. Treat arrhythmias—avoid calcium channel blockers
8. Sodium bicarbonate—1 to 2 mEq/kg prn
9. Monitor temperature, electrolytes, arterial/venous blood gases, CK, urine output, coagulation studies
10. Monitor for recurrence—continue dantrolene 1mg/kg q 4 to 6 hours $\times$ 24 to 48 hours
11. Refer for genetic counseling/in-vitro muscle contracture testing

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Definition: The "difficult airway" can refer to difficulty with any combination of the following: difficult BVM ventilation, laryngoscopy, intubation, placement of a supraglottic device (eg, LMA), or difficult cricothyrotomy.

Clinical Features

According to the ASA Closed Claims Database, failure to evaluate the airway and predict difficulty is the single most important factor leading to a failed airway

Predictors of difficult bag-valve-mask ventilation:

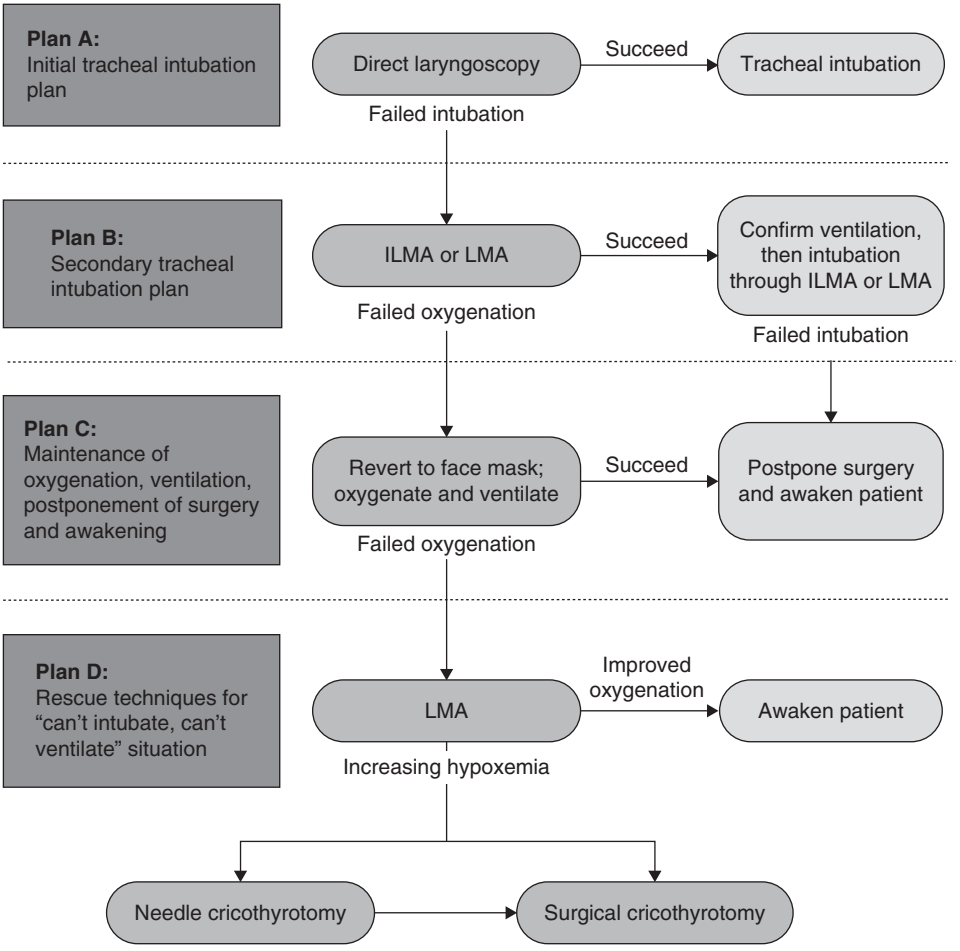
1. History of neck radiation
2. Increased body mass index (>26 kg/m^2)
3. Presence of beard
4. Lack of teeth
5. Age >55 years
6. Limited mandible protrusion test (bottom teeth advanced below top teeth)
7. History of OSA/snoring
8. ↓ Pulmonary compliance (opioids, pulmonary edema, asthma)
9. Airway tumors, hematomas, foreign bodies
10. Male sex

Predictors of difficult laryngoscopy & intubation:

1. History of difficult intubation
2. Mallampati class III or IV
3. High-arched palate, large tongue, prominent incisors, receding mandible, small mouth opening, short and/or thick neck
4. Mouth opening <2 to 3 cm, thyromental distance <6 cm
5. Acromegaly, diabetes, rheumatoid arthritis
6. Limited cervical spine movement

Management

1. Assess likelihood of difficult ventilation and/or laryngoscopy as above
2. If difficulty is anticipated, consideration of an awake intubation is indicated; awake FOB intubation or awake surgical airway
3. The following aspects should also be considered:
   a. if direct laryngoscopy is evaluated as likely to be difficult, is the anesthesiologist skilled in an alternative technique likely to be successful in this situation?
      i. if not, awake intubation is strongly encouraged
      ii. if yes, consideration of intubation after induction of GA using the alternative strategy is acceptable, presuming that there is no anticipated difficulty with BVM ventilation or the use of a supraglottic device
   b. is there an aspiration risk?
      i. multiple attempts at direct laryngoscopy are associated with ↑ risk, so combo of difficult laryngoscopy and full stomach may prompt consideration of an awake intubation



Approach for unexpected difficult airway as per the Difficult Airway Society.

Reference

Difficult Airway Society: www.das.uk.com

Massive Blood Transfusion

Definition: Replacement of patient's total blood volume (approximately 10 units of blood) in 12 to 24 hours.

Clinical Features

1. Hypovolemia shock:
   - hypotension
   - low cardiac filling pressures
   - tachycardia
   - pallor
   - peripheral vasoconstriction
   - altered mental status
2. Thrombocytopenia
3. Coagulation factor depletion
4. Hypocalcemia
5. Hyperkalemia
6. Acid/base alterations
7. Hypothermia

Differential Diagnosis

1. Surgical bleeding
2. Bleeding disorder—vWD, hemophilia A/B
3. Trauma
4. DIC—sepsis/infection, malignancy, hepatic failure, toxins
5. Post-CPB coagulopathy
6. Obstetrical hemorrhage—atony, retained placenta, laceration, abruption

Management

1. Assess need for airway management and initiation of cardiopulmonary resuscitation
2. If during surgery, consider risk/benefit of aborting procedure
3. Call for assistance, notify transfusion service/blood bank
4. Consider need for invasive hemodynamic monitors and rapid transfusion devices
5. Blood transfusion: rarely indicated if Hb >10 g/dL (100 g/L), usually indicated if Hb <7 g/dL (70 g/L)
6. Minimize hypothermia with fluid warmers and external forced-air warming devices
7. Labs: CBC, electrolytes (potassium, calcium), ABG, PT/PTT/INR, fibrinogen
8. Monitor platelet count, PT/PTT, and fibrinogen level to assess need for component therapy of platelets, fresh frozen plasma, and cryoprecipitate respectively
9. Replace calcium as required, treat hyperkalemia
10. Consider use of recombinant factor VIIa if persistent coagulopathy

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Metabolic Acidosis

Definition: ↓ pH and bicarbonate concentration in tissues caused by either the accumulation of excess acids stronger than carbonic acid or by abnormal losses of fixed base from the body; blood pH is <7.35 and HCO_3^- <21 mEq/L.

Clinical Features

1. Hyperventilation (or overbreathing ventilator if mechanically ventilated)
2. Dysrhythmias
3. ↓ Cardiovascular response to catecholamines
4. ↓ Myocardial contractility and cardiac output
5. Hypotension, shock

Differential diagnosis

1. Body is producing too much acid:
   - chronic renal failure
   - lactic acidosis from shock/hypoperfusion, cardiac arrest, hypoxemia, release of tourniquet, cyanide/CO toxicity
   - malignant hyperthermia
   - diabetic ketoacidosis
2. Body has been poisoned with acids:
   - methanol ingestion (formic acid)
   - ethylene glycol ingestion (glycolic and oxalic acids)
   - aspirin
3. Body is losing HCO_3- or cannot excrete H^+:
   - renal tubular acidosis
   - pancreatic, biliary, or intestinal fistulas
   - diarrhea
   - acute renal failure
4. Body has received too much normal saline (hyperchloremic metabolic acidosis)

Management

1. Ensure adequate oxygenation and ventilation:
   - 100% O_2, ensure clear airway, consider mild hyperventilation to provide short-term compensatory respiratory alkalosis
2. Ensure adequate oxygen delivery to tissues:

   REMEMBER: $DO_2 = CO \times (Hb \times SaO_2 \times 1.34) + (PaO_2 \times 0.003)$

   - fluid boluses with crystalloid/colloid
   - inotropes/vasopressors as guided by diagnosis (eg, sepsis) and hemodynamic parameters
   - transfusion if indicated (usually indicated if Hb <6 mg/dL, usually *not* if >10 mg/dL)
   - use lactate levels, SvO_2 to provide indication of tissue perfusion
3. Treat cause of acidosis:
   - review anesthetic record, patient problem, and medication list
   - check electrolytes, anion gap, osmolar gap, ketoacids, tox screen
   - consider cyanide poisoning if sodium nitroprusside administered recently: Rx—sodium nitrite (produces methemoglobin, which combines with CN^- to produce cyanomethemoglobin), and sodium thiosulfate (catalyzes the conversion of CN^- to thiocyanate)
4. Other resuscitative measures:
   - hemodialysis if severe, not responsive to above measures and not contraindicated
   - bicarbonate therapy:
     - only if pH <7.1 (level associated with dysrhythmias and ↓ response to catecholamines)
     - controversial due to concern regarding complications including:
       - CNS acidosis
       - hypercapnia
       - tissue hypoxia due to left shift of oxy-Hb dissociation curve
       - volume overload
       - hypokalemia
       - alkali stimulation of organic acidosis (lactate)

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Definition: Myocardial ischemia occurs when myocardial oxygen requirements exceed myocardial oxygen supply. Myocardial infarction is myocardial necrosis in the clinical setting of ischemia.

Clinical Features

1. Asymptomatic (or unable to assess symptoms if under general anesthesia)
2. Symptoms—angina, dyspnea, diaphoresis, nausea, syncope, altered mental status
3. Hypertension, hypotension
4. ECG changes—ST-T changes, new LBBB, arrhythmias
5. Echocardiography—new regional wall motion abnormality
6. Blood tests—elevated troponin I or T

Differential Diagnosis

1. Cardiac:
   - partial or complete coronary artery occlusion due to plaque rupture/erosion
   - ischemia/infarction due to increased oxygen demand or decreased supply (coronary spasm/embolism, anemia, arrhythmias, hypertension, hypotension)
2. Other cardiac—aortic dissection, pericarditis
3. Chest wall trauma
4. Pulmonary embolism
5. Severe asthma
6. Esophagitis

Management

1. Assess need for airway management and initiation of cardiopulmonary resuscitation
2. If during surgery, consider risk/benefit of aborting procedure
3. Restore coronary oxygen supply:
   - supplemental oxygen
   - treat anemia
   - optimize blood pressure, heart rate/rhythm
4. Decrease coronary oxygen demand:
   - analgesia
   - nitrates
   - beta blockers
   - optimize blood pressure, heart rate/rhythm
5. Monitor ECG, consider need for invasive hemodynamic monitors
6. Depending on underlying cause consider aspirin or anticoagulants (heparin, LMWH)—if not contraindicated by surgical procedure
7. Cardiology consultation re: thrombolytic therapy, coronary angiography/angioplasty, referral for emergency cardiac surgery
8. Postcardiac arrest—referral for induction of therapeutic hypothermia

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Definition: Excessive bleeding at time of delivery. Normal blood loss is 500 mL for vaginal delivery and 1000 cc during a cesarean section.

Clinical Features

1. Hypovolemic shock:
   - hypotension
   - low cardiac filling pressures
   - tachycardia
   - pallor
   - peripheral vasoconstriction
   - altered mental status
2. Fetal distress:
   - non-reassuring heart rate
   - acidosis
3. Associated with massive hemorrhage:
   - thrombocytopenia
   - coagulation factor depletion
   - hypocalcemia
   - hyperkalemia
   - acid/base alterations
   - hypothermia

Differential Diagnosis

1. Uterine atony:
   - multiple gestation
   - grand multiparity
   - fetal macrosomia
   - polyhydramnios
   - prolonged labor
   - chorioamnionitis
2. Vaginal or cervical tears
3. Retained placental fragments
4. Placenta previa, placenta accreta
5. Placental abruption
6. Uterine rupture
7. Other:
   - bleeding disorder—vWD, hemophilia A/B
   - trauma
   - DIC—sepsis/infection, malignancy, hepatic failure, toxins

Management

1. Assess need for airway management and initiation of cardiopulmonary resuscitation
2. Call for assistance and potential need for neonatal resuscitation
3. Factor replacement for underlying coagulation abnormality
4. Notify transfusion service/blood bank
5. Uterine atony:
   - bimanual uterine compression, uterine massage
   - medications to improve uterine contraction:
     - oxytocin
     - methylergonovine
     - $PGF_{2\alpha}$
   - surgery—uterine vessel embolization/ligation or hysterectomy
6. Emergency cesarean section/hysterectomy may be necessary for placenta previa, placenta accreta, placental abruption, or uterine rupture
7. Consider need for invasive hemodynamic monitors and rapid transfusion devices
8. Blood transfusion: rarely indicated if Hb >10 g/dL (100 g/L), usually indicated if Hb <7 g/dL (70 g/L)
9. Minimize hypothermia with fluid warmers and external forced-air warming devices
10. Labs: CBC, electrolytes (potassium, calcium), ABG, PT/PTT/INR, fibrinogen
11. Monitor platelet count, PT/PTT, and fibrinogen level to assess need for component therapy of platelets, fresh frozen plasma, and cryoprecipitate respectively
12. Replace calcium as required, treat hyperkalemia
13. Consider use of recombinant factor VIIa if persistent coagulopathy

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Oliguria

Definition: Urine output <0.5 mL/kg/h (adult) or <1mL/kg/h (neonates).

Clinical Features

1. Prerenal—thirst, hypotension, tachycardia
2. Renal—recent use of contrast dye, aminoglycoside antibiotics, NSAIDs
3. Postrenal—bladder distension, urge to void

Differential Diagnosis

1. Prerenal—absolute or relative decrease in renal perfusion (bleeding, hypovolemia, heart failure)
2. Renal—glomerular, tubular, vascular, or interstitial (acute tubular necrosis)
3. Postrenal—obstruction (blocked catheter, prostatic hypertrophy)

Management

1. Avoid further renal insults:
   - maintain euvolemia—CVP 10 to 15 mm Hg
   - maintain adequate renal perfusion—MAP >65 to 70 mm Hg
   - avoid nephrotoxins—contrast dye, aminoglycoside antibiotics, NSAIDs
2. Intravascular volume assessment may require invasive monitors (CVP, PAWP, or TEE)
3. Identify and treat underlying cause:
   - replace intravascular volume
   - optimize cardiac output, avoid hypotension
   - correct outflow obstruction
   - stop nephrotoxic medications
4. Renal failure—may require hemodialysis

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Pneumothorax

Definition: Air or gas in the pleural cavity. Tension pneumothorax is air in the pleural cavity under positive pressure.

Clinical Features

1. Chest pain—acute onset, increases with inspiration
2. Dyspnea
3. Anxiety, fatigue
4. Diaphoresis
5. Cyanosis
6. Tachypnea, tachycardia, pulsus paradoxus, hypotension, cardiovascular collapse
7. Distant/absent breath sound, percussion—hyper-resonance
8. Ventilator—high-peak airway pressures, decreased tidal volume

Differential Diagnosis

1. Spontaneous pneumothorax (no underlying lung disease)—cigarette smokers, Marfan syndrome, pregnancy
2. Secondary pneumothorax (underlying lung disease)—COPD, asthma, pneumonia, malignancy, TB, CF, ARDS
3. Iatrogenic pneumothorax—central line placement, barotrauma
4. Traumatic pneumothorax
5. Other—myocardial ischemia, pulmonary embolism, esophagitis

Management

1. Observation may be appropriate for a small, asymptomatic pneumothorax
2. Treat immediately if hemodynamically stable:
   - oxygen
   - support hemodynamic parameters
   - needle aspiration (2nd or 3rd intercostals space, midclavicular line)
   - chest tube

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Postoperative Stridor

Definition: High-pitched sound due to turbulent airflow in the upper airway. Indicates acute upper airway obstruction.

Clinical Features

1. Anxiety
2. Restlessness
3. Hoarseness
4. Respiratory distress—cyanosis
5. Nasal flaring
6. Use of accessory muscles, upright posture

Differential Diagnosis

1. Anatomic/acquired malformations—foreign body, large tonsils, or epiglottis, congenital abnormalities
2. Infection—epiglottitis, acute tonsillitis, retropharyngeal abscess, croup
3. Neoplasms—larynx, trachea, or esophagus
4. Neurologic disorders—bulbar palsy, myasthenia gravis
5. Trauma—to larynx or neck, airway edema or subglottic stenosis due to instrumentation or intubation
6. Allergic reaction
7. Vascular rings
8. Other—laryngospasm, asthma

Management

1. Narrow differential diagnosis based on focused history and physical exam
2. Assess need for emergent airway management—endotracheal intubation or surgical airway (cricothyrotomy or tracheotomy)
3. Consider flexible fiberoptic bronchoscopy or other imaging to assess pathology as situation permits
4. If conservative management:
   - supplemental oxygen
   - nebulized epinephrine (adrenaline) for airway edema
   - dexamathasone 4 to 8 mg IV for airway edema
   - inhaled heliox (70% helium, 30% oxygen)—reduces turbulent flow through airways
5. If airway management required, follow difficult airway algorithm:
   - supplemental oxygen
   - prepare difficult airway equipment
   - assess likelihood of difficult ventilation, difficult intubation, difficult tracheostomy, and patient cooperation
   - consider awake intubation versus intubation after induction of anesthesia
   - consider endotracheal intubation versus surgical airway
   - consider preservation of spontaneous ventilation versus ablation of spontaneous ventilation
6. In most clinical situations, awake fiberoptic intubation is indicated with surgical airway as backup plan

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Pulseless Cardiac Arrest

Definition: Sudden cessation of heartbeat and cardiac function, resulting in the loss of effective circulation.

Clinical Features

1. Sudden collapse
2. Unconsciousness/unresponsiveness
3. Respiratory arrest
4. Cool periphery, pallor

Differential Diagnosis

1. Ventricular fibrillation
2. Ventricular tachycardia
3. Asystole
4. Pulseless electrical activity. Consider:

Hypovolemia	**T**oxins
Hypoxia	**T**amponade, cardiac
Hydrogen ion (acidosis)	**T**ension pneumothorax
Hypo/Hyperkalemia	**T**hrombosis (coronary/pulmonary)
Hypoglycemia	**T**rauma (hypovolemia)
Hypothermia	

5. Monitor artifact (electrical interference from cautery or EKG lead detachment)

Management

1. Quickly verify arrest is real: check for EKG disconnection (mimics asystole), pulse oximeter waveform
2. Initiate BCLS (check responsiveness, open airway, check for breathing)
3. Breathing? If no, two breaths using BVM (or if necessary, mask to mouth or mouth to mouth)
4. Take no more than 10 seconds to check for pulse
5. If no pulse, start chest compressions at 100 per minute. Cycle compressions/ventilations at rate of 30:2 until advanced airway in place (eg, ETT/LMA), then do not stop compressions (100/min) for ventilation (8-10/min)
6. Attach defibrillator and check rhythm
7. Shockable (ie, VF/VT)?
   a. give one shock. Biphasic 12 to -200 J, monophasic 360 J
   b. resume CPR immediately × 5 cycles (or 2 minutes, if airway in place)
   c. secure airway (ETT if possible) and confirm placement
   d. check rhythm again. Shockable?
   e. give another shock. Resume CPR × 2 minutes
   f. give vasopressor q 3 to 5 minutes: epinephrine 1 mg IV/IO or vasopressin 40 U IV (this can replace the first or second dose of epinephrine and should only be given once)
   g. check rhythm again. Shockable?
   h. consider antiarrhythmics → give during CPR between shocks:
      i. amiodarone 300 mg IV
      ii. lidocaine 1 to 1.5 mg/kg IV
      iii. magnesium 1 to 2 g IV for torsade
   i. repeat CPR and shock cycles
   j. if at any time a non-shockable rhythm appears (eg, sinus rhythm), check for pulse. If pulse, begin post-resuscitation care
8. If initially or at any time NOT shockable and pulseless (ie, asystole or PEA):
   a. resume CPR × 5 cycles (or 2 minutes)
   b. give vasopressor q 3 to 5 minutes: epinephrine 1 mg IV/IO or vasopressin 40 U IV (this can replace the first or second dose of epinephrine and should only be given once)
   c. consider atropine 1 mg if PEA and rate slow
9. Search for correctable cause (eg, tension pneumothorax, hypovolemia) and treat as appropriate

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Respiratory Alkalosis

Definition: pH >7.45, $PaCO_2$ <35 mm Hg.

Clinical Features

1. Respiratory—tachypnea
2. Neurologic—confusion, apathy, tetany, seizures
3. Cardiovascular—tachycardia
4. Metabolic—hypokalemia, hypocalcemia

Differential Diagnosis

1. Iatrogenic—excessive ventilation
2. Hypoxia (compensatory tachypnea)—pneumonia, pulmonary edema, aspiration, pulmonary embolus
3. Pregnancy
4. Fever/sepsis
5. High altitude
6. Pain/anxiety
7. CNS disease—stroke, meningitis
8. Hepatic failure
9. Salicylate overdose

Management

1. Identify underlying condition
2. If on ventilator, decrease minute ventilation
3. Follow ABG

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Serotonin Syndrome

Definition: A potentially life-threatening drug reaction that is associated with increased levels of serotonin.

Clinical Features

1. Consists of a triad of neuroexcitatory features:
   - neuromuscular:
     - tremor, clonus, myoclonus, hyper-reflexia, pyramidal rigidity
   - autonomic:
     - diaphoresis, fever, tachycardia, tachypnea, diarrhea/ hyperactive bowel sounds, shock
   - altered mental status:
     - agitation, excitement, confusion
2. Association with drugs or combinations of drugs that ↑ serum serotonin levels:
   - SSRIs (eg, sertraline, paroxetine)
   - MOAIs (eg, phenelzine)
   - other antidepressants (eg, trazodone, buspirone)
   - antiemetics (eg, ondansetron, metoclopramide)
   - drugs of abuse: MDMA ("ecstasy"), amphetamines, cocaine
   - phenylpiperidine opioids: meperidine, fentanyl, sufentanil, alfentanil, remifentanil, dextromethorphan

Differential Diagnosis (and differentiating features)

1. Malignant Hyperthermia:
   - ↑$ETCO_2$
   - temporal relationship to inhalational agents or succinylcholine
   - rigor-mortis like rigidity
   - hyporeflexia
2. Neuroleptic malignant syndrome
   - idiopathic reaction to dopamine antagonists evolving over several days
   - "lead-pipe" rigidity
   - bradykinesia
3. Anticholinergic syndrome:
   - normal reflexes
   - mydriasis, delirium, dry oral mucosa, hot skin, urinary retention, absent bowel sounds

Management

1. Stop the offending drug
2. Supportive care:
   - IV fluids
   - correction of vital signs with *direct*-acting vasopressors (NE, phenylephrine), short-acting beta blockers (esmolol) and/or dilating agents (nitroprusside)
3. Give benzodiazepines to treat agitation and blunt hyperadrenergic component (eg, diazepam or midazolam). Physical restraints may increase mortality by ↑ isometric contraction ⟶ lactic acidosis and hyperthermia
4. Give 5-HT_{2A} antagonists:
   - cyproheptadine 12 mg po/NG, then 2 mg q2h if symptoms continue. Daily dose 24 to 32 mg
5. Control hyperthermia:
   - surface cooling
   - if severely ill with temp >41.1°C, patient should be paralyzed, sedated, and intubated (no succinylcholine as ↑ risk of hyperkalemia)
   - no role for antipyretics. Hyperthermia is due to muscle activity, not alteration in hypothalamic set point

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Sinus Tachycardia

Definition: A heart rate of >100 beats per minute (in adults) where each narrow-complex QRS complex is preceded by a P wave.

Clinical Features

1. Rapid heart rate (>100 bpm)
2. Dizziness, syncope
3. Chest pain, palpitations
4. Dyspnea

Differential Diagnosis

1. CNS:
   a. light anesthesia
   b. pain/anxiety/stress
2. Cardiovascular:
   a. reflex tachycardia following hypotension/hypovolemia
   b. supraventricular or ventricular tachycardia
   c. atrial fibrillation/flutter
   d. restrictive cardiomyopathy
   e. pulmonary embolism
   f. sick sinus syndrome
3. Pulmonary:
   a. hypoxemia
   b. hypercarbia
   c. tension pneumothorax
4. Metabolic:
   a. malignant hyperthermia
   b. fever, sepsis
   c. hypoglycemia
   d. carbon monoxide poisoning
5. Endocrine:
   a. thyrotoxicosis
   b. pheochromocytoma
   c. carcinoid tumor
6. Drugs:
   a. anticholinergic: atropine, glycopyrrolate
   b. other vagolytics: pancuronium, desflurane, meperidine
   c. sympathomimetics (eg, ephedrine, epinephrine, cocaine)
   d. histamine-releasing drugs

Management

1. Ensure adequate oxygenation and ventilation:
   - establish/maintain patent airway
   - ensure gas exchange with high-flow/100% oxygen
2. Check EKG tracing to rule out supraventricular tachycardia or wide-complex tachycardia
3. Check depth of anesthesia (vaporizers, TIVA pumps, BIS monitor, etc)
4. Check core temperature, end-tidal CO_2
5. Assess volume status:
   - check blood loss, urine output, review ins and outs
6. Review anesthetic:
   - more opioid needed? Drug error?
7. Review chart and consider differential diagnosis in context of particular patient

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Supraventricular Tachycardia

Definition: A heart rate of >100 beats per minute (in adults) associated with narrow-complex QRS complexes (<0.12 milliseconds).

Clinical Features

1. Rapid heart rate (>100 bpm)
2. Dizziness, syncope
3. Chest pain, palpitations
4. Dyspnea

Differential Diagnosis

1. See wide differential for sinus tachycardia on separate card
2. Other narrow-complex tachycardias:
   - atrial fibrillation
   - atrial flutter
   - AV-nodal reentry tachycardia
   - accessory pathway-mediated tachycardia (eg, WPW, LGL)
   - atrial tachycardia (ectopic and reentrant)
   - multifocal atrial tachycardia (MAT)
   - junctional tachycardia
3. Misdiagnosed wide-complex tachycardia
4. Monitor artifact

Management

1. Check pulse $\longrightarrow$ if not present, immediately switch to pulseless arrest algorithm (see Pulseless Cardiac Arrest card)
2. Ensure adequate oxygenation and ventilation
   - establish/maintain patent airway
   - ensure gas exchange with high-flow/100% oxygen
3. Check EKG tracing to identify rhythm if possible; confirm rate with oximetry tracing
4. Assess patient stability. Unstable signs include:
   - altered mental status
   - chest pain
   - hypotension/shock
   - dyspnea
5. If unstable, perform immediate synchronized cardioversion:
   - use 100 to 200 J with a monophasic waveform (atrial fibrillation), 50 to 100 J for atrial flutter and other SVTs
   - escalate subsequent doses of energy as needed
   - if using biphasic waveform, 120 J is appropriate
6. If stable, check rhythm again. Irregular? If yes:
   - probably atrial fibrillation (or possibly atrial flutter or MAT)
   - control rate with diltiazem (0.25 mg/kg IV) or β blockers (esmolol 0.5 mg/kg or metoprolol 5 mg IV $\times$ 5 minute up to 15 mg)…careful if CHF
7. If regular rhythm:
   - may attempt vagal maneuvers (Valsalva, carotid massage), but frequently unsuccessful
   - give adenosine 6 mg rapid IV push. If no conversion, give 12 mg. Repeat once more if needed
8. If successfully converted, probably reentry SVT:
   - observe for recurrence
   - treat recurrence with more adenosine or AV-nodal blockers (diltiazem, β blockers)
9. If rhythm does NOT convert with adenosine:
   - possible atrial flutter, ectopic atrial tachycardia, or junctional tachycardia
   - rate control with diltiazem, β blockers
10. Search for correctable cause:
    - surgical manipulation of the heart/pericardium
    - digitalis overdose
    - electrolyte imbalances (esp. hypokalemia)
    - hypothermia
    - acidosis
    - toxins
11. Observe and arrange for cardiology follow-up

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Total Spinal

Definition: High or total spinal occurs with excessive cephalad spread of local anesthetic in the subarachnoid space. Increased incidence in obstetric population.

Clinical Features

1. Loss of consciousness
2. Respiratory depression
3. Hypotension
4. Dilated pupils

Differential Diagnosis

1. Anxiety, panic attack
2. Drug overdose
3. Pulmonary embolus
4. Amniotic fluid embolus
5. Pulmonary edema
6. Seizures
7. Hypoglycemia

Management

1. Assess for total spinal with repeated examination. High neuraxial blockade suggested with loss of ability to phonate or squeeze hand
2. Supportive care:
   - assess need for intubation or cardiopulmonary resuscitation
   - maintain oxygenation/ventilation
   - support hemodynamic parameters: left uterine displacement, intravenous fluids, vasopressors
3. Caution with reverse Trendelenburg position. It will not prevent cephalad spread of local anesthetic and may lead to cardiovascular collapse due to venous pooling

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Definition: Adverse reaction to transfused blood or blood components, ranging from mild to life-threatening.

Clinical Features

1. Fever
2. Allergy:
   - rash
   - flushing
   - urticaria
   - dyspnea
   - bronchospasm
   - edema, angioedema
   - hypotension
   - anaphylaxis/shock
3. Inflammation:
   - chills/rigors
   - hypotension
   - shock
4. Respiratory:
   - asymptomatic
   - dyspnea
   - CXR—bilateral infiltrates
   - hypoxemia

Differential Diagnosis

1. Immunologic:
   - hemolytic—fever, pain, flushing, anxiety, hypotension, hematuria
   - febrile nonhemolytic—fever, chills
   - urticarial—urticaria
   - anaphylactic—hypotension/shock, angioedema, bronchospasm, rash
   - TRALI—transfusion-related acute lung injury—hypoxemia, bilateral infiltrates on CXR
   - graft versus host disease—fever, rash, diarrhea, hepatitis
2. Nonimmunologic:
   - TACO—transfusion-associated circulatory overload—dyspnea, tachycardia, hypertension, increased PAWP
   - nonimmune hemolysis
   - air embolus
   - hypocalcemia
   - hypothermia
   - sepsis

Management

1. Stop transfusion
2. Determine diagnosis
3. Acute hemolytic reaction:
   - maintain hemodynamic parameters
   - maintain urine output (fluid, mannitol, diuretics, alkalinization)
   - monitor for hyperkalemia
   - blood for CBC, coagulation, fibrinogen, haptoglobin
   - send blood back to blood bank for re-cross-match
4. Febrile nonhemolytic:
   - stop and determine if acute hemolytic reaction
   - treat fever with acetaminophen
   - leukoreduction of blood products can reduce incidence
5. Urticarial:
   - antihistamines—diphenhydramine (pretreatment with steroids ineffective)
   - washing PRBCs in saline will reduce allergic reactions
   - can continue transfusion
6. Anaphylaxis:
   - intravenous fluids
   - epinephrine bolus/infusion as required
   - monitor peak airway pressures
   - antihistamines and corticosteroids
7. TRALI:
   - supplemental oxygen
   - endotracheal intubation/ventilation
8. Graft versus host disease:
   - no effective treatment
   - prevention—irradiated blood components
9. TACO:
   - supplemental oxygen
   - diuretics
   - prevention: volume reduction of PRBCs

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Venous Air Embolism

Definition: The introduction of air into the vascular system, resulting in a spectrum of clinical effects from the asymptomatic patient to shortness of breath to cardiovascular collapse.

Clinical Features

1. A sudden drop in end-tidal carbon dioxide ($EtCO_2$) may be the first sign, as pulmonary dead space ↑; concomitant rise in $PaCO_2$
2. If CVP in place, may be able to aspirate air from right atrium; also, pressures may be elevated
3. Precordial Doppler ultrasound over right atrium is very sensitive ($2 \times ETCO_2$)
4. Best monitor (but requires operator experience) is TEE
5. The classic "mill-wheel" continuous murmur is rare and usually only heard with massive VAE
6. ↑ End-tidal N_2 concentration

Differential Diagnosis

1. Pulmonary thromboembolism, fat embolism, amniotic embolism
2. Brain-stem retraction/ischemia
3. Other causes of hypotension
4. Entrainment of air into respiratory gas analyzer

Management

1. Prevent further air entry:
   - inform surgeon of probable diagnosis
   - flood wound with saline, compress wound if possible
   - discontinue nitrous oxide (will increase functional impact of bubbles)
   - increase venous pressure by adding positive end-expiratory pressure (PEEP) and/or a fluid bolus
2. Minimize the possible cardiovascular impact:
   - change position if possible: left lateral decubitus may aid in preventing or dislodging right ventricular outflow obstruction airlock
   - aspirate air from circulation if CVP catheter is in place
3. Cardiorespiratory support:
   - maintain cardiac output: fluids and inotropes (eg, epinephrine) as indicated
   - ventilate with 100% oxygen, and maintain PEEP

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Wide-Complex Tachycardia

Definition: A heart rate of >100 beats per minute (in adults) associated with wide-complex QRS complexes (>0.12 milliseconds).

Clinical Features

1. Rapid heart rate (>100 bpm)
2. Dizziness, syncope
3. Chest pain, palpitations
4. Dyspnea

Differential Diagnosis

1. See wide differential for sinus tachycardia on separate card
2. Misdiagnosed narrow-complex tachycardias:
   - atrial fibrillation
   - atrial flutter
   - AV-nodal reentry tachycardia
   - accessory pathway-mediated tachycardia (eg, WPW, LGL)
   - atrial tachycardia (ectopic and reentrant)
   - multifocal atrial tachycardia (MAT)
   - junctional tachycardia
3. Supraventricular tachycardia with aberrancy
4. Monitor artifact

Management

1. Check pulse → if not present, immediately switch to pulseless arrest algorithm (see Pulseless Cardiac Arrest card)
2. Ensure adequate oxygenation and ventilation:
   - establish/maintain patent airway
   - ensure gas exchange with high-flow/100% oxygen
3. Check EKG tracing to identify rhythm if possible; confirm rate with oximetry tracing
4. Assess patient stability. Unstable signs include:
   - altered mental status
   - chest pain
   - hypotension/shock
   - dyspnea
5. If unstable, perform immediate synchronized cardioversion:
   - for monomorphic VT using monophasic waveform → initial shock at 100 J. Increase dose as needed stepwise (eg, 200 J, 300 J, 360 J)
   - for biphasic waveform, use 120 to 200 J
6. If stable, check rhythm again. Irregular? If yes:
   - probably atrial fibrillation with aberrancy, but could be pre-excited atrial fibrillation (eg, WPW) in which case AV-nodal agents (eg, diltiazem, adenosine, verapamil, digoxin) are contraindicated as they can INCREASE the rate of transmission through the accessory pathway and cause ↑↑ ventricular rates or VF. If any doubt, cardiovert electrically or use procainamide (17 mg/kg at 50 mg/min maximum infusion rate) or amiodarone 150 mg
   - torsade de pointes? Magnesium 1 to 2-g load followed by infusion
7. If regular wide-complex rhythm:
   - amiodarone 150 mg IV
8. Observe closely and arrange for cardiology follow-up