A Guide to UCSD Anesthesiology for Residents

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Leon Chang, MD

Clinical Director

Associate Clinical Professor

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Chief editors: Geoffrey Langham, MD; Sonia Nhieu, MD

Assistant editors: Blake Fowler, MD; Nathalie Hernandez, MD; Jason Meeks, MD
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Chapter 1. Introduction to the UCSD Anesthesiology Clinical Curriculum

Welcome to the UCSD Department of Anesthesiology. This guide has three goals: to provide an “insider’s look” or a “resident’s perspective” on our daily lives at UCSD, to describe common approaches to common surgeries we do at UCSD, and to describe physiology and pathophysiology pertinent to those surgeries. It provides basic information on the various general and subspecialty clinical rotations and other aspects of the anesthesiology residency. As such, it is intended to serve as a reference for incoming and current residents. This guide is not intended to replace existing syllabi for the various rotations and it does not describe anesthetic physiology or pharmacology in any great depth. Rather, it will provide the kind of help that one resident wants from another when he or she asks, “Hey, what can I expect out of this month, rotation, case, or situation?”

Our Philosophy

In many ways, the residents here make this program what it is. We pride ourselves on being a tight program with great camaraderie and significant autonomy. Hopefully you knew this already and it was part of why you decided to join UCSD. If the work gets done and things run smoothly during the day, 99.9% of the time it’s because the residents did their jobs, and did them well. This is not a place where your hand will be held and things will be done for you. Often you will have to take the initiative and responsibility yourself to see that things are done right. The reward for all of this is that our graduates are tremendously skilled, fully independent anesthesiologists, and recognized as such around the country.

Your fellow residents are there to help you, and with time you will be there to help them. We routinely help each other out by doing pre-ops, helping with case starts, giving breaks, and so on. Possibly the best example of this occurs during call. Often the senior resident will be supervising or helping the junior resident, while the whole team continues setting up rooms for each other, pre-opping the next patient, etc. If you are ready and willing to lend a hand to your colleagues, that kindness will be visited back to you tenfold in the future. Likewise, if you shirk responsibility, or leave others to fend for themselves, it will be noticed.

It’s important to remember that we’re all in this together. A stressful, intense case, workweek, or call will get balanced out by the more laid-back rotations and the fact that you’re in a friendly work environment. A week where you are late pre-call and have to do a keyword presentation is a tough one, but there will be days when you are sent home at 1100 prior to a 1500 start call. It all works out in the end, and if things are ever too much to handle on your own there’s always a friend ready to lend a hand.

The theme which will be repeated throughout your residency is that of resident responsibility. There is tremendous autonomy in this program, which inevitably can be accompanied by periods of stress. Throughout the various rotations and experiences here at UCSD you will hear emphasized time and time again: “It’s your case. The work is up to you. It’s your responsibility.”

This is a resident-driven program, and to remain so, it depends on residents taking the initiative in patient care and getting the work done. There will be many times where “going the extra mile” will make someone’s day easier or someone’s anesthetic safer. This effort will be rewarded. Conversely, laziness can only hurt you and your fellow residents, and will be noticed.

The beauty of this program is that the responsibility we are given enhances our education and our ability to function independently. By treating our duty with the utmost respect this tradition will continue.
Chapter 1A. Rotations at UCSD Anesthesiology

Each year at UCSD is comprised of thirteen 4-week blocks. Of these blocks, nearly all are required rotations. The required rotations are:

- Main OR (MOR), including night float
- VA (a main OR rotation)
- Obstetric anesthesia (2 months)
- Neuroanesthesia (2 months)
- Cardiothoracic anesthesia (2 months; a 3rd month is optional)
- Pediatric anesthesia (2 months; done at Rady Children’s Hospital San Diego)
- Airway management
- Regional anesthesia (2 months)
- Pain medicine
- ICU (3 months, this is in addition to any ICU experience taken during the PGY-1 year)
- Pre-op clinic and PACU (½ month each)

Two months of electives are taken in the CA-3 year. Available electives include:

- Transesophageal echocardiography
- OR management
- Research
- Advanced regional anesthesia
- Benumof’s “Lessons Learned” book
- NICU
- CA-3 pediatrics (1 month at Rady Children’s Hospital San Diego)
- CA-3 pediatrics (2 months at LA Children’s Hospital)
- Away rotation

Detailed descriptions of each rotation and the expectations are provided in the individual chapters. The exact timing of when you will do specific rotations varies among residents, but in general the years break down as follows:

- CA-1 year: MOR and VA provide a solid base and exposure to daily OR anesthesia. Most residents will complete at least one advanced rotation, e.g. OB or cardiac anesthesia, during this year. ICU, pre-op clinic, and PACU rotations complete the year.
- CA-2 year: Primarily subspecialty rotations (OB, Neuro, Cardiac, Regional, Pain, Peds, ICU, Airway), interspersed with MOR and the VA.
- CA-3 year: Primarily MOR, with the 2nd of 2 months each of Neuro, Peds, and OB; 2 months of electives.

Chapter 1B. A Typical Day in the Main OR

The following is what a typical day in the main OR is like. There are individual variations between Hillcrest, Thornton, and the VA; these will be discussed as applicable.

You should always plan on having your room set up by 0640 to be at morning conference promptly at 0640. Wednesday is the exception; QI conference (a.k.a., M&M) starts at 0630. Allow yourself ample
time to gather everything you need. Depending on the case, this can be simple or quite time-consuming. Setup activities can include preparing drugs, airway equipment, drips, special monitors, and so on. Residents vary in how fast they can do this, so you should always allow yourself plenty of time. The responsibility to get things properly set up is yours and yours alone. For new residents, allow around 40 minutes for a basic case. As you get your style down, setup for an uncomplicated GA case may take as little as 10-15 minutes.

Often the anesthesia technicians (a.k.a., “anesthesia monitoring”) can provide invaluable assistance to your setup. However, don’t count on this. They are busy, spread thin, and have their own morning responsibilities. They are most helpful if you need a special item like pressure transducers or a fiberoptic bronchoscope, or if you’ve discovered an equipment problem during your routine check. Learn from them as much as you can, so that you know where equipment is located and you’ll be able to grab it when you need it on call at 0200, for example.

Morning conference occurs between 0640 and 0700. After conference, you will meet your patient, review the plan with him or her, confirm NPO status and morning medications taken, and confirm a functioning IV. (At Hillcrest and Thornton, the pre-op nurses place the IVs; at the VA this is your responsibility). Your goal is to have the patient in the OR by 0720 at Hillcrest and Thornton, and by 0730 at the VA. However, to do this, the surgical H+P must be done, consent signed, surgical site marked, surgeons available, blood products available, pertinent pre-op labs available, surgical equipment available, circulator and scrub tech available, etc. It’s amazing how many times these things aren’t ready to go, and it is rare that the anesthesiology team is the rate-limiting step here. However, your goal is to never be the rate-limiting step. So, before you roll the patient back to the OR, check with the circulating nurse for your room, who will have the best idea if everything else is ready to go. If you are aware of any time-sensitive issues with your patient (blood bank needs another blood sample for type+screen, patient needs po premedication), see your patient and start this process prior to 0640.

After you’ve started your case, your day is underway. You can generally expect a morning break, no more than 15 minutes long. The timing of this might vary depending on how busy things are throughout the OR, availability of CRNAs or your attending, and whether or not you’ve “displeased” the powers that be.

You should prepare drugs and equipment for your next case (as much as feasible and safe) during your current case, and enter your patient’s post-op orders. After you finish a case, typically you will drop off your patient in the PACU, although patients who will remain intubated post-op will go straight to the ICU. Once you’ve tucked away your patient and given report to whoever will assume care (typically, PACU nurse), you should see your next patient, set up for the next case, and so on. This process will depend on housekeeping and anesthesia monitoring staff to clean and prepare the room as well as the availability of surgical equipment. So, if you’ve prepared during your last case and you’re fast, you can often get 5-15 minutes or more between cases to relax.

As a new CA-1, you are expected to page your attending when planning to start and finish all OR cases. Your supervision for this will be extremely close during the initial stages of residency, and will likely be trimmed back as you mature. As well, you are expected to contact your attending with any significant intraoperative events throughout your residency, e.g., “The surgeon has just lost 800ml blood and we are going to transfuse 2 units.” This is not only for the patient’s benefit, but for your medico-legal protection as well.
Your caseload will typically flow as originally scheduled, but cases and personnel do get moved around quite a bit, and emergencies happen. So don’t be surprised to get moved to another room, or to suddenly have to do an emergency or interesting case that just came in. Flexibility and a positive attitude are the goals when this happens.

Lunch breaks are no more than 30 minutes. Lunch is provided by the department every weekday and it is typically delivered around 1100. If you are industrious, you may well have time to squeeze in a quick lunch between cases and still be offered your 30 minute break later in the day. Breaks in the afternoon, no more than 15 minutes, do happen but are more variable due to cases winding down and staff availability. Your typical, non-call, non-late day will end around 1600-1700. This is by no means written in stone, and there are “regular” days that go until 2100 and “late” days that end at 1500. Again, this is residency, and having a positive attitude and being part of the team are key. Always keep in mind that hard work is rewarded, while complaining or laziness are remembered for a long time.

When your room is done, ask the attending running the floor if there’s any more work for you. Often the attending will find you or let you know as you’re wheeling your last patient to the PACU. Never, ever leave without being told you can, even if it seems all the work is done. There might be a case pending that you simply didn’t hear about. You might need to take over another room.

Once you’re free of clinical duty, you still must consult tomorrow’s schedule and find the pre-ops for those patients, so that you can call your attending in the evening to discuss the plan. This could mean photocopying a completed pre-op, tracking down five inpatients and pre-opping them all, or contacting a resident at another location to email or text you the pre-ops. See the pre-op section for more information.

Don’t forget to do something fun with what remains of your day. Residency, especially the early parts of anesthesiology residency, are tiring. It’s hard and stressful at first to do the clinical work, prepare for the next day, and read, but will get better.

**Chapter 1C. The First Four Weeks**

or, “I’m going to be doing this on my own in a month?”

The transition from the clinical base year to the CA-1 year is probably the most striking and jarring moment many of us have faced in our medical careers. In plain English, you go from a confident intern to a completely green anesthesiology resident in a matter of days. Skills which were carefully honed during the intern year, such as writing good notes, rounding in an efficient manner, and learning how to do discharge paperwork, largely fall by the wayside and become irrelevant. All of a sudden you are surrounded by equipment that you don’t know how to use, drugs that you don’t know how to give, and physiology of such an acute and dynamic nature that you can easily think you never went to medical school. Hopefully, these challenges which are largely unique to anesthesia are what drew most of us to the field in the first place.

Here at UCSD we know that people’s exposure to anesthesiology and what it entails vary greatly. Some will have had extensive shadowing experience while others may have had as few as 2 weeks of clerkship time during medical school. The department knows all of this and expects no prior knowledge or ability, save a good fundamental grounding in medicine itself. However, we do expect that you know and appreciate one piece of information prior to embarking on your anesthesiology career. It is so important, and so vastly different from any prior experience any doctor has had that it bears special mention. If you never forget this fact, it will serve you well. Here it is:
In anesthesia, you have the potential to kill patients on a daily basis.

Let’s take a hypothetical example to make this more clear. As a surgery or medicine resident, you prescribe the wrong medication to Mr. X. For Mr. X to actually receive this lethal medication, the following has to happen:

1. You write the wrong order, and
2. Your senior resident or attending misses the fact this is a wrong order, and
3. The pharmacist misses the fact that this is a wrong order, and
4. The nurse misses the fact this is a wrong order, and
5. The nurse physically goes to the medication dispenser, and administers it to the patient.

Here is the equivalent example for an anesthesiologist:

1. In the OR, you decide Mr. X needs a drug, and
2. You draw up the medication, with no one to confirm you’re doing it correctly, and
3. You give the drug to Mr. X, who can’t object to what you’re giving, and
4. You don’t know you did up anything wrong until the HR is 250 or the etCO$_2$ is zero.

There are many, many other potential sources for error or harm. You could flick a switch, turning a machine or monitor off without even knowing you flicked it. You could let air into your IV tubing. You could commit a drug swap. Any of the myriad procedures we perform on patients can have catastrophic outcomes. The bottom line is, as an anesthesiologist, you have a unique responsibility to the patient.

In light of all this, we have a system to transition people from interns to fledgling anesthesiologists within the first month. This system is steeped in tradition and has withstood the test of time. It is designed both to maximize learning and independence and to give new trainees a support structure during this intense transition phase.

**The First Two Weeks**

During the first two weeks, each new CA-1 is paired up with a CA-3. The pair is assigned daily cases in the OR. The CA-3 is expected to supervise the CA-1 with all aspects of work during the day and to start teaching the basics of anesthesia. In this way, the CA-1 can start learning in a supervised yet informal environment and begin meeting other fellow residents. Each day the pair is assigned to work with different attendings to allow the faculty and new residents to get to know one another.

The CA-3 is the primary resource for the CA-1 during these two weeks. The basics of setting up an OR, checking the anesthesia machine, finding and preparing equipment, preparing drugs, and innumerable other pieces of information will all be covered during this time. The vast majority of CA-3s will allow the CA-1s to do things “on their own” once they show they understand, in order to immediately facilitate the independence which is a hallmark of UCSD.

The CA-1 is responsible for calling the attending to discuss the cases for the next day. However, during the first 2 weeks, the CA-3 will be instrumental in preparing for this discussion by going over and formulating a coherent anesthetic plan with the CA-1.

By the end of the first two weeks the CA-1 should have a small knowledge base from which to build, some level of comfort in the OR, and some familiarity with his fellow residents and attendings. The
groundwork for hands-on, routine anesthesia care, plus a general approach to intraoperative emergencies or issues, will be laid during these first 2 weeks.

The Second Two Weeks

At this point, the CA-1 is paired one-on-one with a faculty member. The resident and attending work together every day for the next two weeks. This allows the faculty to intensely train the resident and to build off of prior knowledge and topics. It’s worth noting that the attendings who do this one-on-one training have volunteered to do so and are doing it out of their interest in resident development and education; they are some of the department’s finest educators. Typically the resident is allowed more responsibility and independence during these second two weeks, but, as always, the attending must be called every night to discuss the next day’s cases.

During this whole month the new CA-1s have a daily lecture at 1500. These lectures are designed to provide the “nuts and bolts” of anesthesiology. These lectures are mandatory, and the CA-3s and faculty know you must attend them. Therefore, you will always be released in time to make the lectures. In general, CA-1s are not required to come back to the OR after lecture is finished, although this is always up to the attending running the floor. Of course, the day concludes with getting the next day’s pre-ops and calling the attending.

At the conclusion of these first four weeks, the CA-1 is ready to join “the crew” as a functioning, independent resident. Starting from this point, for the rest of your resident career, you will be the sole resident in any given case. In addition, you will join the overnight call pool and start taking in-house call. While all this may seem daunting and more than you can handle after a mere four weeks of anesthesia training, rest assured that you can handle it. Generations of anesthesiology residents have come before you and flourished under this system. Furthermore, there are multiple support systems in place for you to lean on: your classmates, fellow residents, and the faculty. Remember, we’ve been there before and are always willing to lend a hand.

Chapter 1D. Call Duties

MOR Call

This is the first type of call a resident takes, and it is also the least predictable and the most likely to cause anxiety. You are eligible to take this call right at the beginning of August after your 1st four weeks of one-on-one training. You may not feel ready for it – nobody does, at first – but remember there are senior residents and your attending ready to help you out.

For weekday calls, the day will typically start at either 0600 with a first-case-start OR case, or at 1445. If the call starts at 1445, you must be immediately available on your pager to come in as needed. A call that starts around 0900 in pre-op clinic in the 1st floor of Hillcrest is also a possibility, depending on staffing needs; see the section on pre-op clinic. In general, the attending making the schedule makes every attempt possible to avoid a 0600 start for the call resident, or at least to relieve you early to rest. This in order to maximize your learning:work ratio, but this is not always possible.

Again, flexibility and a positive attitude are the name of the game here. Hard work is noticed and rewarded, whereas complaining is noticed and, ultimately, can be punished. Residents are by no means entitled to or “default” to a 1445 start, but they should always trust that the attendings who make the schedule and run the floor are looking out for their well-being.
On your pre-call night, always check for updated schedules. Things change. Checking your email should be the last thing you do before you go to bed and the first thing you do when you wake up. It’s a good idea to have your pager nearby on your precall night, so if things change, and the front desk decides to page you, you’ll know.

Call duties include your OR clinical work, completing any inpatient pre-ops for the next day (in conjunction with the ICU resident), and sending inpatient and outpatient pre-ops to those residents who need them for the next day. Obviously, these duties may be in conflict with each other; you can’t email pre-ops when you’re sitting in a 6-hour finger reimplantation. However, by coordinating with your attending and those residents who may be contacting you for pre-ops, and by using your time wisely, you can achieve most or all of the above duties, help your colleagues, and become a resident known for hard work, industry, and helping others out – that’s the UCSD way.

As the MOR call resident, you have different types of backup depending on the time of day. Currently there is a CRNA who works 1030-1900, another who works 1500-2300, and the night float resident who works 1900-0700. Depending on the quality and quantity of case load, you may have a call where you are in an OR from 0700 to 0700 the next day (with appropriate breaks), or you may wrap up your cases by 1800 and have no remaining work to do. The best way to not be disappointed is to have low expectations, so that you’re not surprised when you finish every case on the board by 0300 and still get paged for a lap appy at 0500. The best part of an anesthesia call is that once 0700 rolls around you will be relieved.

Weekend MOR call is a little different from weekday call. There are two residents on call: MOR1, and MOR2. The call officially starts at 0700 and lasts 24 hours. However, nearly every weekend day, there are elective cases scheduled, and if there is a first-case-start scheduled, you may need to be in-house earlier than 0700 to set up.

You should treat elective cases on the weekend the same way you would a weekday: you pre-op the patient or obtain the pre-ops, you call your attending to discuss the cases, and you’re ready to be in the OR with the patient at 0720. If there is just one OR scheduled to be running, the MOR1 resident is responsible for pre-ops and calling the attending. If it’s scheduled for 2 concurrent ORs to be running, the MOR1 and MOR2 resident will decide amongst themselves which cases they’d like to do, and call the attending with their respective pre-ops. The brunt of the work during call falls on the MOR1, independent of seniority. But again, in an atmosphere of cooperation, the MOR2 must always be there to give breaks, start cases, or alternate cases with the MOR1 resident should there be many cases scheduled to run back-to-back in a single OR.

Finding out if there are weekend cases scheduled is easy enough via PCIS in the hospital or from home or by calling the OR front desk. A bright spot of weekend call is that the attending on call traditionally buys the whole call team dinner from some nearby restaurant, and the call team usually coordinates to send someone out for lunch takeout as well.

**Weekend MOR Pager Call**

Every weekend there is one resident assigned to “backup” pager call for the Hillcrest MOR. It is relatively uncommon for the pager-call resident to be called in, however, if you are this resident you should have your pager nearby starting the night before so that you’ll be prepared if you’re needed at 0700 the next day. If you look on PCIS or talk with the MOR call residents and see that there are two OR’s already scheduled to be running, be prepared and available to come in. You must be able to be in scrubs and in
the OR within 1 hour of being paged in on this pager call. A perk here is that this call counts as a “work” weekend whether you are called in or not.

**MOR, Thornton, and VA Late**

The late resident is the second-to-last resident to leave the hospital, before the call resident. You are frequently scheduled to be in an OR that is expected to run late, and you will likely also take over ORs from CRNAs or precall or senior residents, at the discretion of the floor attending. A “late” day may end as early as 1500 or as late as 2200. As such, a “late” day is not a good day to schedule a fancy dinner at 1900 with your significant other! One implication of being the “late” resident on Friday is that you are typically presenting a keyword that morning as well. Lastly, be aware that the floor attending is not always 100% aware of who the “late” resident is, and if you are this person and are sent home while multiple non-call, non-residents are still in ORs, you are effectively doing your colleagues an injustice. So, be aware.

**Thornton Call (1500 start)**

This is a Monday-Friday, 1500-start shift where the resident comes in by 1445 and is ready to physically go into an OR no later than 1500. The resident stays in-house until all the cases are done, and is then free to go home on pager call until 0700. Breaks and dinner are arranged with the call attending. The exact time when the cases are all finished can be quite variable at Thornton. Having your pager on during the day, before you shift actually starts, is encouraged, because very infrequently you get called in earlier than 1500 (e.g., an add-on emergency case that there is no one else available for). The floor attendings will respect the “10 hour rule,” such that, if you are in a room until 0700, you will not be expected to be back at Thornton until 1700.

**VA Call**

On weekdays, this call starts at 1000. Call duties include completing inpatient and add-on pre-ops, seeing inpatient post-op evaluations (QA/QI sheets, a.k.a. “blue sheets”), carrying the code pager and responding to codes/airways/IVs. At the floor attending’s discretion, you may be asked to help in the ASU (the VA’s pre-op clinic) or to give breaks. Your in-OR duties, i.e., starting or finishing cases, typically do not start before 1700, but you should certainly be prepared to do so if asked. If your duties are complete, it’s always welcome to lend a hand with a tough case start, observe or help out with a regional block, or go over the TEE with the cardiac anesthesia attending.

One critical difference between VA and MOR call is that at the VA, when all the cases in the OR are done, the call attending will leave the building. This means that for any codes or overnight airway emergencies, the resident is on his or her own. For any OR case that is added on – and at the VA, these are typically true emergencies – you must notify the call attending as soon as possible to allow them travel time to the hospital. The resident does not do OR cases on their own. The same applies for airway “urgencies” – if you do not feel comfortable with an intubation that you are asked to do, you are certainly advised to call in the attending.

Weekends at the VA start at 0700, and are typically very light. The same call duties as during the week apply, with a few additions. Scheduled cases are rare, which means the attending will not routinely come in. After taking over for the outgoing resident, call duties include seeing inpatient pre-ops (added on to the main OR board), “blue sheets,” holding the code pager. You are also responsible for regional anesthesia follow-ups as well as any new pain medicine consults or follow-ups over the weekend. There
is a pain attending on call who will be able to help you with this.

The specifics of the VA, including the computer system, how to write a note, where to get food on weekends, etc., will all be explained to you when you get there. You are advised to bring plenty to keep you busy and to make arrangements for lunch – the cafeteria is not open, and as of June 2012, only breakfast and dinner are delivered to room 5095 at 0600 and 1800.

**OB Call**

Details about the duties on OB will be discussed further in the OB section. During the week, OB call starts with MOR duties: either at 0700 for first-start MOR cases, or at 1445 where you help with finishing up MOR cases at the discretion of the floor attending. When the MOR duties are completed, you relieve the day OB resident.

Once you have taken over the OB pager, you become responsible for any OB issues that may arise. Epidurals and C-sections are foremost, but difficult IV access, resuscitations, or D+E of retained placenta in a patient in hemorrhagic shock may come along as well. The OB anesthesia attending is the person you answer to, and you should notify him or her if you are doing anything. The MOR attending is always a helpful resource as well. Typically, the OB call resident is one of the more senior residents in house and thus may be called upon to hold the code pager, to supervise a junior resident in the MOR, or to help out in any number of ways.

On weekends the procedure is fairly simple: come in at 0700 and take over for the outgoing resident. Often times the OB call resident will help out with MOR duties (case starts, lots of pre-ops) with the other residents, since there is usually some downtime during the day when on OB call. That said, it’s not advised to expect the OB call resident to have done any pre-ops; it’s just a nice bonus if it happens.

Lastly, the OB day and OB call residents have been given the informal responsibility of keeping our lounge clean.

**SICU Call**

This will be described in detail in the SICU section. Your duties to the anesthesiology department include helping with inpatient pre-ops; often the ICU call resident is the only one available to do so during the day. Also, you will hold and respond to the code/airway pager, make sure the two code bags are stocked and ready, make sure the emergency rooms (OR7 and OR11) are stocked and ready, and respond to any emergency OR resuscitations.

**Night Float**

After your cardiac and OB anesthesia rotations are complete, you become eligible for night float. This is a 2-week, 1900-0700, Monday-Friday rotation. The night float resident is available to do OR cases, help with the MOR duties (pre-ops, breaks, code pager, case starts), etc., at the discretion of the MOR and OB attendings. The night float resident is also the primary liver transplant resident during the week.

The role of the night float resident is different from that of the MOR call resident. The night float resident is preferentially kept out of the OR, while the MOR call resident is preferentially put in the OR – this is because the night float resident will be back at 1900 the next day while the MOR call resident is resting at home. This role – assisting, but not primarily responsible for, MOR duties – are independent of
the seniority of the residents who are respectively on MOR and night float. Occasionally a CA-3 on MOR call will be up all night doing cases or holding the code pager while a CA-2 night float resident is in their call room resting.

Although the shift is from 1900 to 0700, the responsibility for liver transplants that start at, say, 1800 or 0500, has never been clearly defined. Some attendings will attempt to use available day personnel to do a daytime transplant, but sometimes lack of available people does not allow this. The best solution is to have your pager with you at all times in case you get called. If you happen to work during the day you will be given the night off, no exceptions, but this event is exceedingly rare.

**CA-1 Heart Call**

This is a pager call that primarily pertains to nighttime, add-on, and weekend cardiac and thoracic cases. Typically the resident on their first month of the cardiac anesthesia is on call a majority of the weekdays. This means if there are cardiac cases running late, or a case gets taken back to the OR after hours they do the case. This can make for a tiring month, but in general our attendings will try to relieve you if you get called in at night. Sometimes elective heart cases are scheduled on the weekend. The CA-1 heart call resident will do these cases, and be expected to fulfill all the usual duties: pre-opping the patient, setting up the room, calling the attending, etc.

**Pediatric (“Diamond”) Call at Rady Children’s Hospital, Pain Call, and Regional Anesthesia Call**

The details of these calls are described in the section giving an overview of these rotations, and will also be explained to you at the relevant rotations. They are considered outside rotations with their own responsibilities. You will not be called in for another duty such as MOR when you are on these rotations.

**Chapter 1E. A Basic OR Setup**

There are certain basics that will apply to every OR setup you do before a case. The room setup becomes modified as the case requires, but all of have a checklist that we do not deviate from. Having, and sticking to, your checklist will obviate embarrassment or, worse still, potential compromise of patient safety. There are many mnemonics for a basic room setup; one example is “MOM SAID.” Briefly:

- **M:** machine
- **O:** oxygen
- **M:** monitors
- **S:** suction
- **A:** airway
- **I:** IV
- **D:** drugs

**Machine:**

Perform a machine check and adjust ventilator settings, etc., as desired. This includes case-specific equipment, e.g., appropriate sized mask, circuit extension for a case with the head away from the anesthesia machine, etc.

**Oxygen:**
Verify that you have a backup E-cylinder of oxygen in the OR, and that at least 1000 psi remains. The tank has a bag full of simple O₂ facemasks and Mapleson circuits, which you must verify. This is your way to save your patient in the event of catastrophic machine failure. It’s important.

**Monitors:**

Ensure that the standard ASA monitors: BP cuff, EKG leads, pulse oximeter probe, and temperature probe are ready to go. A twitch monitor should be ready as needed for your case. If you will use special monitors (e.g., arterial line, EEG), set up or get the necessary equipment.

**Suction:**

Verify that the suction is on and readily available. If you are going to place an OG or NG tube, now is a good time to get that out.

**Airway:**

Prepare your “Plan A” airway equipment for your case, whether it is a laryngoscope, ET tube, LMA, or other. Ensure that oral and nasal airways are available in the cart, that a Bougie is attached to the anesthesia machine, and that there are backup LMAs in the anesthesia machine drawer. This ensures that you have not only Plan A but B, C, and possibly D in the event of an emergent, “cannot ventilate/cannot intubate” situation.

**IV:**

Prepare extra IV fluids or IV start equipment as needed for your case.

**Drugs:**

Prepare appropriate drugs for the case. The basic drugs most of us have drawn up for a general endotracheal anesthetic are fentanyl, midazolam, propofol, lidocaine, succinylcholine, rocuronium or vecuronium, phenylephrine, and ephedrine. Depending on the case, you may want many other drugs ready to go. Prepare any special drips the case requires.

**Chapter 1F. The Code Pager, the Code Bags, and the Emergency OR Setups**

Part of anesthesiology resident duties include carrying the Code Blue/airway pager, ensuring that the code bags are always set up, and that there are ORs set up for an emergency craniotomy and an emergency OR trauma resuscitation. Both these duties will be described in detail.

**The Code Pager**

There are four basic scenarios for which the code pager will be paged:

1. A true cardiac arrest (Code Blue)
2. An airway or respiratory emergency or urgency
3. An inbound trauma or burn patient that might require airway management
4. Other: usually a request for help with a central or arterial line or difficult peripheral IV. These are usually less urgent. Occasionally the ER, ortho, or trauma will request “procedural sedation” for a
short, painful procedure (dislocated shoulder); we typically do not involve ourselves in direct patient care in these situations, but it’s wise to bring these concerns to your attending’s attention.

The person holding the code pager varies on a daily basis and during the day. During the day, typically one of the anesthesiology residents on the SICU team will have the pager. If there is someone on call in the SICU, they will hold it for the rest of the day and night. If there is no SICU resident on call that night, the person with the pager will distribute it to either the MOR or OB call person at the end of their duties, or, rarely, to an attending if none of the above is available. Later at night, the MOR person generally has the pager since the OB person is often occupied with OB work, but it may pass to either the night float resident or MOR or OB attending, as circumstances dictate.

There’s no need to ever go find the code pager; it will find you if you’re the person who should be holding it. When these people are in-house, the order of “priority” of the code pager is:

SICU resident > MOR call > night float > OB call > MOR attending > OB attending

The Code Bags

It is our responsibility to ensure that the code bags at Hillcrest and at the VA are fully stocked and ready to use. As the airway experts of the hospital, we are first and foremost responsible for that aspect of patient care during a code, or during urgent/emergent airway management consults. The code bag contents reflect this, with a large part of the equipment being devoted towards airway management. However, the anesthesia provider is often the most experienced or senior physician present, not to mention the most level-headed. Thus, while ostensibly we are only responsible for airway management during a code, often times we find ourselves assisting with other aspects of the resuscitation, or even running the code itself. The bag is therefore stocked with other useful pieces of equipment such as drugs and IV lines.

At Thornton, we are not responsible for either stocking code bags or for responding to codes. However, there are times when we might be called for an urgent intubation. Typically the floor attending at Thornton will be notified of this.

So, who specifically stocks the code bags? Whoever is currently holding the code pager is responsible for the contents of the bags. It must be noted that the code pager will change hands multiple times a day; see above. So, an attending who is holding the pager for 10 minutes while all his residents are in ORs cannot really be expected to check and stock the bags. So, at handoff of the pager, the recipient must ask, “Are the bags good to go?” If you are handing off the pager, it is your responsibility to verify that they are, both from a patient care and a personal-pride standpoint.

The fact that they truly are stocked and ready depends on mutual trust between donor and recipient of the pager, and if you have the pager for any amount of time (MOR call, SICU resident, night float, OB call, not in an OR for hours at a time) it is upon you to make sure the bags are “good to go” by going through both of them and stocking as necessary.

Any time you use the code bag, you must immediately and completely restock all used items. This ensures the legitimacy of the mutual trust that happens with the pager handoff.

At the VA, the person responsible for stocking the code bags is the call resident from 1000-0700. Typically the resident on regional anesthesia or a CRNA will be holding the pager between 0700 and
At Hillcrest, the bags are stored in the anesthesia workroom, first shelf as you enter, bottom row. They are large and “danger orange” in color.

Specific things to be stocked in the bags will be described to you in more detail later, but include:

- **Drugs:** these come in a premade pharmacy kit that is located on right side zippered compartment. It is sealed until use. After it is opened, it is returned to the Pyxis and a new sealed bag removed from the Pyxis. It contains etomidate 2mg/ml, propofol 10mg/ml, rocuronium 10mg/ml, succinylcholine 20mg/ml, phenylephrine 100mcg/ml, ephedrine 5mg/ml, vasopressin, and “Abbojects” of code doses of epinephrine, lidocaine, calcium, atropine, and sodium bicarbonate.
- **Airway:** this is the large “roll-up” package in the main code bag compartment. It must include a short and long laryngoscope handle, and a variety of blades (MIL2, MIL3, MAC3, MAC4). Styletted and with 10ml syringe attached are a 6, 7, and 8 regular ETT, and a 6, 7, and 8 silver-impregnated ETT. Tongue blades, nasal airways, oral airways, lube, Magill forceps, tape, twill ties, Bougie, and Yankauer suction are included.
- **Airway rescue devices:** a size 3.5 and 4.5 Cookgas LMA, a Combitube, and Fastrach LMA, and a variety of nasal RAE tubes are located in the compartment that forms the “lid” of the code bag.
- **A Cook Airway Exchange Catheter** is located in the main compartment, below the airway pouch.
- **Mapleson circuit** with at least one each of size 4, 5, and 6 masks is located on the left side zippered compartment.
- **Airway confirmation devices:** in the main compartment must be a colorimetric an end-tidal CO$_2$ detector (“EZ-Cap”) and an esophageal detector bulb device.
- **Miscellaneous:** IV equipment, arterial line equipment, gloves, and billing/documentation paperwork are located on the front and back compartments of the bag.

**Remember to restock the bags with whatever you have used.** This this will save a lot of headache – and possibly a life – later.

The airway rescue devices for “cannot ventilate, cannot intubate” situations are numerous, and this is not intended to be definitive instruction on how to use them. Most of us consider the LMA the essential and the first line option in the situation. You may ask, “Why do we need several types of LMAs in the code bag?” Any properly placed LMA will allow you to ventilate and oxygenate in nearly all CV/CI situations, but there are differences between the various types. Briefly,

- **The standard LMA (“Classic”)** is usually the one we are most familiar with, and thus will probably be easiest to place in a crisis. However, it is difficult to intubate the trachea through a standard LMA (requiring either a nasal RAE or a bronchoscope and specific training) and it is nearly impossible to remove a standard LMA over an ETT if one is placed.
- **The Fastrach LMA** is intended to facilitate blind tracheal intubation in a crisis, and readily accommodates its specific ETT and can be used with a bronchoscope.
- **The Cookgas LMA** is designed to facilitate bronchoscope-assisted tracheal intubation.

You will learn how to use these devices in time. Do not fear.

A word on the tracheal intubation confirmation devices: we use the EZ-Cap CO$_2$ detector and the esophageal detector bulb device.
• The EZ-Cap is placed on the endotracheal tube adaptor and detects expired CO$_2$. A successful intubation will be confirmed with a color change on the cap which roughly correlates with the amount of expired CO$_2$. Ventilating a tube in the esophagus will not result in sustained expired CO$_2$ and EZ-Cap color change. However, in a situation where the patient has no cardiac output (e.g., cardiac arrest with ineffective chest compressions) the EZ-Cap will not change color. In these circumstances the esophageal detector device is warranted.

• The esophageal detector device is also attached to the adaptor of the ETT after intubation. When deflated, attached to the ETT, and allowed to reinflate, it creates suction on the tube and whatever lumen the tube is sitting in. If in the esophagus, this suction will cause the pliable folds of tissue to collapse on the tube and the bulb will not reinflate. If in the trachea, the bulb will rapidly reinflate since the trachea walls are rigid and will not collapse.

Both these devices are needed to cover all situations where one needs objective confirmation of tube placement. The gold standard is, arguably, visualizing seeing the ETT passing between the vocal cords, but this can be nearly impossible during chest compressions. Fiberoptic bronchoscopy is an excellent option, but requires both time and equipment, which may not be available. Breath sounds and chest rise are notoriously inaccurate measures. Although the trend of priorities in ACLS is toward Circulation (i.e., early chest compressions) and away from tracheal intubation, Airway and Breathing are still high priorities, and a missed esophageal intubation is quite frankly unacceptable.

**Responding to the Code Pager**

Until about 6 months into your CA-1 year, you must call your attending if you’re going to a code or other emergency. After that, it’s on a case-by-case basis, depending on the clinical situation, the attending, and your own comfort level. There is never any shame in asking for help from a senior resident or letting the attending know what’s going on. In general, as a CA-1, you should never go to a code or intubation without someone else from our department. It is a good thing to have multiple people from our department present and cooperating, both for patient safety and so that multiple residents can learn from whatever situation comes along. We are a team and we help each other out; an extra set of helping hands is always welcome. All senior residents can recall multiple times when they were glad an attending was with them for what at first seemed like a routine intubation.

Most calls for airway management are semi-elective or merely urgent. If you are called for such, talk to the primary doctor about the clinical situation and get a current set of vitals including height and weight, so you can plan to bring the most appropriate equipment (Glidescope, bronchoscope, extra help, etc.). Emergency calls are usually clear, e.g., “Code blue, 10 East, room 1021,” but might simply say, “anesthesia stat to 10E” or even worse, just a number and a frantic nurse on the other end. If it seems urgent, you should just go see what’s going on for yourself and ask questions later.

This syllabus is not meant to teach ACLS or the myriad skills needed in a code/emergency. That being said, here are some tips:

1. Try to size up the situation when you first walk in. This can be difficult, especially when there are 20 people in the room, most of which whom are running around like chickens with their heads cut off. Is it a true code? Is the patient responsive? Is the patient ventilating? Is anyone clearly in charge? Are any monitors on the patient?

2. If you need to get to the patient, be assertive. There’s likely to be a whole bunch of people of dubious utility around the patient. Calmly but forcefully let people know you are present and your
intentions, e.g., “I’m the anesthesia resident, I need to get to the head of the bed, please move.”
3. If a code situation, establish early who is running it, and identify yourself to that person. All commands should come from that person and ideally there should be little other talking. Sometimes that person will be you, especially when no one else “volunteers” to run the code.
4. Be calm. It’s amazing how often a composed, competent demeanor sets the whole situation at ease.
5. Prepare the appropriate equipment according to your mnemonic, “MOM SAID” or “MSDAMIT,” etc.
6. Prepare as much as you can before you do something. For example, if you go to an semi-urgent intubation and have some time, get everything you’ll need out and organized beforehand so you’re not struggling to find it later. This might mean an LMA, the EZ-Cap, drugs, suction, etc., all within easy reach or with someone you can trust prepared to hand them to you.
7. ABC! So much of what we do comes back to that. If you constantly think “ABCs” during most emergencies you will be fine. Since A is first, if the patient needs an airway all other people’s activities become secondary and yours become of the utmost importance.
8. If someone is truly in cardiac arrest, you don’t need to give any drugs before laryngoscopy and intubation.
9. After you’ve secured the airway, see how else you can make yourself useful. This might involve assisting with the code, starting an arterial line, or obtaining (better) IV access. Many times the code leader or primary team may ask for your help, or may be resistant to additional monitors or access. Often, if you think the patient will need something, you’re right, even if the primary team feels differently. So make your case forcefully but politely; this might avoid a situation where you are sent away only to be called back 5 minutes later to assist with an “urgent arterial line.”
10. Before you leave a code, you must ask the code leader if you’re needed anymore.

The Emergency OR Setups: OR7 and OR11

This information applies only to Hillcrest. At the VA and Thornton, we do not have preset rooms for emergencies due to the low volume of trauma and emergency cases. The responsibility of ensuring these rooms is shared by no more than two people: the person holding the code pager (typically, the SICU resident) and the MOR call resident. Whoever has the time and opportunity to check or set up these rooms must do so; sometimes this is not the MOR call resident or the SICU resident, but rather the night float, OB call, or MOR2 resident. Communicate with each other to make sure that the rooms get set up.

OR7, Emergency Craniotomy Room:

One OR is traditionally reserved for emergency craniotomies after “main” OR hours. This is almost always OR7, but sometimes cases from the day run late in OR7, necessitating another room to be designated as the “crani room.” Check with the OR front desk staff when in doubt. The room is set up with the goal of emergently inducing anesthesia so that the surgeons can open the cranium and dura. Standard room setup applies with the following additions:

- The bed is turned 180° so that the head is already away from the anesthesia machine. This is the natural position of most craniotomies and will allow a truly emergent crani to be positioned without having to turn the table. The yellow “donut” foam pillow for head position should be at the far end, where the head will be.
- Because the head is away from you, a Mayo stand with all relevant airway equipment must be in the OR. This tray lives in OR7. It must include EVERYTHING you might need on the stand for intubation and ventilation: masks, oral/nasal airways, blades and handles, various ETTs, tape, etc.
• Because the head is away from you, you must have a long circuit extension available in the room or attached to the circuit so the circuit can physically reach the patient’s head. It is very embarrassing to intubate a patient and then not be able to connect your circuit because it’s too short.

• Have mannitol and several 100ml bottles of propofol in the room.

• Arterial and venous pressure transducers, pre-zeroed, must always be in the room.

• Equipment to rapidly start an arterial line and central line must always be in the room.

• Have at least one IV ready, prespiked and attached to a fluid warmer, for immediate hookup to whatever IV access you have or place.

**OR11, Emergency Trauma (“OR Resus”) Room**

This room should be set up at all times for an emergency surgical/trauma resuscitation. This room is where life-threatening penetrating and blunt trauma cases will be brought emergently by EMS. The only time it will not be set up is when it is either currently in use or has just been used and is being cleaned. The standard OR setup again applies, with the following additions:

• Airway equipment, e.g. blades, tubes, oral/nasal airways, and circuit, prepared as it would be in the “crani” room or the code bag, must always be ready.

• Two IVs pre-spiked and attached to fluid warmers.

• Emergency resuscitation drugs are drawn up and available. The cart has a standard drug tray plus code drugs like epinephrine, atropine, and bicarbonate Abbojects.

• Arterial and venous pressure transducers, pre-zeroed, must always be in the room.

• Equipment to rapidly start an arterial line and central line must always be in the room. This means the actual kits, as well as sterile gowns and gloves, must always be ready.

**Chapter 1G. The Pre-Operative Evaluation: General Comments**

A safe and high-quality anesthetic begins with proper pre-operative evaluation of the patient. Nothing can better illustrate the interaction between a patient’s comorbidities and their anesthetic implications than a thorough pre-operative evaluation. There will be times during your anesthetic career where conditions preclude a full pre-operative workup (e.g., emergency situations, non-verbal patient) but in general every case demands a full pre-op evaluation.

The difference between a good pre-op and a poor one is like night and day. A well done pre-op conveys precisely the information you want and need to deliver a high-quality anesthetic. A poor one can often leave you scratching your head or hunting for more information. At worst, a poor pre-op can compromise patient safety. All of us have experienced the phenomenon of the poorly-done pre-op and in fact most anesthesiologists can guess the level of a person’s experience by the quality of their pre-ops. The following questions are common when first starting anesthesiology residency:

**When will I be doing pre-ops?**

The following are the typical scenarios in which residents will do a pre-op.

**1. The Pre-op Clinic at Hillcrest, Thornton or the VA:**

The dedicated pre-op clinic experience at Thornton is described above in the “call duties” section. Briefly, scheduled patients come in throughout the day, usually on a day anywhere from 1-14 days prior
to their elective surgery, when they are also seeing their surgeon and having pre-op testing done.

At the VA, the call resident is infrequently asked to seeing patients in the VA pre-op clinic (referred to as ASU). There are multiple full-time CRNAs assigned to ASU, who typically shoulder the pre-op burden.

2. Inpatient Pre-ops

At Hillcrest, a substantial percentage of all surgical cases are for inpatients with acute or subacute issues, including ICU patients and ortho-trauma patients. These patients clearly cannot go to our pre-op clinic and thus must be seen on their ward. Ultimately, it is the responsibility of the resident who will be doing the case to pre-op his or her own patient. That said, in the spirit of mutual assistance and camaraderie, you may find that your resident colleagues will have your inpatient pre-ops completed, especially if you are at a different site the day prior, which is very common. Generally the SICU resident, in conjunction with the MOR call and OB call residents, will have the opportunity to pre-op these patients, and in the case where a SICU resident pre-ops a SICU patient, they may know the patient quite well. This is worked out on a case-by-case basis, but in general we all try to help each other out and get each other’s inpatient pre-ops done. CA-1s should be doing their own inpatient pre-ops for the first six months, independent of the prior day’s assignment or schedule, to help them gain more experience.

Occasionally, a CRNA will be assigned to an OR with inpatient for the next day. When this happens, typically the resident helping with the other residents’ pre-ops will be responsible for these as well. Life goes on.

The call resident at the VA does all the inpatient pre-ops for that location. The list of patients who need pre-ops will be provided when you arrive on call at 1000. Usually, several cases are added on during the day as well. Thus, a major difference between Hillcrest and the VA is that the call resident is technically responsible for any inpatient pre-ops you may need at the VA for the next day.

However, at Hillcrest you could finish up in the OR only to find three inpatients waiting for you to be seen. Again, every resident has been in this situation, it is not common, and the best tack is to complete the work efficiently and move on.

3. Pre-ops “On the Fly”

All that happens in the OR is not scheduled or predictable. For truly emergent, or very urgent, cases, the patient is not likely to have a pre-op already done, e.g., a patient with mesenteric ischemia in septic shock who requires an emergent exploratory laparotomy. Time is of the essence, and the most you may be able to obtain is a quick history asking about major cardiopulmonary issues, medications, allergies, an airway examination, and pertinent lab results. Similar situations often arise in OB anesthesia, in which there is a rapid turnover of patients, some of whom need our services immediately on arriving to the hospital. Always be as thorough as the situation allows, which is very different, depending on if you have 2 minutes or 20 minutes.

Some elective cases are for patients who were not scheduled or did not show up to our pre-op clinic. You may find out about this patient just a few minutes before you take them back to the OR. Your attending will typically assist with this endeavor (particularly if you two have a previous case running). No one will expect you to have been able to do a thorough pre-op beforehand, but you have the opportunity to do one at that point, immediately pre-op.
What does a basic pre-op entail?

Philosophically, the pre-op serves several functions. First, it provides a brief history and physical, describing the patient, their current health status, and their chronic comorbidities, especially those that pertain to anesthesia. Secondly, it provides a framework and plan for the actual delivery of anesthesia. Clearly, as one’s experience with pre-ops and anesthesia grows, it becomes significantly easier to identify which information is pertinent and which is superfluous. The goal is to provide a brief, concise, and relevant summary of the patient and their medical problems. A pre-op which is agonizingly thorough but is twenty pages long is of no use to anyone.

It is impossible to list here every condition and situation which may have anesthetic relevance. Indeed, mastery of such implies mastery of both medicine in general and anesthesia in particular, an unlikely combination for any resident. That being said, the following will help guide you through each section of the pre-op, pinpoint areas to focus on, and give the rationale behind why an anesthesiologist would want this information. Refer to the blank pre-op templates (one for Hillcrest and Thornton, another similar one for the VA). The templates are intended to be easy to read and follow and to minimize writing – hence, the checkboxes. This also serves as a “primer” so that certain key questions are unlikely to be missed.

Who can I turn to for questions about pre-ops, or whether a patient needs further evaluation?

Any faculty or senior resident will be happy to assist you. Often, your own peers are your best resource. When in doubt, ask. There’s no such thing as a stupid question.

Of note, all pre-ops done in any of the pre-op clinics need to be signed off on by an attending. This is an assigned, daily duty for the attendings and will usually be the late attending at Hillcrest and the floor attending at Thornton and the VA.

If I feel a patient needs further workup, another test, etc., who orders it?

This is an important question. In general, the primary service should be the ones ordering any additional workup. This may be the surgery service or the surgeon’s office (for elective patients) or whichever primary team an inpatient is on. Since the surgical team follows the patient pre- and post-operatively, they are in the best position to both order a test and initiate any necessary follow-up. Additionally, there is a medico-legal perspective which we are lucky in anesthesia to be largely shielded from – namely, if you order a test and find an abnormal result, you are obligated to do something about it. For example, if you order a chest X-ray on a patient and a mass is found, it becomes your responsibility as the requesting physician to make sure this doesn’t fall through the cracks. If no one else follows up with the patient, and an issue arises in the future from said mass, you can be held responsible. Thus, in general, you don’t want to be ordering tests yourself.

The best way to go about obtaining further workup is to contact the surgical team, office, or attending, and tell them what you feel is necessary. They can then go about obtaining this information. Sometimes the information you want is broad, e.g., “please evaluate this patient for coronary disease.” There are several ways to test this and the political thing to do is to leave this at the discretion of the service performing the test. In this example the surgical service would consult cardiology, who can then choose one of several ways to evaluate the patient. Other times the information you will want will be quite specific, and then you can ask for it, e.g., “please obtain flexion/extension X-rays of the neck to evaluate for atlanto-occipital instability.” This fulfills our role as perioperative consultants and frees us from
having to do much of the leg work ourselves.

**Chapter 1H. The Pre-operative Evaluation: Section-By-Section**

1. **Age, Weight, Vital Signs, PO Status, Proposed surgery, and Diagnosis**

This section is mostly self-explanatory. Age and sex can have profound implications on anesthetic technique and sequelae. Less obviously, the weight and height of the patient can dictate the airway management plan, drug dosing, the size of an endotracheal tube, and sometimes the whole anesthetic.

Elective patients must be NPO for solid foods for at least 8 hours prior to undergoing anesthesia. “Solid food” includes any food with fat or protein, to differentiate it from a “light meal” as below. This applies to all anesthetics, including non-general anesthetics, such as neuraxial techniques and MAC. There is always the potential to convert to a general anesthetic as “Plan B,” e.g., failed spinal, or patient who becomes uncooperative under MAC. Small amounts of clear liquids may be taken as recently as 2 hours prior to induction of anesthesia. A “light meal,” i.e., one that doesn’t contain fat or protein, or milk alone, can be taken as recently as 6 hours prior to anesthesia. The recommendations for children are different: 2 hours for clear liquids, 4 hours for breast milk, and 6 hours for formula or solids.

The proposed procedure and diagnosis will determine the anesthetic technique. As obvious as it sounds, it is not uncommon to find yourself preparing to perform one type of anesthetic, only to have to switch at the last minute because the procedure you were ready for (on the pre-op) is different from the one the surgeon actually intends to do. Accuracy is important.

2. **The Cardiovascular System**

There are several checkboxes here pertaining to HTN, CAD, CHF, arrhythmias, etc. Certain items, if positive, deserve more clarification. For example, if the patient has a “hx of MI” it should be stated that the patient has CAD, when the MI occurred, what interventions were done, current management, etc. Similarly, if the patient has heart failure, is it compensated? What is their EF? What is their diastolic function? What are the patient’s symptoms? In general, use your best judgment about when to provide more information.

The question of exercise tolerance is perhaps the most useful and important of the entire pre-op. By assessing the patient’s ability to tolerate physical activity or exercise, and using the threshold of 4 METs, we can risk-stratify them and determine if further cardiac workup is necessary. Four METs is roughly the activity of climbing a single flight of stairs, walking a few blocks at 4mph, doing light householdlike dusting or washing dishes, or running a short distance. One MET is, very simply, your resting metabolic rate, i.e., your metabolic rate while sitting quietly.

The “ACC/AHA 2007 Guidelines on Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery” are the reference for decision-making in this area, and we apply these guidelines on a daily basis. The short version is that if the patient can perform 4 METs of activity without symptoms of chest pain or shortness of breath, or if the case is an emergency, you should proceed with the appropriate anesthetic plan. If neither of those is true, then depending on the patient’s medical conditions and the type of surgery, further testing or therapy may be indicated. In short,

- If the surgery is emergent
  - Go to the OR, monitor as appropriate
• If the patient can perform 4 METs without symptoms
  ➢ Go to the OR
• If not emergent, and the patient has “active cardiac conditions” e.g. acute coronary syndrome, decompensated heart failure, uncontrolled arrhythmia, or untreated valvular disorder
  ➢ Delay going to the OR, evaluate and treat conditions as appropriate
• If not emergent, and the surgery is low-risk
  ➢ Go to the OR
• If not emergent, and the surgery is moderate- or high-risk and the patient cannot do ≥ 4 METs
  ➢ Consider additional testing or β-blockade depending on the type of surgery and the patient’s “Clinical Risk Factors” (see below)

Generally speaking, the guidelines are crystal-clear for the vast majority of planned surgeries. However, the most common situation in which the AHA/ACC Guidelines need to be applied and decisions made about further testing or perioperative β-blockade is the 5th bullet point, where patients with 0, 1, 2, or 3 “Clinical Risk Factors” along with either moderate- or high-risk surgery may or may not need additional testing or perioperative β-blockade. The five Clinical Risk Factors are CAD, heart failure, DM, CKD, or cerebrovascular disease. This decision-making tree is easiest to understand visually, and so the algorithm follows below.

![Algorithm Diagram](image-url)

**Figure 1.** Cardiac evaluation and care algorithm for noncardiac surgery based on active clinical conditions, known cardiovascular disease, or cardiac risk factors for patients 50 years of age or greater. *See Table 2 for active clinical conditions. †See Class III recommendations in Section 5.2.3. Noninvasive Stress Testing. ‡See Table 3 for estimated MET level equivalent. §Noninvasive testing may be considered before surgery in specific patients with risk factors if it will change management. Clinical risk factors include ischemic heart disease, compensated or prior heart failure, diabetes mellitus, renal insufficiency, and cerebrovascular disease. ¶Consider perioperative beta blockade (see Table 2) for populations in which this has been shown to reduce cardiac morbidity/mortality. ACC/AHA indicates American College of Cardiology/American Heart Association; HR, heart rate; LOE, level of evidence; and MET, metabolic equivalent.
Note that:

- **Low-risk surgery**: endoscopy, skin surgery, cataract surgery, breast surgery, ambulatory surgery
- **Moderate-risk surgery**: anything intraabdominal or intrathoracic, carotid endarterectomy, head & neck surgery, orthopedic surgery, prostate surgery
- **High-risk surgery**: aortic vascular surgery or peripheral vascular surgery

The bottom line is that when you are doing a pre-op, there should be certain “red flags” that you are listening for. Any of the “active cardiac conditions” or multiple “clinical risk factors” along with a planned moderate- or high-risk surgery should certainly grab your attention. In practice, surgeons, who generally do not want to have their cases cancelled on the day of surgery, are not shy of ordering stress tests (whether nuclear or echocardiography-based) or consulting cardiology. So, in many instances, the appropriate tests have already been done, and/or the patient may have been started on β-blockers. Nonetheless, vigilance is the name of the game here, so take a detailed history and review the medical record carefully.

**Coronary Stents**

Patients with prior coronary stents demand special attention. Current guidelines are designed to minimize the risk of perioperative stent thrombosis and perioperative MI, which carries a mortality rate of around 50%. A few items you **must** know about your patient’s coronary stents:

- When they were placed
- What type of stent (drug-eluting vs. bare-metal)
- Which vessels they are in
- The type of antiplatelet therapy and the intended duration of dual antiplatelet therapy

Timing of placement influences the timing of the dual antiplatelet therapy regimen, which is typically aspirin and clopidogrel. Generally speaking, a patient should not be taken off clopidogrel for at least 12 months following DES and for at least 6 weeks following BMS. And since almost no surgeon will perform surgery on a patient taking clopidogrel, this effectively means that elective surgery should not happen for at least 12 months following DES and 6 weeks following BMS.

Where the stents are located influences your choice and placement of ECG leads. A patient with a BMS placed in the LAD 8 weeks ago, off clopidogrel for an elective surgery, will certainly benefit from monitoring of lead V₄ or V₅.

Ultimately, decisions about timing of elective surgery following coronary stenting and management of antiplatelet agents are made mutually by the patient’s cardiologist, surgeon, and anesthesiologist. Be sure to contact an attending with any questions regarding coronary stents and medication management.

**Pacemakers and Automated Implantable Cardioverter-Defibrillators (AICD)**

These devices represent another unique challenge to the anesthesiologist. Current guidelines are designed to allow safe management during the entire perioperative period, as well as assessment of the device itself. The “old days” of simply placing a magnet on the device during surgery have gone by the wayside. In short, these devices are very time-consuming and managing them can be quite complex. The guidelines for this topic are available from the ACC/AHA website. A few items you **must** know about
your patient’s implanted device:

- Where it physically is (right/left) and how many leads it has
- Model and manufacturer of the device (e.g., Medtronic, Boston Scientific)
- If a pacer,
  - Indication for its placement (heart block vs. sick sinus/tachy-brady)
  - Pacing settings (DDD? VVI? AAI?)
  - What happens when a magnet is applied? I.e., what is the “magnet mode?”
  - Single-chamber, dual-chamber, or biventricular?
- If an AICD,
  - Indication for its placement (actual VT/VF vs. low-EF heart failure)
  - What the magnet does when applied (it usually disables antitachycardia therapy)
  - Backup pacing function
- Phone number for the device representative

Depending on the location on the patient’s body of the surgery, the need for electrocautery, the patient’s position, and device characteristics, the device may need no special management perioperatively, or it may require the representative to come to the hospital to reprogram the device pre-op and then re-reprogram and interrogate the device post-op. Add in the challenge that most, if not all, patients have little to none of the above information. So, obtain everything you can, and write it down on the pre-op form. Calling the device representative ahead of time and/or scheduling a day-of-surgery visit is the most important. All the major manufacturers have national phone numbers and local representatives on call 24/7/365. Most patients at least have this phone number on them.

You will learn much more about these devices throughout your residency and especially during the cardiac rotations. The above simply represents the minimum information which should be gathered during a pre-operative visit.

3. **Respiratory System**

COPD (emphysema and chronic bronchitis) and asthma are conditions that need further description, primarily in delineating the severity of the condition and any recent changes in symptoms. For both COPD and asthma, you must know the patient’s current medication regimen, and any previous hospitalizations or intubations for their condition. A patient who has exercise-induced asthma and uses albuterol once a week is very different from a COPDer who wears 3L oxygen 24 hours a day and uses daily inhaled steroids and long-acting bronchodilators. What are their usual symptoms, and have these changed in the prior days or weeks? Many patients with COPD, or even just those suspected of having COPD, will have pre-operative pulmonary function tests done. Note the absolute values and the ratio of FEV₁ and FVC.

Previous significant instrumentation or changes to the airway bear further explanation. Examples of these might include history or presence of a tracheostomy, significant radiation to the head and neck, or prior surgeries in these same areas. Clearly an ongoing, active issue with the patient’s airway warrants further investigation and explanation.

Obstructive sleep apnea has profound implications in the perioperative period. The “Practice Guidelines for the Perioperative Management of Patients with Obstructive Sleep Apnea,” published by the ASA in 2006, is an excellent reference. Dr. Benumof has been an instrumental member of the taskforce that
created these guidelines. At the minimum, the presence or suspicion of OSA must be noted on the pre-op form. Changes in anesthetic technique, planned postoperative monitoring, discharge requirements, and even whether to proceed with surgery can occur in the presence of OSA. At UCSD, generally speaking, patients with known OSA who have undergone GA will be monitored in the hospital overnight with continuous pulse oximetry, to monitor for life-threatening hypoxemia. However, there are exceptions. Refer to the guidelines for specific recommendations.

4. Neurologic System

History of cerebrovascular disease is important to note, in that patients may have neurologic deficits or significant vascular disease as described above. Patients with seizure disorder should always maintain their medication regimen throughout the perioperative period. It is essential to carefully document preexisting neurologic dysfunction or deficits, because perioperative neurologic dysfunction makes up a substantial amount of claims brought against anesthesiologists, whether theoretically due to positioning-related neuropathies or manipulation of the cervical spine.

5. Hepatic and Renal Systems

Preexisting hepatic and renal disease can profoundly influence the choice of individual anesthetics and timing of surgery. Patients with advanced disease in either of these organ systems will have decreased metabolism and/or elimination of drugs, in addition the effects on other organ systems. If the patient is on peritoneal or hemo-dialysis, note the normal schedule of dialysis and when the patient’s pre-operative dialysis will be.

6. GI, Hematologic, Endocrine, and Musculoskeletal Systems

If the patient has GERD or symptoms of heartburn, be sure and clarify the situation. Are the symptoms more indicative of simple heartburn, or that of true GERD (worse when supine, acidic taste in the mouth or throat). Additionally, note if the symptoms are controlled on any medications or not.

Dysfunction of the hematologic system is important to note, especially preoperative anemia or coagulopathy and any known causes or treatment regimens.

Diseases of the endocrine system, particularly diabetes mellitus and thyroid disorders, can have profound implications in the perioperative period. At minimum, note the type of diabetes (1 vs. 2) and whether a type 2 diabetic is insulin-dependent. Hypo- or hyperthyroidism is typically controlled with medication to an asymptomatic state prior to elective surgery. Here, you can also note any comments about morbid obesity, or pheochromocytoma.

Preexisting musculoskeletal problems can also be noted here. Be sure to clarify between osteoarthritis and rheumatoid arthritis, as these have very different implications for anesthesia.

6. Cancer, Infectious Diseases, History of Postoperative Nausea and Vomiting (PONV), Smoking, Alcohol, and Other Drug Abuse

Indicate as appropriate. Pay special attention to cancers of the head and neck, cancers that have been irradiated, or exposure to bleomycin or adriamycin (hydroxydaunorubicin) as they may have profound implications for anesthetic management.
If a patient uses recreational drugs, note if they are an active user. Patients who have abused tobacco, ethanol, cocaine, or methamphetamine and have only recently stopped may still have altered physiology from substance abuse.

8. Family History of Problems with Anesthesia

This section is asking for a history of malignant hyperthermia or pseudocholinesterase deficiency. Often patients will know this themselves, and can also be asked about a history of “high temperatures” or “bad reactions” anesthesia. A positive answer warrants further investigation and planning; see the malignant hyperthermia section.

9. Previous Surgeries, Medications, Allergies

Indicate as appropriate. Certain medications must have their doses noted. An occasional vicodin 5/500 as needed is different than someone taking 8 tabs of percocet 10/325 a day. The same goes for antihypertensives – lisinopril 5mg daily vs. 40mg daily, for example. Some which will alter the anesthetic (e.g., narcotics, antiepileptics). Certain herbal medications bear mentioning. If applicable, try and describe the allergic reaction a patient has.

10. Physical Exam

Brief and focused should be the goal. Note the presence of any indwelling catheters or instrumentation. “General” inspection and auscultation of heart and lung sounds is the minimum here.

11. Airway Examination

Note the Mallampati class, hyomental disease, neck and head range of motion, quality and quantity of dentition, and the ability to prognath the jaw. There is room to provide further description if necessary: does the patient have a profound overbite? A thick neck? Scarred submandibular tissue due to prior radiation? If the patient has an endotracheal tube or tracheostomy, you must note the size and type of airway tube. Any available information about past intubation attempts and/or their success must be noted. The importance of a good airway exam cannot be overemphasized.

12. Labs and Other Studies

Note chemistry, hematology, coagulation, and blood gas studies as applicable. Some patients will have had substantial workup prior to their pre-op visit. Men over the age of 40 and women over 50 need a baseline EKG. Note the results of a chest X-ray if available and pertinent.

13. Assessment and Plan

Here is where you indicate a one-line summary of the patient, a pertinent problem list or items in the medical history, and a description of the anesthetic plan. A quick example might be:

42 y/o F with cholelithiasis s/f lap chole. PMH: morbid obesity, DM2, HTN. Plan: NPO > 8h, routine monitors, GA with ETT.

In general, it is good to be broad with the anesthetic plan and it is not necessary to define specifics (e.g., “propofol for induction”). For one, plans change. Secondly, anesthesia providers change, and for
medico-legal reasons you don’t want to create potential problems by being too specific on the pre-op evaluation form. For example, if use of an arterial line for the case at hand would be a judgment call of the anesthesiologist on the day of surgery, you might indicate “Routine monitors ± arterial line.”

Be sure to the ASA Physical Status Classification (“ASA class”). The ASA class attempts to assign a numeric value to the overall health status of the patient and has been shown to correlate with perioperative outcomes. Billing and reimbursement have historically been adjusted for ASA status, in recognition of increased complexity of the patient, but this is always changing. The following definitions are the strict definitions taken from the ASA, with our own examples in bulleted below:

- ASA 1: A normal healthy patient
- ASA 2: A patient with mild systemic disease
  - controlled HTN, controlled hypothyroidism
- ASA 3: A patient with severe systemic disease
  - CAD with stents, morbid obesity, advanced COPD
- ASA 4: A patient with severe systemic disease that is a constant threat to life
  - end-stage heart failure with AICD; an ICU patient intubated with ARDS
- ASA 5: A moribund patient who is not expected to survive without the operation
  - A patient with mesenteric ischemia in septic shock; a patient with an expanding base-of-tongue mass that could occlude the airway
- ASA 6: A brain-dead donor whose organs are being removed for donation purposes

Any of the above can have an “E” added to them, e.g. ASA 2E, to identify surgeries that are emergent. For example, if a patient has just eaten a meal and the surgeon says “We cannot wait 8 hours to do the surgery,” you would do well to indicate an “E” status in this case.

The VA Pre-op

The pre-op form at the VA is essentially the same as the one used at Hillcrest and Thornton. Notably, the VA administration insists that the question of TB “yes or no” must be answered, so check the appropriate box.

The pre-op must also be reviewed and signed off by an attending, without exception. For practical purposes, this means all pre-ops must be signed before they are scanned, including pre-ops done on inpatients after the attendings have left the building.

Lastly, the pre-ops at the VA are scanned into the electronic system when completed. You will be shown how to do this when you rotate through the VA. So, if you see an inpatient after all the attendings have left for the day, don’t scan it in until after an attending has read and signed off on it.

Chapter 1: Presenting Pre-Ops to Attendings

One ritual which you will grow intimately familiar with as a resident is calling attendings, typically in the evening between 1700-2000, to discuss the next day’s cases. This is done around the country at virtually every anesthesiology residency, so take heart in the fact that you are not alone. The case presentations are an opportunity for the resident to formulate a plan and iron out any potential issues before they arise, as well as to learn about the case in general. They are also an opportunity for the attending to know what they will be doing the next day, to teach, and to make sure that they are on the same page
as the resident.

Usually the resident photocopies the pre-op and takes it home to call the attending in the evening. However, depending on your work schedule, you may just call with it from the hospital, physically talk to the attending if he or she is in-house, have the pre-op photo-texted or photo-emailed to you by an on-call or otherwise friendly resident if the pre-op is at a different hospital than you are, or, as a last resort, examine the patient’s medical record and clinic notes on EPIC from home.

The number of attendings’ styles is almost as varied as the attendings themselves. Some attendings expect a thorough understanding of the physiology and pathophysiology of whichever medical condition(s) your patient has, or an intricately detailed anesthetic plan. Others are more laid back and might expect a 30 second presentation, to which they might respond “OK, see you tomorrow.”

Experience will help you determine who falls into which camp, but as a junior resident, you should always be prepared for the former (intense) rather than the latter (laid-back), both in the interest of your education and in the interest of not embarrassing yourself. Furthermore, the attendings as a whole tend to instruct or “grill” the junior residents more than the seniors during the presentations. In many ways this is only natural; it is natural to assume that the junior resident knows less, has less (or no) experience with the case at hand, and is in more need of teaching. So, treat this as the learning opportunity that it is.

Presenting a case is an art form. Done poorly, they can expose gaps in knowledge, waste time and create frustration for both resident and attending. Done well, they can be a great learning opportunity in a very brief space of time. With experience the ease and quality of presenting a case improves dramatically.

Thus, there is a good general format for presenting to attendings. Again, the goal is brevity and clarity. Certain additional pieces of information may be useful from time to time, but in general most attendings do not want to hear a 45 minute description of the patient. For example, it is usually not necessary to go into an exhaustive list of medications and doses during your case presentation. Below are a few examples of case presentations, varying with the complexity of the hypothetical patient.

“Hello Dr. X, this is resident Y. I am calling to discuss tomorrow’s cases with you. Our first case is…”

Case 1: “A 45 year old man having a left inguinal hernia repair. He is otherwise healthy and the airway exam is normal. My plan is general anesthesia versus neuraxial versus MAC/local and standard monitors, depending on the size of the hernia and the patient’s preference.”

Case 2: “A 50 year old woman having a laparoscopic cholecystectomy. She has a significant history of asthma, for which she takes albuterol and steroid inhalers daily. She has never been hospitalized or intubated for her asthma. Otherwise she has no medical problems. Airway exam and pre-op labs are normal. Recent PFTs show a mild obstructive defect consistent with her asthma. My plan is general anesthesia with an ET tube, standard monitors, and to have her use her inhalers immediately pre-op.”

Case 3: “A 66 year old man having a right carotid endarterectomy. He has 90% stenosis of his right carotid and recently had a stroke that left him with residual left sided weakness. He also has 50-69% stenosis of his left carotid. His past medical history is significant for HTN, DM2 and peripheral vascular disease. He does not exercise but a recent AMIBI showed no evidence of CAD and good ventricular function. Airway examination and pre-op labs are all normal. My plan is general anesthesia with an ET tube, standard monitors with an arterial line, and EEG. I will be maintaining with desflurane and nitrous oxide along with remifentanil infusion, with the goal being a quick wakeup so the surgeons can perform
a neuro exam. I'll keep the patient’s blood pressure at normal-to-slightly-elevated to maintain cerebral perfusion, especially during carotid clamping. Hopefully the EEG will allow us to monitor for possible ischemic episodes. Lastly, I will administer a longer acting narcotic like fentanyl at the end of the case to bridge the patient from remifentanil.”
Chapter 2. Anesthesia Equipment and Pharmacology

Chapter 2A. Anesthesia Equipment

I. Medical Gases
   A. Oxygen
      • Stored as a compressed gas at room temperature or refrigerated as a liquid.
      • Oxygen stored in the hospital central supply is at high pressure (2000psi); this cylinder pressure is reduced by valves to line pressure (~55psig).
      • A standard E-cylinder of oxygen is green and contains 650L of gas when full at a pressure of 2200psig.
      • Cylinder pressure falls in direct proportion to content; thus, a half-full E-cylinder contains 325L of oxygen, at a pressure of 1100psig.
      • Most anesthesia machines have 1 or 2 “backup” E-cylinders attached; standard practice at UCSD is also to have a separate E-cylinder in the room, with a Mapleson circuit for positive-pressure ventilation. All post-anesthesia patients are transported with oxygen to PACU.
   B. Nitrous Oxide
      • Can be liquefied at room temperature by storing under pressure.
      • A full E-cylinder contains 1590L of gas (liquefied) at a pressure of 745psig and is blue.
      • The cylinder pressure does not fall as nitrous oxide is consumed until the cylinder reaches below 400L. This is because nitrous oxide will vaporize at the same rate it is used and will thus exert a constant pressure (745psig). Only below 400L (¾ empty) will the cylinder pressure fall.
      • Thus, the only way to determine the volume of nitrous oxide in a cylinder is to weigh it.
   C. Air
      • Stored as a gas in yellow E-cylinders.
      • A standard E-cylinder of air shares the same characteristics as oxygen with respect to capacity and pressure.

The Pin Index Safety System is designed to prevent incorrect cylinder attachments to the anesthesia machine. Each type of cylinder has holes that lock with pins in the anesthesia machine. The spacing and position of pins/holes is unique for each type of gas, which generally prevents erroneous connections.

II. Delivery of Anesthetic Gases to the Patient (Breathing Systems)
   A. Insufflation
      • Gases are blown across a patient’s face; no direct contact is made between the circuit and the patient.
      • Potentially useful in children who may resist a face mask touching them.
      • Can also be used in situations where the patient’s head and neck are draped to avoid carbon dioxide buildup under the drape.
      • Limitations: cannot control ventilation, entrainment of room air, and unpredictable delivery of gases.
   B. Mapleson circuits (semiopen system)
      • Comprised of a breathing tube, a fresh gas inlet, an adjustable pressure limiting valve and a reservoir bag.
      • The positioning of these components determines the type of Mapleson circuit and how it performs.
• The efficiency of the circuit is determined by how much fresh gas flow is necessary to prevent rebreathing; there is usually some rebreathing in any Mapleson system.
• The APL valve should be completely open during spontaneous ventilation but must be partially closed to allow positive pressure during controlled ventilation.
• The longer the breathing tube, the larger the dead space in the system. Longer circuits increase the difference between volume delivered to the circuit and volume actually delivered to the patient during controlled ventilation. This is because the circuit has some inherent compliance and expands during positive pressure ventilation.
• Advantages: low resistance, low dead space, small and portable, little equipment and thus room for error.
• Disadvantages: constant loss of heat and humidity, need high flows to prevent rebreathing, difficult to scavenge waste gases.
• Best systems for spontaneous ventilation: A, D, C, B (“A Dog Can Bite”).
• Best systems for controlled ventilation: D, B, C, A (“Dog Bites Can Ache”).
  • See the diagram below for characteristics of the various Mapleson systems under both controlled and spontaneous ventilation.

C. Circle systems (semi-closed)
• The anesthesia machine is a semiclosed system; the addition of unidirectional valves and a carbon dioxide absorber convert a semiopen to a semiclosed system.
• These additions allow better conservation of heat and humidity (due to rebreathing of alveolar gas) and scavenging; however, there is more resistance during spontaneous ventilation, more dead space and more components, making the system both larger and more prone to malfunction.
• Unlike Mapleson circuits, the length of the circuit has essentially no impact on dead space. Longer circuits still increase the difference between delivered circuit volume and actual delivered volume to the patient; see above in the Mapleson section.
• CO₂ absorbers: exhaled carbon dioxide reacts with water to form carbonic acid. This acid is
neutralized by hydroxide salts (CO$_2$ absorbent), forming water, calcium carbonate, and heat.

- Soda lime is the most common absorbent; barium hydroxide lime is also seen but both forms come with an indicator dye that changes color with pH (as the lime becomes more exhausted, the dye will change color). Ethyl violet, the most common, is white when fresh and purple when exhausted. Be aware there are other types of dye with different colors. Further, exhausted lime that is allowed to rest can revert back to its original color. It is recommended the lime be changed when it is more than 50% exhausted. The anesthesia monitoring technicians typically will replace the lime daily, ensuring a fresh supply, or it can be replaced when the inspired CO$_2$ exceeds 5mmHg.

- Drier lime has a propensity to absorb and degrade volatile anesthetics. Absorbed volatile agent can delay induction and emergence. Degradation products include sevoflurane to compound A (seen only with fresh gas flows < 1L/min) and desflurane to carbon monoxide (barium lime only); see the section on volatile anesthetics. High flows running through an unused anesthesia machine increase the likelihood of dry lime. The so-called “Monday morning effect” comes from a hypothetical anesthesia machine that someone has inadvertently left with high flows going on a Friday afternoon. If the OR is unused the whole weekend, by Monday, the soda lime can be highly desiccated.

- The patient’s tidal volume should not exceed the volume between the granules, as this could result in rebreathing of carbon dioxide.

- Unidirectional valves: the inspiratory and expiratory valves should open only during the corresponding phase of the ventilatory cycle. Warped or cracked valves, or mis-seating of the valves can lead to incompetence and rebreathing of CO$_2$. Malfunction of either valve can result in rebreathing.

- Note the diagram of a circle system above. The fresh gas inlet should be between the absorber and the inspiratory valve. Were the FGI to be distal to the inspiratory valve, during exhalation fresh gas would be vented out and wasted. If the FGI came before the absorber, it would dilute with expired gas, and would be partially absorbed by the soda lime.

- Placing the pop-off valve immediately before the absorber conserves the absorber (exhaled gas vents before passing through the lime) and minimizes the venting of fresh gas.

- The reservoir bag should be in the expiratory limb. This reduces resistance to exhalation during spontaneous ventilation, and tends to vent exhaled gas through the popoff valve.
• With low flow rates, the difference between fresh gas concentrations and actual inspired gas concentrations can be markedly different. This is because the actual inspired gas is a mixture of fresh gas and the exhaled gas that has passed through the absorber. For example, take a fresh gas flow with a concentration of 100 units of gas X and exhaled gas with a concentration of 0 units. If the fresh gas flow and exhaled gas flow are both 1L/min, then the mixed (inspired) gas will contain \((100 + 0)/2 = 50\) units of gas X. However, if the FGF is 4L/min, then the concentration of gas X in the mixed gas will be \((100 + 100 + 100 + 100 + 0)/5 = 80\) units of gas. Thus, higher fresh gas flow rates will cause the inspired gas to more closely reflect the fresh gas itself, as well as speeding induction and emergence. High flows can also compensate for leaks in the system.

D. Closed systems
• Primarily of historical interest now.
• In a closed system, all gas except CO\(_2\) is rebreathed; no gas is evacuated through a popoff valve. The amount replaced by fresh gas flow is nearly equal to that taken up by the patient.
• By contrast, open systems have a fresh gas flow that exceeds minute ventilation (no rebreathing). Semiopen and semiclosed systems feature partial rebreathing, where the gas supplied exceeds that taken up by the patient, but is still less than total minute ventilation.
• Technique: the predicted oxygen consumption, minute ventilation and anesthetic uptake are calculated and then the exact flows are delivered to achieve this delivery. The goal is an unchanging circuit volume.
• Advantages: excellent conservation of heat and humidity, cheap, little or no waste gas to scavenge.
• Disadvantages: the amount and concentration of gas supplied must be precisely calculated; tedious, difficult and potentially dangerous; cannot rapidly change anesthetic concentration.

E. Nasal cannula, “Simple” Face Masks, Non-rebreathing Masks
• Nasal cannula: this is a low-flow system. Each additional liter of O\(_2\) increases F\(_{\text{IO}}\)\(_2\) by 4-5%, to a max of 6L/min or about 45% F\(_{\text{IO}}\)\(_2\). Significant entrainment of room air occurs, further diluting the oxygen in the nasopharynx. Peak flow rates during tidal breathing are around 40L/min, far exceeding that delivered by the nasal prongs. Thus, the actual F\(_{\text{IO}}\)\(_2\) which the lungs “see” is much lower than the maximum deliverable by NC.
• Face masks: deliver approximately 50% F\(_{\text{IO}}\)\(_2\) at flows of 6-10L/min.
• Non-rebreathing masks: have a reservoir bag, and can achieve > 80% F\(_{\text{IO}}\)\(_2\) at flows of 10-15L/min.
• None of these systems allows for positive pressure ventilation.

III. The Anesthesia Machine

The anesthesia machine is perhaps the most complex piece of equipment that we use on a daily basis. In fact, it is probably inaccurate to think of the machine as a single piece of equipment, as it is comprised of a multitude of components and serves simultaneously to deliver anesthetic gases to the patient and as multiple monitors.

One can literally finish an entire residency in anesthesia and still only have a basic understanding of the machine and its components. The complete description of the machine and its function is beyond the scope of this text. Rather, the following pages will serve to describe the essential features of the machines and detail the basic elements of a “machine check.” There are three machines currently in use at UCSD: the Datex-Ohmeda Aestiva 3000, which is the most common machine in use at Hillcrest, Thornton, and the VA; the newer Datex-Ohmeda Avance, in select ORs at Hillcrest and the Sulpizio
Cardiovascular Center; and the Draeger Apollo at Thornton. The newer Datex-Ohmeda Avance is a digital anesthesia machine and is the same machine that is used at Rady’s Children’s Hospital. Subtle differences between the machines will be described here.

A. Gas Supply
- All machines receive the supply of O₂, N₂O and air from two sources: the central hospital pipeline and the E-cylinders physically attached to the machine. Depending on the machine, there may be a fourth pipeline connection for helium/oxygen, or rarely, CO₂. Some machines do not have an air E-cylinder. The E-cylinders should be considered as backups to the primary source, the pipeline.
- The E-cylinders attach to the machine via the Pin Index Safety System (PISS) described above. Similarly, the anesthesia machine receives pipeline input via color-coded connecting hoses using the Diameter Index Safety System (DISS), whereby each pipeline connection has a specific and unique diameter of locking pins to prevent incorrect attachment.
- Before gas from the E-cylinders reach the flow valves, a pressure regulator reduces gas pressure to ~45psig for safety. The pressure is usually lower than pipeline pressure, so that if an E-cylinder is inadvertently left open, gas will still be preferentially drawn from the pipeline.

B. Flow valves and safety
- Before reaching the flow control valves, all gases except oxygen must first pass through a safety device. These devices will only allow the gases to be delivered if there is sufficient oxygen pressure, thereby reducing the chance of delivering a hypoxic mixture to the patient.
- Proportioning safety devices reduce the flow of other gases as the flow of oxygen falls. This is also to ensure against delivery of a hypoxic mixture. If you have both N₂O and O₂ flowing and suddenly reduce the flow of O₂, the flow of N₂O will automatically be reduced as well once the FIO₂ reaches the preset critically low value.
- A low oxygen-pressure alarm is also present which sounds whenever oxygen inlet pressure falls below a preset value.
- The flow valves are specifically designed and arranged to maximize safety and minimize the chance of delivering a hypoxic mixture in the event of a leak. For the non-digital machines, there are knobs for each gas, and the knob for the O₂ valve is always furthest to the right (downstream), is larger and protrudes more than the other knobs, and has ridges that can be felt even when not looking at the knob.
- The flow meters on the older machines are glass tubes in series (Thorpe tubes). The indicator ball or bobbin float rises as the flow of gas creates pressure underneath. Thorpe tubes get progressively wider near the top (“variable orifice”) so that as the float rises higher, more gas is allowed to escape around the sides of the float. The tubes are specifically calibrated for each gas.
- Typical board question: the rate of flow depends on the gas’s viscosity at low, laminar flow and its density at high, turbulent flow.
- The oxygen flow valve delivers a mandatory minimum flow of 150ml/min as long as the machine is turned on, ensuring some oxygen is present even if the anesthesiologist forgets to turn the oxygen on.

C. Vaporizers
- Each vaporizer has an “lockout” device that prevents more than one vaporizer being on at a time.
- Each vaporizer is calibrated to a specific agent, and is designed to deliver a consistent concentration regardless of temperature or flow changes. Each vaporizer must only be filled
with the intended anesthetic, and specific ports and caps for filling are designed to prevent incorrect attachment of the wrong agent to vaporizer.

- Basic mechanism: a certain portion of gas flow is diverted through a chamber containing liquid volatile anesthetic. This gas becomes saturated with anesthetic vapor, and the combined gas flow leaves the chamber where it dilutes with the rest of the unchanged (bypassed) gas flow. The vaporizers are therefore “variable bypass” vaporizers.

- The amount of “gas + vapor” diluted with the gas flow determines the concentration of anesthetic delivered to the patient.

- Desflurane vaporizer: desflurane has a very high vapor pressure, and a low potency. This creates two problems, both of which are addressed by the vaporizer. Due to the high level of vaporization, there is a tremendous cooling effect, because vaporization of the liquid agent requires heat which cools the vaporizer housing. This cooling must be compensated for by direct warming by the vaporizer, which is why the desflurane vaporizer is warm to the touch. Also, because of such high levels of vaporization, the amount of fresh gas flow needed to dilute the carrier gas would be excessive. Thus, small amounts of pure desflurane vapor are added to the fresh gas flow, which does NOT enter the vaporizer chamber itself. The desflurane vaporizer is therefore not a variable-bypass vaporizer. Lastly, the vaporizer cannot compensate for changes in elevation (ambient pressure). Elevation does not decrease the amount of anesthetic delivered, but it does decrease the partial pressure of the agent. Thus, at high altitude, a higher concentration must be delivered manually by the anesthesiologist.

D. High-flow oxygen flush valve

- Provides high flow (30-55L/min) of oxygen directly to the common gas outlet, bypassing the vaporizers and flowmeters.
- Is useful for rapidly refilling or flushing the circuit.
- Risk of barotrauma: the oxygen is supplied at line pressure; use the flush valve cautiously when attached to the patient. When the ventilator is off, ensure the popoff is completely open, or when the ventilator is on, ensure that the bellows are not on an inspiratory cycle.
- The button is recessed in the machine, making it more difficult to inadvertently trigger the flush valve.

E. Oxygen analyzer

- Mandatory; turns on when the machine is turned on.
- Should be placed in the inspiratory or expiratory limb of the circuit, but not the fresh gas line.

F. Pressure sensor

- Mandatory; placed somewhere in the circuit (varies by machine); generally reflects airway pressure.
- The closer to the Y-connector the sensor is, the more closely it reflects airway pressures.
- Changes in airway pressure may reflect obstructions, disconnections or changes in compliance and must be investigated.

G. Adjustable pressure-limiting (APL, “popoff”) valve

- Should be fully opened during spontaneous ventilation; however, closing it slightly can be used to add CPAP to the circuit.
- Designed to have an upper limit (~70cmH₂O) so that the valve can never be truly “closed,”limiting the risk of inadvertent barotrauma.
H. Humidity
- Delivered gases are room temperature and low in humidity, which can cause drying of the patient’s airways and loss of heat both from warming of the gas itself and from vaporizing water to increase humidity (heat of vaporization, the more important phenomenon with respect to heat loss).
- This heat loss represents ~10% of total intraoperative heat loss and is more significant with longer procedures (>1hr).
- Passive humidifiers can be added to the circuit; they function by trapping exhaled water vapor. They are cheap and simple to use but can increase circuit resistance and rarely can become plugged when excessively saturated.
- Active humidifiers add both water and heat to inhaled gases; they are quicker than passive humidifiers but also bulkier and more expensive. Downsides include the possibility of thermal inhalational injury, infection, increased chance of circuit disconnection, and increased dead space. These are typically used only in pediatrics, where airway heat losses can contribute significantly to overall heat flux.

I. Ventilator
- In Volume-Controlled Ventilation, the machine aims to deliver a set volume with each breath. High pressure limits will automatically “cut off” the breath if excessively high peak pressures are encountered. The machines also have the ability to deliver Pressure-Controlled Ventilation.
Here, a set pressure will be delivered for a certain length of time, depending on the set rate and the I:E ratio. The tidal volume delivered will vary with inspiratory time as above and with the patient’s pulmonary/thoracic mechanics.

- The newer digital Datex-Ohmeda Avance has a new mode of ventilation called “Pressure Control Ventilation-Volume Guarantee (PCV-VG).” This mode combines the advantage of pressure-controlled ventilation (the ability to limit peak airway pressures) with the ability to deliver a “guaranteed” tidal volume. A goal tidal volume is set and the machine will use only the minimal amount of pressure needed to deliver that tidal volume.

- I:E ratio: this determines the amount of time the vent will spend in each phase of ventilation. A typical ratio is 1:2. **Increasing** the I:E ratio (e.g. to 1:1) means there will be more time spent in inspiration and less in exhalation. Increasing the I:E ratio during volume control will typically lower the peak inspiratory pressures. Increasing the ratio during pressure control will result in a smaller volume delivered. **Decreasing** the I:E ratio (e.g. to 1:3) is a commonly-cited strategy to facilitate expiration in obstructive lung disorders such as COPD or asthma. Here, allowing more time for exhalation can overcome intrinsic expiratory gas-trapping.

- All machines have a switch to change from bag to mechanical ventilation; on the Drager, confirmation of your selection is confirmed by pressing the dial a second time.

- In ventilator mode, the popoff valve and reservoir bag are excluded from the circuit.

- Ventilator bellows: pneumatically driven (typically by oxygen, sometimes by air). During an inspiratory cycle the driving gas will fill the plastic chamber “outside” the bellows themselves, compressing the bellows and delivering a breath to the patient. If the chamber housing is cracked or incorrectly seated, pressure will be unable to build and the bellows will not drive. Similarly, if there is a leak in the bellows itself, high pressure gas normally used to drive the bellows can be transmitted to the patient.

- Phenomenon of ventilator/fresh gas flow coupling: during an inspiratory cycle, the ventilator will deliver both the preset tidal volume, and a certain percentage of the fresh gas flow itself. This additional amount is dependent on the number of breaths/min the vent is delivering, the time spent in the inspiratory phase, and the fresh gas flow itself. The equation for calculating this is:

\[
\text{Extra volume delivered} = (\text{FGF}) \times (\% \text{ of time in inspiration})/\text{(respiratory rate)}
\]

- Thus, if the FGF is 5L/min, the I:E ratio is 1:3 (25% of time in inspiration), and the machine is delivering 10 breaths/min, the extra volume delivered is \(5 \times (0.25)/10 = 0.125\text{L}\), or 125ml extra per breath.

- High fresh gas flows increase the magnitude of this phenomenon, as is typical during emergence, when we often turn the oxygen flow very high to wash out the anesthetic agent while the vent is still on, and actual delivered volumes can exceed the set volume.

- Potential reasons for discrepancies between set and delivered tidal volumes include leaks in the circuit, breathing circuit compliance (less with stiffer circuits), compressive gas losses, gas sampling from the capnograph, and ventilator/fresh gas flow coupling.

**J. Scavenging systems**

- Remove gases that vent from the popoff valve (when the machine is set to “bag”) or the spill valve (when the vent is on).

- “Closed” scavenging systems empty into a reservoir bag, which has a positive pressure relief valve (prevents excessive buildup of pressure if the scavenging line is occluded) and a negative pressure relief valve (prevents excess negative pressure from the wall suction system to be
transmitted to the patient).

- “Open” scavenging systems empty into a canister with vents, which prevents positive pressure or negative pressure being applied to the patient due to scavenger occlusion, but which can send scavenged gas into the OR.

A Quick Anesthesia Machine Check Out

The full machine checkout list is available from the FDA or our anesthesia monitoring technicians. In reality, it is not practical for most of us to perform a “full” machine check every time we are about to use an anesthesia machine. Thus, most practitioners have a truncated list that hits the most important points in a machine checkout. This list can be further abbreviated for subsequent cases during the day (after a more thorough check has been done earlier).

1. Check appropriate alarms turn on when the machine is turned on.
2. Check high pressure system: physically disconnect the oxygen pipeline from the wall. Line pressure should drop to zero, and a low oxygen pressure alarm should sound. Now open the E-cylinder of oxygen and verify both that the alarm goes away, and that the cylinder pressure is adequate. Close the E-cylinder and reattach the pipeline.
3. Check the low pressure system: occlude the end of the Y-connector, close the popoff valve, and ensure gas flows are off. Flush the high flow oxygen system to build a pressure of at least 30cmH₂O. The circuit, if leak free, should hold this pressure. Opening the popoff while keeping the Y-piece occluded should release the pressure, verifying that the popoff valve opens and closes appropriately.
4. Check the valves: there are different ways to accomplish this. One way is to place the reservoir bag at the end of the Y-piece, and turn the ventilator and fresh gas flow on, and observe for a few breaths. The valves should move with inspiration and expiration. This also checks your ventilator.

Tips for the Anesthesia Machine

1. Extra circuit tubing and breathing bags are in a drawer in the anesthesia machine. Whenever our anesthesia monitoring technicians are not available to “turn the machine over” for another case (e.g., weekends) you must change the circuit tubing and breathing bag yourself. (You also have to clean the machine and monitors.)
2. Circuit disconnections are very common. Typically your first clue will be the ventilator alarming and the bellows not refilling, accompanied by a loss of etCO₂ on the capnograph. Common places for disconnections are: where the circuit meets the machine, between pieces of the circuit (if using an extension), at the CO₂ sampling port, and at the ETT or LMA connector to the circuit.
3. Common sources of leak: the bag, the circuit tubing, the CO₂ canisters (improperly seated) or at any of the connection sites. Others include the bellows cover or the endotracheal tube itself (leak around an underfilled or ruptured cuff). Leaks within the machine itself are very uncommon. When in doubt, think of places where the equipment is often changed or disconnected/reconnected (e.g., where the circuit tubing meets the machine).
4. Make room for yourself prior to starting a case. The anesthesia machine can be pushed back or to the side if necessary to allow better access to the patient.
5. If the anesthesia machine is malfunctioning or there is a problem you just can’t figure out, you can connect the patient’s airway (ETT, LMA, etc.) to a Mapleson circuit and backup E-cylinder oxygen tank and hand-ventilate. This will serve to exclude the machine entirely as a potential source of the problem.
6. Sudden collapse of the bellows and inability to ventilate just after placing an NG or OG tube likely indicates placement of the OG/NG tube in the trachea.

7. Avoid plugging accessories into the outlets in the back of the machine. This includes many items in the OR such as the warmer for heating blankets, fluid warmers or forced-air blankets, electrocautery, etc. A short or overload is possible, potentially leading to malfunction of the machine.

8. The color-coded “caps” that connect the volatile anesthetic bottles to their vaporizers are reusable. Do NOT throw them away!

**Chapter 2B. Anesthesia Monitors**

Vigilance is one of the most defining characteristics of an anesthesiologist, and is in fact the motto of the American Society of Anesthesiologists. There is no better reflection of our constant attention to detail and to the patient’s status than in our use of monitors. Indeed, an outsider with little experience inside an OR would probably identify an anesthesiologist as the “person who is constantly watching the screen and listening to the beeps.” In fact, our duty to the patient demands that we be constantly vigilant of our monitors and the information they provide. Anesthesia profoundly impacts the patient’s physiology while rendering the patient incapable of telling us if something is “wrong.” Vigilance is thus the essence of preventing, detecting, and treating adverse events.

The following section will discuss the basic monitors as outlined by the ASA, as well as other more advanced monitoring equipment. The basic science or engineering concepts behind the monitors will not be discussed. For more complete information, consult a textbook or manufacturer’s guide.

The ASA standards for basic monitoring stipulate certain expectations for patients undergoing anesthesia. As “standards,” they are intended to be universal among anesthesia practice. By definition, standards are what we are all expected to do or employ, and deviation from them requires unusual and extenuating circumstances. The ASA does recognize this fact and several times in their standards mention that requirements may be waived in unusual circumstances, or that sometimes it is not possible to hold to these standards. That being said, the following are the expectations which we attempt to uphold with every anesthetic:

1. A qualified anesthesia provider will be continuously present for all anesthetics. “Continuous” is defined as “prolonged without any interruption at any time.” The wording does allow for absences in extenuating or emergency circumstances, at the provider’s discretion.

2. Oxygenation, ventilation, circulation and temperature shall be continually measured. Continually is defined as “repeated regularly and frequently in steady rapid succession.”

   A. Oxygenation
      - F\textsubscript{O}\textsubscript{2} is measured and a low oxygen alarm is employed during general anesthesia.
      - A quantitative measure of blood oxygenation such as pulse oximetry is used.

   B. Ventilation
      - Adequacy of ventilation must be measured. Capnography should be used unless circumstances do not allow it.
      - If intubation or LMA placement occurs, correct position must be verified by capnography, and end-tidal CO\textsubscript{2} must be continually monitored.
      - Ventilation by a machine must have an audible disconnection alarm.

   C. Circulation
      - EKG must be employed throughout the anesthetic.
• Blood pressure and heart rate should be ascertained at least every 5 minutes.
• Every patient receiving general anesthesia must have an additional continual measure of circulation such as pulse oximetry, auscultation, or palpation of a pulse.

D. Temperature
• Temperature must be measured whenever clinically relevant changes are expected or suspected.

I. The Non-invasive Blood Pressure Cuff

The NIBP cuff is the most commonly employed device to measure blood pressure during anesthesia. For most cases, the cuff is the sole measure of blood pressure. BP cuffs can be placed at a variety of locations, including the upper and lower portions of the arms or legs.

The BP cuff can be set to automatically inflate and measure pressures at various time intervals. The cuff typically inflates to suprasystolic pressures and then deflates in small increments, measuring oscillations in cuff pressure caused by arterial pulsation. These oscillations increase markedly at systolic pressure, are maximal at MAP, and decrease sharply below diastolic pressure. The NIBP measures the pressure at which these oscillatory changes occur and using a proprietary algorithm is able to calculate MAP, systolic and diastolic BP.

Sizing a NIBP cuff is important. Cuffs that are too small tend to overestimate SBP (more pressure is needed to occlude an artery) while cuffs that are too large will underestimate pressure. The cuff itself should be 20-50% wider than the width of the extremity being measured.

A word to the wise: make sure the cuff is placed to your satisfaction and that things are functioning properly before draping is done and surgery begins. It is a major headache to troubleshoot a BP cuff after the drapes are on and surgery has begun. Some patient factors may preclude placement of a BP on a particular extremity, such as presence of an AV fistula or a history of lymph node dissection on that side (which is controversial). Clearly, placing a cuff on an extremity that will be operated on is not ideal.

II. Pulse Oximetry

A pulse oximeter actually employs two scientific principles in its function: oximetry and plethysmography. Oximetry measures the ratio of red and infrared light absorption in blood. Specifically, deoxygenated blood tends to absorb red light more than oxygenated blood, which absorbs more infrared light. The ratio of oxygenated to deoxygenated blood will produce characteristic amounts of light absorption. Using standardized computations, the pulse oximeter can measure the spectrum of light absorption and calculate the percent oxygen saturation of hemoglobin. The oximeter is linked to an audible tone that rises and falls with saturation, giving us a way to know the saturation or detect changes without even having to see the monitor.

The plethysmographic component identifies arterial pulsations, which help differentiate absorption from tissue and non-pulsatile venous blood. Functionally this is displayed on our monitor as a wave corresponding to arterial pulsation. When the signal is clean, one can readily make out features of an arterial pulse on the plethysmography, sometimes including the dicrotic notch. Conversely, when the signal is distorted or poor, the tracing is poor. Not surprisingly, the reported saturation during these times can be erroneous due to poor signal.

Many different types of “sat probe” exist, including preshaped plastic finger probes, stickers, and
smaller probes which can be placed on the earlobe, forehead, or tongue. Picking the right kind of probe is a function of the patient and type of surgery (e.g., pediatric patient, severe peripheral vasoconstriction).

Two common types of artifactual readings from other hemoglobin species commonly show up on the boards. Carboxyhemoglobin absorbs red light to the same extent as oxyhemoglobin, which can produce falsely high $S_pO_2$ readings. Patients with carboxyhemoglobin poisoning will typically show a high $S_pO_2$ but low oxyhemoglobin saturation on ABG/co-oximetry. Methemoglobin has the same absorption coefficient for both red and infrared light, which clinically produces a saturation of 85%. Classic questions usually involve some trigger for methemoglobinemia (e.g., benzocaine) and a $S_pO_2$ of 85%.

Common sources of error in $S_pO_2$ readings are motion, ambient light, hypoperfusion (arterial hypotension or peripheral vasoconstriction) or poor placement of the sensor. Methylene blue dye also causes a transient, artifactual drop in $S_pO_2$. As with the NIBP cuff, make sure the probe is functioning after final positioning and before surgery starts. All of us can remember long cases constantly being worried low saturation that was only artifact due to a poorly positioned but inaccessible probe. Surgical personnel leaning on or compressing the probe or movement of the cable can also cause errors in measurement.

III. Capnography

The capnographs we employ are “diverting” capnographs; that is, they continuously aspirate small samples of gas from the breathing circuit, drawing about 100-200ml/min sample gas into the machine for analysis. By analyzing the infrared light absorption of aspirated gas, the capnograph can determine not only CO$_2$ concentration but also the concentration of inhaled anesthetics and oxygen in the sample. The gas lost to sampling is not usually clinically significant.

Confirmation of sustained end-tidal CO$_2$ following intubation is the gold standard for confirming correct placement of an endotracheal tube or LMA. Furthermore, patterns and changes in the sampled gas provide invaluable information throughout the anesthetic. For example, sudden drops in pulmonary perfusion (e.g., pulmonary embolism or drop in cardiac output) will be reflected as a drop in etCO$_2$. Given below are some very common examples of intraoperative problems and their presentation on the capnograph. (Images of waveforms and explanations from Miller’s Anesthesia, 6th ed.)

The capnograph has an audible alarm that will sound for a whole host of situations, such as apnea, abnormally low or high etCO$_2$, or high inspired agent. It is imperative that these alarms not be disabled. Because gas is actively aspirated, the tubing or sample chamber can become saturated with water vapor or even occluded which can produce false measurements. In these situations it might be necessary to change the tubing, chamber or both. This equipment can be found in the top drawer of our anesthesia machines.

At times alternate methods of oxygenation are employed, such as a face mask or nasal cannula. Our nasal cannulas have a second channel that can be plugged into the sampling chamber of the capnograph. For face-mask oxygen, it is common to connect the sampling line to a 16g angiocath that has been cut short and inserted through one of the holes in the face mask. These measures will be more qualitative than quantitative due to entrainment of room air or a mouth-breathing patient with a nasal cannula.
Examples of capnograph waves. **A**, Normal spontaneous breathing. **B**, Normal mechanical ventilation. **C**, Prolonged exhalation during spontaneous breathing. As CO₂ diffuses from the mixed venous blood into the alveoli, its concentration progressively rises. **D**, Increased slope of phase III in a mechanically ventilated patient with emphysema. **E**, Added dead space during spontaneous ventilation. **F**, Dual plateau (i.e. tails-up pattern) caused by a leak in the sample line. The alveolar plateau is artificially low because of dilution of exhaled gas with air leaking inward. During each mechanical breath, the leak is reduced because of higher pressure within the airway and tubing, explaining the rise in the CO₂ concentration at the end of the alveolar plateau. This pattern is not seen during spontaneous ventilation because the required increase in airway pressure is absent. **G**, Exhausted CO₂ absorbent produces an inhaled CO₂ concentration greater than zero. **H**, Double peak for a patient with a single lung transplant. The first peak represents CO₂ from the transplanted (normal) lung. CO₂ exhalation from the remaining (obstructed) lung is delayed, producing the second peak. **I**, Inspiratory valve stuck open during spontaneous breathing. Some backflow into the inspired limb of the circuit causes a rise in the level of inspired CO₂. **J**, Inspiratory valve stuck open during mechanical ventilation. The “slurred” downslope during inspiration represents a small amount of inspired CO₂ in the inspired limb of the circuit. **K** and **L**, Expiratory valve stuck open during spontaneous breathing or mechanical ventilation. Inhalation of exhaled gas causes an increase in inspired CO₂. **M**, Cardiogenic oscillations are commonly seen with patients who have had their pericardial sac surgically opened in the past, or may occur with sidestream capnographs for spontaneously breathing patients at the end of each exhalation. Cardiac action causes to-and-fro movement of the interface between exhaled and fresh gas. **N**, Electrical noise resulting from a malfunctioning component. The seemingly random nature of the signal perturbations (about three per second) implies a nonbiologic cause.

**IV. Temperature Monitoring**

Most temperature probes we employ are disposable. Temperature can be measured in a variety of places, the most common being the esophagus, nasopharynx, axilla, tympanic membrane, bladder, rectum and blood. Skin temperatures are prone to inaccuracy and often do not reflect a patient’s core body temp. Likewise, rectal temperatures are often slow to reflect changes in core temp (insulating effect of feces). Esophageal probes are most commonly employed for routine cases. The probe can
double as an esophageal stethoscope by connecting the end of a cheap stethoscope to the proximal part of the probe.

Hypothermia is a very common problem during surgery. Part of this is due to the cold environment and nature of the OR. Compounding these effects, during general anesthesia, compensatory mechanisms such as vasoconstriction and shivering are ablated by anesthetic inhibition of the hypothalamus. Regional anesthetics also contribute to heat loss by peripheral vasodilation and altered temperature sensation in blocked dermatomes. Thus, anesthetized patients cannot actively warm themselves and compensate for hypothermia. Problems associated with hypothermia include surgical site infection, coagulopathy leading to blood loss, decreased drug metabolism, prolonged PACU stay, adverse cardiac events, and postoperative shivering and its associated increased oxygen consumption (up to 5x baseline).

Techniques used to maintain body temperature include warming the OR, forced-air warming blankets (“Bair Huggers”), warming and humidifying inspired gases, warming IV fluids and surgical irrigation fluids, and minimizing exposure of the patient’s body surface area. Conservation of heat in general is much more efficacious at keeping a patient warm than trying to replace “lost heat,” which can take much longer. Try to keep patients warm from the start of a procedure.

V. Electrocardiography

Typically, a three-lead or five-lead EKG is employed in the OR. A three-lead EKG consists of a R arm (white), L arm (black) and a L leg (red) lead. This allows us to monitor the electrical axis of lead II, which is the best lead to observe the basic rhythm and P waves of the heart. Using a five-lead adds a R leg (green) and precordial V (brown), and adds the ability to monitor lead V lead V_3, V_4, or V_5, any of which are more sensitive in detecting left ventricular ischemia than lead II.

One helpful mnemonic to remember where the leads are placed is: “white on the right, black is opposite from white (left), snow over grass (white over green), smoke over fire (black over red).

The EKG leads are very prone to artifact from motion or electrocautery. Furthermore, the monitor displays HR by counting QRS complexes, which it sees as the highest voltage during a cardiac cycle. Abnormally tall T waves, sometimes due to incorrect lead placement, can give an erroneous heart rate, a.k.a. “double counting.” The EKG pads can be a potential area for burns if the electrocautery ground pad is dysfunctional.

VI. Arterial Pressure Monitoring

Arterial lines provide beat-to-beat information about a patient’s blood pressure. Indications for placing an invasive arterial monitor include anticipated wide swings in blood pressure, need for precise beat-to-beat knowledge of pressure (e.g., heart disease, intracranial aneurysm), arterial blood sampling, repeated blood sampling, failure of the NIBP cuff, or precise titration of blood pressure (vasopressor use or deliberate hypotension).

The transducers for an arterial line are found in our workroom and often are paired with a transducer for central venous pressure. Some rooms will often have a transducer already hooked up to the machine and zeroed thanks to our anesthesia monitoring technicians. One end of the transducer tubing is connected to a heparinized saline flush bag, and the other end should be connected to the arterial (or central) line. The transducer must also be plugged into monitoring cable of the machine (color coded).
After the cable is plugged in, a colored waveform line should appear on the screen, indicating the monitor is online. It now must be zeroed. To zero a transducer, the cap should be taken off the transducer and the stopcock closed to the patient. Hit the zero button and do not move the transducer. The monitor will beep twice to indicate that zeroing is complete.

After a transducer is zeroed, moving its height in relation to the patient will produce artifacts in pressure. A transducer that is too high will produce a pressure that is artifactually low. Conversely, a transducer that is too low with produce an erroneously high pressure. The transducer should be placed at the height of the organ whose perfusion is most relevant to the case; this may be the heart, or it may be the brain, depending on positioning and the surgery.

There are many different ways to place an arterial line. Techniques to place a radial A-line will be discussed here. The most important principle to remember is that adequate preparation is essential for success with any procedure. You will encounter situations where an A-line is needed emergently and preparation is minimal, but these situations are few and far between.

To begin, ensure that the patient’s wrist is extended and secured. This can be facilitated with an armboard or a rolled towel. In an awake patient, infiltrate the area liberally with local anesthetic to make the procedure more tolerable and reduce the possibility of vasospasm. The lidocaine we are usually supplied with is 2% and tends to burn; experiment with dilution to 1% or alkalinization. The method below describes placement with a 20g angiocatheter, the most commonly used catheters in the OR. Arrow catheters with a built-in wire are also available. The basic technique for cannulating the artery is similar with these two devices, but most people in our department prefer the 20g pink BD brand angiocatheters due to longer length and ergonomics.

To directly cannulate the artery, palpate the artery with your fingertips and advance the needle at approximately a 30-45° angle directly in line with the palpated path of the artery. Avoid the distal wrist if possible—the artery tends to be torturous and it may be difficult to thread the catheter. After getting a flash of blood, drop the angle of the needle and advance 1-2mm more to get both the tip of the catheter and the needle within the vessel lumen. This is very important; most blown IVs and arterial lines occur after a flash of blood is obtained, but attempts are made to thread the catheter before it is within the vessel. Blood flow should still be evident at this stage. Gently attempt to thread the catheter off, possibly with a “twirling” motion; it should go easily and blood should come up inside the catheter. If it does not advance easily, stop. Either do a new stick or attempt to salvage it with the wire-guided technique below.

An alternative method is to use a guidewire to thread the catheter, known as the “transfixation” or “through-and-through” technique. If direct cannulation has failed, you can advance the whole needle another 3-5mm, also going through the back wall of the artery. This can also be the technique intended from the beginning of the procedure, by getting a flash of blood and then initially advancing the needle through the vessel without trying to thread the catheter. The needle is then removed with the catheter in place. Slowly backing up the catheter should now result in arterial blood flow when the tip of the catheter resides in the vessel lumen (the tip has “backed up” into the vessel). At this point a sterile guidewire can be placed through the catheter, and the catheter can then slide over the wire into the artery. This guidewire should pass easily. If it does not, don’t force it – the wire will not end up in the vessel.

Arterial lines can be very challenging, especially in certain patients (obese, edematous, vasculopathic).
Often multiple practitioners can spend a lot of time trying to obtain an A-line with no success. Remember this rule: **don’t force it.** If the guidewire or catheter doesn’t go in easily, the line won’t work or be in the right place. It’s probably better to simply restick and try again. And again, it is best to have all position and equipment optimized for your first attempt; your first shot is your best shot.

**VII. Central Venous Catheters**

Central lines are indicated for monitoring central venous pressure, to infuse certain medications (TPN, hypertonic saline, potassium), to provide large-bore venous access, to provide access for placement of other monitors or tools (e.g., pulmonary artery catheter or transvenous pacing leads), to aspirate venous air emboli, or when peripheral access is not possible.

**VIII. Pulmonary Artery Catheters**

Our first major exposure to these catheters often comes during our cardiac anesthesia rotation. The indications for placing a pulmonary artery catheter include precise measurements of cardiac output, right heart pressures, or mixed-venous oxygen saturation.

**IX. Peripheral Nerve Stimulators**

These are more fully discussed in the section on neuromuscular blockers. They are found in the drawers in our anesthesia machines.

**X. EEG**

For specific cases we periodically employ either “true” (16 lead) EEGs or processed EEG devices such as the BIS monitor or SEDline. Use of any of these devices is not routine here at UCSD. You will most often encounter the EEG during carotid endarterectomy, where it is employed to monitor electrical activity and detect possible ischemic events in the brain. The BIS and SEDline are sometimes used to measure depth of anesthesia, although the data they provide is of controversial value. One common place where the BIS or SEDline is used is in the heart room, when sometimes low levels of anesthetic and increased possibility of patient awareness are a recognized phenomenon. The anesthesia monitoring technicians are invaluable in helping us set up and use these monitors.

**XI. Urine Output**

A Foley catheter is often placed by the circulating nurse or surgical personnel for cases where it is needed for surgical exposure (e.g. C-section) or cases longer than about 2hrs. Monitoring urine output is also useful any time major fluid shifts are expected, end-organ perfusion measurement is desired, keeping in mind the many factors that affect intraoperative urine output. Some Foleys also have temperature sensors included.

**XII. Transesophageal Echocardiography (TEE)**

TEE is used routinely for every cardiac surgery and most liver transplants at UCSD, and occasionally for less major surgeries when major hemodynamic disturbances are expected or occurring. An example would be an open AAA repair or a patient who develops myocardial ischemia and/or shock intraoperatively. All of our cardiac anesthesia attendings are experts at TEE and many of our non-cardiac attendings are skilled with it also. TEE is a powerful and useful monitor that you will gain experience
with during your cardiac anesthesia months.

XIII. Cardiac Output Monitors

Several non-invasive or minimally-invasive cardiac output monitors exist, including those based on esophageal Doppler, thoracic impedance, transpulmonary thermodilution, and arterial pulse contour analysis; TEE can also be used, but is considered “invasive.” At UCSD, we most commonly use the Edwards Vigileo-Flotrac system. This device analyzes the shape of the A-line waveform and uses patient data to calculate stroke volume; this is then used to calculate cardiac output. This monitor needs an A-line, a special proprietary transducer, and another display monitor. Since it is somewhat expensive, it is typically reserved for cases with expected large fluid shifts, where monitors in addition to a standard A-line and/or CVP may be needed.

Chapter 2C. Medications Used in Anesthesia

The following is a list of the drugs we commonly use in anesthesia. It is by no means all-inclusive or meant to replace definitive texts or manufacturer’s guidelines. I offer the list as a quick reference for 99% of the drugs we encounter and administer on a daily basis.

I. Vasopressors
   A. Ephedrine
      - Uses: hypotension, especially with slower HRs.
      - Mechanism of action: indirect adrenergic agonist, causes release of endogenous catecholamines. Mild increases in BP, HR, and contractility. Affects both α and β receptors.
      - Duration of action: minutes if given IV, up to 1hr IM.
      - Usual dose: 5mg bolus IV, 25-50mg IM. Can also be given SC or PO (not common).
      - Notes: considered first line agent for hypotension in pregnancy as it theoretically spares uterine blood flow. Tachyphylaxis develops with repeated administration. Avoid with MAO inhibitors (risk of malignant hypertension due to too much endogenous catecholamines). Potentially ineffective in catecholamine-depleted states (i.e. chronic methamphetamine or cocaine use).
   B. Phenylephrine
      - Uses: hypotension, especially from low SVR state with a higher HR.
      - Mechanism of action: direct α₁ agonist, causing marked vasoconstriction, rise in BP and SVR. May cause reflex bradycardia.
      - Duration of action: minutes.
      - Usual dose: 50-100mcg IV bolus, or run as an infusion 10-200mcg/min.
      - Notes: must be diluted from its packaged concentration which is 10mg/ml. Most of mix 10mg in a 100ml bag of saline to make 100mcg/ml concentration. Safe in pregnancy.
   C. Epinephrine
      - Uses: cardiac arrest, anaphylaxis, bronchospasm, cardiogenic shock, refractory hypotension, reduced CO.
      - Mechanism of action: direct agonist at α₁, β₁, and β₂ receptors, depending on dose. Increased HR, SVR, BP, contractility and bronchodilation.
      - Duration of action: minutes.
      - Usual dose: 10mcg/kg SC, 0.03-0.2mcg/kg IV bolus, 0.01-1 or more mcg/kg/min infusion, 0.5-1mg IV bolus for cardiac arrest.
      - Notes: can cause tissue necrosis if extravasates from IV; cardiac arrest doses can cause profound
hypertension; if given via ETT, give 2-3x IV dose, diluted to > 5ml.

D. Vasopressin
- Uses: alternative to epinephrine in cardiac arrest, catecholamine-resistant hypotension.
- Mechanism of action: activates $V_1$ receptors, causing direct peripheral vasoconstriction and raising SVR independent of adrenergic receptors.
- Duration of action: minutes.
- Usual dose: 40 units IV bolus for cardiac arrest, 1-2 units IV boluses for hypotension, can also be given as infusion, typically 1-4 units/hr. Can also be given via endotracheal tube.
- Notes: very potent. Can cause splanchnic hypoperfusion, lactic acidosis, or myocardial ischemia (especially with infusions). Causes unpleasant symptoms in awake patients.

E. Dopamine
- Uses: hypotension, primarily due to low CO state.
- Mechanism of action: has mixed effects depending on dose. At low dose (1-3 mcg/kg/min), has primarily DA receptor effects, at 3-10 mcg/kg/min $\beta_1$ effects predominate, and >10 mcg/kg/min primarily $\alpha_1$ effects are seen.
- Duration of action: minutes.
- Usual dose: IV infusion, 1-20 mcg/kg/min.
- Notes: preferred 1st line agent for coming off cardiopulmonary bypass by our CT surgeons; low “renal” doses may improve renal perfusion and will cause diuresis. Must be diluted. Drip is usually 400 mg in 250 ml.

II. Antihypertensives
A. Nitroprusside
- Mechanism of action: converted to nitric oxide, a potent vasodilator. Nitric oxide activates guanylyl cyclase, increases cGMP, decreases intracellular calcium, and thus produces smooth muscle relaxation.
- Properties: causes arterial > venous dilation, reducing BP by reduction in afterload > preload.
- Duration of action: quick onset and offset, allowing precise titration.
- Usual dose: 0.5-10 mcg/kg/min infusion, or small (10-20 mcg) boluses.
- Notes: Can cause “coronary steal” (dilation of normal coronaries, stealing flow away from stenotic, maximally-dilated areas). Reduces PVR. Increases cerebral blood flow which can be attenuated by hyperventilation.
- Metabolism: this is a commonly tested question on the boards. Nitroprusside is essentially an iron atom bound to nitroso and cyanide moieties. It oxidizes hemoglobin in RBCs, producing methemoglobin and cyanide ions. Cyanide ions do one of three things:
  1. Bind to methemoglobin, forming cyanomethemoglobin
  2. Combine with thiosulfate to form thiocyanate
  3. Bind to cytochrome oxidase, interfering with oxygen utilization
- Signs of cyanide toxicity include metabolic acidosis, increased mixed venous $O_2$ (less $O_2$ is used), arrhythmias and tachyphylaxis. Cyanide toxicity is unusual in durations less than 2 days and cumulative doses less than 0.5 mg/kg/hr. Supportive treatment of toxicity includes stopping the drug, oxygen, thiosulfate and sodium nitrate. Thiosulfate will divert cyanide ions and produce thiocyanate (above). Sodium nitrite converts hemoglobin to methemoglobin, which can then react with cyanide ion.
- Excess thiocyanate can also produce toxicity, characterized by weakness, hypoxia, thyroid dysfunction and agitation. This risk is increased in renal failure because thiocyanate is cleared by the kidney. Lastly, methemoglobinemia can be treated with methylene blue, which reduces
methemoglobin back to hemoglobin.

B. Nitroglycerin
- Mechanism of action: donates NO, like nitroprusside.
- Properties: primarily venodilation, reducing preload and BP.
- Duration of action: quick onset and offset, allowing precise titration.
- Usual dose: 0.5-10mcg/kg/min, or as small boluses 10-40mcg IV. Can also be given sublingually or transdermally.
- Notes: Relieves coronary vasospasm and does not possess the steal properties nitroprusside does. Reduces preload and myocardial oxygen demand while increasing supply. Pulmonary and cerebral vasodilation, can cause headaches. Also used for uterine relaxation in OB procedures.
- Metabolism: metabolized to nitrites, which can cause methemoglobinemia (see above).

C. Hydralazine
- Uses: hypertension, especially on OB for pregnancy-induced hypertension.
- Mechanism of action: causes direct arteriolar vasodilation.
- Usual dose: 5-20mg IV. Onset is within 5-20min and duration is 2-6hrs, making it difficult to titrate.

III. Neuromuscular Blockers and Reversal Agents
A. Vecuronium
- Mechanism of action: competitive antagonism at ACh receptors in the neuromuscular junction
- Duration of action: 45-90min for intubating dose
- Usual dose: 0.1mg/kg IV for intubation, 0.01mg/kg IV boluses for maintenance
- Notes: hemodynamically unremarkable, cheap. Primarily excreted in bile, 25% by kidneys. May have prolonged block in patients with renal failure. When given as a long-term infusion, can see prolonged blockade lasting for days, possibly due to a polyneuropathy. Forms a precipitate with thiopental (avoid giving concomitantly in same line).

B. Rocuronium
- Mechanism of action: competitive antagonism at ACh receptors in the neuromuscular junction.
- Duration of action: 20-60min for lower doses, up to 2hrs for 4x ED95 rapid sequence dose.
- Usual dose: 0.6mg/kg for intubation, 1.2mg/kg for RSI.
- Notes: quick (1min) onset when given in rapid sequence doses. Rapid (20min) offset when given at lower doses. Used as an alternative to succinylcholine for RSI. Anecdotally, is more “resistant” to reversal, especially after large doses or redosing.

C. Cisatracurium
- Mechanism of action: competitive antagonism at ACh receptors in the neuromuscular junction.
- Duration of action: 30-60min for intubating dose.
- Usual dose: 0.2mg/kg for intubation, 0.02mg/kg for maintenance.
- Notes: eliminated via Hoffman degradation, an organ-independent process, thus is useful in liver/renal failure patients. Unlike atracurium, there is no significant histamine release. Laudanosine is a potentially toxic metabolite (causes CNS excitation, less laudanosine than seen with atracurium, probably clinically insignificant).

D. Pancuronium
- Mechanism of action: competitive antagonism at ACh receptors in the neuromuscular junction.
- Duration of action: 60-120min for intubating dose.
- Usual dose: 0.1mg/kg IV for intubation, 0.01mg/kg IV boluses for maintenance.
- Notes: can cause tachycardia due to vagolytic effects and sympathetic stimulation. Long acting. Excreted primarily by kidney leading to prolonged action in renal failure. May inhibit
pseudocholinesterase, resulting in a prolonged block from succinylcholine or mivacurium.

E. Succinylcholine
- Mechanism of action: ACh receptor agonist. Causes depolarization of the muscle-end plate, then prevents end-plate repolarization, blocking further depolarization.
- Duration of action: 5-10min. Onset is within 30 seconds.
- Usual dose: 1-1.5mg/kg IV. Boluses of 0.1mg/kg IV for maintenance, or 2-15mg/min infusion. Can be given IM (4-5mg/kg).
- Notes: the only depolarizing muscle relaxant in use today. Metabolized by pseudocholinesterase. Most rapid onset and offset of all muscle relaxants. Repeated doses may cause prolonged phase II block or arrhythmias (often bradycardia, more pronounced in children). May cause hyperkalemia, raise intragastric and intraocular pressure, masseter muscle rigidity, can trigger malignant hyperthermia. Can be used in renal failure provided no baseline hyperkalemia. Causes fasciculations which may lead to myalgias (can pretreat with a small amount of nondepolarizing muscle relaxant).

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Dose (mg/kg)</th>
<th>Onset (minutes)</th>
<th>Length (minutes)</th>
<th>Advantages</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Succinylcholine</td>
<td>Depolarizing</td>
<td>1-2</td>
<td>Immediate</td>
<td>3-5</td>
<td>Short action (intubation)</td>
<td>Hyperpotassemia Fasciculations</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>Non-depolarizing</td>
<td>Initial bolus: 0.08-0.2 INF: 0.08-0.2 mg/kg/h</td>
<td>2-4</td>
<td>20</td>
<td>No cardiovascular effects</td>
<td>Muscle weakness</td>
</tr>
<tr>
<td>Pancuronium</td>
<td>Non-depolarizing</td>
<td>Initial bolus: 0.1 INF: 0.1 mg/kg/h</td>
<td>2-4</td>
<td>30-45</td>
<td>Longer action</td>
<td>Tachycardia, hypertension Increase in ICH</td>
</tr>
<tr>
<td>Atracurium</td>
<td>Non-depolarizing</td>
<td>Initial bolus: 0.3-0.6 INF: 0.3-0.6 mg/kg/h</td>
<td>2-3</td>
<td>25-30</td>
<td>Not metabolized by the liver and kidney</td>
<td>Bronchospasm Bradycardia</td>
</tr>
<tr>
<td>Rocuronium</td>
<td>Non-depolarizing</td>
<td>Initial bolus: 0.6-1.2 INF: 5-15 µg/kg/min</td>
<td>1-2</td>
<td>30-40</td>
<td>No cardiovascular effects</td>
<td>Tachycardia at high doses</td>
</tr>
<tr>
<td>Mivacurium</td>
<td>Non-depolarizing</td>
<td>Initial bolus: 0.1-0.2 INF: 10-14 µg/kg/min</td>
<td>2-4</td>
<td>12-18</td>
<td>Short action</td>
<td>Bronchospasm Coughing</td>
</tr>
<tr>
<td>Cisatracurium</td>
<td>Non-depolarizing</td>
<td>Initial bolus: 0.15 INF: 1.5 µg/kg/min</td>
<td>3-4</td>
<td>30</td>
<td>Not metabolized by the liver and kidney</td>
<td>No cardiovascular effects</td>
</tr>
</tbody>
</table>

ICH = intracranial hypertension; INF = continuous infusion.

F. Neostigmine
- Use: to reverse non-depolarizing neuromuscular blockade and treatment of myasthenia gravis.
- Mechanism of action: an acetylcholinesterase inhibitor, neostigmine increases the concentration of ACh available in the neuromuscular junction.
- Duration of action: more than 1hr. Peak effect is within 5-10min.
- Usual dose: 0.05-0.07mg/kg, to a max of 5mg. Giving more could result in paradoxical weakness.
- Notes: lipid-insoluble so cannot cross the blood-brain barrier. Administer with an antimuscarinic
to block cholinergic side effects (usually glycopyrrolate, since onset is similar). Paradoxical potentiation of neuromuscular blockade occurs when excessive doses are used. Side effects include those of muscarinic stimulation: bradycardia, bronchospasm, secretions, CNS excitation, bowel spasm/defecation, urination, miosis. Can result in a prolonged block with succinylcholine due to decreased activity of pseudocholinesterase.

G. Edrophonium
- Use: reversal of neuromuscular blockade, diagnosis of myasthenia gravis (“Tensilon test”).
- Mechanism of action: same as neostigmine (see above).
- Duration of action: Quick onset, within 1-2min but lasts shorter than neostigmine, about 15min. Up to 1 hr with higher doses.
- Usual dose: 0.5-1mg/kg.
- Notes: does not cross blood-brain barrier. Similar side effect profile as neostigmine. Muscarinic effects are less pronounced, requiring half the amount of anticholinergic as an equipotent dose of neostigmine. Atropine should probably be used as the anticholinergic, since its quick onset will parallel that of edrophonium.

H. Physostigmine
- Use: penetrates the blood-brain barrier, making it useful to counter anticholinergic toxicity (e.g., scopolamine). Not used to reverse neuromuscular blockade, and thus not usually given with an anticholinergic.
- Mechanism of action: similar to neostigmine.
- Usual dose: 0.01-0.03mg/kg.
- Notes: the only available cholinesterase inhibitor that crosses the blood-brain barrier.

I. Atropine
- Use: treatment of bradyarrhythmias, “slow” PEA, block muscarinic side effects of acetylcholinesterase inhibitors.
- Mechanism of action: antimuscarinic, blocks the ACh receptor.
- Duration of action: rapid onset, lasts up to 30min.
- Usual dose: 0.01-0.02mg/kg. Can also be given IM or via ETT.
- Notes: most potent and quickest-acting anticholinergic for serious bradycardia. Crosses the blood-brain barrier but CNS effects are usually minimal. Also causes bronchodilation. Avoid in narrow-angle glaucoma, bladder neck obstructions or prostatic hypertrophy.

J. Glycopyrrolate
- Use: decrease airway secretions, block cholinergic side effects of acetylcholinesterase inhibitors, treatment of mild bradycardia.
- Mechanism of action: same as atropine.
- Duration of action: up to 2hrs, slower onset that atropine.
- Usual dose: 0.005-0.01mg/kg.
- Notes: does not cross the BBB, thus OK to use in narrow angle glaucoma.

K. Scopolamine
- Use: premedication because of the sedative effect, decreases airway secretions, good for motion sickness or PONV prophylaxis.
- Mechanism of action: same as atropine.
- Usual dose: same as atropine. Usually given IM. Available as transdermal patch.
- Notes: used to be widely used as a premedication. More sedating than atropine or glycopyrrolate. Pronounced ocular effects; avoid in narrow angle glaucoma. Occasionally used for trauma patients when volatile or IV anesthetic is contraindicated due to hypovolemia.

L. Sugammadex
- Use: reversal of neuromuscular blockade from rocuronium.
- Mechanism of action: a cyclodextrin, it encapsulates and binds rocuronium molecule, rendering it unavailable to bind to acetylcholine receptors in the neuromuscular junction.
- Duration of action: onset within 5-15min.
- Usual dose: 2mg/kg.
- Notes: Only available in Europe. Lower affinity for vecuronium. Does not need to be administered with antimuscarinic agents. When insufficient doses are administered, risk of agent wearing off because neuromuscular blocker is incompletely cleared.

IV. Inhalational Anesthetics

General Considerations

- Blood:gas partition coefficient reflects the blood solubility of an agent. The more insoluble an agent is, the less it is taken up by the bloodstream, the faster it raises the partial pressure, and the faster the induction time. Conversely, the higher the blood:gas partition coefficient, the more soluble the agent is.
- The $F_a/F_i$ ratio is an expression of how much an agent is taken up by the bloodstream. As gas fills the alveolar space it is taken away by pulmonary blood flow. Thus, the $F_a$ is less than the $F_i$. More soluble agents are taken up more avidly, so the $F_a/F_i$ ratio is less than for a relatively insoluble agent. Conversely, the $F_a/F_i$ ratio is greater for more insoluble agents. The $F_a$ determines the partial pressure of anesthetic in the alveoli, and ultimately the brain. Thus, more insoluble anesthetics will have higher $F_a/F_i$ ratio and faster induction times.
- Low cardiac output states speed induction because less anesthetic will be taken up by the bloodstream and the $F_a/F_i$ ratio rises rapidly. This effect is less pronounced for insoluble anesthetics since minimal amounts are taken up anyway.

- A right-to-left intracardiac shunt will slow induction, because a portion of blood flow will bypass the lungs, not become saturated with anesthetic, and lower the arterial partial pressure. Similarly, a mainstem intubation will also slow induction, since half of pulmonary blood flow will go to a non-ventilated lung. MAC, or minimum alveolar concentration, is an expression of an agent’s potency. 1 MAC has been defined as the concentration to which 50% of patients will not move to surgical incision. At 1.3 MAC, 95% of patients will not move to that same stimulus.
Lastly, 0.3 MAC is considered MAC_{awake} (awakening from anesthesia). It is important to remember that MAC is additive, and that other agents may decrease MAC requirements (e.g., opioids, propofol). MAC decreases by 6% for each decade of life.

- The agents available at Hillcrest and Thornton are sevoflurane, isoflurane and nitrous oxide. The VA has all of the above plus desflurane. There is also a desflurane vaporizer in OR7 at Hillcrest. Halothane is not used at any of the three locations, although you may encounter halothane on trips to under-served areas, e.g., Mexico.

A. Desflurane

- **General:** very fast onset and offset, nearly as fast as nitrous oxide. Requires a special vaporizer because of its high vapor pressure; see the anesthesia equipment section. MAC is 6%; not potent, and relatively expensive.
- **Cardiovascular:** minimal cardiac depression, but does cause decrease in SVR and MAP. May cause a significant increase in HR if its concentration is rapidly raised above 6%, not mediated by airway irritation.
- **Pulmonary:** decreases hypoxic respiratory drive and increases apneic threshold. Very pungent, so not ideal for inhalation induction. Induction and awakening can be associated with coughing, bronchospasm, or laryngospasm.
- **Neurologic:** increases CBF but decreases CMRO$_2$. “Uncouples” cerebral autoregulation, rendering CBF proportional to MAP.
- **Other:** degraded by dry CO$_2$ absorbent (especially barium hydroxide) to carbon monoxide, classically in the “Monday morning” scenario described above. Trigger for malignant hyperthermia.

B. Sevoflurane

- **General:** fairly rapid onset and offset (second to desflurane). MAC is 2%.
- **Cardiovascular:** minimal cardiac depression, but does cause decrease in SVR and MAP. Dilates coronary arteries.
- **Pulmonary:** decreases hypoxic respiratory drive and increases apneic threshold. Is a very potent bronchodilator. Nonpungent, suitable for inhalational inductions.
- **Neurologic:** increases CBF but decreases CMRO$_2$. “Uncouples” cerebral autoregulation, rendering CBF proportional to MAP.
- **Renal/hepatic:** decreases blood flow to both systems.
- **Other:** can be degraded by dry barium hydroxide or soda lime to compound A, a potentially nephrotoxic compound. This risk is increased with low flows (<1L/min), or high concentrations of sevoflurane; the recommendation is to run 2L/min or higher if more than 2 MAC-hours are used. Trigger for malignant hyperthermia.

C. Isoflurane

- **General:** slow onset and offset (compared to desflurane and sevoflurane). MAC is 1.1%. Inexpensive.
- **Cardiovascular:** minimal cardiac depression, but does cause decrease in SVR and MAP. Associated with tachycardia, which tends to maintain cardiac output in the face of decreased SVR. Dilates coronary arteries.
- **Pulmonary:** decreases hypoxic respiratory drive and increases apneic threshold. Pungent, so not suitable for inhalational induction.
- **Neurologic:** increases CBF but decreases CMRO$_2$. “Uncouples” cerebral autoregulation, rendering CBF proportional to MAP.
- **Renal/hepatic:** decreases blood flow to both systems.
- **Other:** partially metabolized to trifluoroacetic acid by the liver (metabolism inhibited by
disulfiram), which is potentially nephrotoxic but probably not clinically relevant. Trigger for malignant hyperthermia.

D. Halothane
- General: inexpensive. MAC is 0.7%.
- Cardiovascular: causes direct myocardial depression. Dilates coronary arteries. Blunts baroreceptor response to hypotension. Can sensitize the myocardium to catecholamines and predispose to arrhythmias.
- Pulmonary: decreases hypoxic respiratory drive and increases apneic threshold. Suitable for inhalational inductions.
- Neurologic: increases CBF but decreases CMRO$_2$. “Uncouples” cerebral autoregulation, rendering CBF proportional to MAP.
- Renal/hepatic: decreases blood flow to both systems.
- Other: partially metabolized to triflouroacetic acid by the liver (metabolism inhibited by disulfiram), which is potentially nephrotoxic but probably not clinically relevant. Halothane hepatitis is extremely rare (1:30000 cases) and is associated with multiple halothane exposures, obese women, and family history. The lesion is centrilobular necrosis and is also associated with hypoxia. Halothane does not seem to worsen preexisting liver dysfunction. Trigger for malignant hyperthermia.

E. Nitrous Oxide
- General: colorless and odorless. Supports combustion. MAC is 105% (greater than 1 atmosphere needed to produce 1 MAC).
- Cardiovascular: weakly stimulates the sympathetic nervous system. Increases pulmonary vascular resistance.
- Pulmonary: decreases hypoxic drive.
- Neurologic: mildly increases cerebral blood flow and CMRO$_2$.
- Renal/hepatic: decreases blood flow to both systems
- Other: inhibits methionine synthase, a B$_{12}$-dependent enzyme, which is necessary for DNA synthesis. Prolonged or repeated exposure can result in B$_{12}$ deficiency with megaloblastic anemia and peripheral neuropathy. Possible teratogen; avoid in pregnancy. Will rapidly fill air-filled cavities, potentially creating hazardous increases in pressure or volume- examples include pneumothorax, air embolism, bowel gas, or intraocular air bubbles. Can cause PONV.
V. Hypnotics
A. Barbiturates
- Mechanism of action: potentiates GABA at the GABA<sub>A</sub> receptor.
- Route of administration: typically IV. Thiopental and methohexital can be given PR, and pentobarbital and secobarbital can be given IM.
- Pharmacokinetics: rapid onset when given IV. Rapid offset due to redistribution. Elimination half-life is actually on the order of hours; repeated doses can saturate peripheral compartments, making recovery dependent on metabolism (and thus much slower).
- Cardiovascular: decreases BP and CO, mostly due to peripheral vasodilation, pooling of blood and decreased preload, reflex tachycardia with thiopental.
- Pulmonary: causes respiratory depression and apnea. May not fully depress airway reflexes, resulting in bronchospasm or laryngospasm in “light” patients.
- Neurologic: profound decreases in CMRO<sub>2</sub> and CBF. Considered good agents in the setting of increased ICP. Can be used to induce electrical silence on EEG which may offer cerebral protection from ischemia. Also used as antiepileptic.
- Other: induces cytochrome P450 enzymes which may speed metabolism of some drugs. Can stimulate the formation of porphyrin; avoid in patients with acute intermittent porphyria.
B. Benzodiazepines
- Midazolam will be discussed since it is the benzodiazepine most commonly used by anesthesiologists.
- Mechanism of action: enhances activity of GABA receptor.
- Route of administration: PO, IM or IV. Only IV is suitable for inducing general anesthesia.
- Pharmacokinetics: rapid onset when given IV. Elimination half-life is 2hrs. Large doses can have prolonged effects resulting in slower wakesups.
- Cardiovascular: minimal effects when given alone. Often combined with an opioid to induce general anesthesia in tenuous (e.g., cardiac) patients.
- Pulmonary: can cause respiratory depression. Usually not significant when given alone, however when combined with another agent such as an opioid the effect is synergistic.
- Neurologic: decreases CMRO<sub>2</sub> and CBF. Causes anterograde amnesia.
- Other: useful premedication due to anterograde amnesia and useful in children who cannot tolerate an IV.
C. Etomidate
- Mechanism of action: enhances activity of GABA<sub>A</sub> receptor.
- Route of administration: IV.
- Pharmacokinetics: rapid onset. Rapid offset as well, due to redistribution.
- Cardiovascular: maintains cardiac output, contractility and SVR, almost unique amongst induction agents. Does not blunt response to intubation.
- Pulmonary: typically does not cause apnea when given alone. If given in conjunction with other agents, can cause profound respiratory depression.
- Neurologic: decreases CBF and CMRO<sub>2</sub>. Can activate epileptic foci.
- Other: can cause adrenal suppression with even a single dose, but this is more of a concern when given as an infusion or to those who are critically ill. Significant incidence of myoclonus, which can be disturbing. May cause nausea/vomiting. Burns on injection.
D. Propofol
- Mechanism of action: enhances activity of GABA<sub>A</sub> receptor.
- Route of administration: IV.
- Pharmacokinetics: rapid onset. Rapid offset as well, due to redistribution.
• Cardiovascular: decreases MAP by decreasing SVR, causing venodilation, and reducing cardiac contractility.
• Pulmonary: causes respiratory depression all the way to complete apnea depending on dose.
• Neurologic: decreases CBF and CMRO$_2$. Considered one of the best agents to reduce ICP or brain size, or as the anesthetic for craniotomies. Antiepileptic.
• Other: Antiemetic. Long term infusions can cause propofol infusion syndrome: cardiac failure, renal failure, rhabdomyolysis, lactic acidosis. Lipid emulsion is also a good growth medium for bacteria, use strict aseptic technique and within 6hrs of opening. Burns on injection. Contains soy and lecithin; found in egg yolks, not egg white. Most people with egg allergies are probably allergic to the albumin found in egg whites, not lecithin.

E. Ketamine
• Mechanism of action: antagonizes NMDA (glutamate) receptors.
• Route of administration: IV or IM.
• Pharmacokinetics: rapid onset. Rapid offset as well, due to redistribution.
• Cardiovascular: is a sympathomimetic and thus typically maintains SVR, CO and BP. Also increases HR. However, is actually a negative inotrope in vivo, and thus in patients who are already maximally sympathetically driven or have depleted catecholamine stores (e.g., end-stage shock), there may be profound myocardial depression.
• Pulmonary: does not affect respiratory drive when given alone and works well as a bronchodilator. Increases secretions.
• Neurologic: increases CMRO$_2$ and CBF. The dogma is to avoid ketamine any time increased ICP is an issue. May cause delirium or illusions, less if pretreated with a benzodiazepine. Can cause myoclonus and nystagmus.
• Other: is a “dissociative” anesthetic, in that patients may appear awake but do not respond to sensory input. Also has weak opioid activity, and can be a profound analgesic, or used as an infusion to augment analgesia or reduce post-op opioid requirement. Small doses can be useful for sedation/analgesia in various settings.

VI. Opioids

Opioids are excellent analgesics and mild sedatives. They do not reliably produce amnesia. Although there are many different opioids, only the most commonly used ones will be discussed here: morphine, fentanyl, alfentanil, sufentanil, remifentanil, hydromorphone, and meperidine. Opioids have a very wide therapeutic index and dosing can vary tremendously based on tolerance, general state of the patient, and other medications that may be coadministered. Thus, it is difficult to provide “standard” doses.

Morphine is the prototypical drug against which the other IV opioids are measured. The relative potencies of the various drugs in relation to morphine are:

• Hydromorphone: 5-7x more potent than morphine
• Meperidine: 1/10 as potent as morphine
• Fentanyl: 100x as potent as morphine
• Alfentanil: 1/5 as potent as fentanyl (20x morphine)
• Sufentanil: 10x as potent as fentanyl (1000x morphine)
• Remifentanil: 2x as potent as fentanyl (200x morphine)

Comparison of doses between the opioids generally reflect these relative potencies. For example, you
might consider giving 5mg morphine, 50mcg fentanyl, or 0.8mg hydromorphone to the same patient for postoperative pain.

A. Morphine
- **Dose:** 0.05-2mg/kg IM, 0.03-0.5mg/kg IV.
- **Route of administration:** IV, PO, intrathecal, epidural.
- **Pharmacokinetics:** longer onset and duration of action due to low lipid solubility. Onset typically within 5-15min, peak plasma levels within 60min, duration of action around 4hrs.
- **Cardiovascular:** by blocking sympathetic output, may cause decrease in BP and HR. Reduces SVR secondary to histamine release; can be profound. Minimal changes when given slowly or in small doses.
- **Pulmonary:** causes respiratory depression, depression of hypoxic drive and increases apneic threshold.
- **Neurologic:** may decrease CMRO₂ and CBF to small extent, although associated respiratory acidosis may outweigh this. Can cause nausea, vomiting and pruritus. At equianalgesic doses, tends to be more sedating than other opioids.
- **Other:** slows GI motility and gastric emptying. Is the “equivalent” against which other opioids are measured (“morphine equivalents”). Primary products of metabolism are morphine 3- and 6-glucuronide which are active metabolites; these metabolites and morphine itself can accumulate in renal failure patients, causing prolonged respiratory depression.

B. Fentanyl
- **Dose:** varies depending on tolerance and state of patient; a typical IV dose to block sympathetic response to intubation is 2-5mcg/kg. Doses can be up to 50mcg/kg for cardiac anesthesia.
- **Route of administration:** IV, intrathecal, epidural, intranasal via a spray, transdermal via a patch, or transmucosal via a “lollipop.”
Pharmacokinetics: highly lipophilic, crosses the blood-brain barrier easily. Rapid onset within 1-3min, peak plasma levels 3-5min, short duration of action due to redistribution (30min-1hr), longer duration with larger doses.
Cardiovascular: less histamine release than morphine.
Pulmonary: similar to morphine, can also cause chestwall rigidity when given in large doses, which may compromise ventilation.
Neurologic: similar to morphine.
Other: slows GI motility and gastric emptying. Repeated doses or infusions can cause saturation of peripheral redistribution sites, increasing the time to offset (context-sensitive half-time). Chestwall rigidity can be managed with neuromuscular blockers.

C. Alfentanil
- **Dose:** 10-50mcg/kg, varies depending on length of procedure.
- **Route of administration:** IV.
- **Pharmacokinetics:** Although less lipid soluble than fentanyl, onset/offset is more rapid due to low pKₐ. Thus, most of alfentanil exists in non-ionized, lipophilic form. Onset within 1-2min, duration of action varies depending on dose given but typically 10-30min.
- **Cardiovascular:** less histamine release than morphine.
- **Pulmonary:** similar to morphine, can also cause chestwall rigidity with large doses.
- **Neurologic:** similar to morphine.
- **Other:** slows GI motility and gastric emptying. Repeated doses or infusions can cause saturation of peripheral redistribution sites, increasing the time to offset (context-sensitive half-time), although the effect is less pronounced than with fentanyl. Chestwall rigidity can be managed with neuromuscular blockers. Excellent for situations requiring intense, short-lived analgesia (e.g. rigid bronchoscopy).

D. Sufentanil
- **Dose:** given in doses typically 1/10 that of fentanyl; intubation dose 0.2-0.5mcg/kg.
- **Route of administration:** IV, intrathecal, epidural (in some countries, only indicated for epidural use); a transdermal sufentanil patch is in clinical trials.
- **Pharmacokinetics:** when given IV, onset within 1-3min, duration of action around 3hrs.
- **Cardiovascular:** less histamine release than morphine.
- **Pulmonary:** similar to morphine, can also cause chestwall rigidity with large doses.
- **Neurologic:** similar to morphine.
- **Other:** slows GI motility and gastric emptying. Repeated doses or infusions can cause saturation of peripheral redistribution sites, increasing the time to offset (context-sensitive half-time), but less than either fentanyl or alfentanil over an 8-hour infusion. Chestwall rigidity can be managed with neuromuscular blockers.

E. Remifentanil
- **Dose:** induction dose 1-3mcg/kg followed by 0.1-0.5mcg/kg/min infusion for GA, 0.02-0.2mcg/kg/min for MAC.
- **Route of administration:** reconstituted from powder form and given IV.
- **Pharmacokinetics:** onset within 1min and duration of action of 5-10min. Metabolized by red blood cell and nonspecific esterases. This unique mode of metabolism makes the pharmacokinetics very predictable and titratable. Furthermore, the context-sensitive half-time (3-7min) for remifentanil does not change for long infusions.
- **Cardiovascular:** less histamine release than morphine; very high incidence of bradycardia due to vagal potentiation.
- **Pulmonary:** similar to morphine, can also cause chestwall rigidity with large doses.
- Neurologic: similar to morphine.
- Other: excellent for cases that are very stimulating and have intense analgesic requirements, yet also need precise titration of opioids. Does not have a “tail,” so all analgesic effects will be gone within 10-20 minutes of stopping the drug. Thus, another longer-acting opioid must be used to avoid postoperative pain. Can cause opioid-induced hyperalgesia, possibly due to its intense agonism of opioid receptors.

**Context-sensitive half-time:** the time required for 50% reduction in the plasma concentration of a drug on termination of a constant infusion. This time is determined by both elimination and redistribution, and it varies considerably as a function of infusion duration.

F. Hydromorphone
- Dose: 7x more potent than morphine; typical doses 0.2-2mg.
- Route of administration: IV, PO, SC/IM, PR.
- Pharmacokinetics: when given IV, onset in 5-10min, peak effect in 15-30min, duration of action around 4hrs.
- Cardiovascular: less histamine release than morphine.
- Pulmonary: causes respiratory depression, depression of hypoxic drive and increases apneic threshold.
- Neurologic: similar to morphine.
- Other: has largely supplanted morphine due to less histamine-related side effects and faster onset.

G. Meperidine
- Dose: 0.2-0.5mg/kg IV for post-op analgesia or shivering.
- Route of administration: IV, IM.
- Cardiovascular: often causes tachycardia due to structural similarity to atropine. Can also depress cardiac contractility.
- Pulmonary: causes respiratory depression, depression of hypoxic drive and increases apneic threshold.
- Neurologic: The active metabolite, normeperidine, can cause CNS stimulation, myoclonus and seizures. This risk is increased in renal failure patients.
- Other: uniquely effective among opioids at decreasing shivering via κ receptors. Also has weak local anesthetic properties. Contraindicated in patients taking MAO inhibitors because
combination can lead to serotonin toxicity, hyperthermia, and death.

VII. Local Anesthetics

Local anesthetics function by blocking sodium channels, preventing depolarization and action potentials. The non-ionized form must cross the lipophilic cell membrane and the ionized form must bind the channels on the inside of cellular membranes to achieve their action.

Local anesthetics are weak bases, have a pK\(_a\) above 7.4, and tend to be positively charged at physiologic pH. They are classified as either esters or amides based on the intermediate chain. You can differentiate between an ester or amide by knowing this simple rule: amide anesthetics all have an “I” in the beginning of their name (excluding the “I” in caine). Therefore, lidocaine is an amide, while chloroprocaine is an ester.

True allergies to local anesthetics are rare. Esters tend to be more allergenic because they are derivatives of PABA, which can be an allergen. Some amides are packaged with methylparaben, which is structurally similar to PABA and may also be allergenic. Amides are metabolized by the liver. Esters are metabolized by pseudocholinesterase, and therefore tend to have a shorter duration of action compared to the amide local anesthetics. Their activity is prolonged in patients with abnormal pseudocholinesterase; see the neuromuscular blocker section.

The pharmacokinetics of local anesthetics depend on the pK\(_a\) (the pH at which 50% of the drug molecules are ionized), the lipid solubility, the degree of protein binding, and the concentration of drug. A lower pK\(_a\) means that more drug exists in non-ionized form at physiologic pH and more molecules cross the plasma membrane, making onset faster. More lipid-soluble agents like bupivacaine tend to be more potent and more protein-bound, thus having a longer duration of action. Higher concentration of drug typically creates a “denser” and faster block. A typical example of this is 3% chloroprocaine. Chloroprocaine has a high pK\(_a\), which should confer a slower onset, but the amount administered results in a higher concentration and therefore, a quick onset.

Nerves are affected differently according to size and myelination; this is known as “differential blockade.” Smaller fibers and nonmyelinated fibers tend to be blocked earlier. The order of onset for any group of nerves is autonomic, pain, temperature, touch, proprioception, and motor. Level of block for a spinal or epidural tends to be 1-2 levels higher for the more sensitive nerves than the motor block. So, a T6 motor block may correspond with a T4 sensory block, and a T2 autonomic block.
Epinephrine is often used as an adjunct with local anesthetics. By causing local vasoconstriction, epinephrine decreases systemic absorption of the local anesthetic, prolonging duration and potentially decreasing toxicity. It should be noted that bupivacaine and ropivacaine are not affected, and their long duration of action is a result of high protein binding. Epinephrine can also warn of possible intravascular injection, signaled by tachycardia. It should be avoided in blocks of the distal extremity (e.g., digital blocks, ankle blocks) to avoid excessive vasoconstriction in end-arterial areas.

Acidic environments (e.g., local infection) antagonize block and slow its onset. Premixed solutions of local anesthetic containing epinephrine have a pH around 5, which is needed to maintain epinephrine stability. Adding epinephrine manually to local anesthetics rather than using premixed solutions may speed onset. Similarly, using small amounts of sodium bicarbonate to alkalinize the solution can greatly speed onset of certain local anesthetics. Bicarbonate is not used with bupivacaine since it precipitates above a pH of 6.8.

Toxicity to local anesthetics is a frequently tested topic. Typical reactions are CNS excitation (restlessness, agitation, perioral tingling, dizziness) and depression (drowsiness, slurred speech, unconsciousness) progressing to full-blown seizures. Cardiac complications are the most feared reaction, and include heart block, arrhythmias including ventricular tachycardia, ventricular fibrillation, and cardiac arrest. In general, the excitatory phenomena precede the depressive phenomena, which in turn precede cardiovascular toxicity. The exception to this is bupivacaine, the most toxic local anesthetic, which has a lower CNS-to-cardiovascular effect ratio, and can present with cardiac reactions as the first sign of intravascular injection. Cardiotoxicity from bupivacaine is notoriously difficult to resuscitate owing to its intense binding to cardiac tissue. Lipid emulsion infusion (Intralipid) or cardiopulmonary bypass may be needed; lipids seem to absorb the bupivacaine. The treatment of all types of local anesthetic systemic toxicity is supportive: reassure the patient, support airway and breathing, support circulation, inhibit seizure activity, and provide ACLS as necessary. Esters seem to be less toxic than amides due to their rapid breakdown in plasma.

The most commonly used local anesthetics, as well as those with specific, board-tested issues will be discussed below.

1. Benzocaine (Hurricaine)
   - Uses: topical anesthesia, usually used as a mucosal spray (e.g., airway anesthesia).
   - Duration of action: 1hr.
   - Other: Does not exist in a charged form, so it probably acts by an alternate mechanism; can cause methemoglobinemia via its metabolite O-toluidine, the treatment for which is methylene blue.
2. Procaine
- Uses: spinal, local, regional block.
- Duration of action: 30min-1hr; maximum safe dose: 12mg/kg (like chloroprocaine).
- Other: first synthetic local anesthetic.

3. 2-Chloroprocaine
- Uses: epidural, caudal, local, regional blocks.
- Duration of action: 30min-1hr; maximum safe dose: 12mg/kg.
- Other: when used epidurally, may decrease the efficacy and duration of action of bupivacaine, fentanyl, and morphine; associated with neurologic damage when used in intrathecal space, which may be due to an old preservative, sodium bisulfate.

4. Tetracaine
- Uses: spinal, topical (common for airway anesthesia by pulmonologists).
- Duration of action: 2-6hrs; maximum safe dose: 3mg/kg.
- Other: derivative of procaine; associated with cauda equina syndrome when given intrathecally.

5. Prilocaine
- Uses: dental procedures, topical (EMLA cream).
- Duration of action: 30min-1hr; maximum safe dose: 8mg/kg.
- Other: Can cause methemoglobinemia.

6. Lidocaine (Xylocaine)
- Uses: spinal, epidural, local, regional, airway topicalization, IV regional anesthesia (Bier block), antiarrhythmic, can be given via ETT.
- Duration of action: 1-2hrs (greater with epinephrine); maximum safe dose: 5mg/kg alone, 7mg/kg with epinephrine.
- Other: thought to be neurotoxic in high concentrations. As such, 5% lidocaine given through small-bore infusion catheters is associated with cauda equina syndrome and permanent neurologic damage. Associated with transient neurologic symptoms (TNS) when given intrathecally. TNS includes burning, pain, and aching of the lower extremities, lower back, and buttocks without motor symptoms or signs. It is associated with lidocaine > bupivacaine > tetracaine, the lithotomy position, outpatient surgery, and obesity. Onset of TNS is within 12-24 hours and symptoms typically resolve within 1 week.
- Lidocaine formulations are used for topical anesthesia to the airway for awake FOB intubation. 5% paste is useful for the oropharynx and hypopharynx, and atomized 4% solution is useful for the hypopharynx and trachea.

7. Mepivacaine (Polocaine)
- Uses: epidural, local, regional block.
- Duration of action: 1-2hrs; maximum safe dose: 5mg/kg, 7mg/kg with epinephrine.
- Other: often employed in regional blockade for quick onset. We typically dose our continuous peripheral nerve catheters with mepivacaine for surgical anesthesia, and switch postoperatively to a less dense, more sensory-specific local anesthetic such as ropivacaine for postoperative analgesia.

8. Bupivacaine (Marcaine)
- Uses: spinal, epidural, local, regional blocks.
- Duration of action: depends on dose and site. Locally, 2-4hrs, epidurally 2-4hrs, spinal 1.5-2.5hrs, peripheral nerve block 8-24hrs. Maximum safe dose: 3mg/kg.
- Other: highly potent; cardiotoxicity is cumulative as described above, unique in that it is very difficult to treat. Proven safety in spinal and epidural anesthesia.

9. Ropivacaine (Naropin)
- Uses: same as bupivacaine. Chemical analogue of bupivacaine with similar potency, onset, duration (more predictable), and toxic doses.
- Duration of action: as for bupivacaine; maximum safe dose: 3mg/kg.
- Other: Thought to be less cardiotoxic than bupivacaine. Tends to have preferential sensory blockade over motor, making it ideal for postoperative catheters and analgesia.

VIII. Herbal Medications

Herbal medication use is increasingly common in the general patient population. As a rule, herbal medications have not been thoroughly studied and their long-term effects on the human body are unknown. Further complicating matters is the fact that many of these medications are not regulated in any way with regards to dosing or even purported content of the medications themselves.

There is a small amount of data to suggest certain herbal medications can have unwanted effects in the perioperative period. The following is a brief list of common herbal medications, their effects, and perioperative recommendations. For more information, consult a more detailed reference.

<table>
<thead>
<tr>
<th>Herbal Agent</th>
<th>Purported Benefits</th>
<th>Effects</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valerian Root</td>
<td>Decreases anxiety</td>
<td>May decrease MAC via GABA</td>
<td>Taper weeks before surgery if possible</td>
</tr>
<tr>
<td>Echinacea</td>
<td>Stimulates immune system</td>
<td>Allergic reaction; Hepatotoxicity; Interferes with immunosuppressive therapy</td>
<td>d/c before surgery</td>
</tr>
<tr>
<td>Ephedra</td>
<td>Weight loss Boosts energy</td>
<td>Sympathetic stimulation similar to ephedrine; Increased HR, BP, and arrhythmias</td>
<td>d/c before surgery, avoid use with MAOIs</td>
</tr>
<tr>
<td>Garlic</td>
<td>Reduces BP and cholesterol</td>
<td>Irreversible inhibition of platelet aggregation</td>
<td>d/c 7 days before surgery</td>
</tr>
<tr>
<td>Ginkgo</td>
<td>Improves cognition and circulation</td>
<td>Inhibits PAF</td>
<td>d/c 2 days before surgery</td>
</tr>
<tr>
<td>Ginseng</td>
<td>Protects against stress</td>
<td>Hypoglycemia; Inhibition of platelets and clotting cascade</td>
<td>d/c 7 days before surgery</td>
</tr>
<tr>
<td>Kava</td>
<td>Decreases anxiety</td>
<td>May decrease MAC via GABA</td>
<td>d/c 24hrs before surgery</td>
</tr>
<tr>
<td>St. John’s Wort</td>
<td>Antidepressant</td>
<td>Inhibits reuptake of NE, dopamine, serotonin; Induces cytochrome P450 (increased drug metabolism)</td>
<td>d/c 5 days before surgery</td>
</tr>
</tbody>
</table>

The “G” herbal medications (garlic, ginseng, ginkgo) all inhibit the clotting cascade and/or platelet aggregation. Despite this, the American Society of Regional Anesthesia feels herbal medications do not pose an increased risk of bleeding in the setting of neuraxial blockade.

Chapter 2D. Neuromuscular Blockade

Neuromuscular blockers, or paralytics, are commonly used in anesthesia. Besides preventing movement and facilitating intubation, paralytics can often provide optimal operating conditions for the surgeon. That being said, neuromuscular blockade must be carefully monitored, and the thoughtless use of NMBs can be problematic for anesthesiologists and patients alike. Never forget that NMBs are not anesthetics; they provide no analgesia, amnesia, or hypnosis.
The following is a brief description of the usage, reversal, and monitoring of neuromuscular blockade. Pearls of wisdom concerning NMBs will also be covered. For specific details of each drug, see the section on anesthetic drugs.

**The Neuromuscular Junction**

The neuromuscular junction is composed of the terminal end of a motor neuron and the muscle cell separated by the synaptic cleft. When an action potential depolarizes the terminal end of the neuron, ACh is released, which diffuses across the synaptic cleft. This ACh binds to receptors on the muscle (motor endplate), causing depolarization (endplate potentials). When enough endplate potentials are generated, the whole membrane will depolarize via opening of sodium channels, which subsequently release calcium from the sarcoplasmic reticulum, causing muscle contraction. Termination of the action potential is caused by rapid hydrolysis of ACh by acetylcholinesterase.

**Depolarizing vs. Nondepolarizing Blockade**

Depolarizing NMBs (succinylcholine) very closely resemble ACh and therefore readily bind to ACh receptors and cause conformational changes in the ACh receptor, bringing about ion channel opening and ultimately, prolonged depolarization of the muscle endplate. Thus, succinylcholine is an ACh receptor agonist. By contrast, nondepolarizing NMBs are competitive antagonists at the ACh receptor, binding it but not inducing the conformational changes necessary for ion channel opening.

Succinylcholine’s offset is dependent on diffusion away from the neuromuscular junction and subsequent hydrolysis by pseudocholinesterase, also known as plasma cholinesterase. In contrast, nondepolarizing NMBs must be metabolized and eliminated. They can be outcompeted by additional amounts of ACh, as occurs when we clinically reverse neuromuscular blockade.

**Succinylcholine**

No other NMB is as rapid in onset (within 30 seconds) and offset (usually 5-10min) as succinylcholine. The classic “short” blockade caused by succinylcholine is termed a phase I block. After repeated administration, a phase II block may occur; this resembles the block of nondepolarizing NMBs in duration and response to nerve stimulation; see below.

People who possess abnormal genes for pseudocholinesterase may exhibit a prolonged block from succinylcholine. **This is a frequently tested topic on the boards.** Heterozygotes (one abnormal, one normal gene; 1:50 people) may experience blockade up to 30 minutes. Homozygotes (1:2500 people) may have a profoundly long blockade, on the order of 8 hours. The “dibucaine number” is proportional to the level of normal pseudocholinesterase activity. Normal pseudocholinesterase is 80% inhibited by dibucaine (a local anesthetic), while abnormal pseudocholinesterase is only 20% inhibited. Thus, a normal dibucaine number is 80, while a homozygote for atypical pseudocholinesterase would have a dibucaine number of 20. Heterozygotes fall in the 40-60 range. Beware using succinylcholine in patients with known or family history of atypical pseudocholinesterase, and keep in mind that patients experiencing a prolonged block will need mechanical ventilation and sedation until muscle function returns to normal.

Cholinesterase inhibitors prolong succinylcholine blockade by inhibiting pseudocholinesterase and by providing more ACh at the neuromuscular junction, thereby intensifying depolarization. Other drugs which inhibit pseudocholinesterase include pancuronium, esmolol, metoclopramide, cyclophosphamide,
phenelzine, and organophosphates.

Lithium and magnesium both prolong the onset and duration of succinylcholine. Similarly, quinidine, calcium channel blockers, and certain antibiotics ("mycins" other than erythromycin, aminoglycosides) can prolong blockade. Small doses of nondepolarizing NMB tend to antagonize succinylcholine blockade, i.e., they prevent depolarization.

Succinylcholine is relatively contraindicated for routine use in young children because these patients may have undiagnosed myopathies. It is also contraindicated in patients with a preexisting condition associated with succinylcholine-induced hyperkalemia (examples include, but not limited to, old burns, spinal cord injury, myopathies, etc.) and in patients with a history of malignant hyperthermia. For more info, see the drug section.

**Nondepolarizing Neuromuscular Blockers**

Two classes of nondepolarizing NMBs exist: benzylisoquinolones and steroids. All function via the same mechanism, that being competitive antagonism at the ACh receptor. As such, they can be "outcompeted" by ACh, which is the mechanism of action of NMB reversal agents. However, for reversal of neuromuscular blockade to be effective, some recovery from the NMB must already be present. Recovery depends on metabolism and elimination of the NMB in question.

Typically, 1-2x the ED$_{95}$ dose of a NMB is used for an intubating dose, while 0.1x the ED$_{95}$ dose is used for maintenance relaxation. Higher doses may afford slightly quicker onset of blockade but can also greatly prolong the block.

Certain NMBs, particularly some benzylisoquinolones such as mivacurium and atracurium, can cause histamine release. Histamine release can manifest itself as flushing, bronchospasm, and hypotension. Pretreatment with antihistamines and giving the drug slowly seem to attenuate these effects.

Hypothermia prolongs blockade by decreasing activity of the enzymes that metabolize the drug. Similarly, hypoventilation and respiratory acidosis prolong blockade. Electrolyte imbalances such as hypocalcemia, hypokalemia, or hypermagnesemia will result in abnormally long paralysis. Obviously, hepatic or renal disease or dysfunction can also prolong blockade depending on the particular route of metabolism and excretion of the NMB in question. Other drugs that can prolong nondepolarizing blockade are the same antibiotics that can prolong succinylcholine: ("mycins" other than erythromycin and aminoglycosides), quinidine and calcium channel blockers, dantrolene, and inhalational anesthetics. Drugs that induce enzyme metabolism, such as antiepileptics, can greatly shorten the duration of NMB metabolized by the liver (vecuronium and pancuronium). Mivacurium and cisatracurium are not affected due to their liver enzyme-independent metabolism.

Different muscle groups are more sensitive to neuromuscular blockade than others. The orbicularis oculi, diaphragm, and laryngeal muscles all relatively more resistant than the adductor pollicis or the muscles innervated by the posterior tibial nerve. However, these resistant muscle groups are also highly perfused, and a relatively larger proportion of a bolus of NMB is delivered to these muscles. This explains the phenomenon that the pharyngeal and laryngeal muscles are the first to be blocked but also the first to recover, whereas the less-resistant peripheral muscles are blocked more slowly but recover more slowly. In general, the orbicularis oculi corresponds best with the level of paralysis of the diaphragm and larynx, the two muscle groups we are often most concerned about.
Certain disease states and the changes in response to NMB are often tested on the boards. Myasthenia gravis patients are ultrasensitive to nondepolarizing NMBs, but are often resistant to succinylcholine due to their fewer ACh receptors. (MG patients may have profound weakness with volatile anesthetics alone). Use of either type of NMBs is unpredictable and must be monitored closely. Burn and chronic denervation injury patients have increased extrajunctional ACh receptors, making them resistant to nondepolarizing blockade.

Monitoring Neuromuscular Blockade with Peripheral Nerve Stimulation

Any patient who is given a NMB should have the state of that blockade monitored. The most common way we do this is with a peripheral nerve stimulator. Briefly, the nerve stimulator leads are placed over a peripheral nerve, which, when stimulated, elicits contraction of a muscle group. The three most commonly monitored nerves are the ulnar nerve, the facial nerve, and the posterior tibial nerve. As previously discussed, the facial nerve most closely approximates the diaphragm and laryngeal muscles.

As neuromuscular blockade ensues, the response to peripheral nerve stimulation exhibits a characteristic pattern depending on the agent used. Three types of stimulation are discussed here: train-of-four, single twitch, and tetany. A “twitch” is a single pulse of 0.2ms in duration. “Train-of-four” is a series of four twitches delivered at a frequency of 2Hz (2 twitches per second). “Tetany” delivers a sustained stimulus of 50-100Hz usually lasting 5 seconds. The “twitch height” is a quantitative measure of the level of muscle response to stimulation.

As nondepolarizing muscle blockade is intensified, each successive twitch in a train-of-four will show “fade,” a gradual decrease in the height of each successive twitch. As blockade increases, twitches fade altogether from last to first. Disappearance of the 4th twitch corresponds to ~75% blockade while the disappearance of the 2nd corresponds to a 90% blockade. Thus, if a patient has one twitch present during a train-of-four stimulation, 90% of ACh receptors at the neuromuscular junction are blocked. Fade will also occur during tetanic stimulation in the setting of residual NMB.

- Train-of-four response to nondepolarizing blockade.
- Arrow (↑) represents NMB administration.
- Note the progressive fade, with eventual loss of twitch number 4.

After a tetanic stimulus is applied, a subsequent train-of-four will show supramaximal response. This phenomenon is known as “post-tetanic facilitation.” Facilitation can even be seen when a patient has no response to train-of-four (“no twitches”) and no response to tetany. If a tetanic stimulation is applied and then a train-of-four is checked, it may be possible to see twitches due to this phenomenon. Post-tetanic facilitation is thought to be due to briefly increased levels of ACh in the neuromuscular junction due to repeated stimulation, thereby allowing out-competition of the neuromuscular blocker. The
junction is “flooded” with ACh by the tetanic stimulus, but the fade remains.

Clinically, five seconds of sustained tetany (no fade), the ability of a patient to be able to lift their head up for 5 seconds, or a strong bite on a bite block or tongue depressor correlates with at least 50% of receptors being unblocked. These are the best clinical tests we have to assess recovery from neuromuscular blockade, and represent a train-of-four ratio of no higher than 0.8-0.9, which most would consider a bare minimum threshold for recovery. Inadequate recovery from NMBs poses various risks, including hypoventilation and psychological distress in the case of an obviously-weak patient, and microaspiration in the case of a subclinically-weak patient.

Reversal of neuromuscular blockade is guided by the response to peripheral nerve stimulation. **Reversal should not proceed until at least one twitch is present.** This is because without twitches, the level of neuromuscular blockade present is unknown, and the response to a reversal dose will be unpredictable. The patient could be 5 minutes or 60 minutes from recovery of the blockade, but all we can see clinically is that the patient has no twitches. Reversal at this stage will provide some recovery, but will either result in a patient with incomplete recovery, or worse, a patient who briefly regains strength but becomes weak or paralyzed an hour later due to excessive amounts of paralytic. In general, it is a good idea to keep patients no more paralyzed than “one twitch” so that they are always reversible, should the case end unexpectedly. As in all of anesthesia, the goal is to carefully titrate medications and to give only what is necessary. All of us have been burned at some stage in our careers by giving too much muscle relaxant, only to have the surgery end five minutes later and be stuck with a completely paralyzed, non-reversible patient. The lesson here is: if you have no twitches and the case is over, **wait.** Don’t reverse the patient. Even though it is embarrassing and time consuming, it is the safest way to proceed. The patient should not be put at risk for your impatience.

As previously mentioned, the best evidence of recovery from neuromuscular blockade is 5 seconds of sustained tetany, 5 seconds of sustained head lift, or a strong bite on a bite block. **Return of train-of-four without fade is necessary but not adequate,** because humans are insensitive detectors of fade, with up to 70% of receptors still blocked in this case. Anesthesiologists do not have a better test in clinical practice, so great care must be taken when assessing recovery from neuromuscular blockade.

Blockade with succinylcholine exhibits a different response to stimulation than nondepolarizers. Twitch height during train-of-four and tetany is equally decreased at all stages. **There is no fade and no post-tetanic facilitation.** However, be aware that a phase II block of succinylcholine will respond like a nondepolarizer to stimuli.

- Train-of-four response to depolarizing blockade.
- Arrow (↑) represents depolarizing NMB administration.
- Note the lack of fade, although twitch height is uniformly decreased.
Myasthenia Gravis

This disorder is due to autoimmune destruction of postsynaptic ACh receptors, resulting in skeletal muscle weakness and easy fatigability. Antibodies (IgG) against nicotinic ACh receptors are found in 85-90% of patients with MG. Muscle strength characteristically improves with rest but rapidly deteriorates with exertion. The ocular, laryngeal, and pharyngeal muscles (bulbar muscles) can all be involved, as well as respiratory and proximal skeletal muscles. 10-15% of patients with MG develop a thymoma and 65% have thymic hyperplasia. It is more common in women than men; female incidence is highest during their third decade, while male patients typically present in their sixth or seventh decade.

Anticholinesterase drugs are the usual treatment and work by increasing available ACh in the neuromuscular junction. Pyridostigmine is the most commonly used anticholinesterase. Other treatments for more advanced disease include plasmapheresis, IV immunoglobulin infusions, and steroids. Excision of the thymoma or the hyperplastic thymus greatly alleviates symptoms and is often curative.

Anesthetic considerations include the propensity for these patients to develop postoperative respiratory dysfunction, aspiration (due to weakness of bulbar muscles), and sensitivity to neuromuscular blockade and the relaxing effects of volatile anesthetics. Predictors of the need for postoperative mechanical ventilation include disease duration > 6 years, concomitant pulmonary disease, a vital capacity < 4ml/kg, and pyridostigmine dose > 750mg daily.

The preoperative goals should be to optimize medical therapy. Preoperative respiratory or bulbar weakness should be treated with IV immunoglobulin or plasmapheresis. A classic boards topic is differentiation of myasthenic crisis (acutely decompensated MG) from cholinergic crisis (due to excess anticholinesterase). Patients in cholinergic crises are also weak, but exhibit signs of muscarinic excess such as salivation, lacrimation, miosis, bradycardia, and diarrhea. Edrophonium is short-acting and can be used to differentiate cholinergic crisis from myasthenic crisis. Following a dose of edrophonium, worsening of the symptoms implies cholinergic crisis, while improvement suggests issues arising from myasthenia.

For patients with MG, the response to succinylcholine is very unpredictable. Patients can have prolonged or shortened effects, or a phase II block. Sensitivity to nondepolarizers is profound. These patients need to be closely monitored and reduced doses of NMBs should be used or even entirely avoided.

Lambert-Eaton Myasthenic Syndrome

This disease involves autoimmune antibodies to presynaptic calcium receptors, reducing ACh release from the motor endplate. It is characterized by proximal muscle weakness that typically begins in the lower extremities, but may spread to involve upper limb, bulbar, and respiratory muscles. It is associated with paraneoplastic syndromes, classically small cell cancer of the lung. In contrast to MG, muscle weakness improves with repeated effort. Immunosuppression or plasmapharesis helps improve symptoms to a certain degree, but anticholinesterases have less dramatic effects when compared to treating patients with MG. These patients are very sensitive to both succinylcholine and nondepolarizing NMBs.

Final Thoughts
There is a very common pattern to the use of NMBs as one progresses through training. At first, residents tend to use too much NMB, either because of inexperience with the length of surgery or a fear of the patient moving and upsetting the surgeon. This results in “slow wakeups,” over-paralyzed patients who cannot be reversed, or those who become weak and need reintubation in the PACU. Later, a resident may greatly curtail the use of NMBs, tailoring his anesthetic to a quick wakeup and not worrying about surgical concerns. As experience with NMBs grows, we learn the right times and the right doses to give to patients.

Surgeons often ask for paralysis but sometimes do not understand why they need it or if the patient is paralyzed at all. All of us have had surgeons ask for “more relaxation” when the patient is already maximally blocked with no twitches. Often, the request for more relaxation represents inadequacy of surgical technique. That being said, the challenge for us becomes how to accommodate the surgeon and maintain a good working relationship, while at the same time being responsible and safe for the patient. If the surgeon asks for more relaxation and you know that it is a) not necessary because the patient is already paralyzed, or b) that they will end soon, use your best judgment. Remember, you can always call your attending with questions.

With the above in mind, there are certain cases where NMB is critical. These include craniotomies, most abdominal surgeries, cardiothoracic surgery, or any procedure where patient movement could be catastrophic. A quick way to check is to ask yourself: “If my patient coughed or moved right now, would it be harmless (but annoying) or would it be potentially catastrophic?” Interestingly, the better surgeons seem to be the ones who least often ask for paralysis, whereas surgeons who are constantly struggling with exposure or the size of their incision consistently ask for “more relaxation.” With time, you will learn which are which.

Troublesome spots for us include cases where intense paralysis is desired but the case itself is short, e.g., direct laryngoscopies by the head & neck surgeons. Here, the challenge becomes providing intense blockade (recall that the laryngeal muscles are some of the last to become blocked) in cases that are typically quick. Succinylcholine, with small repeat doses, can often be handy here, but you must watch out for phase II block and bradycardia.

Lastly, be aware when a case might end very quickly and unexpectedly. A typical example of this is an exploratory laparotomy to evaluate a malignancy. Sometimes, the surgeons will open the belly only to find an inoperable tumor or conditions which otherwise preclude surgery. In these cases, they will simply close the patient up and the case is over. This is the so-called “peek and shriek.” Be aware of this possibility. If you stick to the general rule of giving enough drug to get the job done, but no more, things should turn out just fine.
Chapter 3. Anesthesia for Specific Surgeries

The chapters, and the chapters that follow, are not intended to provide an encyclopedic index of anesthetic management for every type of surgery. Rather, the surgeries included are those that are common at UCSD, anesthetically interesting, or frequently tested on board exams.

Chapter 3A. Anesthesia for General Surgery

General surgery encompasses many different types of cases, each with its own anesthetic considerations. Examples include thyroid and parathyroid surgery, soft tissue surgery on the trunk and back, and the gamut of hernia repairs. Intraabdominal surgery is the “classic” general surgery case. Fittingly, the prevailing anesthetic option for general surgery tends to be general anesthesia, although at times other options may exist.

Chapter 3A-1. Anesthesia for Neck, Trunk, and Breast Surgery

Thyroid and Parathyroid Surgery


The usual indications for either type of surgery are neoplasms or hypersecretory glands. For elective thyroid surgery, the patient’s thyroid state must be normalized preoperatively so that the patient is clinically euthyroid; this is typically done by the surgeon or the patient’s endocrinologist. Surgery is withheld until the patient is medically stabilized; there is no such thing as emergency thyroid surgery. A patient in a hyper- or hypothyroid state is managed with methimazole or levothyroxine, respectively.

General surgeons usually perform thyroid and parathyroid surgeries without turning the OR table 180°, in contrast to head & neck surgeons. Because the procedure takes place close to the airway, ensure that the airway is secure. The operation carries the risk of damage to surrounding structures, including laryngeal nerves. Damage to these nerves can manifest themselves in a variety of ways, including postoperative hoarseness and complete vocal cord immobility.

The surgeon will almost always ask for a NIM tube to be placed. This tube has a short monitoring strip, approximately 3cm long and a few centimeters above the cuff, that is capable of detecting vocal cord spasm in the event that the surgeon dissects near or stimulates the recurrent laryngeal nerve. It is very important that the strip be placed precisely at the vocal cords; excellent laryngoscopy conditions or even a Glidescope are needed to confirm this. The tube has several leads which are connected to a special monitor, which is set up by the surgeon.

Because this monitoring technique depends on vocal cord movement, neuromuscular blockade is forbidden during the surgery. Typically, succinylcholine is used for laryngoscopy, but paralytics can be avoided altogether (propofol 2-3mg/kg with alfentanil 30-40mcg/kg or remifentanil 1-3mcg/kg), or a very low dose of nondepolarizer can be used.

The NIM tube is different from a standard ETT in several other ways. It is quite thick, such that the outside diameter of a given size corresponds to that of a standard ETT one size larger; e.g., a 6.0 NIM is as large as a 7.0 standard. The adapter that connects to the circuit is NOT removable, so this tube cannot
pass through a LMA. The NIM tube is also quite “floppy” and requires a stylet for placement.

All neck surgeries carry a risk of postoperative hematoma formation, which can potentially lead to airway compromise. In this event, the airway must be supported while the surgeons are notified.

During parathyroid surgery, we are routinely asked to draw blood to check parathyroid hormone levels prior to excision and at least once after the glands are removed. An IV that draws back reliably is needed, which may necessitate a 2nd IV; phlebotomy might suffice. Removal of all hypersecretory glands reliably causes a reduction in PTH levels within 10 minutes, while sustained high levels of PTH will prompt the surgeon to explore further.

**Superficial Surgery on the Trunk and Back**

These are usually for lipoma excisions or similar soft tissue masses. Anal exams under anesthesia fistulotomies are also included. The surgeries are typically short.

**Technique:** general or local/MAC. Hyperbaric neuraxial techniques ("saddle block") work well for anal surgery. **Monitors:** standard. **IV access:** one IV. **Duration:** 30min-1hr. **EBL:** minimal. **Position:** supine, prone, or prone jackknife depending on operative site. **Special equipment:** prone mask or cushion if indicated. **Special considerations:** none.

**Esophageal Surgery**

These are rare procedures but can be quite long and quite challenging, combining the considerations of thoracic surgery, one-lung ventilation, and a large open abdominal operation. Refer to a textbook for additional information.

**Technique:** general ± thoracic epidural. **Monitors:** standard, plus arterial line. **IV access:** at least 1 large IV ± central line. **Duration:** 2-8hrs. **EBL:** 200-1000ml or more. **Position:** supine, or lateral decubitus, or thoracoabdominal (operative side slightly propped up with the arm airplaned across the body and spine extended). **Special equipment:** equipment for one-lung ventilation. **Special considerations:** Although this is a long and involved operation, blood loss is typically small. Proximity to large vessels makes significant bleeding a rare, but real, possibility.

**Breast Surgery**

These cases range from needle-localized biopsies, to simple lumpectomies, to bilateral mastectomies with abdominal flaps, to major reconstructions or breast reductions (usually in conjunction with plastic surgeons). The extent of the surgery dictates the duration and sequelae of the case.

**Technique:** general ± paravertebral block. Rarely, minor cases can be done under local/MAC. **Monitors:** standard, rarely an arterial line, ± urine output. **IV access:** one IV. **Duration:** anywhere from 30min for simple cases to 8hrs for major reconstruction. **EBL:** minimal to 500ml; most cases are on the low side. **Position:** supine. Many breast cases require the patient to be “sat up” at times to check alignment and symmetry of the breasts. **Special equipment:** none. **Special considerations:** as below.

Position changes may dictate the choice of airway device. For breast reductions or reconstructions, the patient is “sat up” to 45-60° at times, so an ETT may be indicated. Additionally, the surgeon may have specific requests about neuromuscular blockers: either to use them to provide tissue laxity or not to use
them as a form of nerve-monitoring technique for dissection near the axillary and long thoracic nerves.

Even for long breast cases, the blood loss is typically small, due to the relative avascularity of the tissues. If axillary lymph node dissection is planned, avoid placing IV lines or monitors on the side of the affected extremity. The surgeon may ask that you not use pressors like phenylephrine because of the thought that it may decrease blood flow to a flap.

After wound closure, there is usually an extensive bra-type dressing that is applied.

**Chapter 3A-2. Anesthesia for Intraabdominal Surgery**

There are three major categories of intraabdominal surgery: major open, minor open, and laparoscopic/robot-assisted.

**Major Open Surgery**

These types of surgeries are generally lengthy, with the potential for large fluid shifts and blood loss. Large abdominal incisions and exposed bowel cause extensive evaporative and heat losses for the patient. Examples of this type of surgery include:

- Pancreatic, gastric or esophageal resection, including Whipple procedure
- HIPEC
- Partial hepatectomy
- Major bowel resection
- Intraabdominal mass resection
- Major abdominal explorations or lymph node dissection
- Open gastric bypass
- Splenectomy

**Technique:** general ± epidural, depending on the incision location, extensiveness of the surgery, and potential for postoperative coagulopathy. **Monitors:** standard, urine output, usually arterial line. CVP can be helpful. **IV access:** at least two large IVs ± central line. **Duration:** 3-8hrs. **EBL:** 500ml-2L or more. **Position:** supine. **Special equipment:** fluid warmers, forced-air warming blankets. **Special considerations:** as below.

Insensible fluid losses from exposed bowel during these cases can be extensive. Historically, these losses have been replaced with crystalloid at a rate > 10ml/kg/hr. The overall trend is toward fluid restriction and the use of more colloids and fluid boluses; some advocate as little at 4ml/kg/hr as the “insensible” replacement rate. Note that this rate is just for insensible losses and does not begin to take into account volume lost from bleeding.

Patients undergoing bowel surgery usually have a “bowel prep” before surgery which can leave them significantly volume depleted or with electrolyte disturbances. Take this into consideration prior to induction.

Be aware of cases with the possibility of a “peek and shriek,” where the surgeons open the belly and find inoperable conditions, and quickly close. Don’t go overboard on NMBs at the beginning of the case.

Partial hepatectomy cases share the anesthetic considerations above, with the added potential for huge
and rapid blood loss. Central venous access is standard, not only for large-bore access but also for CVP monitoring. Surgeons typically ask us to minimize fluids, with the reasoning that a low CVP causes a low venous back-pressure to the portion of the liver in question and limits blood loss. Have nitroglycerin available for the rare possibility that the surgeon asks for prompt and dramatic CVP reduction to reduce bleeding.

HIPEC

HIPEC stands for Hyperthermic Intraperitoneal Chemotherapy. This procedure is relatively rare outside of UCSD, which is a leading referral center for this surgery. The procedure is used to treat advanced abdominal malignancies, usually peritoneal-based. The patients are typically age 30-50, previously healthy, and relatively free of comorbidities. The surgery involves a large midline abdominal incision, tumor debulking, and heated chemotherapy infusion. The initial stage is identification and resection of as much tumor and tumor-affected organs as possible (“cytoreduction”). Then, the surgeon places perfusion canulas and temporarily closes the abdomen. The chemotherapeutic solution (mitomycin C) is infused at > 40°C throughout the peritoneal cavity for up to 90 minutes. This is called “shake and bake” because the surgeon or assistant will gently shake the abdomen while the chemotherapy drug is infusing. The chemotherapy portion is intended to kill any remaining cancer cells once all visible disease is removed. The chemotherapy solution is then removed, and the incision is closed.

Technique: General with thoracic epidural. Monitors: Standard, arterial line, urine output, ± CVP. IV access: Two large bore IVs usually suffice; difficult access or expected blood loss may require a central line. Duration: 6-10hrs. EBL: 300-2000ml. Position: Supine. Special equipment: fluid warmers and forced-air warming blankets. Special considerations: derangements specific to HIPEC are discussed below.

During the pre-chemotherapy portion of the surgery, fluid warmers and warming blankets are left off and the patient is allowed to passively become hypothermic. This is due to the expected whole-body heating effect that will come later.

During the heated chemotherapy infusion, patients typically develop a sepsis-like hyperdynamic circulatory state that is characterized by a steady increase in oxygen consumption, etCO₂, heart rate, and cardiac output, with a decrease in SVR. Electrolyte abnormalities and fluid shifts become a challenge during the heated perfusion, so frequent blood gases should be monitored during this time. Fluid requirements, already high for this type of abdominal surgery, become profound; it is common for total “ins” to be on the order of 4-5L of crystalloid and 3-4L of colloid and blood products.

Mitomycin C is nephrotoxic. A steady urinary output is required to “flush” the agent and avoid renal injury, so close monitoring of urine output is key. The surgeon may ask for diuretics to improve urine flow if it is inadequate otherwise.

This surgery is exquisitely painful due to the large incision and peritoneal irritation that accompanies the chemotherapy. Thoracic epidural analgesia is the rule for these patients. However, balancing the dosing of the epidural for adequate coverage of the incision without causing hypotension (with fluid shifts ongoing) can be challenging. A good starting dose is 0.0625% bupivacaine at 10ml/hr with patient demand dose of 4ml q30min. The opioid is intentionally left out and the patients are given IV opioid PCAs.

Minor Open Surgery
There is quite a bit of overlap between minor and major abdominal surgery. A simple bowel resection or colectomy tends to be a minor affair, but depending on surgical skill and patient characteristics, it can quickly develop into a major, extensive case. There is still the potential for substantial insensible fluid losses. Examples of minor procedures include:

- Open cholecystectomy
- Small bowel resection
- Colectomy, sigmoidectomy
- Biopsy
- Hernia repair
- Appendectomy

**Technique:** general. Inguinal hernias may be repaired under a variety of techniques, including general, regional, neuraxial, and local/MAC. Patients with mid- and lower-abdominal hernia repairs may have a TAP (transversus abdominis plane) block done for post-op analgesia. **Monitors:** standard, rarely an arterial line. **IV access:** one large IV should suffice for the vast majority of cases. **Duration:** 1-4hrs. **EBL:** < 500ml. **Position:** supine. **Special equipment:** warmers. **Special considerations:** The same comments about keeping patients warm, replacing fluid losses and bowel prep for major procedures apply.

### Laparoscopic and Robotic-assisted Surgery

Many traditionally open procedures are now being performed laparoscopically. A variant of laparoscopic surgery is robotically-assisted surgery. Laparoscopic procedures often reduce the physiologic insult of the surgery as well as fluid shifts and blood loss. However, this is highly dependent on surgical skill, and the procedure can take a very long time.

Robot-assisted surgery is intended to combine the minimally-invasive nature of laparoscopic surgery with a device (robot) that translates the imprecise movements of the surgeon’s hands to smooth mechanical motions and allow instrument manipulations that are not possible with manually-operated instruments. It is absolutely key to remember that **the OR table cannot be moved while the robot is docked**, since the robotic “arms” will not move with the patient and can cause serious injury.

Currently, very few general surgery cases are done with robotic assistance. Examples of laparoscopic surgeries include:

- Cholecystectomy
- Hernia repair
- Appendectomy
- Colectomy, APR, LAR
- Gastric bypass, banding, or Nissen fundoplication

Management for urologic and gynecologic surgery using laparoscopy with or without robotic assistance are discussed in the pertinent section.

**Technique:** general. **Monitors:** standard, rarely an arterial line, urine output for longer procedures. **IV access:** one IV. **Duration:** 1-8hrs depending on the procedure. Use of a robot typically slows down a case. **EBL:** usually minimal. **Position:** supine. Significant Trendelenburg or reverse Trendelenburg may be needed, and may be needed for long durations. **Special equipment:** none. **Special considerations:** as
below.

Steep T-burg or reverse T-burg for long periods of time can have significant impacts on circulatory and respiratory physiology. In short, in T-burg, ventilation and oxygenation are impaired but venous return is high; in reverse T-burg, ventilation and oxygenation are improved but venous return is very low. There have been many cases where steep T-burg position for many hours has resulted in such severe facial edema that the patient had to be kept intubated at the end of the case. Additionally, some laparoscopic surgeries have to be aborted or converted to open due to patient intolerance of the needed position, with prohibitively high peak airway pressures or inadequate oxygenation in steep T-burg.

Insufflation of the abdomen with CO$_2$ creates unique issues for the anesthesiologist. In no particular order, they are: increased CO$_2$ load, decreased venous return, increased afterload, difficulty with ventilation, possibility of CO$_2$ embolism, subcutaneous deposition of CO$_2$, pneumothorax, and pneumomediastinum. Additionally, the anesthesiologist must be vigilant at the time of initial trocar placement, as life-threatening vascular injuries have occurred at that time. From the outset, most of us decrease tidal volumes and increase respiratory rate when the abdomen is insufflated to keep peak airway pressures low and “blow off” the excess CO$_2$. In general, minute ventilation will have to be increased by about 1/3 of baseline. Intravascular injection of CO$_2$ is usually benign but the surgeon should be notified. Typically you would see a sharp rise in etCO$_2$ on the capnograph beginning with insufflation. Pneumothorax or pneumomediastinum should be treated as needed.

Laparoscopic procedures always have the potential of being converted to an “open” procedure if circumstances dictate.

Patients presenting for gastric bypass or banding are, by definition, morbidly obese and with the usual assortment of physiologic derangements that obesity causes. These include the possibility of a difficult airway, full stomach, increased oxygen consumption, decreased FRC, restrictive-pattern lung disease, rapid desaturation during apnea, hypertension, difficult vascular access, and difficult positioning/padding. The surgeon may repeatedly perform upper endoscopy to place a Bougie or investigate suture lines; this creates a risk of ETT dislodgement for which you must be vigilant.

**Chapter 3B. Anesthesia for Urologic and Gynecologic Surgery**

Urologic, gynecologic, and urogynecologic surgery comprise a significant percentage of cases performed at UCSD. Although these are distinct surgical specialties, they will both be covered here due to many intraoperative similarities. While many of the cases are simple, outpatient surgeries, many more complex and invasive procedures are also performed. There is significant overlap between many of these cases and those done by general surgeons, such as laparoscopic/robotic procedures and major pelvic dissections, so for further information, see the general surgery section.

A common position for urologic and gynecologic surgery is the lithotomy position. In this position, the patient’s legs are raised and flexed at the hip and knee. The legs are either allowed to hang freely from soft straps or are placed in padded holders. **This position has the potential for many different peripheral nerve injuries.** Medial compression of the thigh and knee can injure the saphenous nerve, which clinically will present as anesthesia over the medial calf. Lateral compression of the lower leg can result in common peroneal nerve injury and foot drop. Hyperflexion of the thigh can also produce sciatic, femoral and obturator nerve injuries. Usually the surgeons are quite attentive to this, but we are also responsible to ensure that all areas are properly padded and supported.
The lithotomy position increases intraabdominal pressure, leading to reduction of FRC and poor pulmonary compliance. Many of these procedures also employ steep Trendelenburg position, which further increases these problems. In extreme cases, difficulty with ventilation and oxygenation may cause the case to be aborted. Venous return tends to be high.

The intraoperative use of indigo carmine is common in many of these procedures; methylene blue is used rarely. When given intravenously, these dyes are excreted in the urine. They allow the surgeon to “see” damage or holes in the ureter when the dye extravasates. As discussed in the monitoring section, indigo carmine has a vasopressor effect while creating a transient, mild artificial desaturation on SpO₂. Methylene blue has a milder vasopressor effect due to NO scavenging; the artificial desaturation it can cause is transient but may be as low as 60-70% on SpO₂.

The TURP syndrome is a common boards topic and something every anesthesiologist needs to be aware of. It classically is seen during a true TURP, where large volumes of hypotonic irrigating solutions can be absorbed through the prostate’s dorsal venous plexus. This syndrome can also happen in any other case when large amounts of irrigating solutions are used. Absorption of the irrigating fluid leads to hypoosmolality, hyponatremia, solute toxicity, and intravascular fluid overload. The amount of absorption is determined by the pressure (i.e., height) of the irrigation fluid, opening of vascular structures, and the length of the procedure. Absorption is reported to occur at ~20ml/min of irrigation. The problems described above manifest themselves as confusion, agitation, hypotension, and arrhythmias all the way to dyspnea, coma and death. Clearly, most of the signs of TURP syndrome are masked by general anesthesia, and for this reason many of us feel neuraxial blockade is safer any time it is a possibility.

Because polar electrolyte solutions such as NS disperse monopolar cautery and render it useless for TURP, other solutions containing glycine, sorbitol and mannitol are classically used. Each of these solutes can become toxic when significant amounts are absorbed. Glycine toxicity is associated with transient blindness, hyperammonemia, hypotension and neuroexcitatory phenomenon. Sorbitol solutions can lead to hyperglycemia, and mannitol can lead to volume overload. Pure water is generally avoided except in bladder procedures due its marked hypotonicity.

Recently, bipolar cautery for TURP has become more prevalent, which allows the use of NS as an irrigant, and thus avoids the problems of hypotonicity and solute toxicity. However, the risk of intravascular volume overload remains.

The treatment of TURP syndrome is supportive. Usually, cessation of irrigation, volume restriction, and diuresis are sufficient. Severe neurologic manifestations such as seizures or coma should be treated with hypertonic saline according to standard guidelines. Antiepileptic medication is useful for managing seizures, and the airway should be protected as needed.

Coagulopathy during a TURP procedure is another commonly tested board topic. It is thought to result from two etiologies: one is a dilutional coagulopathy from massive blood loss with crystalloid and irrigant in the intravascular space, and the other is systemic fibrinolysis due to prostate tissue release into the circulation.

Urologic and gynecologic procedures can be classified as minor, moderate, and major.

**Minor Surgery**
Examples of this include TURP, cystoscopy with TURBT, cystoscopy with ureteroscopy ± stenting, hysteroscopy, dilatation and curettage/evacuation, extracorporeal shock wave lithotripsy, and minor laparoscopic or diagnostic procedures.

**Technique**: general or neuraxial. **Monitors**: standard. **IV access**: one IV. **Duration**: 30min-2hrs. **EBL**: <100ml for all but D+C and D+E, which are still < 500ml. **Position**: lithotomy, supine, ± Trendelenburg. **Special equipment**: none. **Special considerations**: as below.

ESWL employs high frequency acoustic shocks which can damage or reset pacemakers; these minimally painful, short procedures can be done with an opioid-only spinal. Discuss with an attending on how to proceed. Many of these surgeries are short and outpatient procedures and the anesthetic should be tailored accordingly.

D+Es and less commonly D+Cs have the potential for fair amounts of bleeding due to the highly vascular nature of the pregnant uterus and placenta. The gynecologists will generally inform you if this happens, and additional interventions may become necessary. The gynecologists may sometimes ask for specific agents to aid in uterine contraction, e.g., oxytocin or methylergonovine. These agents and anesthetic implications for the parturient are discussed in the OB section.

**Moderate Surgery**

Examples include open hysterectomy/oophorectomy, vaginal vault reconstruction, surgery for incontinence or organ prolapse, and laparoscopic/robotic procedures such as laparoscopic prostatectomy and laparoscopic hysterectomy/oophorectomy.

**Technique**: general. **Monitors**: standard, arterial line for long cases, urine output. **IV access**: typically one IV will suffice. **Duration**: 2-8hrs. Robotic prostatectomies were notorious in the past for taking 8 hours or longer, but they are typically in the range of 4-5 hours now. **EBL**: 100-500ml, rarely more. **Position**: supine or lithotomy ± steep Trendelenburg. **Special equipment**: fluid warmers and forced-air warming blankets. **Special considerations**: as below.

IV dye such as indigo carmine or methylene blue may be requested; see above.

Urologic, gynecologic, and urogynecologic surgery with laparoscopy and/or robotic assistance are common procedures. These procedures necessitate attention to careful positioning and padding given the steep T-burg and rotational positions that are usually required. Since the arms are usually tucked securely at the sides, it is crucial to verify all lines and monitors function appropriately prior to prepping and draping. Difficulties with oxygenation and ventilation are not uncommon, and your ability to non-invasively investigate these problems is limited. Use of FOB to verify ETT placement or suction secretions is not unheard of. The long duration of the case, fluid shifts, lack of access to the BP cuff in the event of malfunction, plus the potential for intraabdominal vascular injury usually require an arterial line. Facial and/or laryngeal edema resulting from prolonged steep T-burg may preclude extubation. Lastly, it is absolutely key to remember that **the OR table cannot be moved while the robot is docked**, since the robotic “arms” will not move with the patient and can cause serious injury.

Many urologists will ask that the patient be “run dry” for the initial stages of a laparoscopic prostatectomy. The rationale behind this is not fully clear but likely involves their desire for minimal urine output and thus bladder distension during resection of the prostate. While it is true that blood and insensible losses in laparoscopic procedures tend to be small, remember that the patient still has...
maintenance fluid requirements. Objective data such as BP, ABGs and other trends are the best guide to therapy, not the surgeons’ random opinion. Furthermore, these “initial” stages may take anywhere from 2-6 hours depending on the surgeon. When in doubt, be political and defer to your attending, but keep in mind your duty is first and foremost to the patient.

**Major Procedures**

Examples include major pelvic dissection, nephrectomy, open prostatectomy, cystectomy, pelvic lymph node dissection, and typically any surgery for gynecologic or urologic cancer. Many of these are done (or just started) as laparoscopic cases.

**Technique:** general ± epidural. A paravertebral block also may be done for post-op analgesia for lateral abdominal wall incisions. **Monitors:** standard, arterial line, urine output, ± CVP. **IV access:** at least 2 large IVs; Cordis placement is common. **Duration:** 4-12hrs. **EBL:** 500ml-3L, possibly more. **Position:** any position other than prone may be used: supine, lithotomy, lateral, etc., often with Trendelenburg. **Special equipment:** fluid warmers, warming blankets. **Special considerations:** see below.

These cases should be treated like major abdominal cases as outlined in the general surgery section in regard to blood loss, fluid management, and physiologic consequences. Blood loss can be insidious and should be closely monitored. The potential for major vascular injury, diaphragmatic injury, hepatic injury, or pneumothorax is always present. Several patients have suffered IVC or aorta injuries, particularly during laparoscopic and open nephrectomy cases. Be vigilant, and prepare with monitors and IV access appropriately.

Renal tumors are often in close proximity to major vessels including the IVC and may have associated thrombus. Compression or retraction of the tumor and kidney can decrease venous return and cause hypotension. Associated thrombus may be just in the renal vein or extend all the way into the IVC or right atrium. In certain situations, cardiopulmonary bypass and a joint procedure with the cardiothoracic surgeons is indicated. Clearly, the anesthetic management of these cases is profoundly different. These cases are thankfully rare.

Open prostatectomy, now a rare procedure, is included as a “major” case given its association with large blood losses, classically on the order of 1-2L. Reduction of blood loss is one way in which robot-assisted laparoscopic prostatectomy has likely improved patient outcomes.

**Chapter 3C. Anesthesia for Orthopedic Surgery**

Orthopedic procedures comprise a significant percentage of cases at our three hospitals. The patient population is very broad, ranging from the moribund elderly patient with many medical problems coming for a fractured hip to a young athlete with a torn ACL. Typical scenarios for orthopedic cases range from scheduled, elective surgery to urgent or emergent repairs for open fractures. Orthopedic procedures make up a large part of “add-on” or late-night cases. The orthopedic surgeons are likewise heterogeneous in terms of personality and ability. With time and experience you will discover who is who, and what to expect intraoperatively. This guide will hopefully ease the learning process.

Regional anesthesia, or peripheral nerve blocks, will be a viable option for many orthopedic procedures on the extremities. You will gain tremendous exposure to regional techniques throughout your residency, especially so during your CA-2 and CA-3 regional rotations. The relevant and commonly-done blocks will be indicated in each section.
Many orthopedic procedures are “ambulatory” or same-day surgeries. The anesthetic technique for these patients can be challenging and is geared towards quick wakeups, quick room turnovers, and good control of pain and nausea. Any of these components could keep a patient in the PACU for an extended period of time. Be mindful of these short procedures and tailor the anesthetic to the patient’s needs and discharge goals.

Many orthopedic procedures involve use of a tourniquet. The tourniquet is intended to reduce blood flow, reduce blood loss, and improve surgical visualization of the field. You may occasionally encounter a surgeon who asks for controlled hypotension as well, with the idea that a normal or high BP can increase flow past the tourniquet and cause bleeding. As always, weigh the risks and benefits to the patient, be political, and consult with your attending.

Risks of tourniquet use include pain (increases with duration of use, especially with tourniquet times >1hr) and the “reperfusion syndrome” that occurs when the tourniquet is released, due to washout of metabolic waste products and relief of pain. These waste products typically lead to a transient rise in CO₂ levels, potassium, and lactic acid, with a drop in preload and core temperature. Tourniquet pain is also notoriously difficult to treat. Tourniquet use on the lower extremity is associated with a higher incidence of DVT. Lastly, the ischemia produced by a tourniquet makes it a relative contraindication in patients with sickle cell disease.

DVT is a feared and common complication of orthopedic procedures, particularly on the knee and hip. Prophylactic anticoagulation is often given, and compression stockings are the rule. Subcutaneous doses of heparin are not a contraindication to regional anesthesia; see the ASRA guidelines in the section on regional anesthesia. Regional anesthesia may lower the risk of DVT by improving regional blood flow via vasodilation and the reduction of inflammatory mediators. However, the routine use of postoperative anticoagulation can make the placement, use, and removal of an indwelling epidural catheter tricky.

Procedures on fractured long bones carry the risk of emboli, particularly fat emboli. This acute insult to the right side of the heart may manifest as a sudden drop in etCO₂, a drop in arterial blood pressure, and hypoxemia. Other classic signs include mental status changes and petechiae, which typically appear 1-3 days post-op. The surgeons must be notified if this occurs; treatment is supportive.

Joint replacements involve the use of methylmethacrylate to as “bone cement” to hold the artificial joint in place. This cement can cause a variety of intraoperative problems, including hypotension, arrhythmias, increased pulmonary vascular resistance, hypoxia, and debris/fat embolization (the cement itself expands within bone). The cement itself liberates heat via an exothermic reaction, which can burn tissues. Treatment is supportive. The odor of the cement is distinctive; you will know when the surgeons are applying it.

Chapter 3C-1. Anesthesia for Spine Surgery

Spine surgeries can be broadly placed into one of two categories: those with significant blood loss, and those without. The anesthetic management is largely governed by the expected blood loss or fluid shifts. Spine surgeries with the potential for significant blood loss include:

- Instrumented fusion (anterior or posterior), especially multilevel
- Spinal tumor resection
- A combined anterior and posterior surgery
- Corpectomy and fusion, especially multilevel
- “Redo” surgery

Spine procedures with typically little blood loss include:

- Discectomy
- Laminectomy (without instrumentation/fusion)
- Single-level surgery
- Cervical spine surgery
- Isolated “minimally invasive” spine surgery like XLIF or TLIF

Patients coming for spine surgery, particularly those having “redo” or major staged operations, frequently have severe opioid-dependent pain and may be on staggering doses of narcotics. Many of these patients will have been to our preoperative pain clinic. At UCSD, it is common to use a low-dose intraoperative ketamine infusion (around 0.3-0.6mg/kg/h) which the literature supports as a way to reduce post-op opioid requirements in these patients.

**The Prone Position**

A consideration of virtually every spine procedure is the necessity of placing the patient in the prone position. This position demands special padding to protect the eyes, face, chest, breasts, arms, genitals, and feet. The surgeons will attempt to properly pad and position the patient, but it is also our responsibility to the patient that these things are done correctly. Padding of the face and protection of the eyes, ears, and nose are primarily our responsibility. There is a very small but finite incidence of postoperative visual loss following prone surgery. There are various causes including retinal artery or vein occlusion and the much-feared posterior ischemic optic neuropathy (PION). The exact cause of PION is unknown but is associated with long prone surgeries, blood loss, anemia, and hypotension. The retinal vessel-related injuries are more associated with direct pressure on the eyes. At UCSD, most practitioners use the “Prone View” head support which has a headrest, a foam cushion with cutouts for the nose and eyes, and a mirrored base, to allow direct observation of the face. As a general rule, for long surgeries in the prone position, we try to keep the patient’s BP within 20% of their baseline, the hematocrit above 30%, the slightly above the level of the heart, and the saturation as high as possible. Many cases that would otherwise be very lengthy are done in multiple “stages” across several days. An arterial line is very useful in prone spine cases and is almost always indicated.

The prone position has implications for airway management and management of all our monitors and lines. The airway must be carefully secured prior to turning to the prone position; if it is lost in this position, it would be next to impossible to resecure it. One cardinal rule of prone cases is that the patient’s gurney must be readily available after the patient is turned prone, in case of an emergent need to flip them supine again (e.g., for reintubation). Similarly, every one of our monitors and IVs has the potential to snag, kink or otherwise stop working after flipping the patient to the prone position. Extra efforts must be made to ensure everything is secure, for the same reasons that the airway must be secure.

One approach many of us take prior to flipping a patient prone is to disconnect every extraneous line and monitor and then reconnect them after the flip. This minimizes the chance for tangle and snags and allows the anesthesiologist to focus on the airway and proper placement of the eyes/head. The patient is almost always stable enough to go unmonitored for 30 seconds. A typical sequence for this would be:
- Induce anesthesia with the patient supine on their gurney.
- Disconnect and cap off all but one IV line.
- Ventilate with 100% O₂.
- When all team members are ready, disconnect the BP cuff, EKG leads, ± arterial line, and hang up neatly for immediate reconnection.
- Disconnect pulse oximeter and breathing circuit.
- Flip patient.
- Reconnect circuit and confirm ventilation/etCO₂ while verifying face and eyes are free.
- Reconnect pulse oximeter, then all other monitors and IV lines.

Spine Surgery with Significant Blood Loss

**Technique:** general. **Monitors:** standard, arterial line, urine output ± CVP. **IV access:** At minimum, 2 large IVs. Often, 1 large IV plus a Cordis. **Duration:** anywhere from 1-16hrs, depending on the surgeon and the case. **EBL:** several hundred ml to many liters. **Position:** usually prone; supine for anterior cases. **Special equipment:** prone headrest, fluid warmers, forced-air warming blankets. One-lung ventilation may be needed for anterior thoracic spine procedures. **Special considerations:** as below.

Blood loss and resuscitation is the key factor here. Stay on top of blood loss, and give fluids and/or pressors liberally to maintain normal blood pressures. Invasive monitoring, urine output and frequent ABGs can help guide therapy. Our surgeons typically use blood scavenging (“cell-saver”) to reduce need for transfusion. Pay attention to all the potential negative sequelae of massive transfusion, which includes coagulopathy, hypothermia, immune reactions, and electrolyte disturbances.

A surgeon might request an intraoperative “wakeup test,” which is exactly what it sounds like, and is thankfully rare. This plan would be made known to you far in advance, because it requires specific planning to allow timely and smooth patient emergence in the prone position. Monitoring of evoked potentials has supplanted this technique to some degree.

More commonly, the surgeons will employ neurophysiologic monitoring (evoked potentials) to monitor for impending neurologic damage. The choice of evoked potentials (sensory, motor or both) influences the type and amount of anesthetic agent we can use. For example, motor evoked potentials are a contraindication to neuromuscular blockers and a relative contraindication to higher levels of volatile anesthetics, whereas sensory evoked potentials are a relative contraindication to nitrous oxide. Typically, the neurophysiology team, a neurologist and a physiologist, will consult with us beforehand and let us know which of our anesthetics they would like us to avoid. For more information, see the neurophysiology section.

Some spine procedures will have an anterior component. The approach to the anterior thoracic and lumbar spine is generally done by a non-orthopedic surgical team such as trauma surgery. If the procedure has both anterior and posterior elements during the same surgical session, there is the potential for multiple positioning “flips” during the case. As such, it is doubly important to make sure all of our equipment is secure as outlined above. Thoracic anterior spine procedures are a relative indication for one-lung ventilation, which allows the surgeons to have excellent access to the spine. The techniques of one-lung ventilation will be discussed later. Anticipate if there will be a need for one-lung ventilation and choose an appropriate ETT at the start of the case.

Spine Surgery without Significant Blood Loss

In general, there is a low possibility of significant bleeding with these cases and thus, most of the requirements for extensive spine surgeries do not exist. An arterial line may still be prudent depending on the duration of the case and patient comorbidities because the same risk of blindness exists.

Anterior cervical procedures are done supine. Additionally, cervical procedures may be done for impending or preexisting neurologic damage. These cases might involve evoked potentials and the same considerations as above apply. Securing the airway in a patient with an unstable or compromised cervical spine can present challenges. Awake intubation techniques to document stability of neurologic function prior to induction of general anesthesia may be indicated.

Newer “minimally invasive” spinal fusions such as XLIF and TLIF are gaining popularity. Many of these involve multiple smaller incisions and complicated retractor systems, and are done with the patient in a lateral position. They may utilize a modified EMG-sensing instrument that the surgeon uses to detect nerve roots; if so, neuromuscular blockers are contraindicated. Sometimes these low-blood-loss procedures are used as one component of a multi-level fusion case that overall does have the potential for major blood loss; be aware.

Chapter 3C.2. Anesthesia for Lower Extremity Orthopedic Surgery

As previously stated, most lower extremity procedures can be carried out under regional or neuraxial anesthesia as alternatives to general. Notable exceptions include when there is long bone fracture and the possibility of compartment syndrome (e.g., tibial fractures). In these cases we do not place blocks to avoid compromising the surgeons’ ability to monitor for compartment syndrome. We routinely place peripheral nerve blocks in certain types of elective, scheduled cases for post-op pain control and to aid in the rehabilitation process. In these cases, the regional anesthesia team almost always does the block.

Hip Surgery

Technique: general, neuraxial, or both. The “routine” for elective total hip arthroplasty is general with ETT and pre-op epidural, which is then removed early on post-op day 1. Some hip cases can be done under spinal, epidural, or CSE, depending on the duration of surgery, patient factors, and surgeon factors. Monitors: standard ± arterial line, urine output. IV access: one large IV. Duration: 2-3hrs for first-time operations, potentially double for redo or complicated hip cases. EBL: usually < 1000ml, although significant blood loss can go unnoticed within the joint and surrounding structures. Redo operations can involve much more blood loss. Position: lateral or supine. Special equipment: none. Special considerations: as below.

Hip fractures make movement painful for the patient and may preclude easy positioning prior to induction of anesthesia. It may be easier to induce general anesthesia on the patient’s stretcher prior to moving the patient to the OR table. Alternatively, if a neuraxial block is to be used, the block can be placed with the patient already in the lateral position on their stretcher, and then moved after analgesia has occurred.

There are a wide variety of hip surgeries, ranging from the purely elective total hip arthroplasty, to various procedures for fractures, including percutaneous pinning, intramedullary nailing,
hemiarthroplasty, or total arthroplasty. The blood loss usually varies with the invasiveness of the procedure as well as the status of the bones involved: intact vs. completely shattered, younger patient with OA vs. elderly patient with osteoporosis.

**Total Knee Arthroplasty**

**Technique:** general or neuraxial. The “routine” for elective TKA is GETA with femoral nerve block/catheter ± a single-shot sciatic block. **Monitors:** standard, urine output. **IV access:** one large IV. **Duration:** 3-4hrs, longer for redo or repeat procedures. **EBL:** < 200ml; a thigh tourniquet is almost always employed. **Position:** supine. **Special equipment:** none. **Special considerations:** Risks of reaction to cement, use of a tourniquet, and risk of DVT. The surgeons mobilize the leg and make a lot of noise during the surgery, which may bother an awake or lightly-sedated patient. Neuraxial can be done per patient request or patient need. Post-op pain is very significant. The above peripheral nerve blocks are almost always done, and can significantly lower intraoperative anesthetic requirements.

**Other Lower Extremity Surgery**

Examples include knee arthroscopy, fracture repair (ORIF), amputation, and all types of foot surgery.

Many of these surgeries have the potential to be outpatient procedures and the anesthetic technique should be tailored accordingly. For example, we avoid peripheral nerve blocks in patients undergoing knee arthroscopy because they are discharged quickly, and would be unable to bear weight safely on a “blocked leg.” A tourniquet is often employed; see above.

**Technique:** general, neuraxial, or regional. Sciatic, popliteal, femoral, or ankle blocks as applicable. **Monitors:** standard. **IV access:** one IV will generally suffice. **Duration:** 1-4hrs. **EBL:** < 200ml for almost every case. **Position:** usually supine. Certain cases such as talar reconstructions (posterior foot) may be prone or lateral. **Special equipment:** none. **Special considerations:** tourniquet, possible outpatient status.

**Chapter 3C-3. Anesthesia for Upper Extremity Orthopedic Surgery**

**Shoulder Replacement or Reconstruction**

**Technique:** General ± interscalene block for post-op analgesia. **Monitors:** standard. **IV access:** one large IV. **Duration:** 3-4hrs. **EBL:** usually < 500ml. **Position:** beach chair (sitting), sometimes lateral with the arm specially positioned. **Special equipment:** none. **Special considerations:** The beach chair position is commonly employed. Here, the whole OR table is repositioned to place the patient in a sitting position, as if in a chair. Attention must be paid to securing the patient’s thorax and head to the table well, placing appropriate padding, and doubly-securing the airway. Perfusion to the brain is compromised in the head-up position, so BP measurements on the arm or leg may need significant correction to accurately reflect cerebral perfusion pressure.

**Clavicle Surgery**

**Technique:** general. Blocks usually do not apply due to the highly proximal nature of surgery. **Monitors:** standard. **IV access:** one large IV. **Duration:** 3-4hrs. **EBL:** usually < 500ml. **Position:** supine. **Special equipment and considerations:** none.
Other Upper Extremity Procedures

Examples include:

- Humerus fracture repair
- Elbow surgery
- Radius and ulna surgery
- Nerve transposition
- Hand surgery: from minor to extensive

**Technique:** general or regional to include interscalene, infraclavicular, axillary or more distal blocks as applicable, ± continuous catheter for longer post-op analgesia. We tend to avoid regional anesthetics if there is a possibility of nerve injury to avoid complicating the picture (e.g., nerve transpositions). Minor hand procedures such as carpal tunnel release or Dupuytren’s contracture release can often be done under IV regional anesthesia (Bier block). **Monitors:** standard. **Duration:** 30min-4hrs. **EBL:** usually < 100ml; tourniquet is often used. **Position:** supine. The arm may be individually positioned. **Special equipment and considerations:** tourniquet, outpatient status.

Chapter 3C-4. Anesthesia for Debridement and Skin Grafting

Incision & drainage or debridement of infected bone or soft tissue are common surgeries at UCSD, and share some features with minor burn surgery, described below. Patients with osteomyelitis, sacral pressure ulcers, or post-traumatic soft tissue defects often return for multiple surgeries; they may have comorbidities related or unrelated to the surgery. Two examples are a morbidly obese ICU patient who is intubated and on pressors, with a sacral pressure ulcer, or a previously healthy young patient with a leg injury following an MVA. Often, these cases will also employ wound vacuums or skin grafting from another site. In general, the procedures themselves are minor, but some may involve extensive blood loss.

**Technique:** general or regional. Skin grafting from another site may make regional anesthesia either desirable or undesirable. **Monitors:** standard. **Duration:** 30min-4hrs. **EBL:** minimal to < 500ml. **Position:** highly dependent on the position of the lesion. **Special equipment and considerations:** possible tourniquet.

Chapter 3D. Anesthesia for Vascular Surgery

Vascular surgery patients are some of the most ill patients we regularly take care of. Often, the vascular disease co-exists with multiple other serious disorders and comorbidities, such as coronary artery disease, systolic or diastolic cardiac dysfunction, cerebrovascular disease, diabetes, hypertension, kidney disease, COPD, and a long history of smoking, to name a few. A careful and detailed preoperative workup is necessary. These patients should be, and usually are, in their “best medical state” when they present for surgery.

Common intraoperative issues seen with vasculopathies include the tendency for very labile BPs due to their poorly-compliant vascular tree. Essential hypertension and vascular disease may mask hypovolemia. Before induction, they may be normotensive, but upon induction, they may become radically hypotensive, only to then become profoundly hypertensive and after intubation or surgical stimulation. These wild BP swings can be difficult to manage and often occur despite our best efforts.
Another issue is difficulty in obtaining IV access and invasive monitors. The same disease process which necessitates the surgery often affects peripheral veins, making these patients notoriously “tough sticks.” Compounding this problem is that these patients often have frequent blood draws or hemodialysis access grafts/fistulas, all of which make securing IV access even more difficult. Similarly, the arteries can be quite calcified or have had prior instrumentation and are challenging to cannulate. Unfortunately, it is precisely this class of patients who most need an arterial line.

**Minor Vascular Surgery**

These will be considered as a group and include creation or revision of AV fistulas or AV grafts, I+Ds, angiograms, and amputations.

**Technique:** most AV fistulas and angiograms are done under local/MAC or peripheral nerve block with MAC, either of which is well-tolerated by the patient. GA or neuraxial are other options. **Monitors:** standard usually suffices. **IV access:** one IV. **Duration:** 1-2hrs. **EBL:** minimal-300ml. **Position:** supine. **Special equipment:** none. **Special considerations:** I+Ds and amputations are also discussed in the orthopedics section.

Patients in renal failure needing a fistula are typically on the verge of needing dialysis, or have an indwelling dialysis catheter. Ensure medical optimization prior to surgery. With that said, creation of an AV fistula is a minor procedure, and most anesthesiologists feel perfectly comfortable taking a patient to the OR with a potassium < 5.5-6.0 and no EKG changes. Recent trends in the patient’s potassium can be helpful in deciding how to manage any hyperkalemia present.

If an indwelling catheter is to be used, you must be aware that these contain concentrated heparin to keep the catheter from clotting off. The dialysis catheters should be avoided altogether because of the risk of infection and compromising the line, so only use this line in absolutely dire circumstances. If it is used, the heparin must first be withdrawn and discarded. Aspirating from the catheter until undiluted blood appears, at least 2 times the dead space in the catheter, is the best way to accomplish this. Similarly, when finished using the catheter, it must be reflushed with concentrated heparin. Consult with your attending, a nephrologist, or the vascular surgeon.

Angiograms are often done on a special table which can move significantly in all horizontal planes. The design is meant to allow the surgeon to track dye in the patient’s blood vessels by moving the table. Often apnea or the patient holding their breath during these stages is required. Thus, either GA with controlled ventilation or the alternative (a completely responsive, awake patient) is required. It is extremely important to ensure that all lines and monitors be untangled and have significant length, with extensions as necessary, to allow free motion of operating table and avoid snags. Generally, the entire range of motion of the table is tested prior to starting the procedure to ensure that there is enough length on our lines and no obstruction to table movement. Extensions may be necessary.

**Moderate Vascular Surgery**

These include peripheral bypass (e.g., femoral-popliteal bypass), thrombectomy, endovascular AAA repair, and carotid endarterectomy (CEA). The unique requirements of CEA and endovascular AAA repair will be explored further in the special considerations section.

**Technique:** almost always general. Some peripheral bypasses can be done under neuraxial techniques or blocks, but often the length of the procedure and concomitant administration of heparin precludes
them. Similarly, endovascular AAA and CEA can both be done under local/MAC, but possibility of catastrophic rupture and need for emergency GA makes this a poor choice. In addition, our surgeons have very little experience with local/MAC or cervical plexus block/MAC for CEA; see more below. **Monitors:** standard. Usually an arterial line for bypasses, always for CEAs and endovascular AAA repairs. Urine output, EEG for CEAs. Possibly a central venous line for endovascular AAA repair. **IV access:** at least one large IV. Blood loss is usually small, but surgery on large arteries always has the possibility of rapid and significant blood loss. **Duration:** 3-6hrs. **EBL:** typically < 100ml, as vessels and bleeding are rapidly identified and clamped. Vessel damage can generate much larger volumes of blood loss. **Position:** supine. **Special equipment:** EEG for CEAs. Equipment to check ACT for heparin monitoring. Fluid warmers. **Special considerations:** as below.

1. Endovascular AAA Repair

In endovascular AAA repair, the surgeon attempts to place a stent within the diseased portion of the aorta, preventing further growth and/or rupture of the aneurysm. This procedure employs the movable angiography table. This procedure is intended to be minimally invasive and generally does not cause much physical perturbation to the patient. However, the possibility of rupture of the aneurysm during the procedure is a feared complication. Thus, GA and an arterial line are usually employed, allowing for the procedure to transition rapidly to an open one in the case of a catastrophic event. To this end, some people also place a large central line (Cordis) or multiple large peripheral IVs for use in case of rupture. This is usually not necessary. Open AAA repair is a much different procedure and is briefly described below.

2. Carotid Endarterectomy

There are three basic goals in our anesthetic management of CEA: keeping the patient’s blood pressure normal or supranormal (as during carotid clamping), monitoring for neurologic ischemia, and tailoring the anesthetic for a quick wakeup to allow for rapid neurologic assessments. Maintaining the BP at the patient’s baseline is important because the presence of carotid disease also implies generalized atherosclerotic disease within the cerebral and coronary vessels. Cross-clamping of the operative carotid makes the distribution of the ACA and MCA on that side dependent on a “back pressure” from collateral flow through the circle of Willis. This back pressure depends entirely on MAP. To this end, most anesthesiologists employ a phenylephrine or norepinephrine infusion from the outset of the case to allow rapid titration and adjustment of BP. A nitroprusside infusion may rarely be needed to rapidly lower a dangerously high BP. Minute-to-minute monitoring and adjustments clearly necessitate an arterial line.

There are several ways to monitor for cerebral ischemia. The first and perhaps best way is to keep the patient awake, so that they can then act as their own monitor for neurologic insult. Unfortunately, our surgeons are not as comfortable with this technique and generally do not employ it. The EEG allows us to monitor electrical activity as a surrogate for cerebral perfusion. For more definitive information, consult an appropriate text. The monitor itself will be set up by our anesthesia monitoring technicians. Large reductions in electrical activity from a baseline EEG obtained during a stable depth of anesthesia, especially unilateral on the operative side during carotid clamping, are worrisome for ischemia and the surgeon must be notified. At times, the EEG is not employed, depending on the surgeon and/or anesthesiologist. Other methods to maintain or monitor cerebral perfusion employed by the surgeon are creation of a temporary common-carotid-to-distal-internal-carotid shunt to bypass the operative site and maintain flow, or the measurement of “stump pressures” after carotid clamping. The stump...
pressure is the pressure in the internal carotid distal to the clamped section, and theoretically reflects the “back pressure” via collateral flow as described above.

A quick, smooth wakeup is a goal for several reasons. The surgeons desire serial neurologic checks as soon as possible. A cough- and buck-free wake up reduces the risk of trauma or hypertension to the freshly-incised artery. To this end, many of us employ techniques that allow for this. One common method is to use a nitrous oxide-inhalational anesthetic, preferably desflurane combined with a fast opioid technique, often remifentanil. All of these agents can be discontinued immediately before surgery ends with a predictably fast recovery. Finding a balance between two concepts—this surgical incision is only mildly painful, but at the same time an agitated, thrashing, hypertensive emergence must be avoided—must be done to allow quick and pain-free awakening.

Monitoring of BP is key in the post-op period; excessive hypertension must be avoided, and many patients are started on antihypertensives post-op. Patients who have had bilateral CEA are prone to the effects of denervated carotid bodies, namely hypertension due to the lack of baroreceptor reflex and loss of hypoxemic respiratory drive. The loss of hypoxemic drive could be particularly devastating in the context of baseline COPD.

**Major Vascular Surgery**

Examples include open AAA repair and aortic, iliac, or mesenteric bypass. They are separated from the surgeries above because the anesthetic management can be very different.

**Technique:** general. Thoracic epidural is often employed for postoperative pain, which can be severe. **Monitors:** standard, arterial line, CVP, often a PA catheter, ± TEE. Urine output. **IV access:** at least two large IVs, and usually a Cordis. **Duration:** 4-8hrs. **EBL:** 500ml to possibly many liters depending on duration and intraoperative events. **Position:** supine. Potentially thoracoabdominal approach for higher aneurysms. **Special equipment:** Warmers are mandatory as blood transfusions are exceedingly likely. Equipment to check ABGs and ACT. Rarely, a spinal drain to remove CSF. Double-lumen ET tube for one-lung ventilation may be necessary if a thoracic approach is to be taken. **Special considerations:** as below.

Open AAA repair is one of the most labor-intensive and difficult cases we do. In the age of endovascular AAA repair, it is also exceedingly rare, but continues to be a commonly-tested boards topic. Areas of interest include:

- The physiologic derangements brought on by aortic cross-clamping and unclamping
- Cardiopulmonary comorbidities such as CAD, CHF, and COPD
- Epidural analgesia in the context of systemic anticoagulation
- Thoracoabdominal aortic aneurysms and the implications for lung isolation
- Organ dysfunction brought on by aortic cross-clamping including renal injury, mesenteric ischemia, and spinal cord ischemia

Given the infrequency of this surgery, a full discussion is outside the scope of this guide. Consult a definitive reference for more details on this demanding case.

Aortic or iliac bypasses typically involve less blood loss and postoperative sequelae than a frank AAA repair, and share many considerations with “moderate” vascular surgery described above.
Chapter 3E. Anesthesia for Ophthalmic and Head & Neck Surgery

These surgeries are grouped together for one reason: they occur in close proximity to the airway, making close communication between the surgeon and anesthesiologist absolutely essential. Few other types of surgeries combine the unique demands and challenges presented by these cases. Other unique issues are also discussed below.

It is common during these surgeries for the surgeon to be closer to the airway than the anesthesiologist. The OR table is usually turned 90° or 180° away from the anesthesia machine, creating a twofold problem: airway issues are both harder to detect and more difficult to correct. Further compounding the problem is that the airway is often in the surgical field, covered under drapes, or under constant manipulation by the surgeon. All of these create the need for effective communication with the surgeon and the need to doubly-secure the airway from the outset to reduce the chance for complications. A circuit extension is often necessary to reach from the anesthesia machine to the patient, and securing any airway tubes (ETT, LMA, tracheostomy) is done with great care.

Many head & neck surgeries involve advanced cancer or other obstructive lesions in or near the airway, which may make ventilation and intubation difficult or impossible. A thorough preoperative airway exam and a careful plan, often after discussion with the surgeon, are needed. Awake intubation or tracheostomy may be indicated. In the situation of a potentially difficult airway where GA is to be induced, the surgeon should be present and equipped to perform an emergency tracheostomy.

Oral or dental surgery necessitates an airway that does not occupy the mouth. A nasal RAE (Right Angle Endotracheal) tube is typically used for this purpose. The nasal RAE is designed to enter the glottis and trachea, exit via a nostril, and take a 90° cephalad bend at the nostril toward the forehead, where it is typically taped. In this way, the circuit can be placed away from the operative field. It is a long ETT, and the distance from tracheal tip to 90° bend is proportional to the ETT diameter. Nasal RAEs can be placed awake after topicalization, or after a standard induction. Usually, the RAE is soaked in warm water to soften the PVC and avoid trauma to the nasal passages, and generous lubrication is used. If placed under direct laryngoscopy, the RAE is advanced blindly into the oropharynx and then advanced past the vocal cords using Magill forceps, or by forming a bend in the tube and guiding the tube towards the trachea. Trauma or bleeding in the oropharynx may make intubation difficult and require an awake technique.

An oral RAE is generally used for nasal surgery. Like the nasal RAE, it is designed to curve caudad away from the operative field via a nearly-180° bend that is meant to reside at the lower incisors. Also like the nasal RAE, the distance from tip to this bend is proportional to the ETT diameter. Oral RAEs can be placed in all the same ways as a standard ETT, with the caveat that the preformed bend may require a rigid stylet to straighten the tube for placement.

Occasionally an MLT (Micro-Laryngeal-Tracheal) tube is used for vocal cord surgery and endoscopic surgeries. This tube is designed to be smaller in diameter and allow the surgeon to work around the tube and vocal cords. The other design components are intended to overcome the traditional disadvantages of smaller standard ETTs. Thus, an MLT tube is longer (will reach the trachea in full sized adults) and is stiffer.

Armored or reinforced ETTs have a metal framework in them that resists kinking or obstruction from external pressure. They are not the same thing as a laser tube; see below.

Airway Fire

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Head & neck surgery routinely employs the use of a laser. Specific issues arise with laser use, the most devastating being an airway fire. The smoke and vaporized gas must be appropriately scavenged by the surgeon as it represents a biohazard, and eye protection is mandatory for personnel and the patient. To prevent an airway fire, the following maneuvers are employed:

- $\text{F}_2\text{O}_2$ as low as the patient can tolerate, preferably 21%.
- Nitrous oxide must be avoided, as it supports combustion.
- Standard tubes (PVC) are highly combustible. A metal laser tube is designed to reflect and disperse laser light and is non-combustible.
- Cuffs should be filled with saline with dye (e.g., methylene blue) to signal rupture from the laser and decrease combustion.
- Saline-soaked gauze should be used to pack all other parts of the airway.
- Water should be immediately available to extinguish fires.

In the event of an airway fire, take the following steps:

- Stop ventilation.
- Remove the ET tube.
- Turn off oxygen.
- Extinguish the flames.
- Ventilate and reintubate the patient.
- Evaluate damage to the airway; chest X-ray, bronchoscopy, ABG, or lavage may be needed.

Surrounding structures are also at risk of fire, including the drapes around the patient’s head. Insufflation of oxygen (nasal cannula, oxygen mask, blow-by oxygen) can combust these drapes if cautery is being used near that oxygen.

Coughing and bucking can raise venous, intraocular, nasal and inner ear pressures; cause bleeding and disruption of suture lines; and even dislodge surgically placed grafts or artificial membranes. Likewise, hypertensive episodes can exacerbate these problems. These problems are most often encountered during induction, intubation, and emergence. Therefore, many ENT and ophthalmic surgeons ask for “deep extubations.” The flipside to this coin is that many ENT procedures produce significant blood, secretions or edema in close proximity to the airway, placing the patient at risk for aspiration or postoperative obstruction. When these surgeons ask for a deep extubation what they really want is a smooth emergence devoid of coughing or hypertensive episodes. Deep extubations may be suitable for some patients, while for others the anesthetic technique is geared towards a smooth but fully awake extubation (e.g., with narcotics).

In sum, these surgeries can test even the most even-keeled person’s patience. It can be difficult to maintain good humor after a day of sharing the airway, constant circuit disconnections by the surgeon, and repeated requests for deep extubations in patients with significant aspiration risk. Remember that our duty is to the patient, and that you can always defer to your attending.

3E-1. Anesthesia for Ophthalmic Surgery

In general, most eye procedures are relatively noninvasive. Postoperative pain is minimal. However, PONV is a significant problem. Most patients should receive some form of PONV prophylaxis. The issues of keeping intraocular pressure to a minimum are discussed above. The following common anesthetic
situations raise intraocular pressure:

- Coughing and bucking via increased venous pressure
- Increased systemic arterial pressure (modest effect)
- Succinylcholine (modest effect)
- Hypercapnia
- Topical anticholinergics, which decrease aqueous drainage from the eye, thereby increasing pressure; IV anticholinergics seem to have little to no effect

Ophthalmologists sometimes place a bubble of gas (usually sulfur hexafluoride) in the eye to assist with healing and immobility of intraocular structures, like a detached retina. These gas bubbles will avidly take up nitrous oxide, potentially leading to disastrous increases in intraocular pressure. Therefore, nitrous is to be avoided before placement of a gas bubble and for up to 2 weeks afterwards. After this point, the bubble has been absorbed into the systemic circulation and those risks do not apply.

Many eye surgeries are performed as outpatient procedures. Quick emergence and discharge times are desirable.

The oculocardiac reflex can result from pressure or traction on the eye or the extraocular muscles. The reflex involves trigeminal nerve afferents and vagal efferents. Bradycardia is the most commonly seen arrhythmia, but ectopy or even asystole can occur. The reflex is usually mild and is controlled in the following ways:

- Tell surgeon to stop whatever traction or pressure is being applied.
- Atropine or glycopyrrolate as necessary.
- Deepening anesthesia.
- If persistent, retrobulbar blocks can be done to block the afferent limb of the reflex.

Minor Eye Surgery

Examples include cataract removal, vitrectomy, and blepharoplasty.

**Technique:** usually topical anesthesia or local/MAC. Some patients may require general anesthesia. Retrobulbar or facial nerve blocks are also a possibility, and are usually performed by the ophthalmologist. **Monitors:** standard. **IV access:** one IV. **Duration:** 30min-2hrs. **EBL:** minimal. **Position:** supine, head away from anesthesiologist. **Special equipment:** none. **Special considerations:** oculocardiac reflex, lack of access to the airway, ambulatory surgery.

Major Eye Surgery

Examples include retinal detachment repair, strabismus surgery, ruptured globe repair.

**Technique:** usually general. These surgeries are more invasive, longer in duration, and often require a completely still surgical field. Retrobulbar or facial nerve blocks are also a possibility, and are usually performed by the ophthalmologist. **Monitors:** standard. **IV access:** one IV. **Duration:** 1-4hrs. **EBL:** minimal-100ml. **Position:** supine, head away from anesthesiologist. **Special equipment:** none. **Special considerations:** oculocardiac reflex, lack of access to the airway, PONV.

Ruptured globes are open-eye injuries and the goal is to avoid elevations of intraocular pressure,
especially during intubation and emergence. Succinylcholine should probably be avoided, and deep anesthesia and paralysis should be achieved before intubation. On the other hand, emergency surgery necessitates rapid control of the airway and minimization of aspiration risk, for which succinylcholine is ideal. Deep extubation is more controversial; see above. Avoiding PONV and its associated increases in intraocular pressure is important.

Chapter 3E-2. Anesthesia for Head & Neck Surgery

Head & neck surgeries can be broadly classified as endoscopy, minor surgery, and major surgery.

Endoscopy

Examples include laryngoscopy, bronchoscopy, and esophagoscopy, or any combination thereof.

**Technique**: general with profound muscle relaxation. **Monitors**: standard. **IV access**: one IV. **Duration**: 30min-1hr. **EBL**: minimal. **Position**: supine, typically with aggressive neck and head extension, head away from anesthesiologist. **Special equipment**: as needed, laser ETT, MLT tube, oral RAE, armored ETT. **Special considerations**: Use of a laser, sharing the airway with the surgeon, instrumentation near the endotracheal tube.

Some of these cases are done with a “non-sealed” airway via oxygen insufflation and spontaneous ventilation, precluding the use of volatile anesthetics, control of the exact F\(_2\)O\(_2\), and positive-pressure ventilation. Many patients have masses or distorted anatomy of the upper airway which may cause difficult ventilation or intubation. A thorough airway exam and plan is needed. Typically, the surgeon has done at least an abbreviated examination, e.g., nasopharyngoscopy with topical anesthetic, in the office. Postoperative airway issues can arise from surgical instrumentation and manipulation, edema, or secretions.

Profound paralysis may be needed to ensure a motionless surgical field. Repeated small doses or an infusion of succinylcholine is an option, bearing in mind the possibility of cholinergic effects or a phase II block; see the neuromuscular blockade section. Alternatively, non-depolarizing blockade can be used, keeping in mind the short nature of most of these procedures.

These procedures can be profoundly stimulating, but typically cause minimal postoperative pain. Short-acting analgesia, deep anesthesia, and muscle relaxation are key to avoid wide swings in BP.

Minor Head & Neck Surgery

Examples include nasal surgery, ear surgery, oral and maxillofacial surgery, and thyroid/parathyroid surgery. Tracheostomy itself is a relatively benign procedure, but is often performed on patients with significant comorbidities.

**Technique**: general. **Monitors**: standard. **IV access**: one IV. **Duration**: 1-4hrs. **EBL**: generally < 300ml. **Position**: supine, head away from anesthesiologist. **Special equipment**: laser ETT, oral RAE for nasal procedures, nasal RAE for oral/mandibular procedures, armored ETT, circuit extension for airway. **Special considerations**: PONV due to blood flowing into stomach, need for smooth emergence for neck incisions, lack of access to the airway. Preexisting airway issues may make ventilation or intubation difficult, e.g., nasal obstruction from polyps, blood in oropharynx from mandibular fracture, limited mouth opening.
Head & neck surgeons will often ask for hypotension to decrease intraoperative bleeding. This must be considered on a case-by-case basis as the patient’s physiology allows. The need to be political and maintain a good relationship with the surgeon should be stressed. The surgeons often place cocaine-soaked pledgets in the nose to cause vasoconstriction, decrease bleeding and provide some anesthesia; seeing the effects of systemic absorption (hypertension, tachycardia, and arrhythmias) is not uncommon.

During many of these procedures significant amounts of blood and secretions can collect near the airway and in the stomach. This can contribute to PONV and risk of aspiration. The stomach should be suctioned prior to emergence. Oropharyngeal “throat” packs are often employed to soak up debris. These fluids are the primary reason that deep extubation is often unsafe, and can also contribute to laryngospasm and bronchospasm.

Procedures on the ear (e.g., tympanoplasty) create situations where closed air spaces can form, creating a hazardous situation with nitrous oxide. Normally, the Eustachian tubes provide a vent for nitrous oxide buildup; however, these are typically obstructed in many patients with chronic ear problems. Nitrous is best avoided in this situation.

Patients with mandibular fractures must be suspected of having a basilar skull fracture, which is an absolute contraindication to placing a nasal ETT. Recognize this possibility, review appropriate imaging, and confer with the surgeon as needed.

Tracheostomy in a patient with an indwelling ETT merits discussion here. Always ensure that the tracheostomy tube cuff has been tested by the surgeons, just as you would for an ETT you are going to place. The surgeons proceed with dissection until they are quite close to making incision into the trachea itself. At this point, the F\textsubscript{O}\textsubscript{2} is reduced as low as possible, ideally < 30-50%, to decrease the risk of airway fire. The cuff of the ETT is deflated to avoid puncture by the surgeons, and the tape securing the ETT in place to the face is loosened. From this point on, ventilation may be difficult or impossible due to large leaks. The surgeons enter the trachea, and will ask you to pull the ETT back until it is just above the incision. The surgeons then place the tracheostomy tube with a flexible airway connector, and will hand off this connector to you, who will then confirm ventilation and etCO\textsubscript{2}. The ETT can then be removed. Depending on the speed of the surgeon and the patient’s oxygenation status, significant desaturation can occur. If necessary, the surgery can be stopped, tracheostomy removed, and the ETT moved distal to the incision and cuff reinflated for further ventilation and oxygenation.

**Major Head & Neck Surgery**

These cases include extensive maxillofacial reconstruction, oral/lingual/pharyngeal/laryngeal tumor resection, laryngectomy, pharyngectomy, radical neck or face dissections, with or without free flap from the chest or other location. They are sometimes done in conjunction with plastic surgeons.

**Technique:** general. **Monitors:** standard ± arterial line, urine output; CVP may be useful. **IV access:** at least two IVs. **Duration:** 4-12hrs. **EBL:** 500ml-2L depending on extent of surgery. **Position:** supine. **Special equipment:** facial nerve monitors often employed; see below. RAE or armored tubes as needed. Awake tracheostomy under local anesthesia may precede general anesthesia and the surgery itself. Fluid warmers. Circuit extension. **Special considerations:** lack of access to the airway, potential difficulty securing the airway due to preexisting disease.

These surgeries in general are lengthy and extensive dissections with the potential for significant blood
loss. Keeping the patient warm and adequately resuscitated is important. Often, because of the proximity of disease and surgery to the airway, the patient will remain intubated or have an intraoperative tracheostomy performed; see above.

If a free flap will be used, the surgeons will often request that no vasopressor be given due to concerns over graft ischemia. Clearly, maintaining the patient’s BP is important, and the effect of small amounts of vasopressor on graft circulation is questionable, but within these parameters, it is probably best to avoid pressors such as phenylephrine or epinephrine. Volume resuscitation and keeping the patient warm will improve graft circulation.

Depending on the location of the surgery, the surgeons may monitor the facial nerve to avoid damage from dissection. They will request that no neuromuscular blockers be used in this situation.

Dissection around the carotid sinus can cause bradycardia, other arrhythmias, or wild swings in BP. Treatment consists of cessation of surgical manipulation, supportive treatment, or infiltration of local anesthetic around the carotid sinus. Bilateral neck surgery, including dissections and endarterectomy, can result in denervated carotid bodies, causing loss of hypoxemic ventilatory drive and baroreceptor regulation of BP.

**Chapter 3F. Anesthesia for Interventional Pulmonology**

The pulmonology department at UCSD is particularly active and their cases constitute a significant portion of the anesthesia services provided in and out of the OR. While pulmonologists typically perform simple flexible bronchoscopy with topical anesthesia and light sedation (without anesthesiologists), the more invasive procedures described below warrant both the provision of general anesthesia and the expertise of an anesthesiologist.

**Rigid Bronchoscopy with or without Tracheal/Bronchial Stenting, Lasering, or Dilation**

Many patients who have had a tracheostomy go on to develop subglottic strictures, obstructive scar tissue, or tracheomalacia. UCSD is a referral center for many patients with chronic periglottic and subglottic airway disorders in this setting. These patients often have rigid bronchoscopy and intervention serially over many months or years, have comorbid pulmonary disease such as COPD or interstitial lung disease, have a tracheostomy or tracheal/bronchial stents in place, or are dependent on home O₂. This is a sick class of patients with tenuous respiratory function undergoing an intensely-stimulating procedure with a shared airway.

**Technique:** general. **Monitors:** standard. **IV access:** one IV. **Duration:** 10min-2hrs. **EBL:** minimal. **Position:** supine with full head extension. **Special equipment:** Bain circuit (provided by the anesthesia technicians), IV infusion pumps, methylprednisolone. **Special considerations:** as below.

Despite their advanced respiratory disorders, many of these patients do not go to pre-op clinic since they have been anesthetized at UCSD before and, in all truth, the anesthetic would not be postponed for “medical optimization;” these patients need the procedure.

The anesthetic most typically employed is TIVA with propofol, lidocaine, alfentanil, and succinylcholine/nondepolarizer. The reasoning follows. Since the airway will be entered with a rigid bronchoscope that is capable of ventilating but that is not sealed, a volatile anesthetic cannot be used. Aggressive instrumentation of the airway also requires profound muscle relaxation to avoid coughing.
and patient movement during crucial maneuvers in the trachea. However, many of these procedures are short (< 15min), and require very short-acting agents. For longer procedures, a nondepolarizing NMB can be used; the pulmonologists will let you know if this is the case.

The typical approach is as follows: preoxygenation, IV induction with propofol and alfentanil, establishment of a mask airway, placement of the rigid bronchoscope by the pulmonologist, and continuation of the propofol infusion. Oxygenation is done by connecting the circuit to the ventilating port of the bronchoscope and bagging manually, sometimes furiously, and often with the high-flow O₂ flush valve since the airway is not sealed. Often, minimal or no etCO₂ is detected but you may be able to see airway distension and fogging via the bronchoscope screen. Alfentanil and succinylcholine are redosed, with attention to bradycardia with succinylcholine. Methylprednisolone is given to reduce airway inflammation and edema. The propofol infusion is terminated as soon as is practical to allow rapid awakening. Emergence is done with a mask airway, and is often “stormy” given the intense pro-tussive effect of the procedure. Humidified O₂ is the rule in PACU given the airway irritation that occurs.

**Endobronchial Ultrasound (EBUS)**

Another common pulmonology procedure we are asked to provide anesthesia for is EBUS. The procedure involves ultrasound-guided biopsy of suspicious lymph nodes via flexible FOB. Often we are asked to place a CookGas intubating LMA instead of an ETT, with the idea that this supraglottic airway allows access to the entire trachea and has a shorter and large-diameter airway tube to facilitate the FOB. However, an ETT may be indicated depending on patient characteristics. We may also be asked to keep the patient spontaneously ventilating in order to reduce the risk of pneumothorax. The pulmonologists are quite good at clearly communicating these preferences. After induction of general anesthesia, the pulmonologists will provide topical anesthesia to the tracheobronchial mucosa.

**Technique:** general. A TIVA is preferred given the frequency with which the airway seal is broken to place/remove the FOB. **Monitors:** standard. **IV access:** one IV. **Duration:** 30min-2hrs. **EBL:** minimal. **Position:** supine. **Special equipment:** CookGas LMA. **Special considerations:** as above.

**Chapter 3G. Anesthesia for Transplant Surgery**

The most commonly performed organ transplantations at UCSD are kidney and liver transplants. Pancreas transplantation is uncommon at UCSD, but shares many considerations with kidney transplantation. Anesthesia for organ procurement will follow at the end of this section. Lung transplantation will be addressed in the chapter on anesthesia for cardiothoracic surgery. Heart transplantation is usually done by our CT anesthesia fellows, and will not be discussed in this guide.

The most important factors to consider in transplant surgery are the physiologic derangements imparted by the patient’s organ failure. In the case of kidney transplantation, the most obvious is renal failure, which predisposes the patient to volume overload, volume sensitivity, electrolyte abnormalities, acidosis, hypertension and anemia. Elimination of many drugs may be impaired. Patients should be medically optimized prior to surgery. Depending on the timing of dialysis, they may be relatively volume overloaded or underloaded, with their electrolyte and acid/base status under varying degrees of control.

End-stage liver patients are some of the most ill patients anesthesiologists care for, and a liver transplantation is one of the largest, most labor-intensive cases that we do. To begin, the patient’s liver dysfunction can cause, or co-exist with, dysfunction of the neurologic, cardiac, pulmonary, GI, renal, and hematologic systems. Poor or non-existent liver function predisposes patients to coagulopathy, anemia,
hypoalbuminemia, ascites, and a high-cardiac-output, low-SVR state. Metabolism of drugs and hepatic synthetic function are deranged. There may be hepatorenal syndrome, hepatopulmonary syndrome, or portopulmonary hypertension. Furthermore, the case itself involves significant and rapid blood loss, fluid shifts, and major physiologic perturbations, which will be considered in the section below.

Associated disease, such as diabetes or hepatitis C, may be the cause or a result of the organ failure, and further complicates these cases. For cadaveric transplants of any organ, there is a limited window of viability. Transplants occurring after this window have a marked reduction in organ function and survival. For the liver, the window is generally 12hrs, and for the kidney, the window is generally within 24hrs (the sooner the better). Thus, there is usually an urgency placed upon transplants and they should proceed in a timely manner. They are not purely elective cases, with the exception of schedule, elective living-related kidney donations and transplantation.

Chapter 3G-1. Anesthesia for Kidney Transplantation

Kidney Transplant Recipient (Living-Related or Cadaveric)

**Technique:** general. **Monitors:** standard, urine output, rarely arterial line, rarely central line for CVP measurement. **IV:** one large IV should suffice. If the surgeon requests thymoglobulin to be given, a separate dedicated IV line will be needed. **Duration:** 3-4hrs. **EBL:** < 500ml. **Position:** supine. **Special equipment:** mannitol, furosemide, methylprednisolone or other immunosuppressive agents; the surgeon will ask for this. Heparin and protamine. **Special considerations:** Fluid management must be judicious, with the conflicting factors of increased insensible losses from an open abdominal case being balanced against oliguria or anuria and poor tolerance of volume shifts.

Prior to anastomosis of the kidney to the iliac vessels, heparin will be asked for. Depending on the duration of anticoagulation, protamine may be needed. See the cardiothoracic section for more information about protamine.

Before reperfusion of the kidney, the surgeons will often ask for mannitol (0.5-1g/kg) to be given to create osmotic diuresis of the new graft. If urine output is not brisk following graft reperfusion, furosemide may be requested. A tinge of blood in the new urine flow is common; overt blood should be brought to the surgeons’ attention. Methylprednisolone will be asked for by the surgeons at the appropriate time; fast administration has been associated with arrhythmias.

While these patients are typically ill, the case itself is not terribly complicated and usually does not require invasive monitoring. They generally go quite smoothly.

Living-Related Kidney Donation

**Technique:** general. **Monitors:** standard. **IV:** one IV. **Duration:** 2-4hrs. **EBL:** < 500ml. **Position:** supine, or in a 45-90° lateral position for laparoscopic procurement. **Special equipment:** mannitol, heparin, protamine. **Special considerations:** Living-related kidney donors are typically otherwise healthy patients and the donor operation is not a major cause for concern. The case can be done either laparoscopically or open. Typically, these cases are timed simultaneously with the recipient, with the goal being the donor kidney being harvested just before the recipient is ready for implantation. This requires two OR teams.

Mannitol will be requested by the surgeon prior to harvest of the kidney to promote diuresis. Heparin
will also be asked for prior to clamping of the renal vessels and may be reversed with protamine.

Chapter 3G-2. Anesthesia for Liver Transplantation

**Technique:** general. **Monitors:** standard, arterial line, usually a femoral A-line, urine output, CVP, PAC, ± TEE. **IV access:** as much as possible. Routine lines for a liver transplant include one peripheral IV, a Cordis (a 9Fr sheath/introducer), and a triple-lumen Edwards catheter (central line with 3 large bore components). Rapid infusion catheters can also be used (essentially a mini-Cordis in a peripheral vein). **Duration:** 4-12hrs. **EBL:** Varies depending on surgeon but can be large. There is no maximum. 100+ unit transfusions were not uncommon in the past, but low blood loss/transfusion cases are becoming more common. **Position:** supine. **Special equipment:** TEE, warming blankets, fluid warmers to warm IV lines, TEG, perfusionist to aid with massive transfusion. **Special considerations:** as below.

Liver patients should be regarded as “full stomachs” due to compression from ascites, the urgent nature of the case, and/or recent GI bleeding. Thus, they should have a rapid-sequence induction with cricoid pressure.

Due to peripheral vasodilation throughout the case, caused by both the patient’s baseline disease and acidosis and hypocalcemia at critical portions, a radial arterial line may not accurately reflect the patient’s central BP. For this reason, a femoral arterial line is often placed after induction.

The goal of a liver transplantation is to keep everything as normal as possible. Frequent ABGs and TEGs are checked, often q30min or more frequently depending on the stage of the case. Acidosis, anemia, coagulopathy, and electrolyte abnormalities are aggressively treated with an attempt at normalization. Similarly, we attempt to maintain all vital signs within the normal range whenever possible.

**Thromboelastography (TEG)**

A thromboelastogram is a technique of monitoring coagulation that has proven invaluable for intraoperative monitoring and treatment. TEG requires a special machine, is run by the hematology lab, and requires at least 1 hour notification prior to use. For this reason, it is typically employed by us only for liver transplantations and for on-pump cardiac surgery. A blood sample (3ml) is sent to the lab and placed in a special cuvette. A small pin is placed in the blood and rotates to and fro. As the blood clots, its viscosity and clot strength influence the rotation of the pin, and the forces detected and recorded by the machine. Many variables are provided, but the ones we are most interested in are:

1. **R value:** this is the time to first clot formation, generally reflects the initial stages of clot formation (factor-dependent) and also reflects overall clot formation. Low R values are generally treated with FFP.
2. **Angle (α):** reflects the rapidity of clot amplification (cross-linking) once formation begins. Low values reflect inadequacy in fibrinogen, and are generally treated with FFP or cryoprecipitate.
3. **Maximum amplitude (MA):** reflective of platelet presence and function, and overall clot strength. Defects may be related to either of the above, or to platelet deficiency/dysfunction.
Each of the parameters on the TEG diagram above has a normal range, and the sample TEG is compared to those normals. These values are evaluated at 30min and 1hr of incubation. Therefore, TEG information is at least 30 minutes old and cannot be used to titrate minute-to-minute therapy. However, every attempt is still made to normalize abnormalities seen on the TEG. Frequent TEGs present a more linear, continuous picture.

The perfusionist is invaluable in assisting us with meeting transfusion and volume requirements. The perfusion machine is capable of delivering very high flows of crystalloid, colloid, RBCs, or FFP. Typically, we provide the perfusionist with two of our large lines, generally the two smaller lumens of the triple-lumen Edwards catheter. In this way, the perfusionist can maintain high flows without preferential flow through one line. Working with the perfusionist requires close communication; the perfusionist generally will not give anything we do not ask for. Common instructions to the perfusionist are to continuously give blood products in a variety of ratios (e.g. 1:1 pRBC:FFP) or flow rates (e.g. 50ml/min), titrating to CVP or PA pressures, or simply at intermittent intervals based on patient needs. It is generally advisable to leave all blood product infusions to the perfusionist, freeing our remaining lines for drips, boluses of drugs, and platelets.

“Cell-saver” (blood salvaging) is also employed unless the patient has a carcinoma or infection. The perfusionist or our anesthesia monitoring technicians usually handle the processing and washing of RBCs before giving the blood back to us to be infused. It should be noted that the bag of Cell-saver blood has air in it. Unlike a bag of PRBCs, a bag of Cell-saver cannot be pressurized due to risk of venous air embolism.

The presence of renal failure, with or without hepatorenal syndrome, often means that continuous veno-veno-hemodialysis (CVVHD) will be employed. The nephrology team will be present in the OR throughout CVVHD, and another central venous line will be needed, often femoral. The CVVHD machine can help with maintaining the patient’s pH and electrolytes (notably potassium) at normal levels, depending on the dialysate used. The nephrology team can also run the patient hypo-, hyper-, or euvoletic depending on our joint plan and patient needs at that time. Clearly, precise communication with the CVVHD team is also needed. Fluid management and acid/base status can be very complicated with surgical losses and the anesthesia, perfusion, and CVVHD teams all contributing to changes in patient status. Citrate is used as an anticoagulant, which contributes to hypocalcemia; see below.

The two most common and most important electrolyte abnormalities seen are hypocalcemia and hyperkalemia. Hyperkalemia results from massive transfusion, acidosis, potassium washout of the
transplanted organ, and/or concomitant renal failure. It should be aggressively managed and is most relevant during reperfusion of the new liver as below, but deaths have occurred from hyperkalemia even hours after the neohepatic phase. Frequent monitoring is thus mandatory and is done via serial ABGs. The perfusionist has an ABG machine in the OR with us, and can run nearly-real-time ABGs when we provide the sample.

Hypocalcemia also results from several mechanisms. End-stage liver patients are frequently hypoalbuminemic at baseline. Massive transfusion and CVVHD impart a high citrate load, which binds calcium. The diseased liver also has severely decreased metabolism of citrate, and during the anhepatic phase, there is no liver whatsoever to metabolize citrate. Frequent ABGs also help us monitor this situation. Massive calcium requirements, over 10g of calcium chloride, are not uncommon. A continuous calcium infusion is often employed.

A liver transplant has three distinct phases:

1. **Pre-anhepatic:** from induction of anesthesia until clamping of all hepatic vessels. The old liver is dissected out, and there may be massive blood loss if there is extensive perihepatic scarring. By the end of this phase, the liver remains connected only via the IVC, portal vein, hepatic artery, and common bile duct. Normalization of all abnormalities proceeds. Volume loading, so that the patient can tolerate the preload reduction that follows, is done.

2. **Anhepatic:** from clamping of the hepatic artery, suprahepatic IVC, infrahepatic IVC, and portal vein until these vessels are fully anastomosed and their clamps are removed (reperfusion). The IVC is typically completely clamped at this stage, with a dramatic reduction in venous return and potential for hypotension. Moreover, venous congestion typically causes engorgement of veins distal to the clamp, resulting in increased bleeding and possibly ischemia of the bowel. Veno-veno bypass is considered at this stage if the patient cannot tolerate complete or partial IVC clamping, and may use any right-sided central lines we have. Thus, the PA catheter and triple lumen Edwards is better placed on the left side. Veno-veno bypass does not require heparin but does carry a high risk of air embolism. During this period, there is zero metabolism of lactate and citrate leading to acidosis and hypocalcemia with possible hyperkalemia. Aggressive resuscitation continues, and typically a “buffer” of mild hypercalcemia, mild hypokalemia, and mild alkalosis is induced prior to reperfusion.

3. **Neohepatic:** from reperfusion to closure of the abdomen. This is the most dramatic phase of the case. The vessels to the new liver are unclamped, reperfusion of the organ commences, and a cholecystectomy is performed. The reperfusion of the liver and unclamping of vessels creates severe physiologic perturbations, not unlike unclamping of the aorta (see the section on open AAA repair). Washout and reperfusion of the new liver and previously ischemic organs introduces high concentrations of acid, high potassium, CO₂, lactate, adenosine, etc. The preservative solution is also washed out, and it (and the liver) were ice-cold until the start of the anhepatic phase. Taken together, acidosis, hyperkalemia, volume, and hypothermia constitute a massive insult to the right side of the heart, and cause profound hemodynamic changes. Acidosis and metabolic waste products cause a massive drop in SVR, a decrease in cardiac contractility, and arrhythmias. Hyperkalemia exacerbates this situation and can cause arrest on its own. The serum potassium concentration generally rises 1-2mEq/L during this phase. Therefore, we typically take the following steps prior to reperfusion of the new liver, always done in conjunction with the surgeons, who are well aware of the dangers of reperfusion:

- **Everything is normalized as much as possible prior to unclamping.**
- Some practitioners routinely start a dopamine, epinephrine, or norepinephrine infusion prior to
unclamping for positive inotropic/pressor effects.

- Slight hypercapnia is allowed during the anhepatic phase, but just prior to reperfusion, a respiratory alkalosis is induced. This will help with combating acidosis and drives excess potassium intracellularly.
- All anesthetics are d/c’d and the patient is ventilated with 100% O₂. Midazolam is useful here.
- Several ampules of sodium bicarbonate and calcium are placed in line, as well as bolus syringes of vasopressor. Of note, bicarbonate and calcium should not be given at the same time through the same IV because calcium carbonate (chalk) will precipitate.
- These ampules are usually given immediately before or after unclamping, knowing that hyperkalemia and acidosis are inevitable. We do not draw labs at this point, as the clinical situation changes literally on a second-to-second basis. Titration to BP and normalizing EKG changes (if any) are the goals.

After the new liver has been reperfused, it will typically start metabolizing waste products as well as synthesizing proteins and factors within the hour. “Typically” is the key word here; sometimes the previous coagulopathy and hypocalcemia persist, depending on graft function and health. It is still important for us to maintain homeostasis and check labs frequently. A slight coagulopathy is typically tolerated at this point. The goal is to avoid thrombosis of vessels of the new liver and return of endogenous synthetic function. Air embolism is also a possibility after reperfusion as air can enter the donor liver during harvesting. Thorough “flushing” by the surgeons decreases this risk.

Due to the massive transfusions, fluid shifts, and length of the case, these patients are usually kept intubated post-op and allowed to recover in the ICU. However, with shorter cases with minimal transfusion requirements and good liver function, extubation in the OR may be considered.

The presence of hepatitis B will necessitate the use of hepatitis B immune globulin (“HBIG”). This is started during the anhepatic phase at the direction of the surgeon.

Similar to kidney transplants, an immunosuppressive such as methylprednisolone is given at induction and after the new organ is implanted; this will be specifically requested by the surgeon.

**Chapter 3G-3. Anesthesia for Organ Procurement**

**Technique:** general. **Monitors:** standard. These patients often have additional monitors that have been placed during their hospital course. **IV access:** one IV. **Duration:** 1-4hrs. **EBL:** n/a, but < 500ml for our portion of the procedure. **Position:** supine. **Special equipment:** none. **Special considerations:** see below.

The most common type of organ donor is a brain-dead, ASA6 donor. These donors have met all criteria for brain death, with no cortical or brainstem function, and in this sense are not actually “patients.” Organs that can be procured include the heart, lungs, pancreas, small bowel, kidneys, and liver. Our involvement consists of transporting the donor from the ICU to the OR and maintaining homeostasis throughout the “warm” phase of procurement, until the surgeons ask for systemic heparinization and perform circulatory arrest by cross-clamping the aorta and cooling the organs as rapidly as possible. These donors are frequently on multiple hemodynamic drips including levothyroxine and vasopressors, which should be continued as the clinical situation dictates. Although no intact cortically- or brainstem-mediated pain pathways exist, spinal cord-mediated reflexes may still be active, producing hypertension (or even movement of the extremities) upon surgical stimulation. This can be treated with opioids if necessary.
The surgeons will make an incision from sternal notch to the pubic symphysis, do as much inspection and dissection as possible, prompt us for a full heparin dose (on the order of 30,000 units), clamp the aorta, and vent the IVC. The surgeons will notify us that we are no longer needed, the ventilator can be shut off, and the circuit can be detached from the airway. Our involvement generally ends here.

Another type of organ donation is “Donation after Cardiac Death,” or DCD. DCD donors have suffered an irreversible neurologic injury, but still preserve some element of cortical or brainstem function, and thus are still alive, i.e. they do not meet criteria for brain death. Nonetheless, patients selected for DCD are expected to have circulatory arrest within minutes of d/c of circulatory and ventilatory support. These situations are quite different than with brain-dead donors, and are exceedingly rare at UCSD. A full discussion is outside the scope of this guide.

Chapter 3H. Anesthesia for Trauma and Burn Surgery

UCSD is both a Level 1 trauma center and the only burn center for the entire county of San Diego. As such, we have our fair share of “OR Resuscitations” and surgeries for severely burned patients. Burn patients in particular often need extensive debridement and prolonged care requiring multiple trips to the OR.

Chapter 3H-1. Anesthesia for OR Resuscitation

Some trauma patients require immediate surgery to have any chance at survival. Our role as anesthesiologists is often focused on primary resuscitation. Broadly, traumatic insults break down into one of two categories: blunt or penetrating. Examples of blunt trauma include MVA or falls, while stab or gunshot wounds are prototypical examples of penetrating injury. “OR Resuscitation” is the term used at UCSD for emergency trauma surgery for patients with life-threatening injuries. They occur exclusively in OR11 at Hillcrest; the setup for OR11 is described in the “Emergency OR Setups” section. On average, we get about 1-2 OR Resuscitations per week.

UCSD’s trauma program uses a complex algorithm to determine which patients need OR Resus and which do not. Patients who are true candidates for an OR Resus are generally brought straight from the field by EMS personnel. Typically, these are patients with known or suspected trauma in the field and unstable or no vital signs. Because resuscitation is begun in the field by the first responders, these patients will often arrive intubated and with IV access already established. Many times, the report from the field (e.g., properly placed ETT) can be quite different from reality.

The primary survey of the trauma patient can be remembered by the acronym ABCDE, for Airway, Breathing, Circulation, Disability, and Exposure. Often these elements happen concurrently, but it is useful to remember the order of importance. The typical scenario of an OR Resus is as follows:

- The trauma surgery team, anesthesiology team, and OR staff are informed of an inbound trauma resuscitation. There is generally a brief report from the field regarding vitals, history, relevant lines and tubes, and ETA.
- The patient arrives in OR11. EMS personnel continue with their report while the patient is transferred to the OR table and connected to monitors. This situation is often quite chaotic and loud, with many people excited, talking loudly, and all pushing to do their jobs. It is vital to remain calm and focused in this setting, and often helpful to try and calm others down and keep the noise level low.
• ABCDE follows. Assess the patient’s airway and adequacy of ventilation.
• Indications for an advanced airway are myriad but include persistent obstruction, apnea, unconsciousness, facial or neck trauma, and chest or head injury. Burns involving the upper airway are particularly dangerous, as rapid swelling and edema can lead to life-threatening obstruction, even if the initial presentation is benign. Consider early intubation in these patients. Depending on the area of trauma, securing an airway via conventional laryngoscopy may be impossible. Options include tracheostomy or awake intubation. If the patient is already intubated, **you must confirm proper placement** via etCO₂ or an esophageal bulb detector.
• Intubating a trauma patient can present many challenges. These patients are full stomachs with potential cervical spine injury. Thus, rapid sequence induction with manual inline cervical stabilization and cricoid pressure must be performed. The inability to properly position these patients combined with the stress of the situation may make intubation difficult. Furthermore, blood, secretions or regurgitated gastric contents make visualization even more difficult. Ventilate with 100% O₂ until the clinical situation allows.
• Assessment of circulation by other members of the team is often concurrent with airway and breathing. Connect all monitors and/or manually check pulse, blood pressure, and heart rate. ACLS should be initiated whenever indicated. Definitive surgical control of hemorrhage is the first priority. Replacement of intravascular volume is often necessary but does not supersede the above.
• Surgical control of bleeding should proceed as rapidly as possible while intravascular volume is replaced. If there is cardiac arrest before or after arrival to the hospital in the setting of chest or abdominal trauma, the surgeons typically perform an emergency thoracotomy. This allows control of bleeding, potentially via aortic cross-clamp, repair of cardiac injuries (if any), and buys time to control the situation.
• Replacement fluids include blood products along with crystalloids and colloids. The current trend in resuscitation and trauma surgery is to use blood products as the primary fluid therapy, and in ratios approaching “whole blood.” A sample ratio of RBC:FFP:PLT may be 6:6:2 or 4:4:1. There is continual debate over the superiority of crystalloids/colloids with no good answer.
• Fluids of all varieties must be warmed.
• Type O negative (“trauma blood”) can be given while definitive type and crossmatch is performed.
• Often there is a SICU nurse assigned to run the Level 1 Rapid Infuser in the OR, which pressurizes and rapidly delivers fluid.
• An early priority is to establish an arterial line for accurate measurement of blood pressure and to allow blood draws. Large venous access is a priority as well. Large peripheral IVs may suffice. Central access is desirable but should not delay the case. Establishing a central line carries increased risk to the patient, but may be necessary if peripheral access is impossible.
• Disability is a rapid assessment of the patient’s neurologic status, and Exposure involves removing the patient’s clothes to allow assessment for injury. Typically these are done while other aspects of the primary survey commence.
• Appropriate labs (e.g., ABG) should be sent as soon as possible.

Other special considerations for an OR resuscitation follow.

Giving amnestic doses of anesthetics is often a secondary priority. Anesthesia can be given only as tolerated, but the first goal is resuscitation of the patient. Trauma victims are often so unstable that they cannot tolerate any volatile anesthetic. Your “anesthetic” may be muscle relaxant ± small doses of midazolam or scopolamine, until circulatory stability is achieved.

Multiple injuries often produce multiple, potentially conflicting anesthetic goals. For example, a patient
with both head and traumatic chest injury needs rapid control of the airway and resuscitation, but care must also be taken to minimize increases in ICP and prevent brain injury. When in doubt, always come back to ABCDE as the first priorities. Specific injuries should be managed as the clinical situation allows (e.g., hyperventilation in the setting of suspected increased ICP).

Constantly reassess the situation and the patient. The patient’s vital signs and labs will guide therapy. Do not hesitate to initiate ACLS if needed. You must avoid the “lethal triad” of hypothermia, coagulopathy, and acidosis. To this end, make sure to keep the patient warm by monitoring temperature, warming the room, warming all fluids, etc.

The more hands available, the better. Typically this will not be a problem, since the OR Resus is truly an “all hands on deck” situation. There will always be an attending present and often another resident if free. Help is invaluable in ensuring the resuscitation proceeds smoothly and expeditiously. Also, remember that an OR Resus demands a team approach, and will usually be performed concurrently with the surgeons playing a very active role such as recognizing the need for ACLS and initiating therapy, requesting specific blood products, etc.

Finally, because it bears repeating: try to remain calm and focused on the task at hand. This may be difficult at first as the gravity and stress of the situation can be overwhelming. Remember that at the most basic level an OR Resuscitation is actually a simplistic case. The priorities are simply A, B, and C.

Chapter 3H-2. Anesthesia for Burn Surgery

As previously discussed, UCSD is a major burn center and the only center in the greater San Diego area. Surgery for burn victims encompasses the realms of plastic and trauma as well as “true” burn surgery. Because there is often need for extensive, continual debridement and skin grafting, many burn patients make repeated trips to the OR. Furthermore, the healing and reconstructive process can take months or even years. Patients may be discharged home following resolution of their initial, perhaps life threatening injury, only to return to the operating room months later for another procedure (e.g., release of contractures) and be otherwise healthy. Thus, burn surgeries break down into roughly two categories: surgery for major or acute burn injuries, and surgery for minor or chronic injuries. The former are critically ill and will be discussed in detail.

Major Burn Surgery

Prototypical examples include excisional debridement, skin grafting, and placement of Wound-Vacs.

**Technique:** general. Rarely, a regional technique may be employed depending on the location of the burn, but possible preexisting or evolving nerve damage may make this choice impractical. **Monitors:** standard, urine output, almost always an arterial line; CVP may be useful. **IV access:** large. May be difficult in extensively burned patients. **Duration:** up to 8hrs, depending on the extent of the injury and complexity of the case. **EBL:** 100ml to several liters depending on the extent of injury. **Position:** typically supine or prone. **Special equipment:** transport monitor, needle electrodes for EKG, warm room, fluid warmers and warming blankets. **Special considerations:** as below.
As discussed in the trauma section, the airway of a burn victim with inhalation injury must be treated with respect. Thermal injury or inhalation insults (e.g., smoke, ash) can cause rapid, life threatening edema in these patients and loss of the airway. Signs of inhalation injury include hoarseness, stridor, singed nasal or facial hair, facial burns, soot near the airway or mouth, and respiratory compromise. If there is any doubt of impending airway compromise, these patients are prophylactically intubated. Signs of airway obstruction necessitate an awake fiberoptic intubation. In general, most of these patients are intubated or have a tracheostomy in place before they come to the OR. At times, we may be called to assist with an acute burn that does not yet possess an airway. Also, because many of these patients will be in the Burn ICU already intubated and ventilated, the anesthesiologist must go and physically pick the patient up in the ICU for transport and monitoring on the trip to the OR: the so-called “anesthesia transport.” This will be addressed further below.

The extent of body surface area involved in the burn correlates with the severity of the injury and likelihood of survival. The “rule of 9s” can be used to estimate the BSA affected. Each arm, the head, and the anterior and posterior aspect of the thorax, abdomen, and legs each represent roughly 9% of TBSA. The perineum is the remaining 1%. In children, the head comprises double the TBSA than in the adult.

Derangement of the pulmonary system is a hallmark of burn injuries. Obviously, direct inhalational injury can compromise lung function. Carbon monoxide inhalation causes a left-shift of the oxyhemoglobin dissociation curve; carboxyhemoglobin is read by pulse oximeters as oxyhemoglobin, so the pulse oximeter reading will be artifactually high. Burns cause an increase in capillary permeability throughout the entire body, predisposing to pulmonary edema and ARDS. Long periods of intubation and ventilation predispose these patients to ventilator-associated pneumonia. Secretions can be profuse and thick. Adequate ventilation and oxygenation can be quite difficult. These patients are often in a profound hypermetabolic state, with increased O$_2$ consumption and increased CO$_2$ production, both of which place additional demands on the respiratory system.

The increase in capillary permeability mentioned above affects the entire body in burn patients. Large amounts of fluid shift from the intravascular to the interstitial space, resulting in massive edema and relative intravascular depletion. Incredible amounts of fluid resuscitation may be necessary to restore intravascular volume. Typically this is initially carried out with crystalloid according to the Parkland formula. After 48 hours post-burn, capillary integrity begins to be restored, and colloid will remain in the intravascular space. For this reason, our burn surgeons prefer us to use blood products or albumin for routine volume replacement, with sparing of crystalloid. Loss of skin integrity allows substantial evaporative losses which must be replaced. The maintenance fluid requirements of a burn patient are often on par with that of a large, open abdominal case.

Loss of skin integrity creates three additional problems: predisposition to infection, evaporative heat loss, and difficulty with monitor placement. Because of the tremendous potential for heat loss, special measures must be taken. The room is warmed to the point of being uncomfortable. This measure is a major downside of being in the “burn room.” All fluids should be warmed, the circuit should be humidified, and warming blankets should be placed wherever possible. In regards to monitors, there
may be little or no skin to place EKG pads on. In these cases, needle electrodes can be used; the anesthesia monitoring techs can assist with this. Finding an appropriate site to place a pulse oximeter and BP cuff can likewise prove challenging; many patients have an arterial line for this reason.

Excision of burned tissue is essentially shaving the tissue until viable tissue bleeds profusely. While this surgical bleeding is often overt, the magnitude of bleeding may be hard to appreciate at first. Frequent administration of blood products is often necessary. Serial ABGs and hematocrits help guide therapy. For this reason, and because of the inherent critically ill nature of most of these patients, an arterial line is mandatory. In fact, there will often be a preexisting arterial line that has been placed by the burn service. As appropriate sites for an arterial line may be limited due to the burn injury itself, it is not uncommon to find the line in an unusual location like the femoral or dorsalis pedis arteries. The large fluid and blood requirements also mandate large venous access. Large TBSA burns can severely limit sites for peripheral access. Furthermore, CVP can be useful for ongoing fluid management. For these reasons, a central line is often indicated and is usually present, courtesy of the burn service.

After about 24 hours, burn patients begin to develop immature, extrajunctional nicotinic ACh receptors on their muscle cells. As a result, dangerous hyperkalemia could follow the administration of succinylcholine, so this drug is contraindicated in patients with burns older than 24 hours.

Because these patients return to the OR frequently, it is quite possible a recent pre-op has been done and the record is available in EPIC. This can save a tremendous amount of time and provide valuable information about prior anesthetics.

“Anesthesia transport” refers to those patients who we physically bring from their ICU to the OR, with continuous monitoring, ventilation, and treatment of the patient. This can be time-consuming and physically demanding. Do not be afraid to ask for help pushing the bed, IV poles or other equipment, either from the circulating nurse, the ICU nurse, surgery personnel, or anesthesia technician. Mechanical ventilation means we must either bring an E-cylinder of oxygen and a Mapleson circuit, or have a respiratory therapist accompany the patient with a transport ventilator. The latter frees up our hands but is bulky and cumbersome. A transport monitor is mandatory, and can either be supplied by our workroom or the BICU. Required items also include airway equipment (mask, ET tube, laryngoscope, LMA), emergency and anesthesia drugs, and an IV bag with Y-tubing for rapid infusion if necessary. These patients often have multiple infusions, delivered through a towering assembly of infusion pumps and a bewildering tangle of lines. For these reasons, it is usually helpful to call the ICU nurse about 15 minutes before you anticipate arriving to pick up the patient. Generally speaking, all non-essential infusions, IV piggyback bags, CVP monitoring lines, etc., should be disconnected, which will make your life easier. Advance coordination with the ICU nurse and the RT can make an anesthesia transport much smoother. Always ask the RT about recent secretions and suctioning needs and do in-line tracheal suctioning yourself. Make sure to preoxygenate with 100% oxygen via the ventilator before disconnecting; most RTs forget to do this.

**Minor Burn Surgery**

Examples include dressing changes, surgery to minimal areas of injury, or surgery in a now healthy patient.

**Technique:** general or regional. **Monitors:** standard, arterial line if present. **Duration:** 30min-several hours. **IV access:** one IV should suffice. **EBL:** < 500ml. **Position:** generally supine or prone. **Special equipment:** warming measures may still be necessary. **Special considerations:** By definition, most of the
considerations above for major burn surgery do not apply. These patients are typically healthier with little to none of the whole-body derangements seen with major burns. If a patient has major burn injuries, but is coming for a minor procedure, they should be treated as a major burn patient. Typically these patients are on the burn ward (as opposed to the ICU) or are outpatients returning for reconstructive surgery.
Chapter 4. Obstetric Anesthesia

The Obstetrics service at UCSD has a high volume of procedures and deliveries. In addition, a significant percentage of its patients are “high-risk.” Because of UCSD’s proximity to Mexico and because UCSD performs many of the functions of a county hospital, a large portion of our patients do not speak English, have received little prenatal care, or both. These factors combined with the high-risk nature of the patients make OB anesthesia particularly challenging. This section will address the structure of the OB anesthesia rotation, uterine and labor physiology, and the physiologic changes of pregnancy that set parturients in a class by themselves. Specific anesthetic techniques then follow, including anesthesia for non-obstetric surgery in an obstetric patient.

Chapter 4A. OB Anesthesia Rotation

Residents begin the rotation midway through their CA-1 year. Each month, a new resident will come onto the OB service until every resident in each class has become “OB trained.” This means that about half of any given class will be OB trained by the end of the CA-1 year, with the remaining people finishing the training during the CA-2 year. No prior knowledge of OB anesthesia is anticipated, although it is expected that some basic fundamentals in anesthesia will have already been ingrained. Indeed, the timing of the rotation is specifically designed this way, so that the resident learning OB anesthesia at least has a firm general skill set from which to draw upon (e.g., airway management, administering general anesthesia).

For the entirety of the rotation, the resident works with an attending that is solely dedicated to OB and does not have any rooms in the MOR to attend to. The OB day attending takes over at 0700 and is there until 1900, at which time the OB call attending comes in. At the end of the second week, overnight call begins, at which point the resident should have a firm grounding in OB anesthesia. There are generally 4-5 overnight calls in the month.

The usual day on OB begins at 0640 with morning conference or at 0630 on Wednesdays for M+M. After conference, the OB day resident receives signout from the outgoing OB call resident. There are usually several scheduled procedures throughout the day that will demand our services. These can be found on the main OB board, which is discussed below. Other than scheduled procedures, the OB day resident is responsible for any labor epidural requests and unscheduled procedures (e.g., urgent Cesarean sections) as well as completing post-op checks on patients that received either intrathecal or epidural morphine for post-C-section analgesia the day before. On the weekends, the call person is responsible for completing the post-op checks. While on the OB rotation, the day resident can expect to receive a daily lecture at some point during the day; the OB attendings are generally quite good at this. The shift generally ends around 1600-1700, depending on when the OB call resident for that night is available to take over the OB duties. For more information on the OB call hours, see the section on call responsibilities.

The Labor and Delivery suite (L+D) is comprised of 9 delivery rooms, a recovery room where pre- and post-procedure patients are held, an additional small holding area (the “OB ER”) and 3 ORs (LDR rooms), all surrounding a central area where the OB board and patient charts are kept. LDR3 is the main room used for Cesarean sections. LDR2 is generally held in reserve for a second section or a minor procedure (e.g., tubal ligation). LDR1 is almost never used for services that require anesthesia, but deliveries may take place there. OB patients are also held on the 4th floor antepartum and postpartum suites, and rarely in the SICU. We rarely have any involvement with patients prior to their arrival on L+D, but may
sometimes be consulted on a patient on the antepartum ward on the 4th floor. Parturients also have the option of giving birth in the birthing center on the 4th floor. This only becomes pertinent if birthing center patients require our services (whether for a C-section or an epidural), in which case they must be transferred down to L+D.

Because of the sometimes emergent nature of OB anesthesia, **LDRs 2 and 3 must always be set up for an emergency C-section.** In this respect they are no different than OR7, OR11, and the code bags; see the emergency OR setup section. The expectation is that the rooms will always be restored after use, and that they will be in order when giving signout or handing over OB duties to another resident. The basic setup and checkout of an LDR is as follows, and is essentially the same as ensuring a standard OR is good to go with a few modifications:

- Machine checked.
- Suction functional.
- Airway equipment is ready to go – generally left on top of our anesthesia carts. Smaller ETTs and rescue devices such as an LMA should be available. See the section on physiologic changes of pregnancy for more information.
- Standard monitors, plus an arterial and central line transducer for LDR3, where it is most likely to be employed.
- Stand-alone E-cylinder of O₂ with Mapleson circuit.
- The routine drugs provided to us daily by pharmacy are ephedrine, phenylephrine, etomidate, succinylcholine, and rocuronium. Ensure that at least 20 units of pitocin are present, if not drawn up. Most residents also like to have cefazolin and antiemetics also pre-drawn given that these can be needed urgently; see the section on C-sections.
- Spinal and epidural kits.

The central OB board is the best place to find out at a glance the patients on the OB service and to learn of any impending crises or critically ill patients. The board contains the location of each patient, brief pertinent information such as estimated gestational age, parity and concomitant disease, type of labor analgesia present (if any), scheduled procedures for the day, as well as pager numbers for staff on-call for that day, including anesthesia. The OB anesthesia resident has a dedicated pager with an unchanging number (5090), but it is our responsibility to update the OB board with the relevant name, as well as the pager number and name of the anesthesia attending so the OB secretary knows who to call in an emergency. Further details of the OB board will be explained during the rotation.

**Chapter 4B. Physiologic Changes of Pregnancy**

Pregnancy produces profound physiological changes, many of which have direct impact and implications on anesthetic care. Only with a complete understanding of these changes can one hope to deliver a rational and safe anesthetic to a parturient. This complex physiology will be covered in detail during the OB rotation and comprises a major percentage of board questions.

I. **Cardiovascular System**

Increased maternal and fetal metabolic demands dictate an increase in cardiac output, up to a 40% increase at term. This increase is caused by an increase in both heart rate and stroke volume. Most of the increase occurs during the first trimester, although the greatest increase occurs during labor itself and delivery (up to 80%).
Blood volume is also increased by 35%. A relative increase in plasma volume (45%) to red blood cells causes a relative, dilutional anemia of pregnancy. Typical hematocrits range from 31-35%.

Systemic blood pressure is decreased in pregnancy. The uterus can be thought of as a gigantic, low-resistance circuit in the circulation that acts as a “pressure sink”. Uterine blood flow is 500 to 700 mL/min at term.

II. Pulmonary System

The increased metabolic demands and oxygen consumption of pregnancy are also met by an increase in minute ventilation (50%). Both tidal volume (40%) and respiratory rate (10-15%) increase. A slight chronic respiratory alkalosis develops.

FRC is markedly decreased (20%) due to larger tidal volumes and decreased expiratory reserve volume. In addition, increased abdominal volume and compression of the diaphragm raise closing volume and predisposes to atelectasis and shunting, especially when in the supine position. Because of increased oxygen consumption and reduced FRC, apneic parturients rapidly desaturate.

Rate of uptake and elimination of inhaled anesthetics is increased due to increased minute ventilation and decreased FRC.

Airway mucosal edema is often present, and even the most gentle laryngoscopy can lead to bleeding and airway obstruction. The additional weight of many parturients can make laryngoscopy doubly difficult, similar to an obese patient. As described above, parturients do not tolerate apnea and failed intubation well. For this and other reasons which will be described, general anesthesia is typically avoided in pregnant patients. Intubation should be done gently and with smaller ETTs available.

III. Neurologic System

MAC decreases throughout pregnancy, up to 50% by term. Maternal hormones, especially progesterone, are thought to play a role. Likewise, sensitivity to local anesthetics is increased. This is especially relevant given the large amount of regional anesthetics that are performed in OB anesthesia. Dosing for epidural and spinal anesthesia is typically 20-30% less than for a comparable non-parturient. Decreased epidural space due to engorged epidural veins may be responsible for the propensity for cephalad spread of local anesthetics and the need for a decreased dose.

IV. Hematologic System

There is both a rightward shift of the oxygen-hemoglobin dissociation curve and an increase in 2,3-DPG levels, both of which favor offloading of oxygen to tissues.

The physiologic anemia of pregnancy is discussed above in the cardiovascular section. Pregnancy is a state of marked hypercoagulability, with major increases in various clotting factors. Remember that pulmonary embolus is a major cause of maternal mortality and that this hypercoagulability is the root cause.

IV. Renal, Hepatic, and GI Systems

GFR is increased by up to 50% of baseline. Think of the kidneys having to filter a solute load for both
mother and fetus during pregnancy.

Hepatic function is maintained. Pseudocholinesterase levels are slightly decreased but do not appear to have any clinical effect.

Pregnancy is associated with relative insulin resistance and a propensity towards diabetes. Many patients on our service have gestational if not outright preexisting diabetes. Diabetes predisposes patients to macrosomic fetuses with associated difficult vaginal delivery and increased rate of Cesarean section.

Pregnant patients are always considered “full stomachs” and aspiration risks due to several factors. First, there is increased intraabdominal pressure from the gravid uterus. Second, acidity is increased due to fetal gastrin secretion, increasing the risk of pneumonitis if aspiration were to occur. Third, there is less lower esophageal sphincter tone due to progesterone. The effect on gastric motility is controversial. Some texts state gastric motility is decreased which would increase the likelihood of a full stomach, while other texts state there is no change. Regardless, all pregnant patients undergoing general anesthesia should receive a rapid sequence induction with cricoid pressure. Pharmacologic agents which can attenuate or decrease the risk of aspiration include H$_2$ blockers (decreased acidity, takes time to work), sodium citrate (works immediately to neutralize stomach acid), and metoclopramide (increases gastric emptying and lower esophageal sphincter tone).

Chapter 4C. Physiology of Uterine Blood Flow

The gravid uterus receives an enormous supply of blood, about 600ml/min or 10% of the cardiac output. Of this, 90% goes to the placenta, while the remaining 10% perfuses the uterine myometrium. This high amount of blood flow makes potential losses from bleeding a major concern. Indeed, the most common morbidity associated with pregnancy is severe hemorrhage.

Many factors can decrease uterine blood flow, potentially to the detriment of the fetus. Maternal hypotension is often the most obvious and correctable cause. Due to lack of uterine blood flow autoregulation, flow is directly proportional to systemic pressures. Abnormal systemic vasoconstriction (e.g., preeclampsia) can also constrict uterine vessels and decrease flow. Uterine contractions themselves decrease flow due to both increased venous pressure and decreased uterine arterial flow.

Aortocaval compression is the phenomenon whereby the gravid uterus can compress the aorta and IVC, compromising blood flow and venous return to the heart. This can result in severe hypotension, especially in the supine position. Treatment for aortocaval compression is left uterine displacement. This maneuver involves placing a roll or “bump” under the patient’s right hip/pelvis, in order to displace the uterus to the left and off of the great vessels (especially the IVC). This is a commonly asked board topic and should be one of the first responses to any hypotensive situation. In fact, it is recommended that term parturients should not be allowed to lie perfectly supine but rather should have LUD instituted as a matter of course. In extreme instances, you may see the obstetricians having the patient on their hands and knees to completely displace the uterus.
### Summary Table of Physiological Changes of Pregnancy

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Change during pregnancy</th>
<th>Normal pregnancy values</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiovascular</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate</td>
<td>Increases 15–20 bpm</td>
<td>75–95 bpm</td>
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<tr>
<td>Cardiac output</td>
<td>Increases 30–50%</td>
<td>6–8 l/min</td>
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<tr>
<td>Mean arterial blood pressure</td>
<td>Decreases 10 mmHg in midtrimester</td>
<td>80 mmHg</td>
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<tr>
<td>Systemic vascular resistance</td>
<td>Decreases 10–15%</td>
<td>1200–1500 dynes/s/cm³</td>
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<tr>
<td><strong>Respiratory</strong></td>
<td></td>
<td></td>
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<tr>
<td>Tidal volume</td>
<td>Increased 40%</td>
<td>700 ml</td>
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<tr>
<td>Minute ventilation</td>
<td>Increased 40%</td>
<td>10.5 l/min</td>
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<tr>
<td>Expiratory reserve volume</td>
<td>Decreased 15–20%</td>
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<td>Functional residual capacity</td>
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<tr>
<td><strong>Blood gas</strong></td>
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<tr>
<td>pH</td>
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<td>pCO₂</td>
<td>Decreased</td>
<td>27–32 mmHg</td>
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<tr>
<td>pO₂</td>
<td>Increased</td>
<td>100–108 mmHg</td>
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<td>HCO₃</td>
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<td><strong>Hematologic</strong></td>
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<tr>
<td>Blood volume</td>
<td>Increases 30–50%</td>
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<td>Erythrocyte volume</td>
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<td>Hematocrit</td>
<td>Decreased</td>
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<tr>
<td>White blood cell count</td>
<td>Increased</td>
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<tr>
<td>Factors I, II, V, VII, VIII, IX, X and XII</td>
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<td></td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>Increased</td>
<td>&gt;400 mg/dl</td>
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<td><strong>Genitourinary</strong></td>
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<tr>
<td>Renal blood flow</td>
<td>Increased 50–60%</td>
<td>700 ml/min</td>
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<td>Glomerular filtration rate</td>
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<td>Serum creatinine</td>
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<tr>
<td>Serum urea nitrogen</td>
<td>Decreased</td>
<td>&lt;13 mg/dl</td>
</tr>
</tbody>
</table>

*Bpm: Beats per min.*
Chapter 4D. Placental Drug Transfer

Most anesthetic drugs cross the placenta. This includes inhalational agents, IV induction agents, opioids, and benzodiazepines. Clinically, there is little uptake of inhalational agent by the fetus below concentrations of 1 MAC. Similarly, most IV agents when given in their usual doses have little or no effect on fetal physiology, probably due to first-pass metabolism and redistribution.

Although all opioids cross the placenta, most have little to no depressant effect on the fetus unless large doses are used. Morphine is a notable exception and higher IV doses have been associated with newborn respiratory depression. Epidural and intrathecally administered opioids seem to have little effect on the fetus.

Local anesthetics vary in their ability to cross the placenta. Highly protein-bound agents such as bupivicaine and ropivicaine are quite restricted in the ability to cross the placenta and are a safe choice in pregnancy. Chloroprocaine does not cross the placenta to any great extent, because it is rapidly metabolized in the maternal circulation by esterases. Lidocaine is also safe but crosses the placenta in greater amounts than the aforementioned drugs. The phenomenon of "ion trapping" refers to a potential buildup of local anesthetics in the fetal circulation during conditions of acidosis. Only the un-ionized form of the anesthetic can cross the placenta. During ion-trapping the local anesthetic diffuses across to the fetal circulation, and then becomes ionized by hydrogen ions and unable to “cross back” to the maternal circulation. Under these conditions potentially toxic buildup of local anesthetics in the fetal circulation is possible.

![Diagram of placenta and fetal circulation](image)

Drug (crosses placenta) → Drug + H⁺ → Drug-H⁺ (now trapped in fetal circulation)

Notable agents which do not cross the placenta are heparin, insulin, glycopyrrolate (ionized structure), neuromuscular blockers (highly ionized large molecules) and succinylcholine (highly ionized). These can be remembered by the mnemonic “He Is Going Nowhere Soon.”
Fetal Circulation

The fetal circulation is an important concept to know because of the anesthesiologists’ role in the peripartum period. Oxygen is transferred across the placenta and into the umbilical vein. It then bypasses the liver through the ductus venosus and enters the right atrium. Because of differential of pressure, this blood mainly travels through the foramen ovale and into the left atrium, where it is pumped by the left ventricle into the systemic circulation. The blood that gets into the right ventricle is shunted away from the lungs via the ductus arteriosus and into the aorta. Deoxygenated blood then enters the umbilical arteries and is carried back to the placenta to be reoxygenated.

Chapter 4E. Stages of Labor

Understanding the basic progression of labor is important for anesthesiologists for several reasons. The stage of labor influences the duration and frequency of contractions and can greatly affect our choice of regional anesthetic, and indeed whether a regional anesthetic is even possible. Understanding the stages of labor also helps the anesthesiologist gauge the overall time course to better plan the anesthetic. Lastly, specific pain pathways differ for each stage of labor and are frequently tested on exams.

The first stage of labor begins with cervical dilation and ends when dilation is complete (10cm). The latent phase is typically from 0-4cm of dilation, where the cervix slowly becomes more effaced and dilated (typically over 8hrs). The active phase of the first stage then begins, with more rapid cervical dilation and more intense and frequent contractions. The entire first stage of labor generally lasts 10-12hrs for nulliparous patients and can be much quicker (4-8hrs) for multiparous ones. Pain during this stage of labor is visceral, related to uterine contraction, and involves T10-L1. Analgesic options other than regional anesthesia include IV medication and paracervical blocks. Paracervical blocks carry a high risk of fetal local anesthetic toxicity which manifests as bradycardia and acidosis. This is probably due to the close proximity of the block to the uterine vessels. Paracervical blocks are infrequently employed and are performed by the obstetrician in this institution.
The second stage of labor starts with full cervical dilation and ends with delivery of the baby. It generally lasts 30min to 2hrs. **Pain from this stage is somatic and secondary to vaginal and cervical distension, via S2-4 (perineal and pudendal nerves).** These nerves are notoriously difficult to completely block with epidural anesthesia due to their rostral position and thickness of the nerve roots/fibers. A pudendal (not paracervical) block is another anesthetic option and can be performed by the obstetrician.

The third stage of labor lasts from delivery of the baby to delivery of the placenta. Typically there is minimal discomfort associated with this stage. Occasionally we may be called upon to dose an epidural to provide anesthesia for procedures immediately post-partum, e.g. repair of perineal laceration. This stage generally lasts 15-30min.

**Chapter 4F. Placement and Management of Epidural, Spinal, or CSE Anesthesia & Analgesia**

Similar to the old saying, “a picture is worth a thousand words,” being shown how to do and walked through a procedure is infinitely more instructive than any attempt to explain it through text. Thus, the following sections will not describe how to place an epidural or spinal per se but rather offer useful tips and advice and describe common management at UCSD.

Briefly, the layers that an epidural needle will pass through on the way to the epidural space are: skin, subcutaneous tissue and fat, supraspinous and interspinous ligaments, and the ligamentum flavum. Standard technique for placing a lumbar epidural is to locate the posterior iliac crests (“hip bones”); a horizontal line at this point crosses through the L3-4 interspace. As the spinal cord ends at L1 in adults and the lower nerve roots of the cauda equina are not fixed, any neuraxial block below L1 should have no risk of spinal cord injury and little risk of nerve root injury. The interspinous spaces are palpated and the epidural needle is advanced until the distinct resistance, “toughness,” or “crunchiness” of the ligamentum flavum is felt. At this point, a loss-of-resistance syringe filled with saline or air is attached. The needle is advanced millimeter by millimeter while gentle attempts at injection of the syringe are made. The ligamentum flavum does not allow injection and will bounce back any attempts to do so (resistance). When the epidural space is encountered, there will be a sudden “loss of resistance” with easy injection of saline. At this point an epidural catheter can be placed.

Proper positioning, as with any procedure, is of paramount importance. Confirming proper positioning of the patient can often yield success in a difficult placement. The majority of the time, if an epidural
placement is difficult, a recheck and correction of position will save the day. The patient’s shoulders should be level and the lumbar spine flexed (lordosis eliminated) so as to maximize the space between the interspinous processes. If the patient can tolerate the position, having her sit “Indian-style” may optimize the position.

By starting with your needle midline and keeping it midline, all while insisting on perfect patient positioning, encountering bone at shallow depths is likely to due to hitting a spinous process or simply being off midline. Hitting bone at deeper depths likely represents hitting the inferior aspect of the superior lamina, and walking the needle caudad may help.

Loss of resistance is rarely equivocal; the majority of the time, the difference between the feel of the needle in LF and the needle in epidural space is as different as night and day. Occasionally, a “pseudo loss of resistance” is encountered, where there is a boggy and indistinct ability to inject saline, making it unclear whether the epidural space has actually been reached. Using a small air bubble in the syringe along with saline can help in this regard. Air in the syringe will definitely “bounce back” if the needle is not within the epidural space. Usually “pseudo LOR” represents the needle being in subcutaneous tissue or interspinous ligament. Using only air in the syringe is not recommended due to the possibility of accidental intrathecal injection of air and pneumocephalus.

After loss-of-resistance is encountered, dilating the epidural space with an additional 2-4ml of saline may help with placement of the catheter. A standard length Touhy needle is 9cm from tip to “wings,” and 11cm from end to end. Be sure and note at what depth loss of resistance is encountered as it will guide depth of catheter insertion. This is easily done by counting the remaining centimeter marks on the Touhy needle. With experience this will become habit and second nature; however most people can recall early experiences where an epidural catheter was placed, the needle withdrawn, and then the dawning realization that the appropriate depth of insertion of the catheter was completely unknown.

Try to have a systematic way of preparing the epidural tray and placing the block. One good way is to open all necessary vials, draw up all drugs and the loss-of-resistance syringe and arrange the kit before starting so that everything is readily available. Try to anticipate special needs you might have before putting on sterile gloves and arrange for them beforehand, such as another 3ml syringe and a long spinal needle for a CSE.

The obstetricians typically will consult us to place a labor epidural once labor is established, generally around 2-3cm of cervical dilation. Epidurals placed after 3-4cm of dilation do not slow progression of labor or make Cesarean section more likely; patients or families may ask you this. In general, the later in the first stage of labor the patient is, the more frequent and intense the contractions and the harder it is to place an epidural.

If placing an epidural in the lateral position, the interlaminar foramen (true midline) is almost always above what appears to be midline from visual inspection. As an example, for a patient lying on her right side, true midline is probably slightly left of what her back may look like, due to sagging and the effects of gravity on the soft tissues, pulling them down (towards the right side).

If you encounter a patient in excruciating pain, another option is to place a combined spinal-epidural (a.k.a., CSE), where you achieve loss of resistance as above, then use a spinal needle to place a small amount of local anesthetic and/or fentanyl intrathecally for immediate relief, and then place the epidural catheter per usual routine. Be sure to use a spinal needle that is longer than the epidural needle. Benefits of this technique include immediate relief of pain and confirmation that your epidural
catheter is just separated from CSF by the dura. However, it runs the risk of “uterine hypertonic syndrome,” caused by the sudden withdrawal of catecholamines and a subsequent loss of β2 agonism by epinephrine. This leads to a hypertonic uterus and can cause fetal distress that, if intense enough or lasts long enough in duration, may necessitate an emergent C-section. Diagnosis is made with palpation of a “rock-hard” abdomen, tetanic contractions on the tocometer tracing, and fetal decelerations on the fetal heart rate monitor. To avoid this, the anesthesiologist must keep the diagnosis in their differential, and have uterine relaxants (tocolytics) ready. Some of these include terbutaline (a β2 agonist), sublingual nitroglycerin, and calcium-channel blockers. The OB anesthesia carts located in the L&D hallways all have sublingual nitroglycerin spray in the drug tray.

**Tips for Spinal (SAB) Placement**

In general, many of the same comments for epidural placement can be said about spinal placement as well. In many respects, placing a spinal (“subarachnoid block,” SAB) is technically easier than an epidural as there is no need to find the occasionally-elusive epidural space, and the endpoint for the block is objective: CSF return. When the dura is punctured, a distinct “pop” is usually felt. At this point, removal of the stylet should produce free-flowing CSF.

Before injection of the spinal anesthetic, aspirate a small volume of CSF. It should be easy and free flowing, and will visually “swirl” in the syringe due to the difference in dextrose concentration. If this is not evident, **DO NOT inject**; it is likely the needle tip is no longer within the subarachnoid space and the block will fail. It is much better to simply reposition the needle or attempt the block again.

For extremely obese patients, a needle larger than the standard 25g may be needed for rigidity, or a longer needle may be needed to reach the subarachnoid space, or both. In extreme instances, it may be useful to use a Touhy needle as an introducer for a spinal needle for added structural stability and stiffness.

In OB anesthesia, spinals are generally reserved for surgical procedures such as Cesarean section, tubal ligation, or potentially a very low dose for repair of perineal laceration. Rarely, a dilute spinal may be used as the sole anesthetic for labor, for example, if the parturient is nearly complete but in extreme discomfort and there is not time to place an epidural. Most, however, would place a CSE in this scenario.

Please consult an appropriate textbook for more complete and thorough explanations of epidural and spinal techniques.

**Epidural Analgesia for Labor**

The following describes common approaches to labor analgesia at UCSD. The primary choice of anesthetic technique is “regular” epidural vs. CSE. Reasons to place a CSE are described above, and include rapid relief for a particularly uncomfortable parturient or short-lasting relief for a particularly late-in-labor neuraxial block placement.

The most common approach at UCSD is as follows. Typically around 3-4cm of dilation, i.e., when the OBs have confirmed the patient is in true labor (contractions with cervical change), you will be consulted to place a neuraxial anesthetic/analgesic. After a brief history and physical, confirmation of pertinent coagulation studies (platelet count ± INR/PTT), and discussion of the risks/benefits/alternatives, the patient is placed in the sitting position. DuraPrep is used as the antisepsis, and an epidural kit with a 17g Tuohy and a 19g flexible single-orifice catheter is prepared. Local anesthesia with 1% lidocaine is done,
loss of resistance is found, the catheter is advanced usually 2-5cm into the epidural space, and a “test
dose” of 1.5% lidocaine with 5mcg/ml epinephrine is given through the catheter to rule out IV and
intrathecal placement. You must always aspirate the epidural catheter prior to giving any medications
through it.

In the case of a negative test dose, the epidural is “loaded” with increments of 3-5ml at a time, to a total
of 5-10ml, of any combination of medications. Choices include 1% lidocaine for fast analgesia, 0.25%
bupivacaine for somewhat slower but excellent analgesia, the “bag solution” of 0.1% bupivacaine with
2mcg/ml fentanyl, or any of the above with 50-100mcg of fentanyl. The patient-controlled epidural
analgesia (PCEA) infusion is programmed and started, with settings most commonly 8-10ml/hr
continuous with 5-6ml q20-30min demand.

In the case of a CSE, at the time of loss of resistance, a 5” 25g spinal needle is passed through the Tuohy
and CSF is encountered. At this time, 1-2ml of 0.25% bupivacaine, fentanyl, or a combination of the two
can be given. Then, the catheter is threaded and tested, with the caveat that intrathecal catheter
placement can no longer be ruled out with the test dose.

Rarely, a CSE is done without giving intrathecal medications; this is known as a “dural-puncture
epidural.” This might be done simply to confirm placement of the Tuohy in the epidural space without
confounding the test dose, or with the idea that the analgesia might be of a better quality with this
technique. Regardless, any time the dura is punctured, the risk of post-dural-puncture headache is
increased.

Approaches to troubleshooting labor pain in patients with epidural catheters will be discussed in great
depth during the OB anesthesia rotation.

Lastly, an in situ epidural catheter can be used for a semi-elective or urgent Cesarean section, as
described next.

Chapter 4G. Anesthesia for Cesarean Section

Cesarean section is the single most common operation in the United States. Indications for Cesarean
section are myriad and range from the innocuous to the emergent. A particular challenge for the OB
anesthesiologist is balancing two demands: the sometimes-frantic requests from the obstetricians to
proceed immediately with Cesarean section, and the best interests of both mother and child. In general,
the indications for Cesarean section fall into one of several broad categories:

I. Urgent or Emergent C-section
   - Bleeding
   - Risk of infection (chorioamnionitis or herpes with ruptured membranes)
   - Fetal distress
   - Maternal death
   - Umbilical cord prolapse

II. Abnormal fetal presentation, or failure of labor to progress

III. Unsafe labor for fetus or mother
   - Abruption
   - Placenta previa, accreta, increta or percreta
   - Previous uterine or vaginal surgery (including prior C-section)
Multiple gestations

IV. Elective (e.g., patient desires)

General Anesthesia and the Emergency C-section

Truly emergent Cesarean sections necessitate the use of general anesthesia. Even if the patient has an indwelling epidural catheter, the time needed to dose and establish a surgical block is unacceptable when the Cesarean section is truly emergent. This situation needs to be discussed on a case-by-case basis with the obstetrician. Because of the risks of aspiration and failed intubation with parturients, as well as the eight-fold higher increase in maternal mortality, it is prudent to avoid general anesthesia unless the need is truly emergent and the benefits (speed) outweigh the risks.

General anesthesia for emergent C-section does not proceed until the obstetrical team is scrubbed and gowned, with the patient’s abdomen prepped for immediate incision. During this time, standard monitors should be placed and the patient preoxygenated. Four vital capacity breaths, although not as effective in total body oxygenation as 5 minutes of breathing 100% O₂, should suffice. An assistant should be present to help with cricoid pressure or with a difficult airway. When all team members are ready, general anesthesia is induced with rapid sequence induction with an ETT and cricoid pressure. Establishment of GA must be clearly and quickly communicated to the OB team, who should make incision as soon as the patient is unconscious and etCO₂ is confirmed. The goal is to deliver the fetus as quickly as possible from the time of induction. Typically, from the time of induction to delivery of the fetus, a high-volatile-agent anesthetic is provided to assist with uterine relaxation. After delivery, a high-nitrous-oxide, low-volatile-agent anesthetic is provided to prevent uterine relaxation.

The remainder of the case can proceed as in a non-emergent Cesarean section.

Anesthesia for the Non-emergent C-section

**Technique:** general, epidural, spinal, or CSE. **Monitors:** standard. Invasive monitoring is generally not necessary unless warranted by concomitant disease (e.g., severe preeclampsia). **IV access:** one large IV is generally sufficient. **Duration:** 45min-2hrs. **EBL:** 800-1200ml, may be more depending on uterine tone or lack thereof. **Position:** supine with left uterine displacement. **Special equipment:** none. **Special considerations:** as below.

The progression of a typical Cesarean section is as follows:

- Regional anesthesia is induced and appropriate sensory level confirmed, patient prepped and draped. Or, the patient is prepped and draped in preparation for general anesthesia, and GA is induced and the airway is controlled.
- Antibiotics should be given prior to surgical incision.
- Skin incision is made and surgery proceeds.
- Uterine incision is made. Times above 3min from uterine incision to delivery have been shown to correlate with lower Apgar scores and fetal acidosis.
- The fetus is delivered, the umbilical cord is clamped, and delivery of the placenta is started.
- Management of uterine atony after delivery consists of pharmacologic and mechanical strategies. Pharmacologic techniques include oxytocin (Pitocin), methylergonovine (Methergine), and carboprost (Hemabate). Mechanical techniques include routine suture closure, uterine massage, and the “B-Lynch” locking suture.
- **Oxytocin** is given with every C-section. The typical dose is 20 units, which is diluted in a 1L IV bag and infused over 20-40 minutes, starting immediately after delivery. Oxytocin induces uterine contractions and helps maintain uterine tone. It lowers SVR in a dose-dependent fashion by relaxing vascular smooth muscle, and can induce systemic hypotension if given in large or fast doses. The OBs may ask you to “double the Pit,” i.e., give 40 instead of 20 units, often before the initial dose of 20 has had any time to have a physiologic effect. Take care with double-dosing given the hypotensive effect of oxytocin described above.

- **Methylergonovine** is an ergot alkaloid which causes sustained uterine contraction. It is only given postpartum and causes smooth muscle contraction throughout the body, potentially resulting in hypertension and bronchoconstriction. Its major contraindication is hypertension, whether pre-existing or pregnancy-induced. The dose is 0.2mg IM; in a patient with a neuraxial block, give this in a leg. Side effects include nausea and vomiting.

- **Carboprost** is 15-methyl-prostaglandin-F₂₀₅, i.e., a prostaglandin analog, which causes uterine contractions. It is given IM, 0.25mg at a time. Its primary side effect is bronchoconstriction, and so its major contraindication is asthma/reactive airway disease. Other side effects include diarrhea, nausea, and vomiting.

- Manual uterine massage can also help with uterine atony, and so does surgical closure of the uterus with sutures, which the OBs should be doing expeditiously in the setting of atonic bleeding.

- The uterus is “exteriorized” to aid with exposure during surgical closure. If regional anesthesia is employed, it is common at this stage for the patient to have an uncomfortable sense of pressure due to the peritoneal traction and unblocked vagal afferents. Additionally, the OBs tend to reach with lap pads toward the upper abdomen (foregut) which is not blocked by a typical T4-T6 sensory level. Reassurance with or without small doses of ketamine or fentanyl or nitrous oxide may be helpful.

- The uterus, fascia, and skin layers are closed.

Regional anesthesia is often preferred due to less risk to the mother as described above, and because it allows the parturient to be awake and alert at the time of delivery. A T4 sensory level is necessary. Advantages of SAB versus epidural include a more profound and reliable block, quicker onset, and perhaps easier placement. Disadvantages include lack of titratability, inability to re-dose in the event the case is longer in duration than expected, and more profound hemodynamic changes.

Typical dose ranges for a SAB are 1.4-1.8ml of hyperbaric 0.75% bupivacaine. Most will add 10-25mcg of fentanyl and 0.1-0.2mg of morphine to the mixture. This can be drawn up steriley beforehand and given to an assistant to inject into the spinal syringe prior to administration. Fentanyl is thought to improve the quality of the block, although firm data are lacking on this point, and morphine provides good analgesia for up to 24 hours. Epinephrine 200mcg is rarely used to prolong duration of the blockade. Prior to placement of any regional block, the patient should have at least 1L of crystalloid fluid bolus, standard monitors, O₂, and the relevant history/physical/labs checked. Hypotension immediately after SAB is common and should be aggressively treated with fluids and vasopressors. Many people often administer nausea and vomiting prophylaxis at this time. Indeed, one of the first manifestations of the onset of hypotension is maternal nausea and vomiting.

When epidural anesthesia is employed, it is usually because the patient has a prior labor epidural in place and then develops an indication for Cesarean section, such as failure to progress, arrest of descent, or fetal distress. Less commonly, it is employed as part of a CSE and as an alternative to a spinal; CSE may be chosen if the operation is expected to take a long time. An epidural also offers
titratability, unlike SAB, which could be beneficial in a patient with severe preeclampsia or severe aortic stenosis.

Two options to speed the onset of epidural anesthesia include the use of 3% chloroprocaine or 2% lidocaine with 5mcg/ml epinephrine alkalinized with NaHCO₃. The mixture is prepared as 1ml NaHCO₃ added to every 9ml of 2% lidocaine. The usual doses are 15-20ml of either medication, titrated in increments of 5ml. Epidural fentanyl can also be added (50-100mcg). Chloroprocaine needs to be redosed about every 45min, lidocaine every 1-2hr, and bupivacaine every 1.5-3hrs. Epidural morphine, for sustained post-op analgesia, is typically given after delivery of the fetus; the dose is 2-4mg. Occasionally, an epidural block may be incomplete or patchy (“hot spot”). Small doses of ketamine (10-20mg IV at a time) or narcotics can be of great assistance during these times, with special care and vigilance as always. Ketorolac 30mg IV should be given to all C-section patients at the end of surgery, in whom it is not contraindicated by allergy, hemorrhage, or renal dysfunction.

If general anesthesia is employed, the patient should not be induced until the obstetrical team is ready as described above for emergency C-section. A rapid-sequence induction with cricoid pressure is necessary. Anesthesia can be maintained with many agents: 50% O₂ and 50% N₂O with 1% sevoflurane is common in our institution. Often, nondepolarizing muscle relaxant is necessary to facilitate surgery and to prevent interference from maternal respirations. Keep in mind the decreased MAC requirements of the parturient. Emergence is typically uncomplicated and usually requires only increased vigilance in regards to the airway and risk of aspiration.

A tubal ligation may be combined with the C-section in patients desiring sterilization. It is performed after delivery of the fetus and uterine closure, and typically adds 10-15min to the procedure. A repeat C-section, or a primary C-section in a patient with previous abdominal surgeries and scar tissue, can take significantly longer than one in a “virgin belly” due to adhesions.

It is very common for spouses or family members to be present for the operation. They generally are brought to the delivery room after the patient is prepped and draped but prior to skin incision. This lone family member – and you are advised to insist that just one person be present – is often placed very near to the anesthesiologist, sitting near the head of the bed and away from viewing the surgical field. Allowing family to be present should always be a balance between optimal care and giving the patient and family what they desire. The extra family member should never be allowed to distract the anesthesiologist from his or her duties. However, there are also times when they can be of great assistance in reassuring and calming a hysterical patient.

Rarely, the OBs may ask for sublingual nitroglycerin to be administered to aid with uterine relaxation. This can be given safely as long as attention is paid to maternal blood pressure.

**Chapter 4H. Anesthesia for Placenta Accreta/Increta/Percreta**

UCSD is privileged to be a center that performs many deliveries for patients with placental abnormalities. There are three types: placenta accreta is when the placenta grows into the uterine endometrium, increta is when it grows into the myometrium, and percreta is when it grows completely through the uterus and may invade nearby structures such as the bladder or bowel. Because the placenta has grown into the
uterus abnormally, it does not separate cleanly during delivery and can cause massive bleeding. Thus, the patient with any of these conditions cannot be allowed to labor but must instead have delivery by Cesarean section. A hysterectomy is almost always indicated to control bleeding and allow removal of the placenta. Other concurrent procedures may be necessary to remove the placenta from surrounding structures.

The Cesarean hysterectomy for these cases is unique, as are the anesthetic goals. Historically, these patients are taken to interventional radiology where uterine artery balloons are placed, and can be inflated during the procedure to control bleeding. An epidural is generally placed by us to provide anesthesia during interventional radiology and for the Cesarean portion of the case. Once delivery is complete, general anesthesia is induced to provide better operative conditions and better control in case of overt bleeding. There is usually a perfusionist on standby in case massive volume resuscitation becomes necessary. Due to the likelihood of severe bleeding and volume resuscitation, an arterial line and large IV access are mandatory. These are placed pre-induction since there is no time to place them intraoperatively once the need becomes apparent.

As these patients are generally young healthy women, they tolerate blood loss and resuscitation fairly well. These cases combine the challenges of a Cesarean section and maintaining maternal and fetal well-being, with the complexity of a large intra-abdominal case with the potential for massive blood loss.

There are several approaches to the anesthesia for Cesarean hysterectomy, including doing the Cesarean portion under epidural anesthesia and the rest of the procedure under general, or the entire procedure under general without an epidural. Often a large incision is necessary to completely expose the uterus and allow the OB’s to work around the placenta. This incision is often poorly covered by an epidural. Furthermore, a neuraxially-induced sympathectomy is undesirable in situations with potentially large blood loss. Inducing GA in a patient with a preexisting T4 sympathectomy is potentially dangerous. About the only advantage of using an epidural for the first portion of the procedure is that the mother can be awake for the delivery of the baby.

As previously stated, uterine contractions and placenta separation can be catastrophic and should be avoided at all costs. Tocolytics may be necessary, and oxytocin should be avoided at all costs. In this regard, GA may be superior to neuraxial anesthesia in causing uterine relaxation.

**Technique:** general ± regional anesthesia. **Monitors:** standard, arterial line. **IV access:** Large. Frequently a large central line is placed. **Duration:** 2-4hrs. **EBL:** 1L and up, potentially many liters. **Position:** supine with left uterine displacement. **Special equipment:** perfusionist available. Fluid warmers for volume resuscitation. **Special considerations:** as above.

Induction of GA for this procedure requires the usual precautions for GA in parturients, including preparing a ramp for intubation, smaller ETTs, and rapid sequence induction with cricoid pressure.

**Chapter 4I. Anesthesia for Other Obstetric Procedures**

This category includes postpartum tubal ligations, cerclage placement or removal, and other minor procedures.

**Technique:** regional or general, with preference again towards a neuraxial technique. **Monitors:** standard. **IV access:** one IV. **Duration:** 30min-1hr. **EBL:** < 100ml. **Position:** supine or lithotomy. **Special equipment:** none. **Special considerations:** Generally a lower sensory level is necessary due to smaller
incisions and the lower nature of the procedures. A T6 level is more than adequate even for a postpartum bilateral tubal ligation. Similarly, intrathecal or epidural morphine is not given, as these patients tend to be discharged the same day.

**Chapter 4J. Anesthesia for Non-obstetric Surgery in the Parturient**

In general, all elective surgeries should be postponed until at least six weeks postpartum. Truly urgent, emergent, or necessary surgery should proceed with the following items in mind.

- The 3rd to 10th weeks of pregnancy are when major organogenesis occurs and when the fetus is most susceptible to teratogens.
- There is ongoing concern about the teratogenic potential of N₂O and benzodiazepines, despite lack of conclusive evidence. These agents should probably be avoided. Most of our other anesthetic agents have been proven safe in clinical concentrations.
- Most clinicians feel a parturient at 20 weeks gestation or beyond should be considered to have all the physiologic changes of pregnancy described above. Others feel that parturients as early as 12-16 weeks should be treated with the same caution with regards to physiologic changes, airway management, risk of aspiration, and so on.
- Any surgery can induce preterm labor, but those at particular risk are lower abdominal surgeries. Typically the OB team will monitor the fetus and uterine activity from induction of anesthesia to the completion of the procedure. Coordination between teams is often necessary. If coordinated uterine activity is detected, β₂ agonists or magnesium may be employed as tocolytics. A plan for delivery of the fetus should be in place should the pregnancy be threatened.
- Regional anesthesia is preferred if possible, for the same reasons as described above. However, the specific anesthetic technique is determined by the type of surgery needed.

**Chapter 4K. Special Topics in OB Anesthesia**

I. **Hypertensive Disorders of Pregnancy**

A. **Gestational hypertension (Pregnancy-induced hypertension (PIH))**

Hypertension in pregnancy is abnormal. As stated earlier, the normal physiology of pregnancy produces a drop in systemic blood pressures from baseline. Hypertension in pregnancy can be preexisting, or due to the pregnancy itself. It is defined as a systolic pressure above 140 or a diastolic pressure above 90 that develops after 20 weeks of pregnancy and resolves within 12 weeks postpartum. By definition, this is a retrospective diagnosis because it can only be established after delivery.

B. **Preeclampsia, Eclampsia and HELLP syndrome**

Preeclampsia is defined by hypertension and proteinuria (>300mg in a 24 hour urine collection). Eclampsia is much rarer and includes seizures. Severe preeclampsia is defined as blood pressure over 160/110, greater than 5g/d of proteinuria, or signs of end organ damage (headache, seizures, vision changes, hepatic tenderness or rupture, pulmonary edema, oliguria). HELLP syndrome refers to the development of Hemolysis, Elevated Liver enzymes and Low Platelets, and is considered a variant of severe preeclampsia.

PIH and preeclampsia is more likely in nulliparous young women and those with a previous history of
PIH. As a rule, these women are prone to vasospasm, are intravascularly volume depleted, and are edematous.

Treatment of PIH includes bed rest, antihypertensive therapy (hydralazine or labetalol are common), and magnesium for seizure prophylaxis. The only definitive treatment is delivery of the fetus and placenta. PIH behaves like an immune reaction to the fetus, with resolution after delivery.

Typically, we as anesthesiologists tend to become involved early in the management of a patient with PIH. Often, the OB service will inform us of any patients with PIH due to their propensity to progress to Cesarean section and their risk of thrombocytopenia. It behooves us to know of these patients so that we can evaluate them early and potentially assist with management. Furthermore, these patients often need invasive monitoring, which the OBs have little to no experience with, and they will consult us in these cases as well.

Anesthesia for the patient with PIH depends on the severity of disease. Epidural anesthesia tends to be the best choice due to titratability and gradual onset of action. Furthermore, a decrease in catecholamines from neuraxial blockade has been shown to improve uteroplacental perfusion. Hypertension and relative hypovolemia should be treated as much as possible before anesthesia. Coagulation parameters and platelet count should be checked prior to the initiation of neuraxial blockade, with particular attention paid to the trend of these factors. In some cases, platelet transfusion may be necessary prior to initiating a block.

Magnesium Therapy

Many parturients that we come across will be on magnesium therapy for a myriad of reasons, including seizure prophylaxis in preeclampsia and neonatal neuroprotection for women in labor with severely premature babies. Magnesium has many effects on the body and our anesthetics. The therapeutic range for magnesium is 5-9 mg/dl. The patients will typically get a bolus followed by a continuous infusion. They are followed by serial magnesium levels and clinical exam to ensure they are not exceeding toxic levels. The first sign of magnesium toxicity is loss of deep tendon reflexes, followed by respiratory depression, and rarely, cardiac arrest. Treatment of suspected magnesium toxicity includes discontinuation of the infusion and administration of 1g calcium gluconate IV. If there is respiratory compromise, ensure that ABCs are being addressed.

Magnesium potentiates neuromuscular blockade, so a patient on magnesium therapy who requires general anesthesia requires less muscle relaxant. Administering a normal dose could potentially result in a longer duration of paralysis. Magnesium can also cause hypotension and decrease uterine tone, putting these patients at higher risk for uterine atony and increased bleeding during a C-section.

II. Uterine Rupture

This is a life-threatening but rare condition (1:2000 pregnancies). Predisposing factors include prolonged labor with a large fetus, prior uterine surgery, augmented contractions (e.g., oxytocin) or external manipulation of the uterus (e.g., version for breech presentation). It typically presents as abrupt onset of abdominal pain with fetal distress and hypotension, and may classically include palpable fetal parts on abdominal exam. Treatment includes resuscitation, immediate laparotomy and delivery of the fetus, and repair of the uterus or frank hysterectomy.

This is a frequently tested topic on board exams. The possibility of uterine rupture in patients
undergoing **VBAC (vaginal birth after C-section)** means that these patients should be identified and evaluated by us as soon as possible. Epidural anesthesia generally will not mask the signs of uterine rupture, and in fact may help in identifying it when a previously comfortable (due to the epidural), laboring patient suddenly develops abdominal pain.

III. **Placenta Previa**

This complication occurs when a placenta attaches to the uterine wall and covers the internal cervical os, either completely or partially. A placenta previa places the patient at higher risk of bleeding during the pregnancy, and if severe enough, necessitates Cesarean section. The placenta can either be low-lying (near the cervical opening but not covering), marginal (touching the os but not covering it), partial (part of the placenta is covering the os) or complete (completely covering the os). Parturients with prior C-sections are at higher risk for placenta previa, as well as placenta accreta, increta and percreta. These patients can bleed profusely. The OB team will usually alert us if there is a parturient with placenta previa because she will usually need a C-section.

IV. **Placental Abruption**

Abruptio occurs when there is abnormal separation of the placenta, with bleeding into the space between the placenta and uterine wall. Abruptio tends to present as **painful** vaginal bleeding and can cause severe fetal distress or demise. Diagnosis is made by ultrasound. Predisposing factors include hypertension, multiparity, drug abuse, and an abnormal uterus.

Vaginal delivery can be undertaken with a mild abruption. However, any sign of fetal distress or large abruption is an indication for emergency C-section. Bleeding can be substantial. Additionally, release of thromboplastins into the maternal circulation can cause frank DIC and coagulopathy. In all these cases general anesthesia and resuscitation must be employed.
V. Amniotic Fluid Embolism

This is a rare (1:20,000) condition in which the mother has an acute and profound immunologic reaction to fetal tissue, usually amniotic fluid, entering the circulation. It has a very high mortality (over 50%). Most AFs occur during labor (90%) but they can also occur during C-section and even postpartum. The presentation is generally a sudden onset of tachypnea, respiratory distress, and circulatory collapse. AFE can mimic pulmonary embolism, but also shares features of circulatory and pulmonary compromise similar to septic shock and ARDS. DIC develops and leads to coagulopathy and massive bleeding. Hypoxemia, shunting, and increased dead space all occur. The treatment for AFE is supportive. Delivery of the fetus must occur for there to be any chance of effective maternal and fetal resuscitation.

VI. Apgar Scores

This is another frequently tested topic on board exams. Apgar scores range from 0-10 and are measured at 1 and 5 minutes. The 1-minute score correlates with survival, while the 5-minute score correlates with neurologic outcome. The five components of the Apgar score are as follows:
### APGAR SCORING SYSTEM

<table>
<thead>
<tr>
<th></th>
<th>0 Points</th>
<th>1 Point</th>
<th>2 Points</th>
<th>Points totaled</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Activity</strong> (muscle tone)</td>
<td>Absent</td>
<td>Arms and legs flexed</td>
<td>Active movement</td>
<td></td>
</tr>
<tr>
<td><strong>Pulse</strong></td>
<td>Absent</td>
<td>Below 100 bpm</td>
<td>Over 100 bpm</td>
<td></td>
</tr>
<tr>
<td><strong>Grimace</strong> (reflex irritability)</td>
<td>Flaccid</td>
<td>Some flexion of Extremities</td>
<td>Active motion (sneeze, cough, pull away)</td>
<td></td>
</tr>
<tr>
<td><strong>Appearance</strong> (skin color)</td>
<td>Blue, pale</td>
<td>Body pink, Extremities blue</td>
<td>Completely pink</td>
<td></td>
</tr>
<tr>
<td><strong>Respiration</strong></td>
<td>Absent</td>
<td>Slow, irregular</td>
<td>Vigorous cry</td>
<td></td>
</tr>
</tbody>
</table>

- **Severely depressed** 0-3
- **Moderately depressed** 4-6
- **Excellent condition** 7-10
Chapter 5. Cardiothoracic Anesthesia Rotation and Cardiovascular Physiology

Chapter 5A. Cardiothoracic Anesthesia Rotation

The cardiothoracic anesthesia rotation at UCSD is an intensive two-month exposure to cardiothoracic physiology, anesthesia, and surgery. Beginning in the middle of the CA-1 year, one resident at a time begins the rotation each month. Thus, by the middle of the CA-2 year, an entire class will have completed their cardiac rotation. The first month is spent primarily at the Sulpizio Cardiovascular Center (CVC), while the second month is at the VA. At the CVC, you will have lots of one-on-one time with the cardiac anesthesia fellow and cardiac attending to cover the most critical information and basics of cardiac anesthesia, especially during the first week. At the VA, your attending will always be covering another room and there is usually no fellow, in accordance with your increased experience.

The cases encountered at Sulpizio will generally be pulmonary thromboendarterectomies (PTEs), on- and off-pump CABGs, and valve replacements. Thoracic cases happen with some regularity but with lower volume. PTEs are not performed at the VA, but CABGs and valve cases are common. It is exceedingly rare for cardiothoracic cases to be done at Hillcrest. In general, the heart resident does any cardiothoracic case available; if there are none scheduled on a given day, you will be assigned a general OR case.

UCSD also hosts visiting anesthesiology residents from the University of New Mexico, who are in San Diego for a month and need the cardiac volume that we have at UCSD to achieve their ACGME-mandated minimum case requirements.

Call during the two months is also discussed in the section on call responsibilities. During the first month you will be assigned “heart call” at the CVC during many days of the week. This is pager call, and you are responsible for returning to the hospital for emergency cases (e.g., “takebacks” for bleeding, lung or heart transplants). On weekends, you will be called in for the same or for elective cases. As the heart call person, you may be asked to finish an ongoing cardiac case at the CVC after you have finished your main OR cases for the day. This is at the discretion of the attendings. **Expect to work hard during these two months** and take these things in stride.

At the VA you will be part of the regular VA call pool and assigned 4-5 in house calls for the month. You will be given any cardiothoracic case available. Cases usually have a 0730 start time, as opposed to the CVC, which start at 0630. There is usually a CT case every day except for Wednesday, so you will typically be on-call on Tuesday and post-call on Wednesday. Call at the VA normally starts at 1000, but on Tuesdays you will come in early to do the CT case for that day. While at the VA, you are not part of the CVC heart call pool, since the resident one month “behind” you is covering those duties.

Cardiothoracic cases are demanding, and require that you “own” your patient. Accordingly, it is expected that you will be more responsible than “usual” for pre-opping your patients. Many elective cardiac patients at CVC go to our regular pre-op clinic, as do all the patients for elective cases at the VA, so the task of pre-opping has been taken care of for you. PTE patients, however, are usually from out of state, are admitted the day prior to surgery, and will not have gone to pre-op clinic. You will have to see the next day’s patient and review the chart prior to calling your attending and heading home for the day. So, a typical day during the first month of cardiac starts early and ends late, after you’ve wrapped up your cases and seen the one for the next day. Again, expect to work hard during these two months.

You should allow at least one hour or more to set up your room when you are first starting the rotation.
There is a lot to do and in addition you will be unfamiliar with many of the necessary steps, like making all the drips. Also, your patient needs a good IV and an arterial line before induction. Add to this the fact that patients at CVC should be in the OR by 0630. So, you may find yourself having to come into the hospital at 0500 in the beginning of your cardiac month. In general, allow yourself more rather than less time in the beginning. You can always make adjustments later as you get faster, but it looks bad when everyone but the anesthesiologist is ready. Specifics of the room setup are covered later, but are best addressed in the departmental cardiac rotation syllabus.

Cardiac surgeons are notorious throughout the country as being “difficult to work with.” In general, at UCSD this is not true. Our CT surgeons are usually quite friendly and will make an effort to learn your name. Keep in mind that the field of cardiac surgery is under pressure to standardize and protocolize care to improve outcomes, so cardiac surgeons who see a new, green anesthesiology resident come on the rotation each month can be somewhat suspicious of us. Also, cardiac surgeons understand the patient’s physiology better than any other type of surgeon; just imagine a hypothetical discussion of valvular disease with an orthopedist. This tends to create situations where the CT surgeons basically tell you what they want you to do with your management and anesthetic. As with all things, being political and working as a team is the best way to go. Remember you can always defer to your attending if you are unsure. With time and greater experience you will learn what things you can let slide and which battles are truly worth fighting.

The cardiac months will be some of the most educational during your entire time at UCSD. Most people feel that after their heart months there is no case that is too big for them. In addition, you should have gained a firm understanding of basic cardiovascular physiology and anesthesia. It is demanding work but well worth it.

**Chapter 5B. Coronary Anatomy and Circulation**

Blood flow to the myocardium is supplied by the right and left coronary arteries. The right coronary supplies the RA, most of the RV, and the inferior wall of the LV. It typically gives rise to the posterior descending artery (85% of the time), which supplies the posterior interventricular septum and the inferior wall. This is termed “right-dominant” circulation. The remaining 15% of the time the PDA arises from the left coronary: left-dominant circulation. The SA node is supplied by the RCA 60% of the time. The AV node is almost always supplied by a branch from the RCA.

The left main supplies the LA and the anterior, lateral, and septal walls of the LV. It quickly divides into the LAD and the circumflex artery. The LAD supplies the anterior wall of the LV and most of the septum and gives rise to diagonals, while the circumflex supplies the lateral wall and gives rise to the obtuse marginals.

Most coronary venous return is through the coronary sinus and anterior cardiac veins into the RA, although a small amount drains into the LA through the thebesian veins, representing a portion of physiologic shunt.

Perfusion occurs anytime there is coronary flow, which in turn occurs anytime aortic pressure exceeds intrachamber pressure. So, the RV (normally < 40mmHg) is perfused during both systole and diastole, while LV perfusion occurs almost entirely during diastole. The endocardium is the area most sensitive to ischemia because the highest transmural pressures and lowest perfusion pressures are encountered here.
Myocardial Oxygen Supply and Demand

Normal coronary blood flow is regulated almost entirely by constriction or dilation of coronary vessels in response to metabolic demand. Myocardial oxygen extraction is ~60%, compared to 25% for most of the body, so myocardium cannot compensate for increased oxygen demand by increased extraction. Flow must increase to meet any increased demands. Pathophysiology that inhibits the coronaries to increase supply (e.g., coronary atherosclerosis, already maximally dilated vessels in response to chronically high demand) can lead to ischemia if metabolic demand increases. Much of cardiac anesthesia is goal-directed therapy to improve myocardial oxygen supply/demand characteristics.

The determinants of myocardial oxygen supply are:

- **Coronary perfusion pressure (DBP – LVEDP)**
  - Increasing arterial pressure, especially diastolic pressure, and reducing LV end-diastolic pressure will increase CPP.
  - The converse of the above also holds.

- **Coronary vessel caliber**
  - Reductions in vessel diameter (e.g., coronary vasospasm or atherosclerosis) reduce the ability to delivery blood flow and oxygen.
  - Similarly, measures to dilate coronary vessels (e.g., nitroglycerin) can improve flow.

- **Arterial oxygen content**
  - Determined by the hemoglobin concentration, oxygen saturation, and \( P_{a}O_2 \).
  - Increases or reductions in either are directly linked to oxygen supply.

- **Heart rate**
  - The LV is perfused in diastole.
  - The slower the HR, the more time for diastole.

The determinants of myocardial oxygen demand are:

- **Heart rate**
  - Each contraction consumes oxygen.
  - Faster heart rates increase myocardial oxygen consumption in a linear fashion.

- **Wall tension**
  - The primary determinants of wall tension are chamber size, chamber wall thickness, and afterload.
  - Chamber size is determined by preload and inherent ventricular size.
  - Chamber wall thickness is fixed at a given point in time.
  - Afterload increases wall tension.

- **Contractility**
  - Increases in contractility increase oxygen consumption.

Chapter 5C. Anesthetic Goals in Cardiac Disease States

Each cardiac lesion has specific considerations for management during anesthesia. These considerations apply to cardiac patients coming for surgery for the lesion (e.g., CAD in a patient coming for a CABG) as well as patients having non-cardiac surgery who have one of these lesions as a comorbidity (e.g., critical AS in a patient coming for hip fracture repair). When multiple lesions coexist and their management is disparate, e.g. AS coexisting MR, the “dominant” lesion takes priority in management. The specific goals
during anesthesia are discussed below.

**Coronary Artery Disease**

Patients with CAD presenting for CABG usually have disease in at least 2 of the 3 major arterial distributions (RCA, LAD, circumflex) or left main coronary disease. In terms of clinical presentation, these patients may have a long history of stable, exertional chest pain or shortness of breath (angina) or have had a recent acute event that prompted investigation, e.g. NSTEMI. Preoperative evaluation may reveal evidence of impaired systolic function with or without diastolic dysfunction and possibly coexisting valvular disease. Most patients coming for CABG have several comorbidities including hypertension, COPD, diabetes, or advanced kidney disease. The underlying principle in patients with CAD is to carefully match myocardial oxygen supply and demand.

The anesthetic goals for a patient with CAD are:

- **Heart rate and rhythm**
  - Tachycardia must be avoided at all costs, since it increases myocardial oxygen demand while impairing oxygen supply.
  - Sinus rhythm is preferred but not critical.

- **Preload**
  - Should be maintained within normal limits.
  - Decreases in preload that could potentially lead to tachycardia, e.g. hypovolemia, should be avoided.
  - Hypervolemia is to be avoided since an increased LVEDV increases LVEDP and myocardial work.

- **Afterload**
  - Extremely high afterload (e.g., arterial hypertension due to pain or anxiety) is to be avoided since it increases myocardial wall tension.
  - Drastic reductions in SVR and aortic diastolic pressure impair coronary perfusion pressure and must be avoided.

- **Contractility**
  - Should be maintained.
  - Increases in contractility increase oxygen demand and must be avoided.

**Aortic Stenosis**

AS is now usually due to calcific degeneration (a process similar to atherosclerosis); congenital defects (e.g., bicuspid valve) and rheumatic disease were previously the most common causes. These processes lead to gradual impairment of LV outflow. In compensation, the LV develops concentric hypertrophy, which serves to both increase transvalvular flow with more “squeeze” and to decrease LV wall tension. As the disease worsens, these patients are not able to increase cardiac output in response to demand because the
stroke volume is maxed and heart rate increases are limited by impaired coronary flow to the thickened myocardium. Myocardial oxygen demand is increased due to increased work of the hypertrophied myocardium, while supply is diminished from a fixed CO. The hypertrophied LV is “stiff,” often with diastolic dysfunction, which causes a decrease in the LA-to-LV diastolic pressure gradient and results in decreased LV filling. This makes these patients very dependent on coordinated atrial contraction (“atrial kick”) for diastolic filling.

The classic presenting signs of AS are exertional syncope, angina, and heart failure. Angina can be independent of true CAD and results from functional ischemia and the inability to perfuse the hypertrophied myocardium. Heart failure, if present, is the worst prognostic sign of the three. The normal aortic valve area is > 2.5cm², while < 0.6cm² is considered critical AS. Typical surgical intervention involves replacement of the valve; percutaneous valvuloplasty is a rare option.

The anesthetic goals for a patient with AS are:

- **Heart rate and rhythm**
  - Avoid tachycardia or excessive bradycardia. Increases in HR result in decreased ventricular filling and increased oxygen consumption, while bradycardia can impair CO because CO is HR-dependent.
  - **Sinus rhythm must be maintained.** These patients are very dependent on their atrial kick for LV filling. Intra-op atrial fibrillation should be immediately cardioverted.

- **Preload**
  - Should be maintained within normal limits.
  - Decreases in preload leading to decreased BP should especially be avoided; see below.

- **Afterload**
  - A normal to high-normal afterload is absolutely essential.
  - Drastic reductions in SVR and aortic diastolic pressure, such as those that accompany a “typical” induction, must be avoided.
  - Raising arterial blood pressure and SVR does not increase the obstruction to flow or ventricular work in these patients, since the obstruction is fixed at the valve itself.

- **Contractility**
  - Should be maintained.

Hypotension can set off a spiral of decreased coronary perfusion, myocardial ischemia, decreased contractility, leading to further hypotension, decreased coronary perfusion, and so forth. In a patient with critical AS this can be impossible to recover from due to the fixed stenotic valve. For the same reason, chest compressions are ineffective.

The choice of anesthetic agent is not as important as maintaining the above parameters. Neuraxial anesthesia is a contraindicated in severe AS due to an unacceptable drop in blood pressure. These patients should have an arterial line placed prior to induction, and any hypotension must be dealt with immediately. Many practitioners choose a titratable agent such as phenylephrine and have it running prior to induction.
**Aortic Regurgitation**

AR can be acute or chronic in nature. Acute causes include endocarditis, aortic dissection, or traumatic injury. Chronic causes include congenital (e.g., bicuspid valve), rheumatic disease, syphilis, Marfan syndrome, and other connective tissue diseases.

The pathophysiology involves LV volume overload as a portion of the stroke volume passively returns backwards into the LV during diastole. This reduces effective stroke volume. In its chronic form, the LV eccentrically dilates to accommodate the increased volume, and the AR can present as insidious CHF or angina. Eventually these changes to the LV become irreversible. Ventricular dilation may also cause MR. Aortic diastolic pressure tends to be low, and LVEDP tends to be high, so coronary perfusion pressure can be tenuous. In the acute situation, the LV is unable to dilate, and the increased volume and pressure are translated back to the LA and pulmonary circulation, manifesting as pulmonary edema and hypotension.

The anesthetic goals for a patient with AR are:

- **Heart rate and rhythm**
  - High-normal rates are preferable. Increases in diastolic time allow for increased regurgitation, so avoiding bradycardia is necessary.
  - Maintaining sinus rhythm is preferable but not essential as in AS (above).
- **Preload**
  - High-normal volumes should be maintained to allow for adequate filling and forward stroke volumes. Excessive preload should be avoided.
- **Afterload**
  - AR patients generally benefit from a reduction in afterload, which improves forward flow.
  - Sudden increases in afterload increase the regurgitant volume.
  - Typically these patients have a very wide pulse pressure.
- **Contractility**
  - Inotropic support may be necessary to maintain forward flow, especially in acute AR.

A good mnemonic for management of regurgitant lesions is “Full, Fast, and Forward.”

**Mitral Stenosis**

MS is almost always due to rheumatic fever and is increasingly rare in this country. The valve leaflets and chordae tendineae thicken, fuse and become calcific, all of which contribute to impaired valvular opening, decreased LV preload, and dilation of the LA to overcome the transvalvular pressure gradient. Dilation of the LA can lead to arrhythmias such as A-fib. Static blood in the LA leads to formation of clot and the potential for systemic emboli. Furthermore, increases in pressure in the LA translate back into the pulmonary circulation, which can lead to pulmonary edema, increased PVR, and RV failure.
The anesthetic goals for a patient with MS are:

- Heart rate and rhythm
  - Maintenance of sinus rhythm is necessary to improve LV filling.
  - Preexisting A-fib and/or clot are common and may make cardioversion unwise.
  - Avoid tachycardia so as to allow sufficient diastolic time and LV filling.

- Preload
  - Adequate filling pressures must be maintained.
  - Excessive volume is poorly tolerated, does not increase LV preload, and is readily transmitted back to the pulmonary circulation.
  - Keep these patients euvoletic.

- Afterload
  - Should be maintained near-normal.

- Contractility
  - Avoid increases in CO, as the LV is poorly able to compensate and has a relatively fixed preload.

Mitral Regurgitation

MR can either be acute or chronic. Chronic causes include rheumatic disease or degenerative dilation or destruction of the mitral annulus. Acute causes include endocarditis, trauma or myocardial ischemia (dysfunction or rupture of a papillary muscle). MR is characterized by backflow of the LV stroke volume into the LA, resulting in decreased forward flow and CO. To compensate for this increased volume, the LA dilates. Severe LA volume overload is transmitted back to the pulmonary circulation. Meanwhile, the LV also eccentrically dilates and increases end-diastolic volume. This compensatory response increases CO in the face of decreased forward EF. Patients with acute MR, often due to ischemia, are unable to compensate in this way, present with pulmonary edema. Under the low-SVR, low-demand state of anesthesia, MR often appears better on TEE than it would in the awake patient.

The anesthetic goals for a patient with MR are:

- Heart rate and rhythm
  - Keep HR at high-normal ranges.
  - Slow heart rates, and long systolic times, increase regurgitant flow.
  - Maintain sinus rhythm if possible.
- **Preload**
  - Adequate preload must be maintained to meet increased filling requirements from the dilated LV.
- **Afterload**
  - Reducing afterload favors forward flow and effective SV.
- **Contractility**
  - Should be maintained.

Again, the mnemonic for management of regurgitant lesions is “Full, Fast, and Forward.”

**Right-sided Valvular Lesions**

Valvular lesions on the right side of the heart are generally better tolerated by patients and only rarely present for surgery. They may be congenital or degenerative in nature or from infective endocarditis. Often, right heart pathology is secondary to pulmonary hypertension resulting from left heart pathology. For further discussion of right-sided lesions, consult an appropriate text.

**Hypertrophic Obstructive Cardiomyopathy**

This disease entity is characterized by idiopathic hypertrophy of the myocardium, most commonly in the LV at the interventricular septum. Diastolic dysfunction is encountered, with elevated LVEDPs reflecting increased LV stiffness. There is outflow obstruction during systole due to narrowing of the subaortic area by the hypertrophied myocardium. Additionally, a systolic anterior motion (SAM) component of the anterior leaflet of the mitral valve may be present. In this phenomenon, the mitral leaflet is drawn by the Venturi effect into the subaortic area during systole, worsening outflow obstruction.

HOCM can present insidiously as dyspnea, fatigue and syncope or near-syncope. It is the most common cause of sudden cardiac death in young patients. Other associated findings include arrhythmias, LVH and Q waves on ECG, and evidence of hypertrophy on echo. Treatment includes β-blockers, calcium channel blockers, and surgical myomectomy if severe. This is a rare indication for heart transplantation. Rationale for medical therapy is below.

The anesthetic goals of a patient with HOCM are aimed at avoided factors which make the outflow obstruction worse. Increased contractility, decreased LV volume and decreased afterload all worsen obstruction. Therefore, adequate preload, maintenance of afterload, and avoidance of increased sympathetic output and increased contractility are key.

**Chapter 5D. One-lung Ventilation: Anesthesia and Physiology**

Single-lung ventilation can be one of the most challenging maneuvers we encounter in anesthesiology. In addition to the mechanics of placing an appropriate ET tube and physically separating the lungs, profound changes in respiratory physiology are created with potentially deleterious consequences.

The indications for one-lung ventilation can be divided into absolute and relative, and are a favorite topic on the boards. The only absolute indications are:
- Isolate bleeding to one lung (e.g., pulmonary artery rupture)
- Isolate infection to one lung (e.g., overwhelming abscess or pus confined to one lung)
- “True” need to ventilate only one lung (e.g., bronchopleural fistula, tracheobronchial disruption,
bronchoalveolar lavage)

Relative indications are mostly related to the surgical procedure or technique:

- Pneumonectomy, lobectomy, thoracoscopy
- Anterior thoracic spine procedures
- Thoracic or high abdominal aortic aneurysms
- Lung transplantation
- Severe unilateral lung disease with hypoxemia
- Esophageal surgery

Several types of ET tubes and equipment are available to create lung separation and one-lung ventilation, including double lumen ETTs and various types of bronchial blockers. Specific techniques for each are covered in detail in the thoracic surgery chapter in Miller’s Anesthesia and during the airway rotation. Advantages and disadvantages of each are discussed below.

- **Double-lumen ETT**
  - This is a large ETT with a bronchial lumen and a tracheal lumen. They come in either left- or right-sided types and in sizes measured in French.
  - Advantages include the ability to suction either lung, the ability to ventilate either lung, relative stability once placed, and the ability to give oxygen and CPAP to the nonventilated lung.
  - Disadvantages include the larger size, relative difficulty of placement, and the fact that it cannot be left in place if post-op ventilation is needed.

- **Univent ETT**
  - This is a large, single-lumen ETT with a Uniblocker-type bronchial blocker incorporated into the ETT wall.
  - Advantages include better ease of placement, the ability to leave it in place at the conclusion of the case, and relative stability.
  - Disadvantages include the inability to suction the nonventilated lung, relative difficulty in switching ventilation between lungs, the lack of pediatric sizes, and the slow rate of lung deflation due to the small efflux lumen.

- **Bronchial blocker through a standard ETT**
  - Examples include the Uniblocker, Arndt, Cohen, and the Fogarty embolectomy catheter. These are similar; each is a “balloon on a stick.”
  - Advantages include coming in many sizes and small pediatric sizes.
  - Disadvantages include relatively poor stability, the inability to suction or provide oxygen to the nonventilated lung, and the slow rate of lung deflation due to the small efflux lumen.

Regardless of the choice of tube, confirming and maintaining the ability to ventilate only one lung is critical. After insertion, proper tube position and the ability to separate the two lungs must be confirmed. After the patient is positioned for surgery (usually lateral), both tube position and lung separation should be rechecked. Any movement or repositioning of the patient can result in tube migration and the inability to separate the lungs or appropriately ventilate the patient. The following is a complete checklist to follow after placement of a DLT, the type most commonly employed for one-lung ventilation. Although there are many ways to confirm proper placement, this checklist is a thorough starting point.
Before starting, have all the appropriate equipment available. This includes the correct DLT size plus one larger and one smaller, a firm clamp, suction catheters (included in the DLT packaging), a working fiberoptic bronchoscope, and a stethoscope.

1. Place DLT at estimated appropriate depth.
2. Inflate both bronchial and tracheal cuffs.
3. Listen for bilateral breath sounds while ventilating through both lumens.
4. Clamp one lumen at a time and listen for absence of breath sounds on the clamped side and continued presence on the contralateral side.
5. Place fiberoptic bronchoscope down tracheal lumen, confirm proper placement of tube and bronchial cuff, and adjust as necessary.
6. Secure with tape or ties.
7. Run the checklist again after positioning.

Confirming Correct Tube Placement with FOB

At UCSD, lung isolation is almost universally achieved with a left-sided DLT, the placement of which is confirmed immediately after placement with FOB. Auscultation alone has a success rate < 80%.

When using a Univent or single lumen ETT with a bronchial blocker, confirmation of correct tube/blocker placement is fairly straightforward. The tracheal rings will be evident on the anterior aspect of the trachea, and the first major division into the right and left mainstem bronchi occurs at the carina. The right mainstem can be further identified by noticing the straighter, more caudal takeoff, the fairly rapid takeoff of the RUL, and the “trifurcation” where the RML, segment 6 of the RLL, and the 4 inferior segments of the RLL take off. The left mainstem bronchus is much longer, and its first division is a bifurcation into the LUL+lingula and the LLL.

DLTs come in either left- or right-sided types, corresponding to the direction of the bronchial lumen and cuff. In practice, a left-sided DLT is almost always chosen as it is technically easier to place with fewer complications and still allows separation of either lung. The main disadvantage of right-sided DLTs is that the RUL bronchus takes off of the right mainstem bronchus very proximally, so it is easy to block with the bronchial cuff; the “margin of safety” is quite low. With correct left-sided DLT placement, the following features will be observed:

- The tracheal rings should be anterior and visible when the FOB is passed down the tracheal lumen.
- The bronchial lumen should extend into the left mainstem bronchus but not all the way to the bifurcation of LUL and LLL. It is possible to measure the distance from lumen tip to bifurcation, keeping in mind that the left mainstem is often 4-5cm long.
- Seen through the tracheal lumen, the inflated blue bronchial cuff should be stably seated just below the carina.
- Again through the tracheal lumen, the right mainstem bronchus and RUL bronchus should be widely patent.

Physiology of One-lung Ventilation

The lateral position is the most commonly employed position for surgery involving one-lung ventilation and is partially responsible for the physiologic changes encountered. The awake, spontaneously ventilating patient in the lateral position generally has preserved V/Q ratios. The dependent lung
receives more perfusion due to gravity, but is better ventilated due to more efficient diaphragmatic movement and optimal position on the alveolar compliance curve. Under general anesthesia, a reduction in FRC and change in ventilatory patterns creates significant V/Q mismatching. The dependent lung now continues to receive more perfusion, but becomes relatively less well ventilated, creating the potential for large amounts of shunting and hypoxemia. Positive-pressure ventilation and one-lung ventilation will correct or abolish many of these effects.

During positive-pressure ventilation with the nondependent lung deflated, i.e., one-lung ventilation, the nondependent lung continues to receive some amount of pulmonary blood flow. This blood does not become oxygenated, so this represents right-to-left shunt and can lead to hypoxemia. Fortunately, hypoxic pulmonary vasoconstriction limits flow to the nondependent lung. This creates a situation where most of the perfusion and all of the ventilation is delivered to the dependent, ventilated, nonoperative lung. Factors which can decrease HPV and increase shunt include hypocapnia, vasodilators, and inhaled anesthetics in doses > 1 MAC.

Decreasing perfusion to the dependent lung can also create shunt and worsen hypoxemia by diverting blood to the nondependent lung. Factors which decrease perfusion include high airway pressures (excessive PEEP, autoPEEP from inadequate expiratory times), low FIO2 causing HPV in the dependent lung, and compression of relevant blood vessels as in surgical manipulation.

Elimination of CO2 is typically not compromised providing minute ventilation does not change. Smaller tidal volumes, on the order of 4-8ml/kg, and faster respiratory rates are usually employed during one-lung ventilation. Hyperventilation and hypocapnia are avoided as they inhibit HPV, and permissive hypercapnia is an accepted technique.

**Correction of Hypoxemia During One-lung Ventilation**

Hypoxemia and oxygen desaturation during one-lung ventilation must be corrected, and strategies to do so are a common boards topic. The first goal is to determine the minimum tolerable saturation for the patient, recognizing that some amount of shunting is inevitable during one-lung ventilation. Many practitioners use an SpO2 of 88-92% as their cutoff, although clearly this varies according to the patient and their disease state. It’s important to recognize that hypoxemia below this threshold is **life-threatening** and is a legitimate reason to change surgical technique or abort surgery altogether. The following steps can be employed to correct hypoxemia:

1. Ventilation with 100% O2. Most anesthesiologists do this anyway for the duration of one-lung ventilation.
2. 5-10cm H2O of CPAP (with oxygen flow) to the nondependent lung. This is an effective maneuver. This can be done with a Mapleson circuit with an in-line manometer. Beware that this can cause reinflation of the operative lung and interfere with surgery.
3. PEEP to the ventilated lung. This can theoretically worsen hypoxemia by decreasing perfusion to the ventilated lung. It can also correct hypoxemia if atelectasis is the etiology.
4. Periodic two-lung ventilation. This also interferes with surgery.
5. In extreme cases, the surgeon can clamp the pulmonary artery of the nonventilated lung, essentially eliminating all shunt from that source.

**Chapter 5E. Other Topics in Cardiothoracic Anesthesia**

**Intra-aortic Balloon Pump**
An IABP is a device designed to improve myocardial oxygen supply and reduce oxygen demand in a failing or ischemic heart. It is often used to wean patients from CPB. The balloon is typically placed in a femoral artery and threaded retrograde to sit in the proximal descending aorta. The balloon inflates and deflates with the cardiac cycle. Inflation is timed to occur just after the aortic valve closes (dicrotic notch), to improve aortic diastolic pressure and therefore coronary perfusion pressure. Deflation is timed to occur just before LV ejection, so that aortic pressure (afterload) at that time is low and myocardial work is reduced. The net result is afterload reduction and coronary perfusion augmentation; which is the more important component is a matter of debate.

Properly timed inflation of the balloon is critical. Inflating the balloon too early results in increased afterload and myocardial work and can cause aortic regurgitation. Inflating the balloon too late negates diastolic pressure augmentation. The balloon can be set to inflate with a variety of timings, such as with every beat (1:1), every other beat (1:2), or less (1:4, 1:8). This adjustability can be used to wean the heart from the IABP. Timing with the heartbeat is accomplished either by sensing the QRS complex or the arterial waveform.

If an IABP is to remain in place for more than several hours, anticoagulation becomes necessary. Complications of the IABP include limb ischemia, mesenteric ischemia, aortic dissection or rupture, infection, bleeding, coagulopathy (especially thrombocytopenia), gas emboli, renal failure, and spinal cord ischemia resulting in paraplegia. Contraindications to IABP placement include AR, sepsis, and severe vascular disease, which can cause problems with placement and raises the risk of thrombosis.

Although invasive, IABP use is becoming increasingly common. They are clearly beneficial in assisting patients in need of LV support and are first-line agents in some centers (not UCSD).

**LVAD/RVAD**

Patients with heart failure refractory to medical management may require a right or left ventricular assist device. Rarely, they may be placed to aid a patient in coming off CPB when all other measures have failed. VADs may be temporary, used for a few months as a bridge to heart transplantation, or as destination therapy in patients not suitable for heart transplant. Essentially, a VAD is a centrifugal pump that takes blood from the ventricle and pumps it to the downstream artery (aorta or PA). Presence of a VAD requires systemic anticoagulation.

CO in patients with a VAD becomes dependent on preload and VAD speed. In low-CO states, volume administration is usually attempted first, before adjustment of the pump speed.

Patients with VADs may have nonpulsatile blood flow. Therefore, non-invasive blood pressure cuffs and pulse oximeters may work poorly or not at all.

**Heparin Resistance and Anticoagulation**

Heparin resistance is generally due to antithrombin III deficiency. In normal patients, heparin binds to ATIII, greatly enhancing its anticoagulant effects. ATIII-deficient patients cannot achieve this anticoagulated state, so administration of FFP (which contains ATIII) or ATIII concentrate will allow heparin to be effective for CPB.

Patients with heparin-induced thrombocytopenia can be a challenge to anticoagulate prior to CPB. In HIT, heparin antibodies bind to platelets, causing thrombocytopenia and possibly thromboembolism. If a
A patient has a history of HIT, blood samples should be sent specifically to check for heparin antibodies. If there are no antibodies present and the history of HIT is distant, heparin may be used for CPB. If antibodies exist, alternative anticoagulation must be used, including hirudin, bivalirudin, and argatroban. Plasmapheresis may be necessary to remove significant amounts of antibodies.
Chapter 6. Anesthesia for Cardiothoracic Surgery

Much of the relevant information and physiology is covered in the preceding section on cardiovascular physiology, and will also be addressed in depth during the rotation. This section is intended to provide a general “game plan” for the CT cases encountered here at UCSD.

Chapter 6A. Basics of Cardiopulmonary Bypass (CPB)

In general, any case involving CPB can be broken down into three broad stages: the pre-bypass, on-bypass, and post-bypass periods. Each stage has different physiologic goals and requirements. CPB is used for any open heart procedure including valve repair and replacement, pulmonary thromboendarterectomies (PTEs), heart and double lung transplants (usually), aortic aneurysm or dissection repair, and many CABGs. Additionally, partial bypass may be used in other rare cases (e.g., extensive tumor involving the IVC). Essentially, CPB diverts blood away from the heart and lungs to the bypass machine, which then oxygenates the blood, removes CO₂, and returns the blood to a major artery, usually the aorta.

The CPB machine is complex and its function is only briefly covered here. At UCSD, a dedicated perfusionist is responsible for maintaining and monitoring the function of the pump at all times, as well as making any adjustments during bypass (e.g., increasing flow, correcting electrolyte imbalances). It is still important for anesthesiologists to understand the CPB mechanism, especially because it is a tested topic on the boards.

The CPB has five basic components: a venous reservoir, an oxygenator, a heat exchanger, a pump, and an arterial filter. The pump is typically “primed” with around 2L of isotonic solution in the venous reservoir, and many different solutions can be added depending on surgical preference or patient needs (e.g., mannitol, albumin, blood products). Venous blood drains from the venous cannula, in the patient, to the reservoir by gravity. Drainage is thus proportional to the difference in height from the patient to the reservoir and also depends on the size and resistance of the venous cannulas. Entrainment of air into the system can result in an air lock, preventing further drainage and proper pump function, with potentially devastating consequences.

From the reservoir, the blood passes through a thin membrane where O₂ is added and CO₂ is removed. Next, the blood is brought to the desired temperature and sent to the pump. Pumps are either roller pumps or centrifugal pumps. The former uses rollers to squeeze blood through large tubing and flow is proportional to speed of the rollers. Increasing the number of revolutions will increase flow. This form of flow is non-pulsatile; this non-physiologic type of flow may be partially responsible for decreased organ perfusion during CPB. Centrifugal pumps spin, “flinging” the blood outward and using centrifugal force to propel blood to the patient. The flow is proportional to the resistance encountered, and increases in SVR require an increase in centrifugal pump speed to create the same flow. This form of pumping may be less traumatic to the blood (no squeezing). Before returning to the patient, the blood passes through an arterial filter, which serves to trap debris such as particulate matter and emboli. The blood then returns via the arterial cannula.

CPB machines have many additional features. Various suction and “vent” lines return blood from the field and the heart, since even on CPB the heart will still receive venous blood from the bronchial and thebesian circulations. A separate pump is used to infuse cardioplegia solution to the heart; see below. Additionally, inhaled anesthetic can be directly added via the oxygenator.
Myocardial Protection

Upon initiation of CPB and aortic cross clamping, all coronary blood flow ceases. Techniques to protect the myocardium must be initiated to prevent myocardial ischemia and cell damage.

Cardioplegia

Cardioplegia solution containing a high potassium concentration is most commonly used. Infusion of this solution “antegrade” via the coronary arteries causes cardiac arrest and cessation of electrical and mechanical activity, reducing oxygen requirements dramatically. Cardioplegia can also be delivered retrograde, via the coronary sinus, to ensure all myocardium is reached, since arterial flow in a patient with CAD is limited. Cardioplegia must be reinfused periodically, and washed out prior to coming off CPB.

Distention of the heart and electrical activity both increase myocardial oxygen demand. Fibrillation is especially energy-consuming and thus detrimental. Satisfactory conditions for myocardial preservation are not met until the heart is both empty and asystolic.

Hypothermia

Systemic hypothermia reduces metabolic oxygen requirements by about 50% for every 10°C reduction in temperature. CPB is carried out under hypothermic conditions, with the patient typically cooled to 25-30°C. Additionally, cold slush solutions are directly applied to the heart and chest cavity to reduce myocardial temperature and assist with cardioplegia. This hypothermia must be corrected before CPB is removed.

Other Techniques

Other factors which may lessen myocardial damage include minimizing bypass time (over 2hrs is
considered suboptimal; minimizing surgical manipulation of the heart; de-airing the heart and grafts prior to termination of bypass; and the use of inhaled anesthetics, which have been shown to attenuate ischemia-reperfusion injury. Unfortunately, anesthesiologists have little control over most of these components.

Chapter 6B. The Pre-Bypass, On-Bypass, and Post-Bypass Periods

The Pre-Bypass Period

This time period starts with induction of anesthesia and ends with insertion of the venous and arterial cannulas. Hemodynamic stability is of paramount importance during induction. Specific agents and goals should be titrated to the patient’s underlying disease state (e.g., pulmonary hypertension, aortic stenosis) and are covered more fully in the section on cardiovascular physiology. The most hemodynamically labile and stimulating times are during laryngoscopy, skin incision, splitting of the sternum, and manipulation and dissection around the aorta.

Almost every cardiac patient will need an arterial line placed prior to induction of anesthesia. After induction and intubation, other lines such as the CVP and PAC are placed. Relevant labs such as the ACT, baseline ABG, and cardiac output data should be obtained. The TEE probe is placed and an exam performed. Antifibrinolytic therapy is used on all CPB cases, with the exception of PTEs, with the goal of limiting blood loss and combating the profibrinolytic effects of CPB. Aminocaproic acid is the agent of choice at UCSD. Typically, 2.5g of aminocaproic acid is given as a slow bolus loading dose, followed by an infusion of 1g/h. Aprotinin was previously used, but was taken off the market in the USA due to concerns about renal injury. More details will be provided prior to starting your cardiac rotation.

CPB requires full systemic heparinization to prevent catastrophic clotting within the pump, which would be fatal. After surgery has begun, the perfusionist will calculate the dose of heparin needed based on body weight and the baseline ACT. The patient is allowed to passively cool in preparation for systemic hypothermia with CPB. A small amount of hemodilution is beneficial to lower blood viscosity, but typically we limit the amount of fluid we give pre-bypass given the volume of prime solution in the CPB circuit. The surgeons do as much dissection as possible pre-bypass to minimize time actually spent on CPB. This may include harvesting of a saphenous vein and dissection of the internal mammary artery.

Prior to insertion of the CPB cannulas, heparin is administered. The surgeons will call for this at the appropriate time. An ACT should be checked 3 minutes later to ensure proper anticoagulation. An ACT > 400 seconds is generally considered safe. The aortic cannula is placed first. During this time, SBP should be 100mmHg or less to facilitate placement of the cannula and reduce the chance of aortic dissection. This can be accomplished in any number of ways, including deepening the anesthetic, slight reverse Trendelenburg, or by using a short-acting vasodilator such as nitroprusside. Next, the venous cannulae are placed, and venous flow to the reservoir is confirmed. When good venous flow is established, the arterial cannula is unclamped and CPB is initiated. Flow is gradually increased as proper cannula placement, venous return and arterial pressures are confirmed. Cooling is begun immediately, and cardioplegia will begin shortly thereafter.

The On-Bypass Period

Physiologic management of the patient is largely turned over to the perfusionist at this point. The ventilator is stopped and infusions other than aminocaproic acid are stopped. Vasoactive drugs are administered by the perfusionist. Anesthesia is maintained with volatile anesthetic given by the
perfusionist via a vaporizer on the CPB machine. Because the CPB “prime” solution dilutes the blood and reduces concentrations of drugs present, additional muscle relaxant or midazolam may need to be administered at this time, and are given by the perfusionist into the venous reservoir. The PAC typically migrates distally as it cools during CPB, and so it should be withdrawn 2-3cm upon initiation of CPB.

The surgeons may pass off a separate line to the anesthesiologist to topically apply a constant stream of cold topical irrigation to the heart. These cold bags of NS will be provided in the room and should be continued for as long as the surgeon desires. Urine output should be monitored and reported to the perfusionist. The perfusionist may also request syringes of vasoactive drugs to manage the patient’s physiologic parameters.

As the surgical portion of the bypass period concludes, the surgeons will call for the patient to be rewarmed. The patient must be rewarmed prior to termination of CPB, but rewarming too soon can negate the protective effects of hypothermia. Vasodilation can improve pump flow and speed the warming process. Overly-rapid rewarming reduces the solubility of gases and can lead to the formation of bubbles, and in turn, gas emboli. Light anesthesia is common during rewarming and most practitioners administer additional muscle relaxant and/or amnestic agents. The perfusionist will calculate and tell you the dose of protamine that will be needed for eventual heparin reversal. **Protamine must not be administered while the patient is on CPB**, for catastrophic clotting and death are the likely results. It is safest to just wait to draw up the agreed-upon dose until the surgeons ask for protamine to actually be given.

Prior to separation from CPB, the patient should be normothermic, the acid/base status and hematocrit should be normalized, a stable cardiac rhythm and rate must be obtained (generally 80-100bpm; pacing may be needed), and ventilation is resumed. Coming off CPB is a critical time, and the attending should and will always be present. The venous return lines are slowly clamped, and the heart fills and begins to eject blood. The cardiac surgeons usually call for an inotrope at this point, typically low-dose dopamine, but the choice of agent may depend on the patient’s physiology. The aortic line is stopped and the patient’s vitals and cardiac output are assessed. A TEE exam is done to evaluate volume and contractile status as well as the function of implanted valves. Assuming all is well, the patient is deemed to be “off bypass” successfully and management of circulation is once again turned over to the anesthesiologist. Once the need for CPB is has been removed and truly terminated and the cannulas removed the surgeons will call for protamine. **Prior to administration, you must announce in a loud, clear voice that you are about to give protamine.** This confirms to the whole team what is about to happen so that everyone is on the same page (e.g., the perfusionist will shut the CPB suction off). Protamine should be administered slowly, either in 3-5ml increments over at least 5-10 minutes, or diluted in a 100ml bag and dripped in slowly. The side effects of protamine include hypotension from vasodilation, pulmonary hypertension, and myocardial depression, all of which are exacerbated by fast administration. Allergic reactions can occur, and may be more common in diabetics who previously received insulin containing protamine (Neutral Protamine Hagedorn, “NPH”).

At times, the patient’s heart will not perform adequately coming off bypass and additional measures may be needed, including the need to reinstitute CPB. These measures include the use of additional inotropes, an IABP, or rarely, a left or right Ventricular Assist Device (LVAD or RVAD). Possible causes for poor myocardial performance include poor myocardial protection leading to ischemic injury, long CPB time, myocardial stunning, ongoing ischemia (e.g., air in the coronaries), continued valvular dysfunction, and poor baseline cardiac function. The goal is always to make the first attempt to separate from CPB the best attempt, since each subsequent attempt becomes more difficult and more taxing on the heart.
The Post-Bypass Period

This period starts with coming off CPB and reversal of heparin and ends with patient transport to the ICU. It consists of establishment of surgical hemostasis, chest tube placement, closure of the sternotomy, and transport of the patient to the ICU. An ACT should be checked 3 minutes after protamine administration to confirm adequate reversal; more protamine may be needed. Cardiac output data are obtained via the PAC. A typical goal blood pressure is around 100mmHg, in order to maintain an adequate systemic perfusion pressure while reducing myocardial work, risk to the aortotomy, risk to coronary bypass suture lines, and the risk of bleeding. Once the chest is closed with sternal wires, another cardiac output should be obtained. Chest tubes are placed to facilitate drainage and to monitor for postoperative bleeding, which could necessitate a trip back to the operating room.

The perfusionist will usually be able to hemoconcentrate a significant amount of processed RBCs (“cell-saver” blood) from the CPB circuit, and this should be given back to the patient. Indeed, most patients post CPB require additional volume, the exact status of which can be guided by the TEE, vitals, and PAC information. The patient should be prepared for transport, with all lines tidied up and a transport monitor available. Full resuscitation drugs and airway equipment should be ready for transport in case an emergency arises.

When surgery is concluded, the patient is moved to their ICU bed and transported. This is another critical time that may appear innocuous for the unprepared. The move to the bed should be smooth and controlled, and the patient should remain fully monitored. There have been cases of patients who were alive on the OR table, moved to the ICU bed, and then when monitors were reconnected the patient was found to be dead. Full monitoring during transport itself is mandatory; hand-ventilation via Mapleson is usually sufficient for all cardiac cases except PTEs, for whom an RT will provide a transport ventilator. Once in the ICU, care may be relinquished to the ICU nurse per standard protocol.

Chapter 6C. Anesthesia for Specific Cardiothoracic Surgeries

On-Pump Surgery

Prototypical cases include CABGs (although most CABGs are UCSD are off-pump) and valve replacements or repairs.

**Technique:** general. **Monitors:** standard, plus arterial line, CVP, PAC, TEE, some form of neurologic monitoring such as BIS or SEDline, urine output, ± femoral arterial line (mostly for PTEs). **IV access:** one large IV to begin, with central access established during the case. **Duration:** 4-10hrs. **EBL:** difficult to quantify secondary to hemodilution, CPB circuit volume, and CPB salvage, but 500ml-2L not uncommon. **Position:** supine. **Special equipment:** multiple IV infusion pumps, cooling jacket for the head in PTEs. **Special considerations:** The basic steps for surgery involving CPB are described above. Additionally, goals for anesthesia in various disease states are described in the cardiovascular physiology section.

Almost every cardiac patient should have an arterial line placed prior to induction of anesthesia. Many centers also place a central line and PAC before induction. At UCSD, the CVP and PAC are generally placed after induction. Premedication should be used judiciously and tailored to the patient’s needs. Pain, anxiety, and an increased sympathetic state are highly undesirable for most cardiac patients, as are hypoventilation, hypoxia, and hypercapnia.

Infusion drugs should be prepared prior to bringing the patient back to the OR. Specific drugs will vary
depending on the practitioner and the patient’s needs, but in general have at least one pressor (e.g. phenylephrine), one inotrope (e.g. dopamine), and one vasodilator (e.g. nitroprusside) ready. Prepare a nitroglycerin infusion for patients with CAD. These infusions are generally attached to the PAC’s RV infusion port after placement.

The term “cardiac induction” is used to imply a gentle and hemodynamically stable choice of anesthetic. Almost any agent at our disposal is suitable, provided they are titrated appropriately and used judiciously. In the past, a high-dose narcotic technique (e.g., 50mcg/kg fentanyl) was favored for induction, but a high incidence of recall and a move towards “fast-tracking” patients postoperatively raised serious issues with this technique. Most practitioners use a balanced IV technique, again with emphasis placed on hemodynamic stability. Etomidate, benzodiazepines, and narcotics are all excellent agents in this regard. Liberal use of narcotic is still recommended to blunt sympathetic surges in response to laryngoscopy and other stimulating events. Muscle relaxant should be given early to facilitate ventilation and intubation and attenuate chestwall rigidity from narcotic administration.

Likewise, anesthetic maintenance should be geared towards maintaining hemodynamic stability and amnesia, and the choice of agent is less important than judicious use of that agent. Most of us prefer to ventilate with 100% oxygen throughout the case. The downside to 100% oxygen in the short term is negligible, while the potential benefits in a class of patient especially intolerant of hypoxemia are substantial. Volatile anesthetic, additional narcotic, IV agents, and muscle relaxant can all be used for maintenance. Unfortunately, recall under anesthesia is more common in cardiac cases, as it is in obstetric and trauma surgery, due to the increased potential for hemodynamic instability and the use of CPB. Patients should specifically be informed of this rare possibility during the pre-op visit.

Other intensely stimulating points during the surgery include skin incision, sternal splitting, and dissection around the aorta, and aortotomy for cannulation. Need for additional anesthetic should be anticipated during these times. Prior to splitting the sternum, the surgeon will request for ventilation to be held, to avoid lung inflation and possible damage from the saw.

“Redo” procedures deserve special mention. As the name implies, the patient has had a previous median sternotomy, with a high likelihood of scar tissue and adhesions of thoracic structures to the chest wall (e.g., L internal mammary artery, ventricular wall). Sternotomy in a virgin chest can be quite fast, but redo sternotomies always proceed slowly and carefully. The surgeons do not blithely saw through the sternum, but rather proceed stepwise in controlled layers, all to avoid inadvertent damage to critical structures. Despite this, the potential for surgical mishap is still high. Therefore, redo sternotomy patients should have the perfusionist immediately available to “crash” onto CPB, and blood immediately available (e.g., in the OR and checked in).

Assuming adequate revascularization and lack of intraoperative mishaps, most CABG patients generally respond well to surgery, with adequate or increased cardiac vigor secondary to increased blood supply. The response of patients with valvular disease upon termination of CPB varies with the preoperative disease. In general, patients with stenotic lesions tend to perform well after replacement of the diseased valve. Longstanding pathology and compensation of the atrium or ventricle enable “supramaximal” performance once a normal valve is in place. By contrast, patients with regurgitant lesions often do not perform as well, and may need considerable support to come off CPB. The previous regurgitant valve had created a low-pressure “pop-off” situation, which is removed when the new valve is placed. In the case of MR, the LV must now eject against the aortic valve and the SVR only, without the low-pressure LA to eject blood into.
Pulmonary Thromboendarterectomy (PTE)

UCSD is one of the few centers in the world to perform PTEs regularly. In fact, the majority of cases you will encounter during your first month of cardiac will probably be PTEs. While the essentials of the case are the same as a standard CPB case, there are enough differences to warrant further description.

As a class, most PTE patients tend to be younger and have fewer comorbidities than other cardiac patients. The basic pathophysiology of chronic thromboembolic pulmonary hypertension (CTEPH) involves chronic formation of clot and intimal hyperplasia in the pulmonary arteries, elevating PVR, and over time, pulmonary hypertension and right heart overload with RV hypertrophy, dilation, and failure. Regardless of the cause of the pulmonary thrombus, elevated PA and right heart pressures are a hallmark of these patients. Indeed, it is not uncommon to see a PTE patient with PA pressures close to or above systemic arterial pressures!

The surgery involves opening the main pulmonary arteries so that clot can be extracted. Standard CPB will not provide adequate surgical conditions because venous blood from the bronchial and thebesian circulations returns to the LA and interfere with visualization of pulmonary capillaries. Circulatory arrest is necessary, and this prompts most of the major anesthetic differences between a PTE and a standard CPB case. Circulatory arrest must be performed under deep hypothermic conditions (16-18°C) for cellular protection, particularly neurologic protection. As the name implies, deep hypothermic circulatory arrest (DHCA) involves cessation of the CPB machine and all flow to the patient for periods of 15-20 minutes at a time.

Measures to avoid increases in PVR or overall sympathetic state are key. Premedication is generally avoided in order to avoid hypercapnia and hypoxemia. A vasopressor infusion is commonly used during induction to avoid decreases in systemic arterial pressure. Placement of the PAC may prove difficult due to RV dilation, and the risk of pulmonary rupture from PAC balloon inflation is increased. Indeed, many practitioners do not wedge the balloon at all in these patients. A femoral arterial line is also placed after induction, because pressures from the radial arterial line are very different from more central arterial pressures after deep hypothermia.

As the case proceeds, a cooling jacket is wrapped around the patient’s head. This should be undisturbed as much as possible and periodically checked to ensure that it is functioning properly. As discussed in the neurophysiology section, hypothermia is the only measure that reduces the basal metabolic oxygen requirements of the brain. Upon initiation of CPB, the patient is cooled to < 20°C. It is important to note that the CPB circuit “prime” for this case includes mannitol and very high doses of methylprednisolone, with the goal of reducing ischemia-reperfusion injury particularly in the brain; these drugs may have consequences such as volume and electrolyte shifts and hyperglycemia that the anesthesiologist will have to address. Once the patient has reached around 18°C, the perfusionist will ask for around 200mg of propofol, to be administered just prior to DHCA, to flatline the EEG. Temperature is monitored in multiple locations, including blood (PAC, CPB machine), tympanic membrane (probably best reflection of brain temperature), bladder (reflects core temperature), and rectum. The bladder and rectal temperatures typically lag the PAC and tympanic temperatures during warming or cooling.

Immediately before DHCA initiation, a checklist will be run through to ensure it is safe for arrest to occur. This list includes confirming the patient is cold, the EEG is isoelectric, all transducer stopcocks are turned off to the patient, the TEE probe is off, and the patient’s lungs are briefly ventilated with unwarmed room air. Turning the stopcocks off to the patient ensures that no fluid from the transducers
can entrain during circulatory arrest. Turning the TEE off prevents inadvertent warming from that source. Ventilating the patient’s lungs expels any remaining blood from the bronchial and pulmonary circulation. This checklist is a time-honored ritual at UCSD and the surgeons expect to hear it repeated out loud prior to DHCA. While it is an important safety mechanism, the condescending way some of the CT surgeons use it can be a source of frustration. In this situation, it is best to conform, play along, and not get upset over a trivial matter, while continuing to act in the best interests of the patient.

DHCA is used in “stints” of no more than 20 minutes, during which the surgeon is quickly and carefully performing the thromboendarterectomy itself. Time is of the essence, so distractions and delays are unwelcome. The surgeons expect us to be especially attentive at this stage, standing at the head of the bed, so surgery can proceed expeditiously. Typically 1 or 2 stints of DHCA are used for each pulmonary artery, so a typical maximum time of DHCA is about 80 minutes. In between, CPB is reinstituted to restore perfusion. Before each stint of DHCA, the entire checklist is repeated.

Once the thromboendarterectomy is complete, CPB is resumed and the patient is actively rewarmed. This stage can be quite lengthy, depending on the patient’s body habitus, and can approach 2 hours. Pulmonary reperfusion injury is an uncommon but devastating complication in PTE patients, and may be signaled by worsening hypercapnia, hypoxemia, pulmonary edema (via ETT) or frank pulmonary hemorrhage.

**Minimally-invasive Valve Replacement**

**Technique, monitors, IV access, duration, EBL, position, and special equipment** as for standard CPB cases. Coronary sinus catheter and introducer. **Special considerations**: see below.

In 2011-2012, CT surgeons at UCSD began to perform minimally-invasive valve replacement, mostly mitral and aortic valves, which can minimize the impact of a full sternotomy on the patient. CPB is initiated through a venous cannula and an aortic cannula, each inserted via its respective femoral vessels. The surgeons place ports for thoracoscopic equipment to assist with surgery, and a right minithoracotomy is used to gain access to the heart and to initiate CPB. The main difference in the anesthetic management involves the possible use of a double-lumen ETT (vs. a bronchial blocker) if lung isolation is desired, as well as the placement of a coronary sinus catheter for introducing retrograde cardioplegia. The process of placing the coronary sinus catheter can be very time-consuming and can contribute considerably to the length of the case. The procedure is as follows: 2 central venous catheters are placed in sequence in the right internal jugular vein. One is the “usual” sheath/introducer used for infusions and the PAC; the other is used to place the coronary sinus catheter. The coronary sinus returns venous blood to the right heart, and can be seen on TEE. Similarly to “floating” a PAC, the coronary sinus catheter must be placed under direct TEE visual guidance into the coronary sinus and the balloon inflated. This can be very difficult at times, due to patient anatomy, pathophysiology, etc. The surgeon does have the option of placing the coronary sinus intraoperatively if it not possible via TEE guidance. The rest of the case is similar to all other valve replacement and CPB cases.

**Off-pump CABG (OPCAB)**

**Technique, monitors, IV access, duration, EBL, position, special equipment, and special considerations** as for CPB cases or as below.

Most of the CABGs done at UCSD are done without CPB. OPCAB presents unique challenges to the anesthesiologist. While previously reserved for select patients with 1-2 vessel disease and good target
vessels for anastomosis, OPCAB is now being offered to patients with more profound CAD. This technique demands a fair amount of surgical skill since the surgeon is working on and manipulating a still-beating heart. CPB and the perfusionist are immediately available in case of emergent need for CPB or the inability to perform the procedure off-pump.

Because the patient never goes on CPB, physiologic management remains in the anesthesiologist’s hands for the entire procedure. Manipulation of the heart has the potential to produce large hemodynamic swings and arrhythmias which must be handled. Suspension or lifting of the heart to allow access to posterior and inferior vessels can dramatically reduce venous return with pursuant hypotension, of which the surgeons should be notified immediately. The cardiac stabilizer device, which makes the coronary artery “target” area motionless during vessel-to-vessel anastomosis, essentially induces regional myocardial dysfunction and may reduce CO as well. When the heart is lifted out of the chest cavity, interpretation of the EKG becomes difficult, if not impossible, and TEE is similarly rendered useless.

The advantages of off-pump surgery are faster surgical times (generally), decreased bleeding, and perhaps better long-term outcome with avoiding CPB (controversial). Since CPB is not intended to be used, patients for planned off-pump procedures should be kept near-normothermic from the beginning of the case with the usual techniques like fluid warmers and warming blankets. In general, anesthetic management of off-pump cases, including induction, maintenance and hemodynamic stability are the same as for CPB cases.

**Extraction of Pacemaker or AICD Leads**

This surgery is typically done to remove and replace infected pacemaker or AICD leads or generators.

**Technique:** general. **Monitors:** standard, arterial line, urine output, TEE. **IV access:** 1 IV plus Cordis placed after induction. **Duration:** 2-3hrs. **EBL:** < 100ml. **Position:** supine. **Special equipment:** see below. **Special considerations:** at the time of extraction of the lead, a TEE exam is done to rule out acute hemopericardium and thus rule out cardiac injury.
This patient population will generally have several cardiovascular comorbidities, one of which was the indication for the device in the first place, plus an acute condition, e.g. systemic infection or malfunctioning AICD. Because these leads are actually “screwed” into the myocardium to fix them securely when they are first placed, extraction of the lead carries a risk of myocardial injury including catastrophic events like ventricular wall perforation and tamponade. This low but real risk creates the anesthetic implications above, and is the reason that a CT surgeon performs the extraction and a perfusionist is present, while the primary work of lead and generator replacement are done by the cardiologists.

**Thoracic Aortic Surgery**

Examples include elective or urgent repair of aortic dissection, aneurysm or trauma, and repair of coarctation.

**Technique:** general. **Monitors:** standard, arterial line, CVP, PAC, urine output, ± TEE, TEG, EEG. **IV access:** multiple large-bore catheters including central access needed. **Duration:** 4-10hrs. **EBL:** 1-many liters. **Position:** depends on location of the lesion; supine or right lateral decubitus are most common. **Special equipment:** ± need for CPB, ± need for DHCA, ± need for lung isolation (e.g., DLT and FOB), ± neuromonitoring. **Special considerations:** see below.

Surgery on the thoracic aorta is rare at UCSD, but is invariably complex. It combines the challenges of aortic cross-clamp with those of one-lung ventilation, large fluid shifts, and surgery around critical structures. Additionally, these cases are often emergent in nature. Lesions involving the proximal aorta or the aortic arch may require CPB to maintain systemic perfusion, or DHCA if flow to the brain must be interrupted. More distal lesions are generally done without CPB and invariably involve aortic cross-clamping. See a text for a full discussion.

**Thoracic Surgery**

Examples include thoracoscopic surgery (VATS), thoracotomy for lobectomy or pneumonectomy, repair of bronchial trauma, bronchoalveolar lavage, and lung transplant.

**Technique:** general. **Monitors:** standard, plus arterial line. More invasive monitoring dictated by patient needs and nature of the case (e.g., double lung transplantation requires the same monitors as any CPB case). **IV access:** at least one large IV. **Duration:** 2-12hrs. **EBL:** 100ml-1L. **Position:** the operative lung is usually up, with the patient in the lateral decubitus position. Occasionally, supine. **Special equipment:** ability to separate the lungs, FOB, ± CPB, ± thoracic epidural. **Special considerations:** indications for and the physiology of one-lung ventilation and use of DLTs is described in the cardiovascular and pulmonary physiology section.

Diagnostic thoracoscopy, tissue biopsy, and pleurodesis are three examples of “smaller” thoracic cases. Generally these cases are shorter in duration and are not associated with large fluid shifts. An arterial line is necessary for blood gas samples and BP monitoring, but further invasive monitors such as the PAC are rarely needed. By contrast, repair of traumatic injury to thoracic structures and lung transplants require full monitoring, are typically long and involved cases, and have wide physiologic swings.

Preoperative lung function is an excellent predictor of operative risk in patients undergoing lung resection. Specifically, patients with an FEV₁ < 2L, a predicted postoperative FEV₁ < 0.8L or < 40% of predicted, FEV₁/FVC < 50%, and room air PₐCO₂ > 45 or PₐO₂ < 50mmHg are at high risk for postoperative
respiratory failure. PFTs are invariably indicated for all but the simplest thoracic cases. Thankfully, the underlying pathology in these patients means that the vast majority have had extensive pulmonary workup prior to an anesthesiology consult.

Bronchoalveolar lavage is occasionally performed with the operative lung dependent to minimize spillage to the other lung. This position reverses the normal V/Q matching in the lateral position and can result in severe V/Q mismatch and shunting.

In any lung resection or transplantation case, careful attention must be paid to fluid administration and airway pressures, which may lead to postoperative pulmonary edema and respiratory failure. Whether for lobectomy or pneumonectomy, the same cardiac output is being applied to fewer total bronchial segments. Add this to the “typical” tissue edema that occurs after surgical manipulation, and it is easy to see how IV fluid can predispose to pulmonary edema. Goals for fluid administration for lung resection cases should be to provide only is physiologically necessary, and no more. Thoracic surgeons typically aim for net even or net negative fluid balance during their cases. For airway pressures, the usual maneuvers to minimize PIP should be taken while also avoiding hypoxemia and hypercapnia.

If patients are to remain intubated postoperatively, which is common in major lung resection cases, a DLT must be changed to conventional ETT at the end. In this regard, a Univent ETT or a bronchial blocker passed through a standard ETT may be advantageous, as either may be left in at the end of the case.

Thoracic incisions are intrinsically painful and also obviously interfere with respiratory mechanics and respiratory effort. This causes patients to take shallow, ineffective breaths (“splinting”) which compromises effective ventilation. The use of opioids to treat this pain favors undesirable hypoventilation, sedation, and atelectasis. Occurrence of any of these issues after lung resection is clearly deleterious. To combat this problem, aggressive pain control is instituted, often in the form of thoracic epidural analgesia. An epidural, with a combination of dilute local anesthetic and opioid, is perhaps the most effective measure at attenuating postoperative pain, and avoids the side effects of IV opioids. The epidural is generally placed immediately before induction, as patient cooperation and comfort is likely to be poor postoperatively. Thoracic epidural analgesia is considered for all true thoracotomy cases, whereas for VATS cases one might consider simple subcutaneous local anesthetic or intercostal nerve blocks.

Lung Transplantation

Lung transplants are common enough that you may expect to do 1 or 2 during your cardiac month, but complex and varied enough that they warrant reference of an authoritative text. Briefly, unilateral lung transplantation is preferred over bilateral for most disease states other than those that predispose to infection and cross-contamination of the new graft lung, e.g. cystic fibrosis. Bilateral lung transplantation can theoretically be done without CPB, by sequentially transplanting each new lung, but in our department’s experience this is almost always attempted only to be abandoned in favor of CPB. For either type of transplant, a double-lumen ETT is always selected to permit excellent lung isolation, suctioning of the graft lung(s), and endoscopic evaluation of the bronchial anastomoses. TEE is an indispensable monitor, especially for bilateral transplant, to allow evaluation of right heart function, volume status, and vascular anastomoses. Strict fluid restriction and minimizing peak inspiratory pressures are necessary goals to prevent pulmonary edema and trauma to the graft lung(s).

For a unilateral lung transplant, many of the considerations for major thoracic surgery such as pneumonectomy apply, including the preexisting pulmonary disease, need for one-lung ventilation, fluid
management, positioning, and need for excellent epidural analgesia.

Bilateral lung transplants are much more involved and more comparable to a complex on-pump cardiac case. Classically, a bilateral inframammary “clamshell” incision is used, which necessitates suspension of the arms above and over the head to allow surgical access. Monitors and vascular access are planned as such.

Antibiotic and immunosuppression regimens will be discussed with the surgeons preoperatively. For further information, refer to a cardiac anesthesia text.

**Technique**: general + epidural. **Monitors**: standard, arterial line, urine output, CVP, PAC, ± TEE, ± BIS/SEDline. **IV access**: large-bore IV access plus CVP/PAC. **Duration**: 6-12hrs. **EBL**: 200ml-1000ml, often difficult to estimate if on CPB. **Position**: lateral for unilateral, supine with arms suspended overhead for bilateral. **Special equipment**: FOB, double-lumen ETT, possibly nitric oxide, possibly 2nd ventilator. **Special considerations**: as above.

**Surgery for Pericardial Disease**

Cardiac tamponade can be managed surgically by a pericardial window via a subxiphoid, thoracotomy, or median sternotomy approach. Procedures to relieve constrictive pericarditis include pericardial stripping or window.

**Technique**: general. **Monitors**: standard, arterial line, ± TEE. Further monitors may be useful but should not delay an urgent case. **IV access**: at least one large IV. **Duration**: 1-4hrs. **EBL**: usually < 100ml, plus evacuation of any existing blood. **Position**: supine. **Special equipment**: as above. **Special considerations**: physiology of anesthetic management of tamponade.

Cardiac tamponade is a physiologic state in which filling of the heart is constricted by the presence of blood or other fluid around the heart. It can be seen in CT patients postoperatively due to continued bleeding. Other diseases which may cause tamponade include those that cause pericardial effusions (cancer, infection, uremia, MI, autoimmune disorders) or hemopericardium (trauma, postop bleeding). Constrictive pericarditis occurs when the pericardium becomes scarred, stiff, and fibrotic, leading to impaired ventricular relaxation and filling. Clinically, the two states can appear quite similarly.

Signs of tamponade are frequently tested and include tachycardia; the classic Beck’s triad (hypotension, JVD, muffled heart sounds); equalization of diastolic pressures throughout the heart (rare); tachypnea; intolerance of the supine position; and pulsus paradoxus, which is simply an exaggerated phenomenon of the normal fall with SBP with inspiration (pathologic is a fall of > 10mmHg). The EKG classically shows “electrical alternans,” which is variation of the R wave height over several cardiac cycles, thought to be from the heart “swinging” in the pericardial sac. Other EKG findings might include decreased voltages in all leads, or diffuse ST segment elevation (more associated with pericarditis).

Anesthetic management of cardiac tamponade is critically important and depends on the severity of the disease. A relatively stable patient with little to no “tamponade physiology” is on one end of the spectrum, and a patient in extremis is on the other. The key goal of anesthesia for tamponade is to preserve CO by avoiding reductions in HR, preload, or contractility. As most of our induction agents depress CO and sympathetic tone, they are poorly suited for a patient with symptomatic tamponade. Etomidate or ketamine are the agents of choice, as both avoid sympatholysis and preserve spontaneous, negative-pressure ventilation. Bear in mind that ketamine is a negative inotrope in vivo, so a
sympathetically “maxed out” patient may have unmasked negative inotropic effects if ketamine is administered.

Typically, IV fluid loading is done to preserve preload, and an arterial line is placed. After induction and achievement of deep anesthesia, intubation is done without muscle relaxant. Other monitors such as CVP, a PAC, or TEE may all be useful in providing further information and assisting with patient management, but unfortunately these are time-consuming and should not delay an urgent case. Severe tamponade should be alleviated with a pericardiocentesis or subxiphoid window prior to induction of anesthesia. Once surgery has begun, a subxiphoid window may be sufficient, or the surgeon may have to enlarge the incision to gain access to the offending fluid.
Chapter 7. Neuroanesthesia Rotation and Neurophysiology

Chapter 7A. Neuroanesthesia Rotation

The neuroanesthesia rotation at UCSD is a two-month, intense exposure to neurosurgical cases and the specific anesthetic demands they entail. You will complete the first month during your CA-2 year and the second month during your CA-3 year. Throughout each month, you will be exposed to craniotomies for aneurysm repair, resection of tumors, and correction of other intracranial pathology. In addition, spine surgeries are often assigned to the neuro resident as they also demand knowledge of neurophysiology. By the time the formal rotation comes around, undoubtedly the resident has had some experience with craniotomies or spine surgeries simply as a function of previous Main OR duties. This rotation will solidify past experience into a formal fund of knowledge.

If available, Drs. Drummond or Patel are often assigned to “true” craniotomies, especially complex cases (e.g., aneurysms, resection of large tumors). This is typically done at the neurosurgeon’s request because, simply put, they are the leaders in their field. Take advantage of their expertise during this month and avail yourself of the knowledge they command. However, many of our faculty are also experts in neuroanesthesia and are often assigned to staff these cases as well. Overall, this should be one of the most educational months in your entire residency.

For the neuroanesthesia residents, the daily schedule assignment is made specifically with the rotation in mind. The resident will be assigned the most complex or interesting neurosurgical case of the day. In some cases, this might be a large spine surgery with the use of motor and sensory evoked potentials. The expectation is that the neuro resident will finish his or her own case, especially since emergence is a critical portion of most neuroanesthesia procedures. This month can entail some long hours and challenging days, but it is well worth it. In general, you will also be assigned three to four in-house calls during the month, similar to a regular Main OR rotation.

The following chapters cover basic neurophysiology and anesthetic techniques for specific procedures. Anesthesia for spine surgery is covered in the orthopedic surgery section.

Chapter 7B. Neurophysiology and Anesthesia

Most, if not all, anesthetics have profound effects on neurophysiology. Delivering a rational anesthetic, particularly for neuroanesthesia, requires a thorough understanding of the effect of the drugs, anesthetic techniques, and the procedure in question. At UCSD, we are fortunate to have world-renowned faculty in the field of neuroanesthesia. Due to our faculty, we enjoy a particularly harmonious relationship with most of the neurosurgeons. This is fortunate because close communication is often critical during neurosurgical procedures. This relationship is predicated on our ability to deliver a safe and superb neuroanesthetic. This section will cover basic neurophysiology and the effects of anesthesia on that physiology. Specific anesthetic techniques for neurosurgical procedures will follow in the next chapter. For more details on the effects of specific agents, see the drug section.

Cerebral Blood Flow and Autoregulation

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Cerebral blood flow (CBF) is usually about 50ml/100g/min and is higher in grey matter, lower in white matter. An average brain mass is about 1500g, so CBF for an “average” human is about 750ml/min. Flow rates lower than 25ml/100g/min are associated with EEG slowing and neurologic impairment. Flow rates below 20ml/100g/min typically produce an isoelectric (flat) EEG indicative of zero cortical electrical activity, and values below 10ml/100g/min are usually associated with irreversible neurologic damage. At typical physiologic MAPs, cerebral blood flow is tightly autoregulated. Classic teachings describe fairly tight control between MAPs of 50-150mmHg. Within this range, autoregulatory mechanisms keep CBF constant despite potentially wide swings in blood pressure. Beyond this range, CBF becomes MAP-dependent, rising or falling with similar changes in blood pressure. The cerebral autoregulation curve is shifted to the right in patients with chronic hypertension.

CBF is directly linked to $P_{a}CO_2$. Each 1mmHg decrease in $P_{a}CO_2$ corresponds with a 1-2ml/100g/min reduction in CBF. Conversely, each 1mmHg increase in $P_{a}CO_2$ corresponds with a 1-2ml/100g/min increase in CBF. This effect is due to CO$_2$ tension within the CSF and is not seen with acute changes in HCO$_3$, which cannot cross the blood brain barrier. Clinically, most practitioners aim for a $P_{a}CO_2$ between 25-30mmHg to achieve favorable and safe reductions in CBF. Prolonged changes in CSF $CO_2$ tension result in a change in CSF bicarbonate concentration, negating any effects on CBF. The reduction in CBF is typically negated after 12 hours.

Hyperoxia only causes a small decrease in CBF. Hypoxemia, on the other hand, causes profound increases in CBF.

Hypothermia decreases both CBF and cerebral metabolic rate of oxygen consumption ($CMRO_2$), as below, while hyperthermia has the opposite effect. CBF changes 5-7% per 1°C change in temperature. Each 10°C decrease in temperature reduces $CMRO_2$ by 50%, and each 10°C increase doubles $CMRO_2$. Hypothermia produces an isoelectric EEG at around 20°C and is the most effective strategy for neuroprotection, making it useful in situations of decreased or absent CBF. See the section on anesthesia for PTE.

$CMRO_2$
The brain typically receives 20% of total CO and consumes ~50ml/min of oxygen. Glucose is the normal source of energy, the vast majority of which is metabolized aerobically. CMRO$_2$ thus parallels metabolic activity and energy consumption. Under periods of starvation, ketone bodies may be consumed. Glucose deprivation and hypoxia have profound and quick impacts on the brain, with cell death occurring within 3-8 minutes of the insult. The hippocampus and cerebellum are the areas of the brain most sensitive to hypoxic injury.

Reduced metabolic needs of the brain correlate with reduced CMRO$_2$. Reductions in CMRO$_2$ are thus **favorable** in reducing CBF requirements.

Of the total CMRO$_2$, about 60% goes toward energy used for the electrical activity of the brain (neuronal conduction), whereas about 40% is used in the basal “housekeeping” cell activities like protein synthesis and maintenance of transmembrane potential. This is important to note because anesthetics can only reduce the electrical component of CMRO$_2$, whereas hypothermia can reduce both the electrical and the “housekeeping” portion of CMRO$_2$.

**Chapter 7C. Intracranial Pressure**

The brain can be thought of as being enclosed within a rigid space (the skull). This space is occupied by brain cells/tissue (80%), interstitial fluid and blood (12%), and CSF (8%). In order to prevent a rise in intracranial pressure, an increase in any of the components that occupy the space (volume) must be offset by an equivalent decrease in another component. Examples of increased “components” include tumor, bleeding, or hydrocephalus from CSF outflow obstruction. Small increases in volume are normally compensated for quite well, with little to no increase in ICP.

Compensatory mechanisms include shifting CSF to the spinal space, increased absorption of CSF, decreased production of CSF, and decreased cerebral blood volume. However, when these compensatory mechanisms are overcome, small increases in volume correspond with large increases in ICP; i.e., the intracranial space develops low compliance. Large increases in ICP can lead to brain ischemia and catastrophic herniation of brain tissue. Herniation may occur at one of four sites, as in the diagram: 1) the cingulate gyrus under the falx cerebri, 2) the uncinate gyrus through the tentorium cerebelli, 3) the cerebellar tonsils through the foramen magnum, or 4) any area beneath a defect in the skull (transcalvarial).

Cerebral perfusion pressure is calculated as **CPP = MAP – ICP (or CVP, whichever is higher)**. Typically, ICP is < 10mmHg. It should be obvious that large increases in ICP have a deleterious effect on CPP. As
discussed above, decreases in CPP below the autoregulatory threshold will decrease CBF. So, CPP < 50mmHg often leads to EEG slowing, whereas CPP between 25-40mmHg typically show a flat EEG. Sustained CPP < 25mmHg results in irreversible brain damage.

In general:

- Decrease in CBF → decrease in cerebral blood volume (CBV) → decrease in ICP
- Increase in CBF → increase in CBV → potential increase in ICP
- Decrease in CMRO$_2$ → decrease in CBF, and vice versa

The underlying goal of all neuroanesthetic strategies is to reduce CMRO$_2$ and safely reduce CBF in order to reduce ICP, while maintaining MAP and CPP.

Chapter 7D. Effect of Anesthetic Agents on CBF, CMRO$_2$, and ICP

I. Volatile Anesthetics

Volatile anesthetics are generally said to “uncouple” the normal matching of CBF with CMRO$_2$ in a dose-dependent fashion. That is, under a dose of about 1 MAC, volatile anesthetics reduce CMRO$_2$ in a dose-dependent fashion and cause a parallel reduction in CBF. However, above 1 MAC, there is no further decrease in CMRO$_2$, but CBF actually goes up due to cerebral vasodilation. By altering this CMRO$_2$-CBF coupling, doses of volatile anesthetics > 1 MAC create a tendency for CBF to parallel MAP. Of the volatile agents, halothane appears to have the greatest “uncoupling” effect. MAC ≤ 1 of desflurane, sevoflurane, and isoflurane generally has little effect on CBF-autoregulation coupling.

- Dose-dependent ↓ CMRO$_2$ = Isoflurane > Desflurane/Sevoflurane > Halothane
- Dose-dependent ↑ CMRO$_2$ = Halothane > Isoflurane/Desflurane/Sevoflurane

Volatile anesthetics are used in many neuroanesthetic cases, according with the concept that doses < 1MAC do not increase CBF and so do not increase ICP. If elevated ICP is a critical concern, i.e., the brain is “tight,” it is prudent to discontinue all volatile anesthetics to eliminate any possibility of autoregulation-uncoupling or unwanted increases in CBF.

- Most IV anesthetic agents ↓ CBF and CMRO$_2$ in a parallel fashion (except ketamine)
- All IV agents preserve cerebral autoregulation and CBF-CO$_2$ relationship
II. Nitrous Oxide

Nitrous oxide typically has minimal effects on CBF, CMRO$_2$, and ICP. It may slightly increase CBF if used alone, but this effect is minimal. Nitrous oxide may even cause a decrease in CBF when combined with other agents, especially IV agents. When not contraindicated, nitrous oxide is routinely employed in neuroanesthesia due to its rapid onset and offset, allowing for quick and “clean” emergence and early neurologic testing.

III. Opioids

Opioids decrease CMRO$_2$ and CBF to a small extent. They are primarily used to blunt sympathetic responses to noxious stimuli, such as surgical incision and laryngoscopy, and to create a profound antitussive effect for a cough- and buck-free emergence. Short-acting opioids (such as remifentanil) are preferable to allow early neurologic examination and avoid prolonged emergence. Opioids are a mainstay of neuroanesthesia.

IV. Barbiturates

Barbiturates are ideally suited agents for neuroanesthesia. They produce profound decreases in CBF and CMRO$_2$, with relatively more reduction in CMRO$_2$. Thus, metabolic supply tends to exceed metabolic demand; this is known as “luxury perfusion.” Furthermore, barbiturates have antiepileptic properties, and can be used in “barbiturate coma” to produce an isoelectric EEG for neuroprotection. Lastly, barbiturates cause an increase in CSF absorption, which helps lower ICP.

V. Propofol and Etomidate

These agents also reduce CMRO$_2$ and CBF and are good agents for neuroanesthesia. Propofol has anticonvulsant properties, while etomidate may activate epileptic foci in patients with seizure disorder. Propofol has a short elimination half-life, enabling rapid neurologic assessment in the postoperative period. Etomidate decreases CSF production and enhances absorption.

VI. Benzodiazepines

These agents reduce CBF and CMRO$_2$, but, similar to the “ceiling” effect they have on depth of anesthesia, this reduction is small. They have significant anticonvulsant properties. Benzodiazepines are used sparingly in neuroanesthesia since they can profoundly prolong emergence, especially when used as an infusion. They are best used as low-dose adjuncts or, more commonly, avoided entirely.

VII. Ketamine

Ketamine increases CBF and may also impede absorption of CSF. The increase in CBF and CSF volume can markedly increase ICP in patients with decreased intracranial compliance. Whether this is clinically true when ketamine is used as part of a balanced anesthetic is a matter of controversy. Nevertheless, dogma persists that ketamine increases ICP, and thus it should be avoided when ICP is an issue.

VIII. Succinylcholine

Succinylcholine can cause transient increases in ICP. A classic neuroanesthetic dilemma is whether or not to use succinylcholine in a situation of increased ICP. Bear in mind that this increase is usually small
and is easily attenuated with anesthetic agents. Additionally, the rapidity of muscle relaxation that succinylcholine achieves is unmatched. So, if rapid control of the airway is necessary to avoid the much-more-potent ICP effects of hypercapnia or hypoxemia, succinylcholine is a reasonable choice.

IX. Dexmedetomidine

Dexmedetomidine is a selective α₂ agonist with sedative and analgesic effects. It decreases CBF via cerebral vasoconstriction.

Chapter 7E. Neuroprotective Techniques

Ischemic brain injury can be classified as either global (complete) or focal (incomplete). Global ischemia includes total circulatory arrest as well as global hypoxia. Focal ischemia includes embolic, hemorrhagic, and atherosclerotic strokes as well as blunt, penetrating, and surgical trauma. With focal ischemia, the brain tissue surrounding the damaged area, the so-called “ischemic penumbra,” may suffer marked functional impairment but may still remain viable. Although these damaged areas are thought to have marginal perfusion (< 15ml/100g/min), these areas may completely recover if further injury is limited and normal flow is rapidly restored. Clinical goals in such instances are usually to optimize CPP, decrease metabolic requirements, and possibly block mediators of cellular injury.

1. Hypothermia
   - This is the most effective method for protecting the brain during focal or global ischemia by decreasing both basal and electrical metabolic requirements.
   - Even mild hypothermia (33-35°C) is protective.
   - Head-injured patients, if cold, should not be rewarmed rapidly.
   - The beneficial effects of hypothermia must be counterbalanced with possible deleterious effects (e.g., coagulopathy, arrhythmias, impaired immunity).

2. Maintenance of CPP and Oxygen Delivery
   - MAP should be maintained in a normal range to ensure appropriate CPP.
   - Oxygen-carrying capacity should ideally be maintained with a hematocrit of ~30% and a normal oxygen saturation and PₐO₂.
   - Reduction of ICP is discussed below.

3. Reduction in CMRO₂
   - Reduction of metabolic demand is beneficial.
   - Many anesthetic agents can produce an isoelectric EEG; this can reduce metabolic demand by a maximum of 60%.
   - The basal metabolic demand can only be reduced by hypothermia.
   - See a text on “barbiturate coma” for more information.

4. Avoidance of Hypoglycemia and Hyperglycemia

Hypoglycemia predisposes to further ischemia and can worsen the neurologic insult, whereas hyperglycemia is thought to worsen cerebral inflammation following injury or ischemia.

5. Avoidance of Hypoxemia
Hypoxemia is harmful in at least two ways: it directly limits oxygen-carrying capacity and thus oxygen delivery, and it also causes cerebral vasoconstriction and reduces CBF.

Chapter 7F. Strategies to Reduce ICP

These techniques all center on reducing the volume of one of the three components of the intracranial space: brain matter, interstitial fluid and blood, and CSF. Some techniques may be impractical, while others are under direct surgical, as opposed to anesthetic, control.

1. Reduction of Brain Tissue Volume

Examples include removal of tumor or offending mass by the surgeon. Typically, this is not under our control and obviously not a factor for reducing ICP prior to surgery. Rarely, a decompressive craniectomy may be performed to create more “space” for remaining tissue and to relieve elevated ICP.

2. Reduction of Interstitial Fluid Volume

Osmotic diuretics, such as mannitol, can be used to reduce ICP. By increasing serum osmolality, mannitol draws intracellular water into the intravascular space and off brain tissue, decreasing brain volume. Being a hyperosmolar solution (and a fluid bolus), mannitol typically induces a transient increase in blood volume, which may worsen ICP, which is then followed by hypotension from its vasodilatory properties and reduced intravascular volume from diuresis. It is thus used with caution. Mannitol works within 30 minutes to reduce brain volume.

Because of the rapid reduction in brain volume, mannitol may be dangerous before the cranium is opened in aneurysms, AVMs or intracranial hemorrhage. In such instances, decreases in brain volume may create more “room” for expansion of an aneurysm, AVM or hematoma, thereby theoretically increasing the risk of bleeding.

Loop diuretics like furosemide are also used, but these are slower in onset than mannitol. They are useful adjuncts since they have a synergistic effect, tend to increase serum sodium and osmolality, and reduce CSF production.

Glucocorticoids, by limiting inflammation and edema, may also reduce interstitial fluid volume.

3. Reduction of Cerebral Arterial Blood Volume

This can be achieved with the following techniques:

- Choice of anesthetic agent to reduce CBF and CMRO$_2$.
- Mild hyperventilation to induce vasoconstriction. A $P_{aCO_2}$ of 25-30mmHg reduces ICP for 24-36hrs without affecting acid-base status and cerebral oxygen delivery.
  - Avoidance of hypoxemia.
  - Avoidance of arterial hypertension.

4. Reduction of Cerebral Venous Blood Volume

This is often overlooked, and can be achieved via avoidance of elevated jugular venous pressure. This includes:
- Keeping the head of the bed up.
- Avoiding extremes of head flexion or rotation.
- Avoiding circumferential ETT ties.
- Avoiding internal jugular venous catheters.
- Avoiding elevated intrathoracic pressure, e.g., coughing, PEEP, pneumothorax, tamponade.

5. Reduction of CSF Volume

Can be achieved with the following techniques:

- Drainage of CSF, e.g. ventriculostomy, lumbar drain.
- Loop diuretics.
- Choice of anesthetic agent, e.g., barbiturates.
- Other agents to reduce CSF production (acetazolamide, steroids).

Chapter 7G. Anesthetics and Evoked Potentials

Evoked potentials are a form of electrophysiologic monitoring used to test the integrity of nerve pathways that may be compromised by the surgical procedure. There are two main categories of evoked potentials used for neuromonitoring: sensory evoked potentials (SEPs) and motor evoked potentials (MEPs). Typically, the surgeons employ a neurophysiologist under the supervision of a neurologist to monitor the evoked potentials during an operation in which neural damage is of great concern.

Somatosensory Evoked Potentials (SSEPs)

SSEPs reflect the ability of a neural pathway to conduct a signal from the periphery to the cerebral cortex. There are three types of SSEPs:

1. **Somatosensory evoked potentials (SSEPs)**: test sensory cortex and integrity of dorsal columns of the spinal cord. Used for surgeries that have the potential to compromise the spinal cord (spinal tumor resection, instrumentation of the spine, aortic surgery, etc.)
2. **Brainstem auditory evoked potentials (BAEPs)**: test integrity of CN VIII and auditory pathways. Used for surgeries around CN VIII, surgery in the posterior fossa, etc.
3. **Visual evoked potentials (VEPs)**: test optic nerve function and upper brain stem function. Used for resection of large pituitary tumors.

Generally, SEP responses rely on stimulating the particular nerve in question and monitoring the cortical response. A “good” or unchanging response implies an intact neural pathway, while changes in the response could be a signal of impending nerve damage. Taking the BAEP as an example, the technician can periodically trigger a sound within the ear canal, which the acoustic nerve should sense. This signal should travel along the afferent pathway from brainstem all the way to the cortex and can be monitored along the entire path.

The SSEPs are classified according to their latency (time from stimulus to response) and amplitude (voltage measurement from peak apex to baseline). Changes in either parameter may be transient and expected (e.g., irrigation of the area near the nerve) or more sustained and worrisome, such as with surgical irritation or damage. Any decrease in amplitude by 50% or an increase in latency by 10% indicates a worrisome disruption of a neural pathway.
Most anesthetics affect the characteristics of the SSEPs and must be adjusted or omitted entirely. Briefly, all inhalational agents decrease amplitude and increase latency. This effect is usually minimal at MAC ≤ 0.5 but varies with the agent in question. Nitrous oxide reduces amplitude but does not affect latency. IV agents also decrease amplitude and increase latency, but to a lesser extent than inhaled agents. Ketamine and etomidate in particular may increase amplitude. Opioids have little to no effect on either parameter.

In summary, anesthetic “interference” with evoked potentials is as follows: VEPs >> SSEPs >> BAEPs.

Other than interventions we can make in response to a worrisome evoked signal (e.g., correct hypoperfusion, acidosis, anemia, hypotension, and notify surgeon), much of our responsibility during the use of SSEPs will be to utilize an anesthetic that has minimal effect on the monitoring and signals. Thus, a typical “SSEPs anesthetic” will be similar to a TIVA (i.e. propofol infusion and opioids) ± volatile agent at a MAC ≤0.5. The neurophysiologists understand the necessary anesthetic limitations, and preoperative discussion can help you plan the anesthetic accordingly and avoid later hassles.

### Motor Evoked Potentials (MEPs)

MEPs test the integrity of dorsolateral and ventral spinal cord pathways. This is accomplished by transcranial stimulation of the motor cortex, which elicits contralateral peripheral nerve signals, electromyelographic signals, and limb movements. MEPs are frequently used in conjunction with SSEPs for surgeries that have the potential to compromise the spinal cord. The major anesthetic consideration regarding the use of MEPs is that neuromuscular blockers must be omitted from the anesthetic regimen in order to observe these motor responses.

Whenever SSEPs or MEPs are to be used for an operation, the specific anesthetic plan should be discussed with your attending, the surgical team, and the neuromonitoring person to make sure everyone is on the same page.

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Chapter 8. Anesthesia for Neurosurgery

The following are brief descriptions of the typical neurosurgical procedures encountered at UCSD, as well as the anesthetic implications. Anesthesia for spine surgery is described in the section on orthopedic surgery. Basic neurophysiology and anesthesia is addressed in the previous chapter.

Chapter 8A. Anesthesia for Intracranial Vascular Surgery

Examples include open aneurysm clippings and resection of arteriovenous malformations.

Technique: general. Monitors: standard, plus arterial line. CVP may be a useful adjunct to guide fluid therapy, especially if mannitol is to be used. Urine output. IV access: at least one large IV; the standard at UCSD is to have one IV for fluids and boluses and another for infusions. Catastrophic bleeding, although unlikely, is a possibility. Duration: 4-6hrs, potentially more for complicated cases. EBL: 100ml. There can be much more if there is unanticipated or uncontrollable bleeding. Position: supine, lateral, or semilateral depending on location of the aneurysm. The head of the bed is 180° away from anesthesiologist. Special equipment: precordial Doppler if increased risk of venous air embolism; see the section on posterior fossa craniotomy. A ventriculostomy or subdural “bolt” may be employed by the neurosurgeons to monitor ICP; see below. Special considerations: as below.

Most neurosurgical procedures take place with the patient’s head away from the anesthesiologist and the anesthesia machine. The implications of this position change are discussed in detail in the sections on head & neck surgery and emergency craniotomy room setup. They include limited access to the airway, increased risk of airway dislodgement due to close surgical proximity, and hazards of turning the OR table 180°.

An arterial line is mandatory for all but the simplest neurosurgical procedures. Nowhere is the necessity of invasive arterial monitoring more evident than in an aneurysm clipping. Broadly, aneurysms can be classified as ruptured or unruptured. For both types of aneurysms, the hemodynamic goals are similar: tight control of BP. Profound hypertension can cause catastrophic rupture of an intact aneurysm, or increase the risk of rebleeding in an already ruptured one. Similarly, hypotension is generally poorly tolerated because of compromised cerebral perfusion. The potential for bleeding, wide swings in levels of surgical stimulation (and thus changes in BP), fluid shifts with the use of osmotic diuretics, and need for blood samples all mandate the use of an arterial line.

Use of central venous catheter (CVC) is not as clear-cut, and many craniotomies are performed without one. Advantages of a CVC include the ability to monitor CVP, the large and reliable central access it provides, and the ability to aspirate air in situations of venous air embolism. The disadvantages of placing a CVC are myriad and include arterial puncture, pneumothorax, infection, and bleeding. Use of a CVC should be tailored to the individual case, but most craniotomies can be performed without one unless there is a clear indication (e.g., high risk of venous air embolism, need for hypertonic saline, etc.). The potential for large blood loss in AVM or aneurysm surgery demands large IVs, but not necessarily a CVC. Similarly, blood products should be available prior to surgery.

Current neurosurgical management of subarachnoid hematoma involves early (within 72 hours) or late (after 2 weeks) clipping of ruptured aneurysms. Between these time periods is the window for cerebral vasospasm, which is generally 4-14 days post-bleed and occurs in about 30% of patients. Vasospasm is thought to be a response to blood around cerebral vessels, and depending on the severity and distribution of the vessels involved, can lead to brain ischemia or infarction. Prevention of vasospasm
involves the use of the calcium channel blocker nimodipine, as well as “triple-H therapy:” hypertension, hypervolemia, and hemodilution. Triple-H therapy is done by volume loading with NS and the use of vasopressors such as dopamine, phenylephrine, or dobutamine to induce a hyperdynamic and hypertensive state; hemodilution usually occurs passively by the crystalloid load and the acute anemia of critical illness. Because triple-H therapy may increase the likelihood of rebleeding, most neurosurgeons attempt to clip or coil ruptured aneurysms in the early period. Typical measures to reduce ICP are generally not employed in an already-ruptured aneurysm until the dura is opened (e.g., mannitol, hyperventilation). The theory behind this is that lowering ICP will increase the transmural pressure in the aneurysm, increasing aneurysm wall tension, and increase the chance of rupture and rebleed. After the dura is opened to atmospheric pressure, these considerations are removed and standard efforts to reduce intracranial volume can be undertaken.

Direct ICP monitoring is often employed in these patients, through use of either a subdural “bolt,” subdural pressure catheter (a.k.a., Camino), or ventriculostomy (a.k.a., EVD). The ventriculostomy can monitor ICP and can drain CSF directly, whereas the bolt and Camino are monitoring devices only. These monitors are generally placed pre-op or intra-op by the neurosurgeons, but are monitored by us in the OR. Close communication with the surgeons is necessary for specific management goals such as goal ICP and CPP.

With all craniotomies, the most stimulating points of the surgery tend to be the same, and it is during these times that BP must be most closely monitored. In chronologic order, they are: laryngoscopy, head “pinning” in the surgical headframe (Mayfield), skin incision, opening of the skull, and opening of the dura. The brain itself is insensate, and after dural opening, levels of surgical stimulation are typically low, and the surgeons liberally infiltrate local anesthetic with epinephrine into the scalp to limit bleeding. With these points in mind, many anesthesiologists tailor the anesthetic in such a way as to blunt the sympathetic discharge and rises in BP with each of these points. Induction is typically carried out with a large dose of narcotic (typically at least 10mcg/kg of fentanyl) and muscle relaxation to eliminate the possibility of coughing or straining. Deep anesthesia is achieved prior to placement of headframe pins by the surgeon. Most of the neurosurgeons here at UCSD are very good at communicating with us and will inform us before the pins are to be placed. BP is closely monitored and rises can be attenuated with more narcotic or a fast acting agent such as propofol. Similarly, a close eye must be kept on the BP during skin incision and cranial and dural opening. Having quick, titratable IV agents in-line is mandatory (e.g., nitroprusside and phenylephrine infusions to lower or raise the BP).

Additional anesthetic goals of a standard craniotomy are avoiding hypercapnia and hypoxemia, avoiding increases in ICP, minimizing brain volume to create an optimal surgical field, preventing spontaneous patient movement, creating a smooth and cough-free emergence, and allowing for early neurologic assessment with a quick wakeup. Clearly, there are times when some of these goals may be difficult to achieve, and sometimes one goal is in direct conflict with another, such as a smooth wakeup vs. a quick wakeup. Strategies to achieve these goals are discussed below.

Measures and conditions that increase and decrease ICP are discussed in the neurophysiology section. As mentioned, all of our anesthetic agents have some effect on ICP. Most anesthesiologists employ a balanced anesthetic approach with a heavy emphasis on IV agents. A typical example would be a MAC ≤ 0.5 of volatile anesthetic coupled with nitrous oxide and propofol and opioid infusions with muscle relaxation. This combination allows relatively quick offset of anesthesia, ensures paralysis, and prevents increases in ICP. Propofol in particular is favored by our neurosurgeons for its beneficial effects on CBF, CMRO$_2$, and ICP. You will often be asked to give more propofol in response to a “tight brain” and to
achieve better surgical conditions and visualization. An alternative is to eliminate the inhaled agents altogether and to run a TIVA with propofol, opioid, and muscle relaxant. A significant opioid base should allow for a smooth emergence, as well as blunt hemodynamic responses to stimulation as above. Depending on the context-sensitive half-life of the opioid chosen, it may be necessary to discontinue the opioid several hours before emergence to allow for a timely wake up. Ensuring paralysis is necessary to avoid potentially catastrophic movement during surgery on the brain.

A smooth emergence is usually achieved with a good base of narcotic, deep neuromuscular blockade with late reversal, and avoidance of stimulation to the patient during Stage II. The classic Stage II of intense coughing, breath-holding, and hemodynamic instability is more associated with emergence from inhalational anesthesia, as opposed to nitrous oxide and propofol. Also, consider that many patients having craniotomy have altered or depressed mental status at baseline, and that all of them will have just had some form of amnesia-inducing cortical injury, i.e., brain surgery. Therefore, elimination of volatile anesthetic relatively early in the emergence while continuing muscle relaxation can be done with a very low risk of recall.

Tailoring the anesthetic to allow for both a rapid and a smooth wakeup requires considerable experience, but in general, propofol infusions should be discontinued at least 30min before the end of the case and fentanyl infusions about 1hr before. This is, of course, variable depending on the doses used, the length of the case, and the patient’s comorbidities and physical status. Coughing or bucking can be attenuated with additional anesthetic or removal of the ETT if conditions allow, but should not be allowed to persist. Rarely, situations may exist that obviate a quick wakeup or immediate neurological assessment, such as severely depressed mental status at baseline, severely ill patient, etc.

**Chapter 8B. Anesthesia for Emergent Craniotomy**

A significant percentage of emergent craniotomies involve situations with acutely raised ICP, usually due to intracranial hemorrhage with or without trauma. These cases present somewhat different challenges in regards to anesthetic management. Some of this information is also covered in the trauma section. Typical cases include acute subdural or epidural hematoma evacuation with burr holes (durotomy) or formal craniotomy, intraparenchymal hematoma evacuation, and chronic subdural evacuation, all potentially in the setting of trauma.

**Technique:** general. **Monitors:** standard, plus arterial line. CVP may be desirable. Urine output. **IV access:** multiple large IVs. **Duration:** 1-4hrs. **EBL:** 200ml to potentially much more depending on the injury. **Position:** typically supine, head 180° away from the anesthesiologist. **Special equipment:** potential ICP monitor placed by neurosurgeons. **Special considerations:** as below.

Several considerations unique to the trauma patient include: aspiration risk or full stomach, potential for cervical spine instability, an obtunded or combative patient, and hemodynamic instability. These problems must all be managed concurrently. Concurrent head injury and known or suspected increased ICP further complicates matters. Typically, a rapid sequence induction with in-line cervical stabilization and cricoid pressure is used. Control of the airway must proceed with attenuation of profound increases and decreases in BP and avoidance of hypercapnia and hypoxemia. Arterial and venous access must also be achieved as soon as possible. Clearly, this is a team task that often involves several members of the department. Do not be afraid to call for help.

Measures to reduce ICP should be undertaken as soon as possible and are discussed in detail in the neurophysiology section. It should be noted that definitive treatment is the opening of the cranium and
Surgery should not be delayed for want of better IV access, an arterial line, etc. The first priority is quickly securing an airway and providing deep anesthesia in order to allow the surgery to rapidly proceed. Other necessary measures such as fluid resuscitation, placing an arterial line, or obtaining venous access can proceed concurrently while surgery is underway. Note that this is much different than in an elective craniotomy in which surgery would not proceed before arterial access was first established.

Unlike “elective” craniotomies, these patients are often left intubated at the end of the procedure. Concomitant injuries, instability, or diffuse brain injury may necessitate ongoing intubation and mechanical ventilation. Similarly, severely increased ICP may require post-op intubation, paralysis, and mechanical ventilation.

Patients with chronic subdural hematomas represent a class where acute lowering of ICP may be undesirable. The longstanding presence of the hematoma may produce a tamponade effect that prevents significant further bleeding. Rapid lowering of brain volume and ICP may remove this effect and create potential for large bleeding.

Chapter 8C. Anesthesia for Craniotomy for Mass Lesion

Examples include removal of supratentorial tumor or infectious mass, and transsphenoidal resection of pituitary tumor.

**Technique:** general. **Monitors:** standard, plus arterial line. Urine output. CVP may be useful but is not necessary. **IV access:** one large IV should suffice. **Duration:** 2-6hrs. **EBL:** 100-500ml, typically less than for vascular malformations and intracerebral hemorrhage. The chance of catastrophic bleeding is lower, but still present. **Position:** generally supine or lateral ± head turned to side. Head 180° away from anesthesiologist, except transsphenoidal surgery. **Special equipment:** usually none. **Special considerations:** Anesthetic technique and goals are essentially the same as those covered in the vascular malformation and raised ICP sections, namely, avoidance of hypercapnia and hypoxemia, tight control of blood pressure, smooth induction and emergence, and early neurological examination. Depending on the size, rate of expansion, and position of the lesion, the patient may have normal intracranial compliance or all the signs or symptoms of increased ICP.

Transsphenoidal resection of pituitary tumor is a special type of craniotomy. In this procedure, the neurosurgeons proceed through an incision through the maxillary gingiva. The incision is small and the procedure tends to have little hemodynamic consequence to the patient. Most of the neurosurgeons here at UCSD perform the surgery with good technique. As such, this operation is sometimes performed without an arterial line, almost a singular exception for neurosurgical procedures. Similarly, large IV access is generally not required. Of course, one can never be at fault for being too prepared for a case. Also, this case is done with the head of the bed facing the anesthesiologist, simplifying matters greatly. Because of large amounts of blood and debris that can fall into the oropharynx, dense throat packs and/or orogastric tube are placed by us prior to surgery. These throat packs serve to catch most of the debris that would otherwise drain into the stomach and potentially cause nausea and vomiting on emergence. They should protrude out of the mouth and be fixed in some way to prevent being lost down the esophagus and removed prior to emergence. Most other considerations for craniotomy still apply, such as potential measures to reduce brain volume and the need for a smooth emergence. Reduction of intracranial volume should be discussed on a case-by-case basis with the surgeon. In certain cases, reduction of intracranial volume may only serve to cause the mass to retract further into
the skull cavity, making surgery more difficult. At other times, some reduction in volume may facilitate surgical conditions.

Other perioperative concerns for transsphenoidal resection of pituitary tumor centers around the mass itself. The most common “functional” pituitary mass secretes prolactin, but masses secreting ACTH, TSH, GH, and others are possible. Many are nonfunctional. Obviously, each type has potential for different effects on the patient’s physiology (e.g., gigantism and difficult intubation in a patient with a GH-secreting mass). Surgery around the pituitary stalk commonly produces a central diabetes insipidus, which is usually transient. A urinary catheter is necessary and frequently helpful. Lastly, transection of the pituitary stalk can lead to panhypopituitarism.

Chapter 8D. Anesthesia for Posterior Fossa Surgery

Examples include any craniotomy for structures in the posterior fossa, including cerebellar or occipital tumor removal and surgery around the brainstem or cranial nerves (e.g., acoustic neuroma removal, microvascular decompression of the trigeminal nerve).

**Technique:** general. **Monitors:** standard, plus arterial line. A specific central venous catheter designed for aspiration of air from the RA can be used. Urine output. Precordial Doppler. **IV access:** at least one large IV. **Duration:** 4-6hrs. **EBL:** usually less than 300ml; there is potential for large hemorrhage. **Position:** lateral, semilateral, prone, or sitting; head 180° away from the anesthesiologist. **Special equipment:** precordial Doppler, potential for monitoring brainstem or cranial nerve potentials. **Special considerations:** The previous considerations regarding ICP and goals of anesthesia all apply to these surgeries as well.

Posterior fossa craniotomies carry an increased risk of venous air embolism (VAE). VAE is possible any time venous sinuses are open to air. The posterior venous sinuses tend to be “tented open” by virtue of being fixed to the posterior dura and bone and thus facilitate entrainment of air. Any time the involved sinuses are above the level of the heart, the pressure in them is low but they remain open, creating a situation where air entrainment can occur. Thus, the incidence of VAE is highest in patients in the sitting position, which is used to facilitate exposure but is thankfully rare in this institution. However, every posterior fossa craniotomy involves some elevation of the head above the heart to facilitate venous drainage and thus carries the risk of VAE.

Signs of VAE include hypotension, hypoxia, increased dead space ventilation, circulatory arrest, and paradoxical embolism if a PFO or other right-to-left shunt exists. Small air emboli typically go unnoticed and have no effect on the patient. VAE can be monitored in several ways. A reduction in etCO₂ and increase in etN₂ may be seen, although this effect may only be noticed with large emboli. A precordial Doppler is useful and can detect even small emboli; the characteristic “whooshing” sound of the normal heart beat is replaced with a whipping-like noise as emboli pass into the heart. TEE is even more sensitive than a Doppler but requires specific operator skill and may have deleterious consequences for the patient if left in for a protracted period of time, especially in the sitting position with neck flexion; see below.

**VAE is an emergency.** Treatment must be immediate and consists of:

1. Notifying the surgeon, who will flood the field with saline and pack with gauze.
2. Discontinuing nitrous oxide and ventilating with 100% oxygen.
3. Raising cerebral venous pressure with IV fluids, lowering the head, “sigh” breath, and jugular vein...
1. Decreasing the head of the bed to 45 degrees
2. Administering a mixture of oxygen and air
3. Compression.
4. Evacuating the air by aspiration via a central venous catheter.
5. Providing supportive measures like CPR and pressors, as needed.
6. In addition to the above efforts, turning the patient with the right side up may keep the air in the RA and decrease passage to the RV and pulmonary circulation, preventing “air lock.”

Other potential complications specific to positioning for posterior fossa surgeries, especially those in the sitting position, include postoperative macroglossia, quadriplegia, and pneumocephalus. In regards to the first two complications, excessive neck flexion is thought to play a role. It is extremely important to ensure that the neck is not completely flexed in these patients prior to the start of surgery (generally at least two fingerbreadths between the chin and chest). Avoiding unnecessary objects in the mouth may be desirable, and a bite block, if used, should be placed as far forward as possible. Pneumocephalus can occur any time the level of the head is raised, which is common in many craniotomies. Nitrous oxide should not be used if there is a known pneumocephalus due to expansion of the gas pocket and mass effect. However, in posterior fossa craniotomies with the head up, it can be used until the intracranial space is completely closed, because prior to this point, there is no trapped gas pocket. Indeed, some feel that nitrous oxide prior to dural closure may actually be advantageous, as N₂O within a potential gas pocket will be resorbed faster than nitrogen. Finally, the sitting position is associated with reduced venous return possibly resulting in hypotension.

Surgery around the brainstem, including direct trauma or pressure from retraction, can have profound physiologic consequences for the patient. Major cardiovascular changes can occur, such as profound hyper- or hypotension, bradycardia, or other arrhythmias. Injury to fundamental respiratory centers in the brainstem can cause postoperative respiratory dysfunction. If any of these changes are noticed intraoperatively, it is imperative to inform the surgeon and treat the condition. Often, removal of surgical instruments and loosening up on retraction is all that is necessary to correct the problem. Bradycardia is particularly common and can be treated with cessation of the offending surgical stimulus followed by ephedrine, glycopyrrolate, atropine, or epinephrine.

Microvascular decompression of the trigeminal nerve is a somewhat uncommon procedure elsewhere but is done fairly frequently at UCSD. It involves a retromastoid craniotomy to remove vascular structures thought to be compressing CN V and causing trigeminal neuralgia. Many of these patients are young and otherwise healthy but may be on a complex regimen of medications for this condition. The craniotomy has the potential for bradycardia as above and calls for the usual assortment of volume reduction techniques such as mannitol and mild hyperventilation. As such, an arterial line is usually placed. BAEPs are monitored, but given that our anesthetics minimally affect BAEPs, no special techniques are required.

Injury to cranial nerves arising from the brainstem is also a potential complication of these surgeries. The postoperative deficit depends on the nerve involved. One feared complication is damage to the glossopharyngeal nerve, which may result in inability to maintain a patent airway. Neuromonitoring may include evoked potentials such as auditory evoked potentials for acoustic neuroma resection or facial nerve EMG for facial dissection, which would preclude neuromuscular blockers.

**Chapter 8E. Anesthesia for Minor Neurosurgery, Including Stereotactic Surgery**

Examples include placement, revision, or removal of a ventriculoperitoneal shunt or lumboperitoneal shunt and “awake” stereotactic surgery including deep brain stimulator placement.
Technique: general, or general followed by local/MAC for stereotactic surgery. Monitors: standard. IV access: one IV. Duration: around 1 hr for shunts, 4-6 hrs for DBS. EBL: < 100 ml. Position: Supine or lateral for shunts, bed 90° or 180° away from the anesthesiologist. Stereotactic surgery is typically done with the patient sitting up 45-60°, head fixed in a rigid frame, and away from the anesthesiologist; see below. Special equipment: none. Special considerations: see below.

VP or LP shunts are minor procedures. Patients scheduled for placement of a shunt generally have chronic hydrocephalus, and placement of the shunt will drain intracranial CSF into the abdomen and reduce ICP. These patients may have signs and symptoms of elevated ICP, which include nausea, vomiting, confusion, ataxia, and papilledema. Shunt revisions are typically performed on previously functioning shunts which have now become obstructed or infected. Because these are minor procedures in patients with chronic obstructive pathology, drastic anesthetic measures to control or reduce ICP are not necessary. Similarly, an arterial line for precise beat-to-beat measurements is not required for purposes of the surgery. Of course, it is always desirable to avoid overt hypercapnia or hypoxemia. The subcutaneous tunneling of the shunt tubing from head to abdomen can be profoundly stimulating.

Stereotactic surgery is performed for diagnostic purposes, intractable epilepsy, for dyskinetic disorders such as Parkinson's disease, or for tumors near eloquent areas of the brain. As the brain itself is insensate and the surgeon specifically desires a lucidly awake patient who can interact and respond to questions, the patient is only minimally sedated during the majority of the operative portion of the case. However, the head must first be pinned. Often, these cases proceed with an initial period of general anesthesia while the surgeon injects local anesthetic into the scalp at the intended frame pinning sites and places the frame. Anesthesia at the surgical incision site is accomplished with local anesthetics by the surgeon, peripheral nerve blocks by the anesthesiologist (“scalp blocks”), or continuation of general anesthesia. The patient is then allowed to awake and is kept minimally sedated with low-dose midazolam, opioids, propofol, dexmedetomidine, or a combination thereof.

Since each of these surgeries has particular needs and protocol, knowledge of the surgery is key, and your attending will be instrumental for this. Notably, deep brain stimulator cases usually start with head pinning done under brief GA in the pre-op area followed by transport to the CT scanner, and then on to the OR for the “awake” procedure.

It is vital to remember that access to the airway in these cases is nearly impossible. The patient’s head is pinned in a frame and is 180° away from the anesthesia machine. Emergently releasing the pinning is not an option with the cranium open. As such, oversedation, hypoventilation, apnea, or upper airway obstruction can be catastrophic. Intense monitoring and judicious use of medications during the sedation phases of the procedure are mandatory to ensure a patent airway and adequate ventilation/oxygenation.

Stereotactic procedures are typified by long periods of boredom – the patient is awake and generally stable, and the surgery is minimally invasive – interspersed with bursts of stress while titrating in sedation and monitoring the patient for airway issues. Maintaining patient comfort and anxiolysis can be quite challenging. Remember, the OR table is hard and flat and the patient cannot readjust his or her position; back pain is common. Likewise, reassurance and psychological preparation of the patient for the ordeal of an awake brain surgery are crucial.
Chapter 9. Overview of SICU, Pain, Regional, Pediatrics, and Pre-op Rotations

Chapter 9A. SICU Rotation

The SICU experience consists of two 4-week blocks completed during the CA-1 and CA-2 years. During this time, you are part of the “SICU critical care team” which typically consists of 1-2 anesthesiology residents, 1 surgery resident, 1-2 surgical/OB-GYN interns, medical students, a trauma/critical care fellow, and an attending. Both surgical and anesthesiology attendings cover the ICU. While on the SICU rotation, you are not part of the MOR call pool and generally are not responsible for the typical MOR duties, with a few notable exceptions that will be explained below. A detailed syllabus is available and the finer points of the rotation will be explained at the start of the rotation.

The defining feature of the SICU is that it is an open unit, and most of the time, the SICU team functions as consultants. All non-primary patients (e.g. trauma, general surgery, neurosurgery, or neurocritical care) are seen, daily notes and plans are created, and recommendations are communicated to each respective primary team. In addition to the “consulting” aspect provided, the SICU team also serves as the primary providers for transplant, ENT, orthopedic, vascular, and OB/GYN patients, and is responsible for daily notes, orders, and administration of care. Therefore, the SICU experience can be less onerous than at other institutions because, at times, we don’t take care of as many primary patients at once.

A typical day on the SICU team is as follows: The residents and medical students show up at the appropriate time to pre-round on all the patients, typically around 0600, but this may differ depending on patient census and number of team members present. Rounds are then generally made with the fellow, the attending, or both, usually starting at 0830, though this may vary with the attending. Notes and recommendations are written, and any orders or management changes are carried out. There is usually a lecture shortly after rounds, often with a member of the team presenting a critical care topic. After the lecture and discussion, the non-call members sign out to the person on call. Thus, most non-call days are generally very light, usually ending around 1200-1300. Certain days, the call resident is an intern and this handoff/signout cannot occur until 2000.

Other responsibilities of the rotation are to receive consults from other teams and assist with placement of lines and airway and ventilator management. At times, certain teams such as neurosurgery get swamped with patients and they request the SICU team’s help in various ways. That being said, no management should be undertaken on another team’s patient without their prior approval.

As anesthesiology residents, we have unique responsibilities while on this rotation that the surgical members do not have. These include:

- Carrying the code pager and responding to it
- Helping or being the provider for OR Resuscitation cases
- Ensuring the code bags and emergency ORs are set up
- Completing all inpatient pre-ops for the next day
- Fulfilling requests our anesthesiology attendings may have for us to help move the day along, e.g. breaks, room setups, etc.

More information on the code pager, emergency OR setups, and OR Resus cases can be found in the appropriate sections. Generally, the SICU person on call is responsible for all of these things. If an emergency OR Resuscitation case arises, the floor attending has the discretion to decide who does the
case—whether it is a MOR resident, the SICU resident, or someone else. Expect to be involved or even to be the primary resident in any OR Resus. Also, our attendings may occasionally ask the SICU resident to give breaks in the MOR or even do a case. Remember that even when on the SICU rotation, you are still an anesthesiology resident, part of our department, and thus subject to its whims. If a conflict arises between anesthesiology and SICU duties, it can be discussed among the appropriate attendings.

The SICU experience represents a good learning opportunity in a fairly relaxed environment. The majority of the attendings are pleasant, easy to work with, and excellent at teaching. Many people find the paucity of primary patients liberating, in that one can still perform the mental exercise of determining the best course of management, without the burden of actually having to write orders or answer pages at 0200. Of course, the downside to this is that the learning experience may not be as rigorous as it would be in a completely closed unit. All that said, make the most of the opportunity to learn from rounds and enjoy the frequent early days.

As of June 2012, CA-3 residents also take a mandatory one-month rotation in the ICU at Thornton (TICU). The department is also becoming increasingly involved with neurocritical care at Hillcrest and Thornton hospitals. These rotations are “works in progress” and will not be described here.

Chapter 9B. Pain Medicine Rotation

The pain rotation consists of one mandatory month during the CA-1 or CA-2 year. It is also possible to complete a pain elective during the CA-3 year. During the 4-week rotation, you will help manage patients on the inpatient pain service, see patients in pain clinic, observe and perform pain procedures, and take pager call. These duties are usually shared between the resident rotating through pain and the pain fellows, with whom you will work closely. The typical week generally consists of daily rounding with the fellow on patients on the pain service at Thornton, followed by morning pain clinics at Perlman Clinic building, and then afternoon procedure clinics either at the VA and Moores Cancer Center. Assignments to various clinics and procedure days are made by the program coordinator and available online. In addition to daily rounding and note-writing on inpatient pain service patients, you are also expected to round on inpatient regional anesthesia patients (epidurals, peripheral nerve blocks, etc.) on Tuesdays and Thursdays. A detailed syllabus and the finer points of the rotation will be explained at the beginning of the rotation.

Clinic patients consist of either new referrals or returning patients. New referrals need a full H+P, with the obvious emphasis being on the patient’s pain history. After these patients are seen, they should be presented to the attending in clinic, who will then complete the interview and decide on the best course of treatment. Follow-up visits are generally less demanding, and are usually in the clinic for post-procedure evaluation, a medication refill, or for general maintenance. In these cases, you can consult the old notes in EPIC to get a quick overview of the patient. While in clinic, you are also responsible for writing the note and entering orders when the visit is complete. There are EPIC templates that the pain fellows will share with you at the start of your rotation.

The various procedures performed to treat chronic pain are numerous, and each attending has their own style as well. Typically, they will show or teach you the procedure first, and after you develop some experience and procedural skills, will let you perform some of the injections on your own. Depending on your performance and the attending, you may eventually get to perform virtually every procedure that comes through the door. The various procedures performed will be explained during the rotation. Listed below are some of the most common:
- Lumbar/thoracic/cervical epidural steroid injections (ESI)
- Trigger point injections
- Intra-articular injections (knee, shoulder, hip, etc.)
- Sacroiliac (SI) joint injections
- Medial branch blocks/facet joint blocks
- Radiofrequency ablation (RFA) of medial branch
- Botox injections
- Sympathetic blocks (stellate, celiac, lumbar, etc.)

Pain call is pager call. Typically, residents usually take pain call on 1 or 2 weekends (Friday 1700 through Monday 0700) during the four-week block. During your call weekend(s), you will be responsible for new consultations and rounding on all inpatients on the pain service at both Thornton and Hillcrest. After prerounding on these patients, you will then be expected to call the on-call attending to discuss the patients and your management plans. You are responsible for writing the daily progress note on these patients. While on call, all inpatient consults will be directed to you. Consults received before 1700 should be seen that same day, while consults received after 1700 can be seen the next day, depending on the urgency. After evaluating the new consult, the attending on call should be contacted for a definitive plan. The attending is also there to answer any questions you may have on any patients or new consults. New inpatient consults should be added to the pain patient list in EPIC, and the other members of the team should be notified so they know who to round on during the weekday mornings (to be explained when the rotation starts). What this means is that when you are on weekend call, the days may be long, as you may get called in to see consults up to 1700, in addition to your rounding responsibilities on pain service patients at both Hillcrest and Thornton.

Patients also call the operator to speak to the pain physician on call, who will then forward the patient’s contact number to you. It is your responsibility to call this patient regarding whatever issue or question(s) the patient has. Always, always dial *67 before dialing the patient’s number so that your phone number will be kept private from the patient. It is the policy of the pain department that pain medication refills or orders cannot be given over the phone, and patients wanting these things will just have to be told to wait until normal business hours and then call the clinic for an appointment. True medical emergencies must be asked about, and if present, the patient should be told to go to an ER. An example would be a patient who had a recent procedure, and now complains of bowel or bladder incontinence. If a patient has intractable pain, generally we do not recommend that the go to an ED for “exacerbations of chronic pain.” However, such a patient cannot be completely evaluated over the phone. Again, the attending on call is there to answer any questions you may have.

**Chapter 9C. Regional Anesthesia Rotation**

The regional anesthesia experience at UCSD consists of two mandatory months: one during the CA-1 or CA-2 years at Hillcrest and one during the CA-3 year at the VA. An additional 1-month “advanced regional” elective is available for CA-3s. During the first one-month block, you will perform peripheral nerve blocks on a variety of patients. Most of the nerve blocks will be for patients going to the OR, but others might be for patients on the ward, e.g., burn patients having twice-daily dressing changes. As the resident, you will perform many or most of the blocks, with the fellows doing the others. There is normally one fellow with you at Hillcrest, with a second fellow if it is an extremely busy day.

Daily duties include performing the blocks, finishing documentation, rounding on inpatients with catheters, writing progress notes, making phone calls to outpatients with catheters or single-shot blocks,
and helping to coordinate the next day’s blocks. Reflecting Hillcrest’s patient population, blocks are done on a mix of elective orthopedic upper- and lower-extremity cases or for trauma-related fractures, debridements, or skin grafting. Most blocks are done pre-op, but occasionally you might “wait and see” if a patient needs a block for adequate analgesia, or you might do a block to help with perfusion to a new flap or graft.

A typical day starts at 0545 in the block area, where you will draw up all the local anesthetic and midazolam/fentanyl that is likely to be required for the day. Patients for whom a block is planned will be brought to the block area, receive their block, and then be taken to the OR pre-op holding area. As such, blocks for the first OR cases of the day should be done by 0700 at the latest. The block RN will place the patient on monitors, conduct a timeout, and program/set-up the infusion pump as applicable. The resident is responsible for placing orders for the infusion in EPIC and managing the inpatient census list in EPIC. “Bread and butter” blocks at UCSD include:

- Brachial plexus: interscalene and infraclavicular
- Forearm (median, ulnar, radial)
- Intercostobrachial
- Transversus abdominis plane (TAP)
- Femoral
- Sciatic: subgluteal, popliteal
- Saphenous

Blocks that are typically reserved for fellows due to complexity, rarity, or risk include lumbar plexus and paravertebral blocks.

Regional call is a one-week call (Monday 0700 – Monday 0700) and is pager call. The resident will field all pages regarding all patients on the regional service. During the week, there is a fellow at Thornton, so questions regarding Thornton patients (and there are many) should be forwarded to that fellow. During the weekend or after hours, the resident covers both locations. On the weekend, you will round at both locations on the regional patients and write the notes after discussing the patients with the on-call regional attending. Any blocks that must be done over the weekend will be done by the call person and the call attending.

The VA regional rotation differs from the rotation at Hillcrest in a few ways. First, there are no regional anesthesia fellows at the VA; the regional resident is entirely responsible for the service on weekdays, and the call resident rounds on regional patients on the weekends. Secondly, the logistics of doing regional blocks are quite different at the VA due to the VA’s inherent bureaucracy and paperwork. Third, the total number of blocks you do is highly dependent on your own initiative.

A typical day starts at 0640 with morning conference. No blocks can be done prior to the “huddle,” which happens no earlier than 0700, so typically the huddle occurs and then the block is done in the pre-op holding area. Also prior to the block, a separate pre-block “timeout” occurs. The blocks and equipment themselves are quite similar to those at Hillcrest, with a few more items of paperwork than Hillcrest. The regional anesthesia patient list is kept in an Excel file, and the daily follow-up phone calls to outpatients and visits to inpatients are done in a similar fashion to Hillcrest. The day typically ends fairly early and the regional pager is handed off to the call resident.

ASRA Guidelines
Neuraxial anesthetic techniques carry a risk of hemorrhagic complications. The worst-case scenario is spinal/epidural hematoma leading to a devastating outcome like spinal cord compression and paraplegia. Peripheral nerve blocks carry a similar type risk to the nerves being blocked. However, the actual incidence of neurologic dysfunction resulting from hemorrhagic complications associated with neuraxial anesthesia is unknown.

The most recent guidelines published by the American Society of Regional Anesthesia and Pain Medicine (ASRA) are recommendations based on evidence-based reviews. These are consensus statements based on case reports, clinical series, and pharmacology. As always, you should use your best judgment as to when neuraxial anesthesia is appropriate for a patient, and remember that removal of an epidural or spinal catheter in a coagulopathic patient can be just as risky as placement of one.

### 2010 ASRA Guidelines (3rd Edition)

<table>
<thead>
<tr>
<th>Anticoagulant</th>
<th>Placement</th>
<th>Removal</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Subcutaneous heparin</strong></td>
<td>• No contraindication with 5000U BID dosing. Reduce risk of bleeding by starting heparin after block. • Safety for patients on &gt;10,000U daily or more than TID dosing has not been established.</td>
<td>• Remove catheters prior to next dose of heparin and restart heparin 2 hours after. • Check platelet count for HIT if patients have been receiving heparin for more than 4 days.</td>
</tr>
<tr>
<td><strong>IV heparin</strong></td>
<td>• Can start heparin 1hr after neuraxial block.</td>
<td>• Remove catheters 2-4hrs after last heparin dose; can restart heparin 1 hour after</td>
</tr>
<tr>
<td><strong>LMWH</strong></td>
<td>• For patients on daily dosing, wait 10-12hrs after last dose • For patients on BID dosing (whether prophylactic or treatment), wait at least 24hrs after last dose • For patients who receive a dose before surgery, recommend against neuraxial techniques</td>
<td>• For BID dosing, first dose should be 24hrs post-op. Remove catheter 2hrs prior to first dose • For daily dosing, remove catheters 10-12hrs after last dose and resume dosing 2hrs after removal</td>
</tr>
<tr>
<td><strong>Warfarin</strong></td>
<td>• Ensure medication has been discontinued and INR has normalized</td>
<td>• INR &lt; 1.5</td>
</tr>
<tr>
<td><strong>Antiplatelet drugs</strong></td>
<td>• NSAIDS: no contraindication • Aspirin: no contraindication • Ticlopidine: discontinue for 14 days • Clopidogrel, prasugrel: discontinue for 7 days • Abciximab: discontinue for 24-48hrs • Eptifibatide/tirofiban: discontinue for 4-8hrs</td>
<td></td>
</tr>
<tr>
<td><strong>Thrombolytics</strong></td>
<td>• Absolute contraindication</td>
<td>• No definitive recommendation for patients who have a catheter and unexpectedly receive these drugs • Consider checking fibrinogen level</td>
</tr>
<tr>
<td><strong>Thrombin inhibitors</strong></td>
<td>• Due to lack of data, ASRA recommends against neuraxial blocks</td>
<td></td>
</tr>
<tr>
<td><strong>Fondaparinux</strong></td>
<td>• Due to lack of data, ASRA recommends against neuraxial blocks</td>
<td></td>
</tr>
<tr>
<td><strong>Herbals</strong></td>
<td>• No contraindication</td>
<td></td>
</tr>
</tbody>
</table>

* For patients undergoing deep plexus or peripheral block, ASRA recommends that recommendations regarding neuraxial techniques be similarly applied.
**Always check the medical record and ensure that the patient is not taking other medications that affect clotting mechanisms.

### Chapter 9D. Pediatric Anesthesia Rotation

The vast majority of our experience with pediatric anesthesia takes place at Rady Children’s Hospital San Diego. While there are sporadic pediatric cases at Hillcrest – and these children tend to be quite ill – RCHSD provides intensive, daily exposure to pediatric anesthesia during the rotation. Two one-month
mandatory rotations are done, one during the CA-2 year and another during the CA-3 year. A third
month, as a CA-3, is an elective option. Additionally, two consecutive months can be spent at Children’s
Hospital of Los Angeles as a CA-3 elective. Pediatric patients differ markedly from adults in physiology,
cognitive ability, and the typical type of case encountered, and that information will not be addressed
here. What follows are the expectations and requirements during the RCHSD experience.

The rotation at RCHSD is quite different than almost every other experience during residency. Nearly all
of the attendings are in a large private practice group with no formal affiliation with UCSD; a few
attendings in our department also work at RCHSD. Each room is staffed by an attending who expects to
be doing the case solo, and there are no formal room assignments for the residents. Residents are
generally free to choose which cases they would like to see during the day, and are not required to stay
in a specific room for the whole day. This affords the luxury of tailoring our experience to our desires for
that day. So, if on one day, two interesting cases are scheduled in different rooms, it is entirely possible
to start one case and then switch to the other room later. If you spent the previous day in the ENT room,
you may spend the next day in the urology room, gaining experience in caudal injections. The flexibility
of being able to tailor your experience is unique to this rotation. Clearly, this is subject to the approval of
whichever attending you happen to be working with at the moment. If you start a case with one
attending, and then ask to go to another room, and they would rather you stay put, you should use your
best judgment. Similarly, if an attending doesn’t feel like working with a resident that day, they’ll let you
know to find another room. Most of the attendings at RCHSD like to work with residents, are more than
willing to teach, and are flexible with letting us jump from room to room.

The exception to the practice above is when you are assigned to be the “diamond” (call) resident on a
particular day; this is assigned when you start the rotation. When you are the diamond resident, you are
paired with the attending running the floor so they have the flexibility to leave the room and take care
of floor/PACU/OR issues. Since the floor runner has to stay close to the main OR board, they are usually
assigned to OR1 or OR2, which means you will be in one of those ORs for the day. The floor runner will
keep the diamond resident until things start to wind down. Sometimes, this could be late afternoon but
depending on the attending, it could also be late evening. Once sent home, you must be available by
pager overnight. Typically, the attendings will only call you back in for an exceptionally interesting case;
if this happens, you will have the next day off. When on call during the weekend, the usual expectation
is that you will show up on Saturday mornings to do cases – and there are always cases – and be
available by pager for the rest of the night and for Sunday. Again, this is dependent on the attending on
call. Some expect you to come in, some will call you if they need you, and others will tell you not to
worry because you definitely won’t be called. It may be possible to establish this beforehand with the
attending you will be on call with, but in general, expect to come in on Saturday, but not on Sunday.

While at RCHSD, you are still expected to attend M&M and return to UCSD for lectures or visiting
professor sessions. If you are the diamond resident that day, remind the floor runner that you must leave for lecture in the afternoon and they will let you know if you need to return after lecture. Most
RCHSD attendings are aware that our residents have lecture on Wednesdays and if you are not the
diamond resident, then they do not expect you to go back.

The downside of the freedom of choosing your own room at RCHSD is that the learning may be quite
unstructured. Often, you have no idea what you’ll be doing the next day and thus can’t tailor your
reading prior to a case. Most of the attendings prefer the residents not participate in the pre-op
evaluation of the patients, since we slow the process down. You will quickly see that RCHSD is devoted
to rapid turnovers and high productivity, and the attendings hate anything that wastes time or slows the
day down. This same mentality causes a small number of attendings to avoid residents, and you will quickly learn who those attendings are. RCHSD is definitely a rotation for adult learners. If you have a lot of initiative and try to get the most out of the rotation it can be a great experience; conversely, if you find ways to hide, don’t expect to learn very much.

There are other notable impacts of the “maximizing productivity” stance of RCHSD. Almost every patient emerges and is extubated in the PACU by a PACU nurse. A typical case will end with the patient breathing spontaneously, still intubated, and brought to PACU unmonitored. After giving report, the anesthesiologist goes to start the next case, while the PACU nurse extubates the patient when appropriate. This serves to cut down on turnover time. While most of the PACU nurses are excellent, there have been several near-disasters when a patient was inappropriately managed with an attending not present. Furthermore, as residents, we may not get much experience with emergence and extubation of the pediatric patient.

Another common impact is the “attending shuffle” which occurs throughout the day. The attending call system at RCHSD defines the order in which the attendings may leave, with highest call having to stay the latest. This is a rotational system that is generally balanced. The upside of being a high call is that the attendings get first choice on high-paying ORs, and can bump any lower call attending during the day. The impact on the residents is that you may work with several attendings during the day even if you stay in the same actual OR. Furthermore, if you want to work with the same attending, you may have to follow him or her from room to room, to the CT scanner, to MRI, and so on and so forth. This can be somewhat frustrating.

A typical morning at RCHSD is quite different than at our other hospitals. There is little to no setup of the room beforehand. Most attendings walk in 5 minutes before the cases start and have little to no knowledge of their patient. Furthermore, they often do not draw up drugs beforehand, or even check the circuit prior to bringing the patient back into the room. As such, the attendings don’t expect the residents to do this either. From the resident point of view, the morning usually begins with inspecting the OR board for good cases, figuring out who the attending assigned to that room is, and approaching that attending. This lack of preparation can be frustrating because from the resident point of view, you are doing new cases on a scary class of patient with little to no experience. This is part of the challenge and the learning value of the RCHSD rotation.

In summary, RCHSD can be a great experience, but it takes initiative and maturity on the part of the resident. A certain comfort level with pediatric anesthesia can only be obtained with repeated, daily exposure and RCHSD does give us that.

Chapter 9E. Pre-op Clinic and Radiation Therapy

This is a 2- or 4-week rotation in the pre-op clinic at the Perlman Clinic building at Thornton. This duty is combined with providing anesthesia for pediatric patients undergoing radiation therapy at Moore’s Cancer Center. The pre-op clinic experience itself is very much a typical outpatient clinic, where you are seeing patients for pre-anesthetic evaluation prior to their scheduled, elective cases, on a day where they are also seeing their surgeon and typically having pre-op labwork drawn. A typical clinic day consists of 20-30 patients, with the resident and 2 NPs seeing those patients. The anesthesiology resident is primarily responsible for seeing patients who have already seen a staff member of the surgery department, and are coming to the clinic purely for anesthesia pre-op evaluation. The NPs typically see the patients who need both surgical H+P and anesthesia pre-op evaluation. The attending
running the floor at Thornton is responsible for signing off these pre-ops, and is the one you can contact with questions or concerns. Clinic starts at 0830, but your ability to attend at this time is determined by the radiation therapy cases, which are discussed next.

Radiation Therapy is provided at Moore’s Cancer Center, and the pediatric patients undergoing these treatments require GA to remain motionless for their irradiation sessions. You will be working very closely with a pediatric anesthesia attending on this rotation, and they will help you with all of its facets.

A couple of quick comments:

- The first case is usually scheduled to start between 0730 and 0800; you should arrive 15 minutes prior to set up.
- The anesthetic typically consists of intermittent propofol boluses (or infusion) to induce and maintain GA while the child continues to spontaneously ventilate via a simple oxygen facemask.
- There is an anesthesia machine in the XRT room itself, but it is rarely used.
- Routine monitors, however, are always used.
- There may be anywhere from zero to 5 or more cases scheduled each weekday, and typically the patients come back several times a week.
- Most of these patients have vascular access ports (“Port-a-cath”) which are used for your IV anesthetic and must be accessed and flushed appropriately.

At the conclusion of these cases, you should report immediately to Thornton pre-op clinic.