A Guide to UCSD Anesthesia for Residents

2nd Edition

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Author’s disclaimer- the majority of this guide was written when I was a CA-3 resident in 2007. As such, some of the information may have changed and no longer be current. As examples, I am aware of new, different cases (e.g., HIPEC) that are now routinely performed at UCSD but were not at the time of writing. Many of the rotations have changed in structure and expectation. Some of the medications that were common in 2007 are no longer employed (e.g., aprotinin). This guide should still prove useful, but it is not meant to replace a current textbook nor supplant current rotation requirements. Thanks.

Leon Chang 3/2011
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Welcome to UCSD anesthesia. This guide is intended to be an “insider’s look” or a “resident’s perspective” on our daily lives here. It provides basic information on various rotations and other aspects of the anesthesia residency. As such, it is intended to serve as a reference to incoming residents. In no way does this guide replace existing syllabi for the various rotations, nor does it explain anesthetic pharmacology and physiology (at least, in any great detail). Rather, it will provide the kind of help that one resident wants from another when he asks, “hey, what can I expect out of this month/rotation/case/situation?”

**Our philosophy**

In many ways, the residents here make this place what it is. We pride ourselves on being a tight program with great camaraderie. Hopefully you knew this already and it’s 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 in time you will be to help them. We routinely help each other out starting IVs, doing preops, giving breaks, and so on- the list is endless. Possibly the best example of this occurs during call- often the senior resident will be supervising/helping the junior resident, while the whole team continues setting up rooms for each other, pre-oping the next patient, etc. If you are ready and willing to lend a hand to your brothers and sisters that kindness will be visited back to you ten-fold in the future. Likewise, if you shirk responsibility and leave others to fend for themselves, it will be noticed. Remember that we’re all in this together, and if things are 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 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.

The underlying message here is this is a resident- driven program. To remain as such it depends on residents taking the initiative in patient care and getting the work done. There will be innumerable 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 the responsibility we’re given enhances our education and our ability to function independently. By treating our duty with the utmost respect this tradition will continue.
Rotations at UCSD Anesthesia

Each year at UCSD is comprised of thirteen 4-week blocks. Of these blocks, the vast majority are required rotations. The required rotations are-

- Main OR (MOR)
- VA (a main OR rotation)
- Obstetrical/labor anesthesia
- Neuro anesthesia (2 months)
- Cardiac anesthesia (2 months total, one at the VA)
- Pain
- Pediatrics (done at Children’s Hospital San Diego)
- SICU (5 months, including any ICU experience taken during the PGY-1 year)
- Airway
- Regional anesthesia

Available elective rotations (taken during the 3rd year) include-

- CA3 cardiac
- Advanced regional anesthesia
- Ambulatory anesthesia
- Research
- CA3 pain
- CA3 pediatrics (at CHSD or LA Children’s)
- CA3 OB @ Mary Birch Hospital
- Potentially others of your own design

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-

CA1 year- primarily MOR and VA provide a solid base and exposure to daily OR anesthesia. During this year you will also have a few advanced rotations, e.g. OB or cardiac anesthesia.

CA2 year- Exposure to all required rotations except regional will be complete by the end of the CA2 year, interspersed with MOR and the VA.

CA3 year- if following the basic clinical track, the resident will be able to choose from 5 of the above electives. 1 of the 5 electives must be “on-site” at UCSD, e.g. ambulatory anesthesia. The required regional rotation is completed during this year. Required MOR and VA weeks round out the year.

If you choose to follow the research track, this year will be significantly different. In general, your elective blocks are usually devoted to research time. The exact nature and timing of your rotations will vary depending on your research project, your preceptor
and program needs. If you intend to follow the research track your research preceptor and advisor will have more information.
A typical day

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 plan on having your room set up by 6:40am (Wednesdays being the exception due to M+M) in order to make morning conference. Thus, you should show up early enough in the morning to allow yourself enough time to gather everything you’ll need. Depending on the case this can be simple or quite time consuming. Set-up activities can include but are not limited to drawing up fresh drugs, getting tubes ready, setting up drips, special monitoring equipment, and so on. Further complicating matters is your own individual speed. Bottom line- allow yourself enough time. The responsibility to get things properly set up is yours and yours alone. For new residents, I would allow around 45 minutes for a basic case until you get your style down.

Often the anesthesia monitoring team can provide invaluable assistance to your setup; however, don’t count on this as they are busy, spread thin, and have their own morning responsibilities. They are most helpful if you need some special item or if you’ve discovered an equipment problem during your routine check. My recommendation would be to learn from them as much as you can, so later if you need to grab something and they’re not around, or it’s 2am and you’re on call you know where to find it.

Once you’ve set up the room hopefully there’s enough time left for you to chat with your patient before conference. Here you can answer any last minute questions, ask some of your own (i.e., are they NPO, have they taken their morning meds?) and confirm IV access. At the U or Thornton IVs will often be done for you- however, it’s your case and your patient. If you feel the patient needs more access to start than they have, it’s your job to make it happen. Also, later in the afternoon staff members start to leave, and you find out how helpful they normally are when you have to put all the IVs in yourself. At the VA you start ALL your IVs from first in the morning on so allow time for this.

Word to the wise- if you don’t do all this before conference, you’re going to have to after, so better to get everything lined up early rather than later…

Conference typically ends around 7:00, +/- 5min. The expectation is you will be back in your room, with the patient ready to go at 7:10 (7:30 @ the VA). However, before your patient can be brought back any number of items must be checked off, including surgical consent, up to date H+P, nurses are ready, equipment is ready, etc. It’s truly amazing how many times these things AREN’T ready to go. So, before you roll back check with the circulating nurse for your room- they generally have the best idea if everything else is ready to go. You should also have some idea about these things, and this will come with experience. After all, you wouldn’t want to bring a patient back for a case that you know will have a lot of blood loss and likely require transfusion without appropriate labs having been sent.

Now, your day is underway. You can generally expect a morning break, 15 minutes long. The timing of this might vary depending on how busy things are
throughout the OR and whether or not you’ve “displeased” the powers that be. After you finish a case, typical drop off point is the PACU, although some cases might go straight to the ICU or other location. Once you’ve tucked away your patient and given report to whoever will assume care (typically, PACU nurse), time to go setup the room for the next case, meet your next patient, and so on. Again, you can’t bring your patient back until everyone else is ready to go- this includes the room being turned over and cleaned. So, if you’re fast about things you can often get a few extra minutes between cases to relax (sometimes more than a few minutes). Your caseload will generally follow the order you expected, i.e. the schedule. However, things get cancelled, moved around, and emergencies happen. So don’t be surprised to get moved to another room, or to suddenly have to do an emergency case that just came in. The name of the game is flexibility. The attendings running the floor try not to disrupt the preparation you’ve put in for your expected cases if they can help it.

Lunch breaks are up to 30 minutes, and the same comments can be made as above regarding timing, etc. Afternoon breaks are more variable due to cases winding down and staff leaving, but will also be 15 min in duration. Typically your day will end around 4-5pm if you’re not late or on call, but remember this is residency. In addition to your education you’re here to work and the work has to get done, and sometimes that comes back to bite you. On the other hand there will be days when you leave at 2pm, because the work is done. So it cuts both ways. Bottom line, if you go into it with a good attitude things will work themselves out. If you don’t expect to leave at a certain time it’s hard to get disappointed. Just remember that hard work is rewarded, while complaining is remembered for a long time. When your room is done ask the attending running the board if there’s any more work for you. At this point, you will find out if there is, or if you’re going home. Simple. Often you don’t even have to do this, as the floor attending has a good idea of what’s going on globally and will simply tell you you’re done 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. Enough said.

Once you’re told you can go, there’s one last thing to do- find out what you’re doing tomorrow and get the preop’s for those patients. This could be as easy as photocopying them or as annoying as tracking down five inpatients and pre-oping them all. See the preop section for more information. Later that night you’ll be discussing the cases and the anesthetic plan with the next day’s attending, and it behooves you to have that information handy.

Don’t forget to do something fun with what remains of your day, in between all the reading and working. Seriously. It’s hard and stressful at first but will get better.
The first four weeks as a UCSD resident, or “I’m going to be doing this on my own in a month?”

The transition from the clinical base 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 medicine or surgical intern to a completely green anesthesia 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 write discharge orders largely fall by the wayside and become irrelevant. All of the 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 anesthesia and exactly what is entailed vary greatly. Some of you perhaps were able to shadow a family friend or mentor in the field for years. Others (as was the case with me) will have had only one, or at best two anesthesia rotations in medical school and have essentially no clue what to expect. The department knows all of this and expects no prior knowledge or ability, save a good fundamental grounding in medicine itself. After all, that’s what the residency is for- to become an anesthesiologist. However, we expect that you know and appreciate one piece of information prior to embarking on your anesthetic career. It is so important, and so vastly different from any prior experience a “pre-anesthesiologist” 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 clearer. As a surgical 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-

a) you write the wrong order
b) your senior resident or attending misses the fact this is a wrong order
c) the nurse misses the fact this is a wrong order, and
d) the nurse has to physically go to the medication dispenser, and administer it to the patient

This example doesn’t even take into account the multiple other checks which usually exist, e.g. pharmacy has to also approve the medication. Here is the equivalent example for an anesthesiologist-

a) you decide Mr. X needs a drug in the OR. You draw it up and there’s no one around to confirm you are doing it correctly. You then give the drug to Mr. X, who can’t even object because he is anesthetized. Your first indication something is wrong is when things starting “hitting the fan”.
There are so many other potential sources for error or harm they would fill volumes (and have). You could flick a switch, turning a machine or monitor off without even knowing you flicked it. 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.

Thus, UCSD has a system to transition people from interns to fledgling anesthesiologists within the first month. This system is steeped in transition 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.

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

Typically, after a few days depending on their comfort level, the CA-1s will begin calling attendings to discuss the cases for the next day. This allows the CA-1 to become familiar with the anesthesia pre-operative evaluation and to start learning how to formulate an anesthetic plan. As always, the CA-3 is available to discuss to plan before the CA-1 calls the attending.

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.

II. The second two weeks

At this point the CA-1s are paired up 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, without having to get to know a new person every day. Typically the resident is allowed more responsibility and independence during these second two weeks. The faculty must still be called every night to discuss the next day’s cases.
During this whole month the new CA-1s have a daily lecture at 3pm. These lectures are designed to provide the “nuts and bolts” of anesthesia. These lectures are mandatory and the CA-3s and faculty know you must attend them. As such 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 du jour. Getting the preops, preparing for the next day’s cases and calling attendings is of course non-negotiable.

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 anesthesia resident in a 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 anesthesia 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.
Call duties

MOR call

This is the first type of call a resident takes and typically the one that causes the most anxiety. Here at UCSD we start off taking call early, right at the beginning of August after your 1st four weeks of one-on-one mentoring are done. You might not feel ready for it (nobody ever does at first), but remember there are senior residents and your attending standing by to help you out.

For weekday calls, typically the day will start at 9am in the preop clinic. The clinic is located on the first floor near the cafeteria, down the hall to the left of the information desk in the front lobby (when facing the desk). This is meant to give you a light as well as a late start to the day. Preop clinic generally runs from 9-4pm or so, whenever the preops are done. See the preop section and syllabus for more information.

Typically one call resident and a Nurse Practitioner are there, rarely a CRNA, and rarely you will be yourself. Periodically if the day in the ORs is light the senior of the two residents (usually, the OB person) will come in at 3pm. Also, there will be times (thankfully, very rare now) when you will have to come in for a 7am case just like a regular day if there aren’t enough people available. If this happens the attending running the floor will make every effort to relieve you as soon as possible to rest or otherwise take it easy for a while before your duties resume. Just realize that if you have to come in early it’s for an unavoidable reason, and thus you should DEFINITELY just suck it up and not complain. As always, hard work will be noticed and rewarded. You will know whether you’re coming in early, at 9am, or hopefully at 3pm by the standard emailed schedule. Always check for updated schedules - things change. It’s probably also a good idea to have your pager on you the night before your call day, so if things change and the front desk decides to page you you’ll know.

After all the preops are done, head up to the MOR and check in with the attending running the floor. They will tell you what is in store for you - be it resting for a while, taking over a room, giving a dinner break, or getting dinner yourself. From this point on you are at the mercy of fate and the attending. Expect to finish the last room running. Obviously any cases that get added on during the night are your responsibility, but the front desk will page you to let you know this. The good news is once all the cases are done, you’re free to relax or go to sleep. The workload is highly variable - we’ve all had calls where we didn’t do anything after 5pm, as well as calls where we came in at 7am, and did 24 straight hours of anesthesia. Bottom line - once 7am the next day rolls around you will be relieved.

Weekend MOR call is slightly different. For one, you come in at 7am and it’s a 24 hour shift. For another, there are often (meaning - the VAST MAJORITY OF THE TIME) elective cases scheduled during the weekend. This means that if you’re on call Saturday, and there are three elective cases scheduled, it’s your responsibility to call the
attending that will be with you that day with the preops the night before, just as if it was a weekday. This can be a real drag b/c often the weekend scheduled cases are inpatients, meaning they have to be preop’d. Furthermore, you have to give yourself time to set up the room for the case in standard fashion, so you end up coming in before 7am. Err on the side of caution with calling attendings- many won’t care if you call or not, or they’ll be happy with a 2 second rundown of the weekend cases. Others will want all the details. You’ll figure out who is who as you go along but play it safe in the beginning. To find out if there are cases scheduled on the weekend, you have many options, including accessing the schedule on PCIS, or calling the OR front desk. One bright spot is that the attending on call traditionally buys the whole call team dinner from some nearby restaurant. This happens with very few exceptions.

**Weekend backup call**

Every weekend there are two residents assigned to “backup” duties for the Hillcrest MOR. These residents alternate between first and second backup on Saturday and Sunday- thus first backup on Saturday will be second on Sunday and vice versa. Back up is called up at the discretion of the MOR attending. Very infrequently, backup may be called to assist with a case at Thornton. Typically you can expect to be called in as a first backup, much less so if you are second backup.

The expectation as backup is that you will have your pager on you starting the night before so that you are reachable, and that the pager remains on even if you get sent home. It is routine to come in for a 7 am case, finish and go home, only to be called back 1 hr later. Furthermore, most attendings expect you to check the schedule on PCIS the night before. If you are first backup and see that there are two rooms that will run simultaneously, you should probably be prepared to just come in without being called. The call resident will be in one room and first backup will be in the other.

In general be prepared to work most of the day as a first backup. The good news is, when the OR cases are down to one room you should be relieved. Also, backup duties count as “working a weekend” and count towards our policy of having at least 2 weekends free.

**MOR/Thornton/VA late**

The late resident is the second to last resident to leave the hospital. You take over rooms, relieving other residents or CRNAs as applicable and deemed by the floor attending. The duration of this shift can be quite variable- people have left as early as 3pm or as late as 10pm.

At times, when you are the late resident the daily schedule does not reflect this and the floor attending is also unaware of your status. It is your duty in these situations to inform the floor attending that you are the late resident. There have been times in the
past when the late resident has been told to go by the floor attending, who was simply unaware that resident was supposed to stay late. These instances are remembered for a long time and unfair to other residents. If you take care of your fellow residents they will take care of you.

**Preop at Thornton**

When assigned to this shift the resident comes in to Thornton preop clinic at 8:30am and, with a Nurse Practitioner, sees the scheduled patients for the day. Other duties include visiting the post-op patients from the day before and calling those that have already gone home. Kelle Cale, Dr. McCarren’s secretary will provide a list of those patients with applicable room and phone numbers. After the clinic is done in the afternoon the resident takes over an OR if necessary. As always, check with the floor attending when you are done.

**3pm – call shift at Thornton**

The resident comes in at 3pm at takes over an OR, and then stays until all the cases are done. This can be quite variable at Thornton. They then remain on pager call overnight. Having your pager on during the day is encouraged, because infrequently you get called in earlier than 3pm (e.g., add-on emergency case that there is no one else available for). The floor attendings will respect the “10 hr rule”, meaning if you work late you will not be called back in for at least 10 hrs.

**VA call**

On weekdays the resident comes in at 10am. Duties include seeing patients in ASU (VA version of preop clinic), seeing post-op patients and inpatient preops, as well as holding the code pager. At the attending’s discretion you may be asked to perform other duties such as giving breaks or starting a case expected to go late.

Once all the patients in ASU are seen (typically 4-5pm) and all other duties are finished, the call resident takes over a room. 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 emergencies, the resident is on his own. If an emergency OR case comes in the call attending should be notified. The resident does not do OR cases on their own.

Weekends at the VA are typically very light. Often there are no cases scheduled which means the attending will not be coming in. After taking over for the outgoing resident, call duties include seeing inpatient preops (added on to the main OR board), post op checks, and holding the code pager. We are also responsible for any new pain
consults and follow ups over the weekend. The pain attending on call 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.

**OB call**

Details about the duties on OB will be discussed further in the OB section. During weekday calls the same basic principles as described above for MOR call apply- you will usually come in at 9am to preop clinic, have your pager on the night before in case you get called in early, and so on. When you are done with preop clinic check in with the floor attending, who will usually have you relive the OB day person.

Once you have taken over the OB pager you become responsible for any OB issues that may arise- epidurals and cesarean sections to name a few. The OB anesthesia attending is the person you answer to and should be notified if you are doing anything. Of course, the MOR attending is always a helpful resource if need be. 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. Take these additional duties as the compliments that they are.

On weekends the procedure is fairly simple- come in at 7am and take over for the outgoing resident. Often times the OB call resident will try and get some inpatient preops done for the other residents, since there is usually some downtime when on OB call. It’s really nice when the schedule comes out for the next day and you find out you have two inpatient preops to do, and then learn that the OB call resident has already done them. That being said, don’t expect the OB call person to have done any preops- it’s just a nice bonus that sometimes happens.

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

**ICU call**

This will be described in detail in the ICU section. In general your duties will be holding and responding to the code pager, making sure the emergency rooms and code bags are stocked, and managing any ICU patients you may be taking care of. Additional duties may include responding to an emergency OR resuscitation or helping out another resident.

**Night float/liver transplant call**
After your cardiac and OB rotations are complete you become available for the night float shift. The shift is typically 1-2 weeks in duration, Monday through Friday, 7pm to 7am. The night float is available to fill whatever need is deemed most pressing by the MOR and OB attendings- from taking over an OR and sending someone home, to helping out on OB, to just holding the code pager when there’s no ICU person present. You are also on liver call during this time. Although the shift is from 7pm to 7am, you should have your pager on during the day as well in case there is a liver transplant that you are needed for. It has never been clearly defined whether the night float resident is responsible for liver transplants during the day. Some attendings will attempt to use available day personnel to do a daytime transplant, but sometimes lack of available people does not allow this. Thus, it’s better to just have your pager with you during the day in case you get called. If you happen to work during the day you will be given the night off, no exceptions.

CA-1 Heart call

This shift covers all basic cardiac cases, including late and weekend cases. The call is home call. Typically the resident on his first month of the cardiac rotation is on CA-1 heart call every day except Saturday. 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 (preoping the patient, setting up the room, calling the attending, etc).

Heart transplant call

This is also a home call shift, typically for more senior residents. It assumes the cardiac rotation has been completed. Responsibilities include lung transplants as well as potentially more “involved” cases when the cardiac anesthesia attending feels he needs more experienced hands. This call is typically very easy, but be aware you can work a regular day in the OR and still be on heart transplant call.

Pediatric (“diamond”) call at Children’s hospital and pain call

The details of these calls will 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.
A “basic” room setup

There are certain basics that will apply to every room setup you do before a case. The room setup becomes modified as the case requires, but in general most of us have a checklist that we do not deviate from. Having a checklist will help you remember what you need and make it difficult to leave out certain critical steps which, if forgotten, can look quite embarrassing and might be dangerous for the patient. There are many mnemonics for a basic room setup. The one I use is “MS DAMIT”. Briefly,

M- machine
S- suction
D- drugs
A- airway
M- monitors
I- IV
T- tank, table, tape

Machine-

Perform a machine check and adjust settings as desired (e.g., a pediatric case might need different ventilation settings). Ensure everything in relation to the case in appropriate with the machine (close enough to the patient, circuit is long enough, etc).

Suction-

Verify that the suction is on and readily available.

Drugs-

Prepare appropriate drugs for the case. The basic drugs most of us have drawn up are a narcotic, midazolam, succinylcholine, vecuronium, ephedrine, phenylephrine, lidocaine 2%, and two syringes of propofol (often you find yourself needing a second at a time it is most inconvenient to draw up). Depending on the case you may want many other drugs ready to go. Prepare any special drips or infusions the case may require.

Airway-

Prepare the ET tubes, LMA or other airway you will be using for the case. Ensure that a backup LMA is available in case of an emergency airway (cannot intubate/ventilate situation). Get the other airway devices that you may use- oral airways, bite block, tongue blade, etc. Ensure there are a variety of functioning laryngoscopes present.

Monitors-
Ensure the standard ASA monitors- BP cuff, EKG leads, saturation probe and temperature probe are ready to go. If you will use special monitors (e.g., arterial line, EEG) get the necessary equipment.

**IV-**

Prepare IV fluids if necessary and equipment to place an IV (often this will be done for you; the nurse in the holding area will usually place the IV at Hillcrest and Thornton).

**Tank, table, tape**

Ensure a backup E-cylinder of oxygen is available with a mapleson circuit and mask and that it has oxygen. Situate the table as you see fit, including any pillows/positioning aids you might need. Get out the tape or other devices you will use to secure the ET tube, lines, etc.
Code bags, the code pager and Emergency Room setup

Part of resident duties include ensuring that the code bags are always set up, and that there are OR’s set up for an emergency craniotomy and an emergency trauma resuscitation. Both these duties will be described in detail.

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. The code bag contents reflect this, with a large part of the equipment being devoted towards airway management. However, often times during codes the anesthesia provider is the most experienced physician around, 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.

The most common situations for which we will be called and which the code bag will be useful are codes themselves, urgent or emergent intubations, and brief out of OR procedures that may require airway management.

At Thornton we are currently not responsible for stocking code bags, nor 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, the question arises- who specifically stocks the code bags? In the past the person responsible for making sure the code bags are stocked has been nebulous. At various points the MOR call person or the person holding the code pager has taken responsibility. While there is no clearly defined person, we feel that whoever is holding the code pager should make sure the bags are “good to go”, b/c they are the ones literally left holding the bag if things aren’t up to snuff. At the VA this will usually be the call resident, but at Hillcrest it might be someone on the SICU rotation, night float, or even the OB or MOR call person.

Any time you take responsibility for the code pager (and thus the bags) you should check to make sure things are in order. If you accept the bags make sure you ask if things are in order- in theory, the bags should ALWAYS be up to date b/c after things are used they should be immediately replaced. Likewise, you should never hand off responsibility of the bags without them being fully stocked, both from a patient care and a personal-pride standpoint.

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-

- Fresh drugs- these MUST be drawn up every 24 hrs. Date and time the labels. At the minimum, the drugs available should be 1) succinylcholine, 20mg/ml, 2) rocuronium 10mg/ml, 3) phenylephrine 100mcg/ml, 4) ephedrine 5mg/ml, 5) etomidate 2mg/ml. This will provide a variety of pressors, muscle relaxants and an induction agent. As of now the pharmacy is providing new drugs for our bags every 24 hrs, but after use or on weekends they must be physically replaced by us.

- Other drugs in the drug pack, not necessarily drawn up- epinephrine, atropine, vasopressin, lidocaine, propofol

- Airway- a variety of blades and handles, and a 6, 7, and 8.0 ETT. Tongue blades, nasal and oral airways, McGill forceps, tape and twill ties. Mapleson circuit with a variety of mask sizes

- Airway rescue devices- a variety of LMAs, nasal RAE tubes, combitube

- Airway confirmation devices- an end-tidal CO2 detector (“EZ cap”) and an esophageal intubation detector

- Miscellaneous- airway exchange catheters, yankauer suction tips, NG tube, IVs, arterial line catheters and setups, billing paperwork (for filling out after going to a code, starting a line, etc)

Remember to restock the bags with whatever you have used- this will save a lot of headache (and possibly a life) later.

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

- A standard LMA 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 among other things a nasal RAE) and it is nearly impossible to remove a standard LMA over an endotracheal tube if you do intubate through one.

- The fastrach LMA and blue Cookgas LMA are intended to facilitate blind tracheal intubation in a crisis. Needless to say the ability to secure an airway is priceless. In addition fiberoptic intubation through these LMAs is considerably easier. The downside is most of us are less familiar with these devices and potentially have not developed the skills needed to use them properly.

- 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 CO2 detector and the esophageal intubation bulb detector.

a) The EZ cap is placed on the endotracheal tube adaptor and detects expired CO2. A successful intubation will be confirmed with a color change on the cap which roughly correlates with the amount of expired CO2. Ventilating a tube in the esophagus will not
result in sustained expired CO2 and EZ cap color change. Clearly, in a situation where the patient has no cardiac output (i.e., cardiac arrest with ineffective chest compressions) the EZ cap will not change color. In these circumstances the esophageal bulb detector is warranted.

b) The esophageal bulb detector is also attached to the adaptor of the ETT after intubation. When deflated 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. The rigid cartilaginous rings of the trachea, by contrast, will not collapse and the bulb will rapidly re-expand.

Both these devices are needed to cover all situations where one needs objective confirmation of tube placement. Breath sounds and chest rise are notoriously inaccurate measures and even visualizing the tube passing through vocal cords can be erroneous (for example, if in fact it was not the cords which were visualized). Airway and Breathing are the first two steps in the ACLS algorithm and a missed esophageal intubation is quite frankly unacceptable.

The person holding the code pager varies on a daily basis. During the day an anesthesia member of the ICU team (i.e., someone on their ICU rotation) will usually have the pager. If there is someone on call in the ICU they will hold it for the rest of the day and night. If there is no ICU person on call that night, when the ICU team is done for the day the person with the pager will distribute it to either the MOR or OB call person in preop clinic, rarely to an attending if both of these are unavailable. Later at night the MOR person generally has the pager since the OB person is often occupied, but if in a case passes the pager to either the attending, the OB resident or the night float resident.

You don’t have to worry about finding the pager- it will find you if appropriate (meaning, someone will pass it on to you if appropriate). On weekday nights there are often a wealth of other people to carry the pager, including-

- the night float resident
- potentially a Navy resident; they rotate through UCSD periodically for trauma
- the attending

Until about 6 months into your CA1 year, most attendings expect you to call them if you’re going to a code or other emergency. Afterwards, it’s on a case-by-case basis depending on the attending and your own comfort level. There’s never any shame in letting the attending know what’s going on or having an extra set of hands present. All senior residents can probably recall multiple times when they were glad an attending was with them for what seemed at the outset like a routine intubation.

There are four basic scenarios for which the code pager will be called-

1. a true cardiac arrest (code blue)
2. an airway or respiratory emergency
3. an inbound trauma that might require airway management
4. other- potentially help with a central or arterial line, IV, or some random issue that the questioner thinks “anesthesia will know”. These are usually less urgent

Regarding emergency calls- in an ideal world when paged we would know exactly what is going on, what is needed of us, and where. Alas, such is not the case. Sometimes the page will be a clear text message- i.e., “code blue 10E room 1021”. Other times it will simply say, “anesthesia stat to 10E” or even worse, just a number. Then when you call the number back you reach a frantic nurse who is unable to describe to you the clinical situation or scenario, but just keeps saying, “come up here”. Basically 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 whom are running around like chickens with their heads cut off. Is it a true code? Is the patient ventilating? Is the patient responsive? Are any monitors on the patient? Etc, etc… A quick gestalt goes a long way.
2. If you need to get to the patient, be assertive. Again, there’s likely to be a whole bunch of people of varying utility around the patient. Calmly but forcefully let people know anesthesia is present and your intentions, e.g., “I need to get to the head of the bed, please move”.
3. If a code situation, establish early who is running it. 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 as much as you can before you do something. For example, if you go to an urgent intubation and have some time, get everything you’ll need out and organized beforehand so you’re not floundering for it later. This might mean an LMA, the EZ cap, drugs, suction, etc all within easy reach or with someone prepared to hand them to you.
6. ABC. So much of what we do comes back to that. If you constantly think “ABCs” during most emergencies you should do ok. And since A is first, if the patient needs an airway all other people’s activities become secondary and yours become of the utmost importance.
7. If someone is truly in cardiac arrest, you don’t need to give any induction drugs before laryngoscopy and intubation.
8. 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. Ask the primary team or whoever is running the code. In our experience, most of the time you think the patient will need something you’ll be right, even if the primary team feels differently. For example, a hypothetical patient codes, is revived and is started on dopamine. He has one 22G peripheral
IV and no arterial line. You politely suggest central IV access and an arterial line... only to be turned down. In these situations, try and make your case forcefully but politely. Usually if the patient needs something, you know it, the primary team declines, and you leave, you’ll just be called back 5 minutes later to assist with an “urgent arterial line”. So try and nip it in the bud early.

9. Ask whoever is running the code if you’re needed anymore before you leave.

**Room 7 and 11 setups (emergency rooms)**

This information applies only to Hillcrest. At the VA and Thornton we do not have preset rooms for emergencies (low volume of trauma/emergency cases). As with the code bags, the responsibility of ensuring these rooms are set up is somewhat nebulous. In general, whoever is on MOR call should ensure these rooms are set up b/c when a case comes in for one of these rooms, it’s typically the MOR call person doing the case. That being said, whoever is holding the code pager (often the SICU or night float person) will have the most opportunity to set these rooms up. Communicate with each other to make sure that the rooms get set up.

**Room 7 (emergency craniotomy room)**

One room at night is traditionally reserved for emergency craniotomies. Often, this is room 7, but sometimes cases from the day run late in room 7, necessitating another room to be designated as the “crani room”. Check with the front desk staff when in doubt. Whichever room is the crani room needs to be set up so as to allow an emergency case to proceed as quickly as possible. Standard room set up applies, such as having drugs ready, suction, doing a machine check, etc. Specific considerations include-

- the bed should be 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 foam pad/pillow for head position should be “down there”, i.e. on the side away from the anesthesia machine.
- Because the head is already away from you, the airway equipment must be placed on a mayo stand that can be moved to the head. It does no good to have the equipment on the anesthesia machine, only to have to run back and forth between the machine and patient’s airway. Put EVERYTHING you might need on the stand for intubation/ventilation- masks, oral/nasal airway, a variety of blades and tube sizes, tape, etc.
- Again, because the head is away from you, you must put a long circuit extension from the 15mm adapter to the machine so it can physically reach the patient’s head. It is most embarrassing to intubate a pt and then not be able to connect your circuit b/c it’s too short.
- Have mannitol, lasix and several 100cc bottles of propofol in the room.
- Have equipment to rapidly start an arterial line and central line in the room. Also, a transducer for said A/C lines should be hooked up to the monitor and zeroed for immediate use.
- Have at least one bag of crystalloid prespiked and run through tubing, including a fluid warmer for immediate hookup to whatever IV access you have/get.
- Basic drugs for the craniotomy room are currently being supplied by pharmacy every 24 hrs. Again, they must be manually replaced by us like with the code bags after they are used. Furthermore, the list of drugs supplied is not comprehensive.

Room 11 (trauma room)-

This room should be set up at all times for an emergency surgical resuscitation. This room is where you will do the vast majority of your gunshot and MVA resuscitations, among others. The only time it will not be set up is when it is a) in use, or b) immediately after being used and being cleaned. Standard room set up again applies, including a machine check, airway, drugs, and so forth. Specifics for this room include-

- Two pre-spiked IV bags, run through lines attached to fluid warmers.
- Emergency resuscitation drugs should be drawn up, with more available. These include epinephrine, atropine, vasopressin, calcium and NaHCO3. ALL drugs, including these, must be drawn up fresh every 24 hrs, just like the code bags. The pharmacy will (should) supply most of these drugs predrawn but the list is not all inclusive.
- Equipment for starting an arterial line and central line, as well as the transducers for both, prezeroed.
The preoperative evaluation and presenting cases to attendings

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

The difference between a good preop and a poor one is like night and day. A well done preop 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, and at worst could compromise the safety of the patient. All of us have experienced the phenomenon of the poorly-done preop and in fact most anesthesiologists can guess the level of a person’s experience by the quality of their preops.

The pre-evaluation clinics have undergone major positive changes in infrastructure and caseload since the last edition of this text. These changes are almost solely due to Dr. Anush Minokadeh who is the Director of Pre-evaluation Services. I have made every attempt to keep this text up to date with our practices. In the event of any discrepancy, please defer to Dr. Minokadeh’s wisdom and decisions.

When will I be doing preops?

The following are the typical scenarios in which residents will do a preop-

1) preop clinic at Hillcrest, Thornton or the VA-

   This is by far the most common. Typically you will be assigned to these clinics during your on-call days at the various locations. Thus, they will be described in more detail in those sections. Briefly, at Hillcrest the MOR and OB call residents will start their day in the preop clinic. Scheduled patients come in throughout the day. Once the clinic is done, generally around 4-5pm, they assume the rest of their call duties. Rarely, one might be assigned to preop clinic as a shift for the day, even when not on call.

   At Thornton there is a preop shift. When assigned to this shift the resident again starts the day in preop clinic. When clinic is over the resident reports to the OR for assignment to a room or is sent home.

   At the VA the call resident has multiple daytime responsibilities, including seeing patients in the VA preop clinic (referred to as ASU). There is a full time CRNA assigned to ASU who shares the preop burden.
2) Inpatient preops-

Sometimes at Hillcrest you will be assigned patients for the next day who are in house already. These patients do not go to our preop clinic and thus must be seen directly. It is typically the responsibility of the resident who will be doing the case to preop his own patient. Often times the on-call residents will have a spare moment to do these preops for you. This is something that 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 preops done for each other. There is an unwritten rule that CA-1s should be doing their own inpatient preops for the first six months to help them gain more experience.

Rarely, a CRNA will be assigned cases for the next day with inpatients. When this happens a random resident might be chosen to preop those people. Life goes on.

The call resident at the VA does all the inpatient preops for that location. The list of patients will be provided when you arrive on call. Thus, a major difference between Hillcrest and the VA is that at the VA you might be assigned cases for the next day that are inpatients, but the call resident will do them for you. 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, and it is not common.

3) Preops “on the fly”

All that happens in the OR is not scheduled or predictable. Many times in your career here at UCSD you might find yourself doing an emergency case. Clearly in these instances the patient is not likely to have a preop already done. Furthermore, time is often of the essence in these situations, precluding a full workup. 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. In these situations be as thorough as the situation allows.

On a less emergent note, some elective cases feature patients who were not scheduled or did not show up to our preop clinic. During these times you might find yourself finding out about the patient a few minutes before you take them back to the OR. Clearly in these situations no one will expect you to have been able to do a thorough preop beforehand (which does not necessarily mean you can’t do one right then).

**What does a basic preop entail?**

Philosophically, the preop serves several functions. First, it provides a brief description of the patient and their 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 preops and anesthesia grows it becomes significantly easier to identify information which is relevant to anesthesia and information which is superfluous. The goal is to provide a **brief, concise and relevant**
summary of the patient and their medical problems. A preop 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 preop, pinpoint areas to focus on, and give the rationale behind why an anesthesiologist would want this information. Refer to the blank preop templates (one for Hillcrest and Thornton, another similar one for the VA). The templates are intended to be easy to read and follow, and thus have sections which can be checked off. This also serves as a “primer” so that certain key questions are unlikely to be missed.

**Who can I turn to for questions about preops, or whether a patient needs further evaluation?**

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

Of note, **all preops done in any of the preevaluation clinics need to be signed off on by an attending.** This is an assigned duty for the attendings and will 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. Since they follow the patients pre- and post- operatively, they are in the best position to both order a test and initiate any necessary followup. 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 a 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, as the requesting physician it becomes your responsibility 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. Ergo, 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 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 would 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 atlanto-occipital instability”. This fulfills our role as perioperative consultants and frees us from having to do much of the leg work ourselves.
The preop evaluation by section-

1. Age, weight, vitals, 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 choice of induction, the size of an endotracheal tube and sometimes the whole anesthetic.

   Elective patients should be NPO for solid foods for at least 8 hours prior to undergoing anesthesia. *This includes non-general anesthetics, such as neuraxial techniques and MAC.* There is always the potential to convert to a general anesthetic even if one was not planned from the outset (e.g., failed spinal). Small amounts of clear liquids may be taken 2 hours prior. Opaque liquids such as cream or milk need a 6 hour window. The recommendations for children are different- 2 hrs for clear liquids, 4 hrs for breast milk and 6 hrs 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 preop) is different from the one the surgeon actually intends to do. Accuracy is important.

2. Cardiovascular system

   There are several points worth noting here in addition to the boxes provided. Certain items, if positive, deserve more clarification. For example, if the patient has a “hx of MI” it is useful when it occurred, and what was done if this information is available. Similarly, if the patient has CHF, is it compensated? Does the patient have active orthopnea? In general, use your best judgment about when to provide more information.

   The exercise tolerance section is perhaps the most useful of the entire preop. Essentially, by quantifying the amount of physical activity the patient can tolerate, we can begin to risk stratify them and determine if further cardiac workup is necessary. The “AHA/ACC 2007 guidelines for perioperative cardiac evaluation for non-cardiac surgery” is what we use on a daily basis. Refer to those guidelines for a more detailed explanation. Newer guidelines are in the process of being evaluated and adopted. A basic explanation is as follows-

   - if the surgery is emergent, no further workup is required go to the OR
   - if the patient has been revascularized within 5 years and does not have recurrent signs/symptoms of cardiac disease go to the OR
If the patient has had recent (within 2 years) coronary evaluation and the result is favorable/there has been no change in symptoms  go to the OR.

If none of the above is true, then further steps are necessary. Examine the patient for the so-called “clinical predictors”. There are 3 categories- major, intermediate and minor.

Major predictors include-
- decompensated CHF
- unstable coronary syndromes
- major valvular disease
- significant arrhythmias

Intermediate predictors include-
- prior MI (older than 30 days)
- compensated CHF
- diabetes
- renal failure
- mild angina

Minor predictors include-
- advanced age
- prior stroke
- rhythm other than sinus
- abnormal ECG
- uncontrolled hypertension
- poor functional capacity

If a patient has major clinical predictors, the surgery should be delayed until further workup/management can be obtained.

If a patient has intermediate, minor or no clinical predictors, it becomes necessary to quantify their functional capacity. Functional capacity is categorized as either poor (inability to do 4 METS of exercise) or moderate to excellent (can do 4 METS or greater). A MET is a metabolic equivalent and provides a way to grade physical exertion when performing an activity. Examples of 1 MET of exertion include very light activities- slow walking, dressing oneself, and eating. 4 MET activities include walking up a flight of stairs, walking four blocks at a time, moving heavy furniture or significant housework, and moderate physical activities such as golf, bowling, or throwing a ball. In practicality many of us simply ask a patient if they can climb a flight of stairs, although it is useful to know other ways to assess the number of METs a patient can perform.

Additionally, it becomes necessary to categorize the risk of the planned surgery. The general breakdown of categories is as follows-

High risk (cardiac risk often > 5%)-
- major vascular procedures
- emergent major surgery
- prolonged surgeries with significant fluid shifts or blood loss

Intermediate risk (risk < 5%)-
- carotid endarterectomy
- head and neck surgery
- orthopedic procedures
- prostatic surgery
- intraabdominal or intrathoracic procedures

Low risk (risk < 1%)-
- endoscopic or superficial procedures
- cataract or breast surgery

Clearly, there is some overlap between these categories. With time and experience, you will learn which procedures entail “major fluid shifts” and which ones will not, and which surgeries pose an intermediate rather than a high risk, and so forth.

To finish, if a patient has minor or no clinical predictors, and can perform 4 METS of work to OR. If this same patient has poor functional capacity, and the surgery is intermediate or low risk, no further workup is required. Only if the surgery is high risk, and the patient has no functional capacity to speak of is further testing warranted.

If a patient has intermediate predictors and the surgery is low risk, go to the OR. If he has poor functional capacity or the surgery is high risk, further testing is warranted. Intermediate risk procedures with a good functional capacity can go to the OR, while a poor functional capacity needs further workup.

Patients with prior coronary stents placed demand special attention. Current guidelines are designed to minimize the risk of stent thrombosis which can be devastating. Our most current guidelines are listed below-

Perioperative Management of Patients with Coronary Stents

Preoperative Evaluation
1. Determine the type (BMS, DES) and location in the coronary circulation of stents placed in the patient and the date of implantation.
2. When possible, obtain catheterization/stent procedure report to identify any high risk factors:
   a. Low EF
   b. Prior coronary brachytherapy
   c. Long Stents
   d. Proximal vessel involvement (i.e. L main)
   e. Multiple Lesions
   f. Overlapping Stents
g. Ostial/bifurcation lesions  
h. Small vessels  
i. Suboptimal stent results

3. Consult with a patient’s cardiologist and, when high risk, with the interventional cardiologist. When no cardiologist is on record, consult:
   a. Hillcrest Cardiology Consult  
   b. Thornton Cardiology On-call

4. Perform the surgery in a center with 24-h interventional cardiology coverage so that stent thrombosis, if occurs, could be treated by PCI.

5. Inform patients of risk of perioperative stent thrombosis on preoperative visit.

6. **Arrive at a joint decision between the anesthesiologists, cardiologist(s), and surgeons about the timing of surgery and the most appropriate management of the patient’s antiplatelet regimen.**
   a. **Make sure you discuss each one of these cases with an Anesthesia attending on the day of the visit.**  
   b. **Make sure you document the ‘joint decision’ on the chart. Depending on the situation, this discussion may need to involve the Anesthesiology attending.**

### Coronary Stents

1. Patients with drug-eluting stents (DES) should defer all elective surgery by at least 12 months.
2. Patients with bare metal stents (BMS) should delay all elective surgery by at least 6 weeks.
3. Patients with BMS and DES should be on dual antiplatelet therapy (thienopyridine and ASA) for 6 weeks and 12 months, respectively.

### Perioperative Coronary Stent Management options (after the 6 week and 12 month period)

1. Hold clopidogrel (Plavix), if taken, for 5 days prior to surgery
2. Continue aspirin during and after the surgery if possible.
3. Consult with cardiologist when plan is inconsistent with #s 1 and 2.
4. Inform patients of risk of perioperative stent thrombosis on preoperative visit.
5. Ensure blood product availability for the case
6. Consider platelet transfusion in the setting of bleeding if anti-platelet therapy continued (3 hours after last Aspirin dose and 8 hours after last Plavix dose)
7. If taken, load (600mg) and restart clopidogrel as soon as possible after surgery.

### Perioperative Coronary Stent Management options (within the 6 week or 12 month period):

1. Continue dual antiplatelet therapy during and after surgery if possible.
2. Consult patient’s cardiologist.
3. Discuss with interventional cardiologist as they may have insight to the patient’s coronary anatomy and optimal/suboptimal circumstance under which stent was placed.
4. If any antiplatelet therapy is held or discontinued, consult with patient’s cardiologist as to the decision and the risk-benefit analysis for the particular patient and procedure.

5. If decision is made to stop clopidogrel (5 days), continue ASA 325 mg through the perioperative period.

6. High risk patients:
   a. Advanced age
   b. Acute coronary syndrome
   c. Diabetes
   d. Low EF
   e. Prior coronary brachytherapy
   f. Renal Failure
   g. Long Stents
   h. Proximal vessel involvement (i.e. L main)
   i. Multiple Lesions
   j. Overlapping Stents
   k. Ostial/bifurcation lesions
   l. Small vessels
   m. Suboptimal stent results
   n. Known stent stenosis or thrombus

7. If clopidogrel is held, load (600 mg) and restart clopidogrel as soon as possible postoperatively.

8. If absolute or relative contraindications to continuing antiplatelet therapy exist:
   a. Contraindications to continuation of perioperative single or dual antiplatelet therapy (refer to options, next section):
      i. Surgery for bleeding
      ii. Bleeding cancer (i.e. colon)
      iii. Spine surgery
      iv. Intracranial surgery or intracranial mass
      v. Hepatic resection
      vi. Eye or Ear surgery
      vii. Other surgery with significant expectant blood loss
   b. Inform patients of risk of perioperative stent thrombosis with holding antiplatelet therapy and bleeding risk with continuing antiplatelet medication
   c. Consult with Cardiology and Surgery as to best perioperative plan based on risk factors, bleeding risk, and urgency of surgery

9. Ensure blood product availability for the case

10. Consider platelet transfusion in the setting of bleeding if anti-platelet therapy continued (3 hours after last Aspirin dose and 8 hours after last Plavix dose)

Patients who have NOT been evaluated preoperatively and present WITHOUT antiplatelet therapy on day of surgery.

1. If within 6 weeks and 1 year of BMS or DES:
   a. Consult cardiology
b. If elective surgery, postpone case until after 6 weeks or 1 year

c. If urgent case, would consult cardiology (interventional cardiology) prior to proceeding as these patients will be high risk.

2. If after 6 weeks and 1 year of BMS or DES:
   a. Administer aspirin 325 mg orally and wait additional 2-4 hours until proceeding with the case.
   b. Continue aspirin postoperatively and if taken, load Plavix (600mg) as soon as surgically safe.

Be sure to contact an attending with any questions regarding coronary stents.

Pacemakers and Implantable Cardiac Defibrillators (ICD) represent another unique challenge to the anesthesiologist. Current perioperative 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. These guidelines are available from the ACC/AHA website. For the resident in preop clinic, the following information must be obtained-

- model and manufacturer of the device (ie, Guidant, Medtronic)
- current settings and default settings (ie, what happens when a magnet is placed on the device)
- phone number for the device representative
- schedule the device representative to come the day of surgery, both preoperatively (for possible reprogramming) and postoperatively (to interrogate for proper postop function).

Clearly, some patients may have little to none of the above information. Obtain what you can and write it down on the preop form. Calling the device representative/scheduling a day-of-surgery visit is the most important- all the major manufacturers have national numbers and 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 preoperative visit.

3. **Respiratory system**

   Asthma and COPD are conditions that could potentially need further description. The severity of the disease is often useful to note. Does the patient use inhalers daily? Have they ever been hospitalized or intubated for their condition? Preoperative pulmonary function testing may be very useful.
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 can have profound implications in the perioperative period. The recently published “Practice guidelines for the perioperative management of patients with obstructive sleep apnea: a report by the American Society of Anesthesiologists Task Force on Perioperative Management of Patients with Obstructive Sleep Apnea” is an excellent reference. At the minimum, the presence or suspicion of OSA should be noted on the preop form. Changes in anesthetic technique, post operative monitoring or discharge requirements and even whether to proceed with surgery can occur in the face of OSA. Refer to the guidelines for specific recommendations.

4. Neurologic system

Preexisting neurologic dysfunction or deficits should be noted. As perioperative neurologic dysfunction makes up a substantial amount of claims brought against anesthesiologists, careful preop documentation is important.

5. Hepatic and renal systems

Preexisting renal and hepatic disease can profoundly influence anesthetics and should be noted (e.g., decreased metabolism and excretion of drugs). If the patient is on dialysis, note the normal schedule of dialysis and when this patient’s last dialysis prior to surgery 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 or not.

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

7. Cancer, infectious diseases, history of Post-Operative Nausea and Vomiting (PONV), smoking, EtOH and other drug abuse
Indicate as appropriate. Pay special attention to cancers of the head and neck as they may have profound implications for airway management.

If a patient uses recreational drugs, note if they are an active user. Patients who have abused cocaine, crack or methamphetamines and have only recently stopped may still have altered physiology from substance abuse.

8. Family History of problems with anesthesia

This section is essentially asking for a history of Malignant Hyperthermia. Often patients will know this themselves, and can also be asked about a history of life threatening reactions to anesthesia. A positive answer warrants further investigation and planning (see the malignant hyperthermia section).

9. Previous surgeries, medications, allergies

Indicate as appropriate. Special attention should be paid to the dose of some medications (e.g., narcotics, antihypertensives) and to medications which could alter the anesthetic (e.g., narcotics, anti seizure medications). 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.

11. Airway exam

In addition to Mallimpati class, HMD, neck ROM, dentition and ability to prognath the jaw, there is room to provide further description if necessary (e.g., does the patient have an overbite? Thick neck?). If the patient is already intubated or has had a recent intubation, that information can also be noted. The importance of a good airway exam cannot be overemphasized.

12. Labs, other studies

Note as applicable. Some patients will have had substantial workup prior to their preop visit. Men over 40 and women over 50 need a baseline EKG.

13. Assessment and Plan
Here is where you indicate a short summary of the patient, and a brief description of the anesthetic plan. In general, it is good to be broad with the anesthetic plan and it is not necessary to define specifics (e.g., will use propofol for induction). For one, plans always can change. Secondly, another anesthesia provider may end up doing the case, and for medico-legal reasons you don’t want to create potential problems by being too rigid on the preop evaluation form.

Be sure and note ASA class. The ASA class attempts to assign a numeric “value” to the overall health status of the patient and has been show to correlate with perioperative outcomes. Additionally, billing and reimbursement is often adjusted for ASA status (in recognition of increased complexity of the patient). The ASA classes are-

ASA I- an otherwise healthy patient  
ASA II- a patient with systemic disease but no symptoms or no functional impairment (e.g., HTN)  
ASA III- a patient with systemic disease causing symptoms or functional impairment (e.g., angina)  
ASA IV- a patient whose disease is a constant threat to life. An intubated, mechanically ventilated patient with no other medical problems is an ASA IV, since his condition is a threat to his life (and would probably cause death if not supported by the ventilator)  
ASA V- a moribund patient not expected to survive without the operation  
ASA VI- a brain dead organ donor  
E- a modifier to note emergent nature of surgery. An ASA II patient about to undergo an emergency operation would be an ASA IIE

14. Other areas

There are areas to sign the form, record lab values intraoperatively, and for attending signatures.

The VA preop

The preop form at the VA is essentially the same as the one used at Hillcrest and Thornton. One notable difference is the question about TB under the respiratory section. The VA administration insists that this question be specifically asked and checked off, even if the rest of the pulmonary system is normal. In other words, you cannot simply check “within normal limits” for the entire system, but must also check off “neg” specifically for TB. Redundant, but…

The preop must also be reviewed and signed off by an attending without exception. This is a relatively new change at the VA. For practical purposes, this means all preops must be signed before they are scanned (see below), including preops done on inpatients after the attendings have left the building.
Lastly, the preops 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. When at the preop clinic the nurses there will typically scan them in for you. We are responsible for scanning in any inpatient preops that are done. 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.

**Presenting to attendings**

One ritual which you will grow intimately familiar with as a resident is calling attendings to discuss the next day’s cases. This is done around the country at virtually every anesthesia program, 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. If you plan on taking the preop information home with you to call the attending with later, make sure and take a *photocopy*, not the originals. Cases and room assignments get moved around a lot and you don’t want to lose the preop, especially for someone else.

The number of attendings’ styles is almost as varied as the attendings themselves. As you might expect, some are very laid back and your presentations might last 30 seconds, at which point you’ll hear “ok, see you tomorrow”. Others are significantly more demanding and will expect you to have read a little before the presentation, or expect a more detailed description of your anesthetic plan. Experience will help you determine who falls into which camp. 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. Treat it 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 I: “A 45 year old man getting a left inguinal hernia repair. He is otherwise healthy. My plan is general anesthesia versus neuraxial versus MAC/local and standard monitors.”

Case II: “A 50 year old woman getting a laparoscopic cholecystectomy. She has a history of daily asthma for which she takes albuterol and steroid inhalers. However, she has never been hospitalized or intubated for her asthma. Otherwise she has no medical problems. Recent PFTs show a mild obstructive defect consistent with her asthma. My plan is general anesthesia, standard monitors and to administer her inhalers preoperatively.”

Case III: “A 66 year old man going for a right carotid endarterectomy. He has a history of HTN, DMII and peripheral vascular disease. He does not exercise but a recent AMIBI showed no evidence of CAD and good ventricular function. He also has had a CVA which has left him with residual left sided weakness. Recent carotid duplex shows over 90% stenosis of his right carotid, while the left has 50-69% stenosis. In light of these problems, my anesthetic plan is general anesthesia, standard monitors, an arterial line, and an EEG. I will be using a balanced anesthetic with remifentanil, nitrous oxide and desflurane, with the goal being a quick wakeup so the surgeons can perform a neuro exam. I will also be using a phenylephrine infusion to maintain the patient’s blood pressure at baseline levels during the anesthetic to maintain cerebral perfusion. Hopefully the EEG will allow us to monitor for possible ischemic episodes. Lastly, I will administer a longer acting narcotic at the end of the case to bridge the patient from the remifentanil.”
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 (2000 psi);
  this cylinder pressure is reduced by valves to line pressure (~ 55psig)
- A standard E-cylinder of oxygen contains 650L of gas when full at a
  pressure of 2200 psig, and is green
- Cylinder pressure falls in direct proportion to content; thus, a half full E-
  cylinder contains 325L of oxygen, at a pressure of 1100 psig
- 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 ventilation. All post-anesthesia patients
  are transported with oxygen

B. Nitrous Oxide

- can be liquefied at room temperature by storing under pressure
- An full E-cylinder contains 1590L of gas (liquefied) at a pressure of 745
  psig 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 (3/4 empty) will the cylinder pressure fall
- Thus, the only way to determine the volume of nitrous oxide in a cylinder
  is to weigh it
- Our anesthesia machines typically have a backup E-cylinder attached

C. Air

- stored as a gas in E-cylinders
- a standard E-cylinder of air shares the same characteristics as oxygen with
  respect to capacity and pressure; the cylinders are yellow

The Pin Index Safety System- designed to prevent incorrect cylinder attachments
to the anesthesia machine. Each type of cylinder has holes which 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; further, longer circuits increase the difference between volume delivered to the circuit during controlled ventilation and volume actually delivered to the patient. 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 (one popular mnemonic is 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 (semiclosed)-

- The anesthesia machine represents 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 maplesons, 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 (CO₂ absorbent), forming water, calcium carbonate, and heat.
- Soda lime is the most common absorbent; barium hydroxide lime is also seen.
- 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 is the most common and 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.
- 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 for more info. High flows running through an unused anesthesia machine increase the likelihood of dry lime, the so called “Monday morning effect”. The term comes from a hypothetical anesthesia machine that someone has inadvertently left with high flows going on a Friday afternoon, and then the OR is unused the whole weekend. By Monday, the soda lime can be highly dessicated.
- The patient’s tidal volume should not exceed the volume between the granules, as this could result in rebreathing of carbon dioxide.
- Unidirectional valves- inspiratory and expiratory, these should open only during the corresponding phase of the ventilatory cycle. Warped or cracked valves, or misseating of the valves can lead to incompetence and rebreathing of CO2. 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. 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 with 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 X.
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\textsubscript{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) and 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 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 concentrations

E. Nasal cannula and face masks

- is a low flow system; each additional L of O\textsubscript{2} increases FiO\textsubscript{2} by 4-5%, to a max of 6L/min or 50% FiO\textsubscript{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 FiO\textsubscript{2} which the lungs “see” is much lower than the maximum deliverable via nasal cannula
- face masks- deliver approximately 50% FiO\textsubscript{2} at 6-8L/min
- neither system 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 two basic machines currently in use at UCSD- the Datex-Ohmeda Aestiva 3000 at Hillcrest and the VA, and the Draeger Narkomed 2C at Thornton. Subtle differences between the two will be described. There are older Datex machines in use on the OB suite but for our purposes they function similarly to the new Datex’s in the main OR.

A. Gas supply

- both machines receive the supply of O₂, N₂O and air from two sources- the central pipeline of the hospital and 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 “backups” to the pipeline, the primary source of gases.
- The E- cyliners attach to the machine via the Pin Index Safety System described above. Similarly, the pipeline connections utilize the Diameter Index Safety System, whereby each pipeline connection has a specific and unique diameter of locking pins to prevent incorrect attachment. The pipelines are also color coded.
- 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 drawn preferentially 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. You can try this on an anesthesia machine- if the flow of both O₂ and N₂O are turned to 5L/min, then the flow of O₂ is reduced, 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. The knob for the oxygen valve is always furthest to the right, is larger and protrudes more than the other knobs, and has ridges (which can be felt even when not looking at the knob).
- The flow meters are two 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.
- Board question- the rate of flow depends on the gas’ viscosity at low, laminar flow and its density at high, turbulent flow
- The oxygen flowmeter is always positioned furthest downstream, closest to the vaporizers to minimize the chance of hypoxic mixture delivery in the event of a leak
- The oxygen flow valve delivers a mandatory flow of 150ml/min the instant 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 exclusion device that prevents more than one vaporizer to be “turned on” at once
- 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 (heat of vaporization, vaporization requires heat which is supplied by cooling of the liquid anesthetic). This cooling must be compensated for by direct warming by the vaporizer. Touch a desflurane vaporizer and you will feel that it 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 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
- 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 vent is on 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 on all machines, should turn on whenever 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-connection the sensor is, the more closely it reflects airway pressures
- changes in airway pressure may reflect obstructions, disconnections or changes in compliance and should be investigated

G. Popoff valve
- should be fully opened during spontaneous ventilation; however, closing it slightly can be used to add PEEP to the circuit
- designed to have an upper limit (~70cm H2O) so that the valve can never be truly “closed”; this limits the risk of inadvertent barotrauma

H. humidity
- delivered gas are room temperature and low in humidity; this 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 even 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 (gas temp should be monitored), infection (water source should be sterile), increased chance of circuit disconnection, and increased dead space

I. the ventilator

- both the Dragers and Datexs can deliver preset tidal volume breaths (volume control). The machine aims to deliver a set volume with each breath. High pressure limits on either machine will automatically “cut off” the breath if excessively high peak pressures are encountered. The Datex also has the ability to deliver pressure controlled breaths. Here, a set pressure will be delivered for a certain length of time (dictated by the number of breaths/min and the I:E ratio), resulting in a certain, variable tidal volume to be delivered.

- I:E ratio- determines the amount of time the vent will spend in each phase of ventilation. Increasing the I:E ratio means there will be less time spent in inspiration and more in exhalation. Increasing the I:E ratio during volume control will typically raise the peak inspiratory pressures (less time available to deliver the same volume). Increasing the ratio during pressure control will result in a smaller volume delivered. One common scenario where increasing the I:E time is useful is in obstructive lung disorders such as COPD or asthma. Here, allowing more time for each breath to be exhaled can be beneficial.

- both machines have a switch to change from bag to mechanical ventilation; on the Drager a second knob at the top of the machine must also be turned to switch the ventilator “on”

- switching from bag to vent excludes the popoff valve and reservoir bag from the circuit

- the ventilator bellows are pneumatically driven (typically by oxygen, sometimes by air). During an inspiratory cycle the driving gas will fill the plastic chamber containing the bellows assembly, in turn 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 dependant 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-

\[(\text{FGF}) \times (\% \text{ of time spent in inspiration}) \div (\text{number of breaths/min})\]
- 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) x (0.25)/10 = 0.125L, or 125 ml extra per breath
- High fresh gas flows increase the magnitude of this phenomenon (typical during emergence, when we often turn the oxygen flow very high to washout the anesthetic agent when the vent is still on)
- This phenomenon can be seen with the Drager machines; the Datex machines have a mechanism to eliminate this from happening
- 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 “vent” is on)
- the positive pressure relief valve prevents excessive buildup of pressure if the scavenging line is occluded
- the negative pressure relief valve prevents excess negative pressure from the vacuum system to be transmitted to the patient

A quick 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- verify both that the alarm goes away, and that the cylinder pressure is engaged. Close the E-cylinder and reattach the pipeline
3. check the low pressure system- occlude the end of the Y piece, close the popoff valve and ensure gas flows are off. Flush the high flow oxygen system build a pressure of at least 40cm H2O. The circuit, if leak free, should hold this pressure. Opening the popoff now while keeping the Y-piece occluded should release the pressure, verifying that the popoff valve opens and closes appropriately
4. check the valves- different ways to accomplish this. One way is to place another bag at the end of the Y-piece. Give a positive pressure “breath” and watch that
only the inspiratory valve moves. Similarly, when the breath is released (simulating exhalation), only the expiratory valve should move.

5. Check the ventilator- switch to ventilator mode and turn flows on with the second bag still in place. The bag should inflate as the bellows drop, and then bellows should reinflate as the bag collapses.

**Tips for the anesthesia machine**

1. Extra circuit tubing and breathing bags are in a drawer in the anesthesia machine.
2. Circuit disconnections are very common. Typically your first clues will be the ventilator alarming and the bellows not refilling, accompanied by a loss of end tidal CO₂ 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 (sampling line off the Y piece), and at the airway/circuit connection itself.
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 incompetent 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 (ET tube, 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 in the setting of placing an NG or OG tube = probable placement of the NG tube in the trachea and inadvertent suctioning out of ventilation
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. Turning the vent on with the Drager machine is a **two-step process**. Switch from bag to vent AND turn the knob on the machine to turn the ventilator on.
9. Ventilator/fresh gas flow coupling is more pronounced on the Dragers as opposed to the Datexs because the Datex machine takes increased fresh gas flow into account automatically.
10. 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. Both are reusable- don’t throw them away but save them (put them on the reusables bucket on the side of each anesthesia cart)
11. The specific anesthetic locking keys for refilling the vaporizers are reusable. Save them- don’t throw them away.
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 point of fact our duty to the patient demands that we be constantly vigilant of our monitors and the information they provide. The vast majority of our anesthetics have profound impact on a patient’s physiology, and at the same time often render the patient incapable of directly telling us if something is “wrong”.

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

The ASA standards for basic monitoring stipulate certain expectations for patients undergoing anesthesia. As “standards”, they are intended to be nearly universal among anesthesia practice. By definition standards are what we are all expected to do or employ and deviation from them requires unusual, extenuating circumstances. In fact, 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-

I. A qualified anesthesia provider will be present for all anesthetics. The word continuous is used, 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.

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

A. oxygenation-
   - FiO2 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 CO2 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 min.
- 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.

The non-invasive blood pressure cuff

The NIBP cuff is the most commonly employed device to measure blood pressure in anesthesia. Most of us use the cuff as the sole measure of blood pressure for the majority of cases. BP cuffs can be placed at a variety of locations, including the upper and lower portions of both the arms and 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 pressure increments. The NIBP cuff measures blood pressure by oscillometry. Pulsations cause oscillations in cuff pressure which are detected by the machine. These oscillations increase markedly at systole, are maximal at the MAP, and decrease sharply after diastole. 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 systolic pressure (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 positioning 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 (controversial). Clearly, placing a cuff on an extremity that will be operated on is suboptimal.

Pulse oximetry

A pulse oximeter actually employs two scientific principle 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 an oxygen saturation. 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 differentiation 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. Conversely, when the signal is distorted or poor the tracing reflects this. 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 or forehead. Picking the right kind of probe is a function of the patient and type of surgery (e.g., pediatric patient).

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 SpO2 readings. Patients with carboxyhemoglobin poisoning will typically show a high SpO2 but low saturation on ABG. Methemoglobin has the same absorption coefficient for both red and infrared light. This clinically produces a saturation of 85%. Classic questions usually involve some trigger for methemoglobinemia (e.g., benzocaine) and an SpO2 of 85%.

Common sources of error in SpO2 readings are motion, a cold extremity, ambient light, hypoperfusion or a poorly placed sensor. Methylene blue dye also causes a transient, artifactual drop in SpO2. 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 about an artifactually low saturation due to a poorly positioned probe, and not being able to get to that probe. Surgical personnel leaning on or compressing the probe can also cause errors in measurement. Ideally the middle link in the probe cable should be easily accessible, to allow a new probe to be easily placed and plugged in to the existing cable.

**Capnography**

The capnographs we employ are diverting capnographs; that is, they actively aspirate small samples of gas from the breathing circuit, drawing the sample up into the machine for analysis. By analyzing the infrared light absorption of aspirated gas, the capnograph can determine not only CO2 concentration but also the concentration of
inhaled anesthetics and oxygen in the sample. This aspiration represents a small leak with is usually not clinically significant.

Confirmation of sustained end tidal CO2 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 EtCO2. 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.)

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 artifactually 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 CO\textsubscript{2} J. Inspiratory valve stuck open during mechanical ventilation. The "slurred" downslope during inspiration represents a small amount of inspired CO\textsubscript{2} 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 CO\textsubscript{2}. M, Cardiogenic oscillations, when seen, usually 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. The CO\textsubscript{2} concentration in gas entering the sampling line therefore alternates between high and low values. N, Electrical noise resulting from a malfunctioning component. The seemingly random nature of the signal perturbations (about three per second) implies a nonbiologic cause.

The capnograph has an audible alarm that will sound for a whole host of situations, such as apnea, abnormally low or high EtCO\textsubscript{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. We have nasal cannula that have a luer-lock channel which can be plugged into the sampling chamber of the capnograph. Likewise, the tubing from the capnograph can be placed in the face mask (one common method is to attach an IV cannula to the end of the tubing and place the cannula inside the mask). These measures allow better qualitative than quantitative measurement of CO\textsubscript{2} due to significant entrainment of room air. In fact, some patients mouth breathe so significantly that little to no CO\textsubscript{2} will be measured via a nasal cannula, although anyone could see the patient spontaneously ventilating.

**Temperature probes**

Most temperature probes we employ are disposable. Temperature can be measured in a variety of places, the most common being nasopharyngeal, esophageal, tympanic, bladder, rectum and blood. Axillary or 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. These probes double as esophageal stethoscopes- the ends of our stethoscopes can be attached to the esophageal probe for auscultation.

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 shivering are ablated by anesthetic inhibition of the hypothalamus. Regional anesthetics also contribute to heat loss by peripheral vasodilation and altered temperature sensation from blocked dermatomes. Thus, anesthetized patients cannot actively warm themselves and compensate for hypothermia. Problems associated with hypothermia include postop shivering and
increased oxygen consumption (up to 5x baseline), poor wound healing, coagulopathy, decreased drug metabolism and even arrhythmias.

Techniques used to maintain body temperature include- warming the OR, heating blankets, forced-air warming blankets (“bair huggers”), warming and humidifying inspired gases, warming IV 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.

Electrocardiography

Typically, a three lead or five lead EKG is employed in the OR. 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 left intercostals lead (brown) and adds the ability to monitor lead V5, which is probably more sensitive in detecting left ventricular ischemia.

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

The EKG leads are prone to artifact from motion or electrocautery. Furthermore, our monitors display heart rate by counting QRS complexes (what it perceives as the largest voltage change from the leads). Abnormally large T waves, sometimes due to incorrect lead placement can give an erroneous heart rate (“double counting”). This becomes obvious when an audible tone for each heart beat is employed. The EKG pads can be an potential area for burns if the electrocautery ground pad is dysfunctional.

Other monitors

Arterial line

Arterial lines provide beat-to-beat information about a patient’s blood pressure. Indications for placing an invasive arterial monitor are anticipated wide swings in blood pressure, need for precise beat to beat knowledge of pressure (e.g., heart disease, intracranial aneurysm), multiple blood samples, inability to obtain measurements from a 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 and/or pulmonary artery 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 (often 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 opened to air (turn closed to the patient). Hit the zero button and do not move the transducer. The monitor will beep and display visual confirmation when 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.

There are many different ways to place an arterial line. Techniques to place a radial A-line will be discussed here. To begin, ensure that the patient’s wrist is extended and secured. This can be facilitated with an armboard. Get all your equipment exactly where you need it before you begin. Infiltrate the area liberally with local anesthetic- this makes the procedure more tolerable for an awake patient, and will reduce the possibility of vasospasm.

To directly cannulate the artery, advance the needle at approximately a 30-45 degree angle, directly in line with the palpated path of the artery. Avoid the distal wrist if possible- although the radial pulsation is often prominent here, the artery itself 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 the tip of the catheter within the vessel lumen (the tip of the needle is slightly longer than the catheter, thus you can get a flash of blood without the catheter itself being in the vessel). 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- it should go easily and blood should come up inside the catheter. If it does not go easily- stop. Attempting to thread the catheter at this point will only result in a failed line. Either do a new stick or see below.

An alternative method is to use a guidewire to thread the catheter. If direct cannulation has failed, you can advance the whole needle another 0.5 cm, going through the back wall of the artery (transfixion or “through and through” technique). This can also be employed from the beginning of the procedure- 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 been “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, vasculopaths). 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.

Central lines

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

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 are when precise measurements of cardiac output or volume status are needed, or when mixed venous oxygen saturation will be measured.

Peripheral nerve stimulators

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

EEG

We periodically employ both “true” (16 lead) EEGs and processed EEG devices such as the BIS monitor or PSA. That being said routine use of any of these devices is not common. As a resident you will most often encounter the EEG during carotid endarterectomies where it is employed to monitor electrical activity and detect possible ischemic events in the brain. The BIS and PSA are sometimes used to measure depth of anesthesia, although the data they provide is of controversial value. One common place where the BIS or PSA 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.

Urine output
A bladder catheter is often placed by the circulating nurse or surgical personnel. Monitoring urine output is useful any time major fluid shifts are expected, end-organ perfusion measurement is desired or the surgery is expected to be lengthy.
A list of drugs common 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-

Mechanism of action- *indirect* adrenergic agonist, causes release of endogenous catecholamines. Mild increases in BP, HR, and contractility. Affects both alpha and beta 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).

Uses- hypotension, especially with slower HRs.

Notes- considered first line agent for hypotension in pregnancy as it theoretically spares uterine blood flow. Tachyphylaxis develops with repeated administration. Avoid with MAO-I inhibitors (risk of malignant hypertension due to too much endogenous catecholamine). Potentially ineffective in catecholamine depleted states (i.e. chronic methamphetamine or cocaine use).

B. Phenylephrine-

Mechanism of action- direct alpha-1 agonist, causing marked vasoconstriction, rise in BP and SVR. May cause reflex bradycardia.

Duration of action- minutes.

Usual dose- 50-100 mcg IV bolus, or run as an infusion 0.5-10 mcg/kg/min.

Uses- hypotension, especially from low SVR state.

Notes- **must be diluted** from its packaged concentration which is 10mg/ml. Most of us take 0.1cc from the vial and mix with a 10cc vial of saline, to make a 100mcg/cc concentration. An alternative is to mix all 10mg in a 100cc bag of saline, thus achieving the same. Also safe in pregnancy.

C. Epinephrine-
Mechanism of action- direct agonist at both alpha and beta receptors. 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 and up mcg/kg/min infusion, 0.5 -1mg IV bolus for cardiac arrest.

Uses- cardiac arrest, anaphylaxis, bronchospasm, cardiogenic shock, refractory hypotension, reduced CO

Notes- can cause tissue necrosis if extravasates from IV; cardiac arrest doses can cause profound hypertension. Can also be given via ETT if need be, 2-3x IV dose, diluted to 5cc.

D. Vasopressin-

Mechanism of action- activates V1 receptors, causing direct peripheral vasoconstriction. Acts independently of adrenergic receptors

Duration of action- minutes.

Usual dose- 40 units IV bolus for cardiac arrest, 2 unit IV boluses for hypotension, can also be given as infusion, typically 1-4 units/hr. Can be given via endotracheal tube.

Uses- alternative to epinephrine in cardiac arrest, hypotension (especially when adrenergic agents are failing)

Notes- very potent. Can cause splanchnic hypoperfusion and lactic acidosis (especially with infusions). Causes unpleasant sx in awake patients.

E. Dopamine-

Mechanism of action- has mixed effects depending on dose. At low (1-3mcg/kg/min) has primarily DA receptor effects, at 3-10mcg/kg/min Beta effects predominate, and at 10mcg/kg/min and above primarily alpha effects are seen.

Duration of action- minutes.

Usual dose- IV infusion, 1-20mcg/kg/min.

Uses- hypotension, primarily due to low CO state.

Notes- preferred 1st line agent for coming off cardio-pulmonary bypass by our CT surgeons; low (renal) doses may improve renal perfusion. Must be diluted.
II. Hypotensive agents

A. Nitroprusside

Mechanism of action: converted to nitric oxide, a potent vasodilator. Nitric oxide activates guanylyl cyclase, cGMP, decreases intracellular calcium and produces smooth muscle relaxation.

Properties: causes both vaso and venodilation, reducing BP by reduction in both preload and afterload. Quick onset and offset, allowing precise titration.

Usual dose: 0.5-10mcg/kg/min infusion, or small (10-20mcg) boluses.

Notable effects on organ systems: can cause coronary steal (vasodilation of coronaries stealing flow away from ischemic areas that are already maximally vasodilated). Reduces PVR. Increases cerebral blood flow which can be attenuated by hyperventilation.

Metabolism: this is a commonly tested question on the boards. Nitroprusside is reduced by 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 O2 (less O2 is used), arrhythmias and tachyphylaxis. Cyanide toxicity is unusual in durations less than 2 days and cumulative doses less than 0.5mg/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 nitrate converts hemoglobin to methemoglobin, which can then react with cyanide ion (see above).

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: same as nitroprusside.

Properties: primarily venodilation, reducing preload and BP. Quick onset and offset, allowing precise titration.
Usual dose: 0.5-10mcg/kg/min. Can also be given sublingually or transdermally.

Notable effects on organ systems: 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 to relax the uterus in OB procedures.

Metabolism: metabolized to nitrites, which can cause methemoglobinemia (see above).

C. Hydralazine

Mechanism of action: causes direct arteriolar vasodilation.

Usual dose- 5-20mg IV. Onset is within 15-20min and duration is 2-4 hrs, making it difficult to titrate

Uses: hypertension, especially on OB for pregnancy-induced hypertension.

III. paralytics and reversal agents

A. vecuronium-

Mechanism of action- competitive antagonism at Ach receptors in the neuromuscular junction.

Duration of action- 45-90 minutes for intubating dose.

Usual dose- 0.1mg/kg IV for intubation. 0.01mg/kg IV boluses for maintenance.

Notes- hemodynamically bland, cheap. Primarily excreted in bile, 25% by kidneys. May have prolonged block in renal failure pts. 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. Cisatracurium-

Mechanism of action- competitive antagonism at Ach receptors in the neuromuscular junction.

Duration of action- 30-60 minutes 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. No significant histamine release unlike its cousin atracurium. Laudanosine is a potentially toxic metabolite (causes CNS excitation, less laudanosine than with atracurium, probably clinically insignificant).

C. Pancuronium-

Mechanism of action- competitive antagonism at Ach receptors in the neuromuscular junction.

Duration of action- 60-120 min 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- prolonged action in renal failure. May inhibit pseudocholinesterase, resulting in prolonged block from succinylcholine or mivacurium.

D. Mivacurium-

Mechanism of action- competitive antagonism at Ach receptors in the neuromuscular junction.

Duration of action- 15-20 min for intubating dose.

Usual dose- 0.2mg/kg for intubation, 0.05mg/kg maintenance.

Notes- metabolized by pseudocholinesterase. Short duration of action. May cause histamine release. Many feel blockade from mivacurium is “weak” or less profound than with other nondepolarizing agents.

E. Rocuronium-

Mechanism of action- competitive antagonism at Ach receptors in the neuromuscular junction.

Duration of action- 20-60 minutes for lower doses, up to 2 hrs for 4x ED_{95} rapid sequence dose.

Usual dose- 0.6mg/kg for intubation, 1.2mg/kg for rapid sequence intubation.

Notes- quick (1min) onset when given in rapid sequence doses. Rapid (20min) offset when given at lower doses. Alternative to succinylcholine for rapid sequence intubation.
F. 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- 5-10 min. Onset is within 30 sec.

Usual dose- 1-1.5mg/kg IV. 0.1mg/kg IV boluses for maintenance, or 2-15mg/min infusion. Can be given IM.

Notes- only relevant depolarizing muscle relaxant in use today. Metabolized by psuedocholinesterase. Most rapid onset and offset of all muscle relaxants. Repeated doses may cause prolonged phase II block or arrythmias (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).

G. Neostigmine-

Mechanism of action- an acetylcholinesterase inhibitor, neostigmine increases the concentration of Ach available.

Duration of action- more than 1hr. Peak effect is within 10 min.

Usual dose- 0.05mg/kg, to a max of 5mg.

Use- to reverse non-depolarizing neuromuscular blockade. Treatment of myasthenia gravis.

Notes- lipid insoluble, cannot cross blood-brain barrier. Administer with an anticholinergic to block muscarinic side effects. Paradoxical potentiation of neuromuscular blockade when excessive doses are used. Side effects include those of muscarnic stimulation- bradycardia, bronchospasm, increased secretions, CNS excitation, GI spasm, increased bladder tone and miosis. Can result in a prolonged succinylcholine or mivacurium block (decreased activity of psuedocholinesterase).

H. Edrophonium

Mechanism of action- similar to neostigmine.

Duration of action- shorter than neostigmine, about 15min. Up to 1 hr with higher doses. Quick onset, within 1-2min

Dose- 0.5-1mg/kg
Use- reversal of neuromuscular blockade. Diagnosis of myasthenia gravis (tensilon test)

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.

I. Physostigmine

Mechanism of action- similar to neostigmine.

Dose- 0.01-0.03 mg/kg.

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.

Notes- only available cholinesterase inhibitor that crosses the blood-brain barrier.

J. atropine

Mechanism of action- anticholinergic. Binds to the acetylcholine receptor, rendering it inactive.

Duration of action- quick onset (within minutes), lasts up to 30min

Dose- 0.01-0.02 mg/kg. Can be given IM or via ETT.

Use- treatment of bradyarrhythmias, “slow” PEA, block muscarinic side effects of acetylcholinesterase inhibitors.

Notes- best anticholinergic for serious bradycardia. Crosses the blood-brain barrier but CNS effects usually minimal. Also causes bronchodilation. Avoid in narrow-angle glaucoma, bladder neck obstructions or prostatic hypertrophy.

K. Glycopyrrolate

Mechanism of action- same as atropine.

Duration of action- up to 2 hrs. Slower onset that atropine.

Dose- 0.005-0.01 mg/kg.
Use - decrease airway secretions, block muscarinic side effects of acetylcholinesterase inhibitors, treatment of mild bradycardia.

Notes - does not cross the blood-brain barrier and thus is ok in narrow angle glaucoma.

L. Scopolamine

Mechanism of action - same as atropine.

Dose - same as atropine. Usually given IM.

Use - premedication (sedative effect). Decreases airway secretions, good for motion sickness.

Notes - used to be widely used as a premedication. More sedating than atropine or glycopyrrolate (potent CNS effects). Pronounced ocular effects - avoid in narrow angle glaucoma.

IV. Inhalational anesthetics

General considerations -
- the more insoluble an agent is, the less it is taken up by the bloodstream, and the faster the induction time. This is expressed by the blood:gas partition coefficient. The higher the blood:gas partition coefficient, the more soluble the agent is.
- The Fa/Fi ratio is also 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 Fa is less than the Fi. More soluble agents are taken up more avidly, so the Fa/Fi ratio is less than for a relatively insoluble agent. Put another way, the Fa/Fi ratio is greater for more insoluble agents. The Fa determines the partial pressure of anesthetic in the alveoli, and ultimately the brain. Thus, more insoluble anesthetics will have higher Fa/Fi ratio and faster induction times.
- Low cardiac output states speed induction. Less anesthetic will be taken up by the bloodstream and the Fa/Fi ratio will rise rapidly. This effect is less pronounced for insoluble anesthetics since minimal amounts are taken up at baseline.
- A right to left intracardiac shunt will slow induction, because a portion of blood flow will bypass the lungs and not become saturated with anesthetic. Similarly, a mainstem intubation will also slow induction, since half of pulmonary blood flow will go to a non-ventilated lung.
- MAC, or the 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 stimuli. At 1.3 MAC, 95% of patients will not move to that same stimuli. 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. Halothane is not used at any of the three locations although you may find old, empty vaporizers still attached to the anesthesia machines. You will encounter halothane at Children’s Hospital or on trips to under-served areas, e.g., Mexico.

A. Desflurane
- general- very fast onset and offset, as fast as nitrous oxide. Requires a special vaporizer (see anesthesia equipment section). MAC is 6- not very potent. Relatively expensive.
- Cardiovascular- minimal cardiac depression, but does cause decrease in SVR and MAP. May cause transient increases in blood pressure and heart rate.
- decreases hypoxic respiratory drive. Increases apneic threshold. Very pungent-induction and awakening can be associated with coughing, bronchospasm or laryngospasm
- neurologic- increases CBF but decreases CMRO2. Uncouples cerebral autoregulation, rendering CBF proportional to MAP.
- Other- degraded by dry CO2 absorbent (especially barium hydroxide) to carbon monoxide. Trigger for malignant hyperthermia.

B. Halothane
- general- inexpensive. MAC is 0.7. Suitable for inhalational inductions.
- Pulmonary- decreases hypoxic respiratory drive. Increases apneic threshold.
- Neurologic- increases CBF but decreases CMRO2. 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 (1:30,000 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.

C. Isoflurane
- general- 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. Increases apneic threshold. Not suitable for inhalational induction.
- Neurologic- increases CBF but decreases CMRO2. 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. Sevoflurane  
- general- Fairly rapid onset and offset (second to desflurane). Suitable for inhalational inductions. MAC is 2. Somewhat expensive.  
- Cardiovascular- minimal cardiac depression, but does cause decrease in SVR and MAP. Dilates coronary arteries.  
- Pulmonary- decreases hypoxic respiratory drive. Increases apneic threshold.  
- Neurologic- increases CBF but decreases CMRO2. 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), high concentrations of sevo, or low anesthetics. 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- stimulates the sympathetic nervous system. Increases pulmonary vascular resistance.  
- Pulmonary- decreases hypoxic drive.  
- Neurologic- mildly increases cerebral blood flow and CMRO2.  
- Renal/hepatic- decreases blood flow to both systems  
- Other- inhibits B12 dependent enzymes, including those necessary for DNA synthesis. Prolonged exposure can result in megaloblastic anemia and peripheral neuropathies. 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.

V. Hypnotics

A. Barbiturates-  
- Mechanism of action- GABA.  
- 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 hrs- repeated doses can saturate peripheral compartments, making recovery dependent on elimination (and thus much slower).
- **Cardiovascular**- decreases BP and CO, mostly due to peripheral vaso and venodilation, pooling of blood and decreased preload. Said to have “killed more GI’s than the enemy” in World War II due to rapid administration in under-resuscitated patients.
- **Pulmonary**- causes respiratory depression and apnea. May not fully depress airway reflexes- bronchospasm or laryngospasm in “light” patients is possible.
- **Neurologic**- profound decreases in CMRO2 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. 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 or versed will be discussed as it is the benzodiazepine most commonly used by anesthesiologists.
- **Mechanism of action**- GABA.
- **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 wakeups.
- **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 CMRO2 and CBF. Causes antegrade amnesia.
- **Other**- useful premedication due to antegrade amnesia. PO or IM administration useful in children who cannot tolerate an IV.

C. **Etomidate**
- **mechanism of action**- GABA.
- **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.
- **Pulmonary**- typically does not cause apnea **when given alone**. If given in conjunction with other agents (e.g., narcotics) can cause profound respiratory depression.
- **Neurologic**- decreases CBF and CMRO2.
- **Other**- may cause adrenal suppression, more of a concern when given as a long term infusion. Significant incidence of myoclonus when given which can be disturbing. May increase nausea and vomiting. Burns on injection.

D. **Propofol**
- **Mechanism of action**- GABA.
- Route of administration- IV.
- Pharmacokinetics- rapid onset. Rapid offset as well, due to redistribution.
- Cardiovascular- decreases MAP and cardiac contractility.
- Pulmonary- causes respiratory depression all the way to complete apnea depending on dose.
- Neurologic- decreases CBF and CMRO2; considered one of the best agents to reduce brain size/anesthetic for craniotomies. Antiepileptic.
- Other- considered to have antiemetic properties. Long term infusions can cause lactic acidosis. Lipid emulsion is good growth medium for bacteria- use strict aseptic technique and within 6 hrs of opening. Burns on injection. Contains lecithin- found in egg yolks, not 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 receptors.
- Route of administration- IV or IM.
- Pharmacokinetics- rapid onset. Rapid offset as well, due to redistribution.
- Cardiovascular- is a sympathomimetic, stimulates the sympathetic nervous system and thus typically maintains SVR, CO and BP. 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) may have profound myocardial depression.
- Pulmonary- does not affect respiratory drive when given alone. Good bronchodilator.
- Neurologic- increases CMRO2 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.
- Other- is a “dissociative” anesthetic, in that patients may appear awake but do not respond to sensory input. Also a profound analgesic. Often useful in small doses during cesarean sections when a spinal anesthetic is inadequate or the patient cannot tolerate peritoneal traction.

VI. Opioids

General- Opioids are intense analgesics. However, they do not reliably produce amnesia. Although there are many different opioids only the most common IV ones will be discussed here- morphine, fentanyl, sufentanil, alfentanil, remifentanil 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 which may be coadministered. Thus, it is difficult to provide “standard doses”.

Opioids are most commonly administered by anesthesiologists for intraoperative anesthesia or postoperatively for pain control. We also provide a wide range of services through our chronic and acute pain consult services where opioids are a mainstay of treatment.
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-

- Fentanyl- 100x as potent
- Remifentanil- 200x as potent
- Sufentanil- 10x as potent as fentanyl (1000x morphine)
- Alfentanil- 1/5 as potent as fentanyl (20x morphine)
- Meperidine- 1/10 as potent as morphine

Comparison of doses between the opioids generally reflect these relative potencies- for example, you might consider giving 4mg of morphine or 50 mcg of fentanyl to the same patient for postoperative pain.

A. Morphine
- Usual route of administration and dose- 0.05-2mg/kg IM, 0.03-1mg/kg IV; can also be given intrathecally or epidurally.
- Pharmacokinetics- longer onset and duration of action due to low lipid solubility. Onset typically within 20 min, peak plasma levels within 60min.
- Cardiovascular- by blocking sympathetic output, may cause decrease in BP and HR. Typically minimal changes when given alone.
- 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 and vomiting and pruritis.
- Other- slows GI motility and gastric emptying. Is the “equivalent” against which other opioids are measured (e.g., 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 with prolonged effects. Large doses can result in significant histamine release.

B. Fentanyl
- Usual route of administration and dose- very wide ranges of doses depending on tolerance and state of the patient. Typically given IV, intrathecally or epidurally, transdermally via a patch or transmucosally via a “lollipop”. A typical IV dose to block sympathetic response to intubation is 2-5 mcg/kg.
- Pharmacokinetics- rapid onset within minutes. Highly lipophilic- crosses the blood-brain barrier easily. Typically short duration of action due to redistribution.
- Cardiovascular- by blocking sympathetic output, may cause decrease in BP and HR. Typically minimal changes when given alone.
- Pulmonary- causes respiratory depression, depression of hypoxic drive and increases apneic threshold. Can see chest wall rigidity when given in large doses which may compromise ventilation.
- Neurologic- may decrease CMRO2 and CBF to small extent, although associated respiratory acidosis may outweigh this. Can cause nausea and vomiting and pruritis.
- Other- slows GI motility and gastric emptying. Repeated doses or infusions can cause saturation of peripheral redistribution sites which can greatly increase the time to offset (context sensitive half time). Chest wall rigidity can be managed with neuromuscular blockers.

C. Alfentanil-
- Usual route of administration and dose- IV. Given in doses 5x that of fentanyl.
- Pharmacokinetics- rapid onset/onset within minutes. Although less lipid soluble than fentanyl, onset/offset is more rapid due to low pKa, thus most of alfentanil existing in non-ionized, lipophilic form.
- Cardiovascular- by blocking sympathetic output, may cause decrease in BP and HR. Typically minimal changes when given alone.
- Pulmonary- causes respiratory depression, depression of hypoxic drive and increases apneic threshold. Can see chest wall rigidity when given in large doses which may compromise ventilation.
- Neurologic- may decrease CMRO2 and CBF to small extent, although associated respiratory acidosis may outweigh this. Can cause nausea and vomiting and pruritis.
- Other- slows GI motility and gastric emptying. Repeated doses or infusions can cause saturation of peripheral redistribution sites which can greatly increase the time to offset (context sensitive half time), although the effect is less pronounced than with fentanyl. Chest wall rigidity can be managed with neuromuscular blockers. Excellent for situations requiring intense, short lived analgesia.

D. Sufentanil-
- Usual route of administration and dose- IV. Given in doses typically 1/10 that of fentanyl.
- Pharmacokinetics- very rapid onset/offset within minutes.
- Cardiovascular- by blocking sympathetic output, may cause decrease in BP and HR. Typically minimal changes when given alone.
- Pulmonary- causes respiratory depression, depression of hypoxic drive and increases apneic threshold. Can see chest wall rigidity when given in large doses which may compromise ventilation.
- Neurologic- may decrease CMRO2 and CBF to small extent, although associated respiratory acidosis may outweigh this. Can cause nausea and vomiting and pruritis.
- Other- slows GI motility and gastric emptying. Repeated doses or infusions can cause saturation of peripheral redistribution sites which can greatly increase the time to offset (context sensitive half time), less than either fentanyl or alfentanil over 8 hr infusions. Chest wall rigidity can be managed with neuromuscular blockers. Excellent for situations requiring intense, short lived analgesia.

E. Remifentanil-
- Usual route of administration and dose- IV. 1mcg/kg induction doses and 0.05-1 mcg/kg/min infusions are common.
- Pharmacokinetics- very rapid onset within 1 minute. Also rapid offset, within ~ 4 min. Pharmacokinetic behavior is due to unique mode of metabolism which is by red blood cell and nonspecific esterases. This metabolism makes the pharmacokinetics very predictable and titratable. Furthermore, the context sensitive half-time for remifentanil essentially does not change for long-time infusions.
- Cardiovascular- by blocking sympathetic output, may cause decrease in BP and HR. Typically minimal changes when given alone.
- Pulmonary- causes respiratory depression, depression of hypoxic drive and increases apneic threshold. Can see chest wall rigidity when given in large doses which may compromise ventilation.
- Neurologic- may decrease CMRO2 and CBF to small extent, although associated respiratory acidosis may outweigh this. Can cause nausea and vomiting and pruritis.
- Other- excellent for cases that have intense analgesic requirements, yet also need precise titration of opioid. Has no “tail”- all analgesic effects will be gone with minutes of stopping the drug. Thus, another longer-acting opioid must be used to transition patients from the stopping of remifentanil to avoid postoperative pain. Must be reconstituted and diluted from powder form.

F. Meperidine-
- Usual route of administration and dose- IV or IM. 0.2-0.5mg/kg IV doses often given for post op analgesia or shivering.
- Cardiovascular- unique to the IV opioids, often causes tachycardia due to structural similarity to atropine. Also unique, can depress cardiac contractility.
- 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.
- Other- uniquely effective among opioids at decreasing shivering. Active metabolite is normeperidine which causes CNS stimulation and potentially seizures. Both meperidine and normeperidine can accumulate in renal failure, compounding this risk. Avoid with MAO inhibitors.

VII. Local anesthetics

Local anesthetics function by blocking sodium channels and preventing depolarization and action potentials. They bind the channels on the inside of cellular membranes and thus must cross the lipophilic cell membrane to achieve their action.

Local anesthetics are weak bases, have a pKa above 7 and tend to be positively charged at physiologic pH. They are either esters or amides. 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. Esters are metabolized by pseudocholinesterase and their activity is prolonged in patients with abnormal pseudocholinesterase (see neuromuscular blocker section). Amides are metabolized by the liver. Esters also tend to have a shorter duration of action than amide local anesthetics.

The pharmacokinetics of local anesthetics depend on the pKa, the lipid solubility and the concentration of drug. A lower pKa means that more drug exists in non-ionized form at physiologic pH, more readily crosses the plasma membrane, and thus onset is faster. More lipid soluble agents tend to be more potent, have more protein binding, and a longer duration of action (e.g., bupivacaine). Higher concentration of drug typically creates a more profound and faster block. A typical example of this is 10% procaine. Procaine has a high pKa which should confer a slower onset, but the concentration is so high that 10% procaine has a quick onset on action.

Nerves are affected differently according to size and myelination. Smaller fibers and myelinated fibers tend to be blocked earlier. Thus, the order of onset for any group of nerves is autonomic, sensory, motor. Level of block for a spinal or epidural tends to be 1-2 levels higher for more sensitive nerves. 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 can prolong duration of the block (less uptake) and decrease systemic absorption and potentially toxicity. It should be noted that bupivacaine and ropivacaine are not affected, and their normally long duration of action is a result of high protein binding, not epinephrine. 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 onset. Premixed solutions of local anesthetic containing epinephrine have a pH around 5 (needed to maintain epinephrine stability). Adding epinephrine manually to local anesthetics rather than using premixed solutions can 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 and fibrillation and frank arrest. In general, excitatory phenomenon precede depressive phenomenon, which tend to precede cardiac involvement. The exception to this is bupivacaine, which can often present with cardiac reactions as the first sign of intravascular injection. Cardiotoxicity from bupivacaine is notoriously long and difficult to resuscitate owing to its protein binding. Cardiopulmonary bypass and lipid infusions may be employed (lipids seem to absorb the bupivacaine). The treatment of all these toxicities is supportive- reassure the patient, protect the airway as necessary, inhibit seizure activity with benzodiazepine, propofol or barbiturates, and support circulation/ACLS as necessary. Esters seem to be less toxic than amides due to rapid breakdown in plasma. Bupivacaine is the most toxic.

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

A. Benzocaine

Uses: topical anesthesia (e.g., airway)
Usual duration: 1hr
Notables: Can cause methemoglobinemia (see monitoring section), the treatment for which is methylene blue.

B. Bupivacaine

Uses: spinal, epidural, local, regional blocks
Usual duration: 2-8 hrs, sometimes more (unaffected by epinephrine)
Maximum safe dose: 3mg/kg (unaffected by epinephrine).
pKa: 8.1
Notables: Very long duration of action, highly potent. Proven safety in spinal and epidural anesthesia.

Unique toxicity behavior as described above.

C. Chloroprocaine

Uses: epidural, local, regional blocks
Usual duration: 30min to 1 hr.
Maximum safe dose: 12mg/kg
pKa: 9
Notables: Associated with neurologic damage when used in intrathecal space, which may be due to an old preservative, sodium bisulfate.

D. Mepivicaine

Uses: epidural, local, regional block
Usual duration: 1-2 hrs.
Maximum safe dose: 5 mg/kg, 7 with epinephrine.
pKa: 7.6
Notables: Often employed in regional blockade for quick onset, potent, medium duration block. 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 post op analgesia.

E. Lidocaine

Uses: spinal, epidural, local, regional topical and IV regional anesthesia.
Antiarrhythmic.
Usual duration: 1-2 hrs (greater with epinephrine)
Maximum safe dose: 5mg/kg, 7 with epinephrine.
pKa: 7.8

Notables:
Associated with cauda equina syndrome and permanent neurologic damage. The described cases involved 5% lidocaine and small bore infusion catheters, which were thought to cause pooling of lidocaine and unacceptably highly-concentrated areas on spinal nerves, leading to damage. These catheters have since been removed but the fear of lidocaine remains. Lidocaine does seem to be more neurotoxic than other local anesthetics.
 Associated with transient neurologic symptoms (TNS) after intrathecal administration of 5% lidocaine. These symptoms include burning, pain and aching of the lower extremities and buttocks. They typically resolve within 1 week. 
Lidocaine, lithotomy position, outpatient surgery and obesity all seem to be risk factors. Many people have abandoned the use of 5% lidocaine for this purpose.

Can be given via ETT.

F. Prilocaine

Uses: dental procedures, topical (EMLA cream)
Usual duration: 30min – 1 hr.
Maximum safe dose: 8mg/kg.
pKa: 7.8
Notables: Can cause methemoglobinemia (see above and monitors section).

G. Procaine

Uses: spinal, local, regional block
Usual duration: 30min -1hr.
Maximum safe dose: 12mg/kg (like chloroprocaine).
pKa: 8.9
H. Ropivacaine

Uses: same as bupivacaine. Think of ropivacaine as similar to bupivacaine in almost all aspects, including protein binding, duration, uses, and type of block.
Usual duration: 2-8hrs
Maximum safe dose: like bupivacaine, 3 mg/kg.
pKa: 8.1 (same as bupivacaine).
Notables: Thought to be less cardiotoxic than bupivacaine and thus safer.

Tends to have preferential sensory blockade over motor, making it ideal for postop catheters and analgesia.

I. Tetracaine

Uses: spinal, topical Usual duration: 2-6hrs. Maximum safe dose: 3 mg/kg. pKa: 8.2
Notables: Also associated with cauda equina syndrome (see above) and small-bore intrathecal catheters
**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 recommendations. For more information, consult a more detailed reference.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Purported benefit</th>
<th>Effects</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valerian</td>
<td>decrease 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 rxn; 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 stim similar to ephedrine; increased HR, BP, arrhythmias</td>
<td>d/c prior to surg avoid MAOI</td>
</tr>
<tr>
<td>Garlic</td>
<td>reduces BP, cholesterol</td>
<td>irreversible inhibition</td>
<td>d/c 7d before surgery of platelet aggregation</td>
</tr>
<tr>
<td>Ginkgo</td>
<td>improves cognition, Circulation</td>
<td>inhibits PAF</td>
<td>d/c 2d 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 7d before surgery</td>
</tr>
<tr>
<td>Kava</td>
<td>decrease anxiety</td>
<td>may decrease MAC via GABA</td>
<td>d/c 24 hr before surg</td>
</tr>
<tr>
<td>St. John’s</td>
<td>antidepressant</td>
<td>inhibits norepi,</td>
<td>d/c 5d before surgery</td>
</tr>
</tbody>
</table>
Wort dopamine, serotonin
reuptake; induces
 cytochrome P450
(increased drug metabolism)

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

References:
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 overuse of NMB’s is a common Achilles’ Heel for many anesthesiologist. Never forget that NMB’s 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 NMB’s 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. When the nerve’s 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 end plate), causing depolarization (end plate potentials). When enough end plate potentials are generated, the whole membrane will depolarize, opening sodium channels, releasing calcium and causing muscle contraction. Termination of the action potential is caused by hydrolysis of acetylcholine by acetylcholinesterase and is rapid.

Depolarizing vs. nondepolarizing blockade

Depolarizing NMB’s (succinylcholine) work by causing depolarization of the muscle-end plate, then preventing end-plate repolarization, blocking further depolarization. Thus succinylcholine is an Ach receptor agonist. By contrast, non-depolarizing NMB’s bind to the acetylcholine receptor, preventing depolarization (competitive antagonism).

Succinylcholine’s offset is dependent on diffusion away from the neuromuscular junction and subsequent hydrolysis by pseudo or plasmacholinesterase. By contrast, the NMB’s must be metabolized and excreted. They can be outcompeted by additional amounts of Ach (reversal of neuromuscular blockade).

Succinylcholine

No other NMB is as rapid in onset and offset as succinylcholine. The classic “short” blockade caused by succinylcholine is termed a phase I block. After repeated administration, a phase II block may occur which resembles that of non-depolarizing 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 30min. Homozygotes (1:2500) may have a profoundly long blockade, on the order of 8 hrs. The dibucaine number is proportional to the level of normal pseudocholinesterase activity. Normal pseudocholinesterase is 80% inhibited by dibucaine, 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 the use of succinylcholine in patients with known or family history of atypical pseudocholinesterase, and keep in mind that patients experiencing prolonged block will need mechanical ventilation and sedation.

Cholinesterase inhibitors prolong succinylcholine blockade by inhibiting pseudocholinesterase, and by providing more Ach at the neuromuscular junction, 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 (prevents depolarization by succ).

Succinylcholine is relatively contraindicated in children b/c of the possible presence of undiagnosed myopathies. It is also contraindicated in patients with a preexisting condition associated with succinylcholine induced hyperkalemia (many of them, examples include old burns, spinal cord injury, myopathies) and in patients with a history of malignant hyperthermia. For more info see the drug section.

Non depolarizing neuromuscular blockers

These are either benzylisoquinolones or steroids. All function via the same mechanism- namely, 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 NMB must already be present. Recovery depends on metabolism and elimination of the NMB in question.

Typically, 1-2x the ED95 dose of a NMB is used for an intubating dose, while 1/10 ED95 doses are used for maintenance relaxation. Higher doses may afford slightly quicker onset of blockade but can also greatly prolong the block.
Certain NMBs, particularly 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 will prolong blockade, either by decreasing enzyme activity (metabolism) or slowing excretion. Similarly, hypoventilation and respiratory acidosis prolongs blockade. Electrolyte imbalances such as hypocalcemia, hypokalemia, or hypermagnesemia will result in abnormally long paralysis. Obviously, liver or kidney disease can also prolong blockade depending on the NMB’s route of metabolism and excretion. Other drugs that can prolong non-depolarizing blockade are the same antibiotics as 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 duration of NMB’s metabolized by the liver such as 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 recover sooner than the adductor pollicus or the muscles innervated by the posterior tibial nerve and are the “last to go” during onset of blockade. In general, the orbicularis oculi corresponds best with the level of paralysis of the diaphragm and larynx, the two muscles 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 blockade, but are often resistant to succinylcholine (fewer Ach receptors). The block with either type of drug is unpredictable and must be monitored closely. Burn and chronic denervation injury patients have increased extrajunctional receptors, making them resistant to nondepolarizing blockade.

Monitoring neuromuscular blockade with peripheral nerve stimulation

Any patient who will be given 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 larynx, the two muscles groups we are often most concerned about.

As NMB 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 200 microsec duration. Train of four is four twitches delivered in 2 seconds. Tetany delivers
very rapid (50 or 100Hz) twitches as long as the button is pressed, resulting in sustained contraction. The “twitch height” is a qualitative measure of the level of muscle response to stimulation.

As nondepolarizing muscle blockade ensues, each successive twitch in a train of four will show “fade”, meaning the height of each successive twitch decreases (see diagram below). As blockade increases twitches fade altogether, from last to first. Disappearance of the 4th twitch corresponds to ~ 75% blockade and the 2nd to a 90% blockade. Thus, if a patient has one twitch only to 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 NMB.

After a tetanic stimuli is applied, a subsequent train of four will show supramaximal muscular 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 now see twitches due to this phenomenon. It is thought to be due to briefly increased levels of Ach in the neuromuscular junction due to repeated stimulation, thereby allowing outcompetition of the neuromuscular blocker.

Clinically, five seconds of sustained tetany (no fade) or the ability to lift one’s head up for five sec correlates with at least 50% of receptors being unblocked, and are the best tests we have to measure recovery from NMB.

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Train of four response to non-depolarizing blockade. The arrow represents administration of NMB. Note progressive fade, with eventual loss of twitch number 4.

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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 with no twitches you have no idea of the level of NMB present. The patient could be 5 min or 60 min from recovery of blockade but you don’t know which- all we see clinically is that the patient has no twitches. Reversal at this stage could result in a
patient who briefly regains strength but becomes weak or paralyzed again some time later due to excessive amounts of paralytic. Thus, 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 with 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 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. It is embarrassing and time-consuming but the safest way to proceed. The patient should not have to pay for your impatience.

As previously mentioned the best evidence of recovery from NMB is 5 seconds of sustained tetany, or 5 seconds of lifting one’s head up. Return of train of four without fade is not adequate, because 70% of receptors could still be blocked in this case. In fact, with return of 5 sec of tetany we can do no more than say at least 50% of receptors are unblocked- meaning up to 50% may still be blocked. Unfortunately anesthesiologists do not have a better test in clinical practice. The reality of the situation is that this should be a safe level of neuromuscular function that the patient will be able to breathe on his own and maintain an airway.

Blockade with succinylcholine exhibits a different response to stimulation than non-depolarizers. Twitch height during train of four and tetany is equally decreased at all stages. There is no fade and no post-tetanic facilitation. However, a phase II block of succinylcholine will respond like a non-depolarizer to stimuli- be aware of this phenomenon if it occurs. See the diagram below.

Myasthenia gravis

This disorder is commonly tested on the boards. It is due to autoimmune destruction of postsynaptic Ach receptors, resulting in weakness. The ocular, laryngeal and pharyngeal muscles (bulbar symptoms) can all be involved, as well as proximal skeletal and respiratory muscles. It is more common in women, has an association with
other autoimmune disorders and thymoma or thymic hyperplasia is often found. The
disease improves with rest and worsens with exercise.

Anticholinesterases are the usual treatment and work by increasing available Ach.
Pyridostigmine is the most commonly used anticholinesterase. Other treatments for more
advanced disease include plasmapheresis, IgG infusions, and steroids. Excision of
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. The preoperative goals should be to optimize
medical therapy. Preop respiratory or bulbar weakness should be treated with IgG or
plasmapheresis. Periodically a patient may present in cholinergic crisis from excess
anticholinesterase. These patients are also weak, but exhibit signs of muscarinic excess
such as salivation, miosis, bradycardia and diarrhea. Edrophonium can be used to
differentiate cholinergic crisis from myasthenic crisis and is short acting. Worsening of the
symptoms implies cholinergic crisis, while improvement suggests issues arising from
myasthenia.

Response to succinylcholine is unpredictable. Patients can have prolonged or
shortened effects, or a phase II block. Sensitivity to non-depolarizers is profound. These
patients need to be closely monitored; reduced doses of NMB should be used or avoided
entirely.

**Lambert-Eaton myasthenic syndrome**

This disease involves autoimmune antibodies to presynaptic calcium receptors,
reducing Ach release from the motor-end plate. It is classically improves with exercise. It
is associated with paraneoplastic syndromes, classically small cell cancer of the lung.
Immunosuppression or plasmapheresis helps to certain degree, anticholinesterases less
so. These patients are sensitive to both succinylcholine and non-depolarizers.

**Final thoughts**

There is a very common pattern to the use of NMB’s as one progresses through
anesthesia 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 more than few “slow wakeups” or over-paralyzed patients who
either have to wait to be reversed, or become weak and need reintubation in the recovery
room. Later, a resident may greatly curtail the use of NMB, tailoring his anesthetic to a
quick wakeup and not worrying about surgical concerns. As experience with NMB’s
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 even 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. One technique many of us employ is to simply agree with the surgeon and pretend to give the NMB. 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, abdominal surgeries (especially laparoscopic where the abdomen will be insufflated) or any procedure where patient movement could be catastrophic. Interestingly, the better surgeons seem to be the ones who least often ask for paralysis, either because they don’t need it or they expect us to know when it is indicated.

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

Lastly, be aware when a case might end very quickly and unexpectedly. A typical example of this is an exploratory laparotomy for possible cancer. Sometimes, the surgeons will open the belly and either find inoperable tumor or conditions which preclude surgery. In these cases they will simply close the patient up and the case is over (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.
Anesthesia for general surgery and intraabdominal procedures

General surgery encompasses many different types of cases, all with their 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.

I. Surgery on the neck, thorax, breast and trunk

Thyroid and parathyroid surgery

Technique: general anesthesia.

Monitors: standard.

IV access: one IV typically suffices.

Duration: 2-3 hrs.

Estimated blood loss: < 100cc.

Position: supine.

Special equipment: possible NIM (Nerve Integrity Monitoring) tube.

Special considerations:

General surgeons usually perform thyroid and parathyroid surgeries without turning the OR table (in contrast to ENT surgeons). Usual indications for either surgery are neoplasms or hypersecretory glands. If a patient is in a hyper or hypometabolic emergency state, surgery is withheld until the patient is medically stabilized (in other words, there is no such thing as emergency thyroid surgery). Occasionally, a parathyroid gland will be reimplanted in a peripheral site such as the arm.

Because the procedure takes place close to the airway, ensure that the airway is secure. The operation can also cause 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 may ask for a NIM tube to be placed. This tube has leads which are connected to a special monitor (run by an outside monitoring technician and set up by the surgeon). The tube has a special monitoring stripe, usually blue, which must be placed between the vocal cords. Other than proper placement and the monitoring leads, it functions as a regular endotracheal tube. Intraoperative nerve injury can be detected early via the monitoring device and allow for a change in surgical technique.
Rarely, when the surgeon has a high suspicion of bilateral vocal cord nerve injury, they may ask for vocal cord function to be visualized before the endotracheal tube is pulled at the end of the case. This necessitates a fiberoptic bronchoscope or some other means of visualizing the cords while the patient is still anesthetized. Hematoma formation can also lead to potential airway compromise.

During parathyroid surgery we are routinely asked to draw blood to check parathyroid hormone levels. Removal of all hypersecretory glands reliably causes a reduction in PTH levels, while sustained high levels of PTH will prompt the surgeon to explore further.

**Esophageal surgery**

Open procedures on the esophagus including esophagectomy necessitate a thoracotomy and one-lung ventilation. Additionally, there is the potential for significant blood loss and surgical time. The indication is almost always cancer. Thoracic epidurals are of proven benefit in managing post operative pain and ventilation in these patients, but unfortunately our present surgeons are “philosophically opposed” to epidurals and do not like them to be placed.

**Technique:** general +/- thoracic epidural.

**Monitors:** standard, plus arterial line.

**IV access:** at least 1 large IV.

**Duration:** 2-6 hrs.

**Estimated blood loss:** 200 – 1000 cc or more.

**Position:** supine, lateral decubitus or thoracoabdominal (operative side slightly propped up with the arm airplaned across the body and spine extended).

**Special equipment:** tubes for one lung ventilation (see cardiothoracic anesthesia section).

**Special considerations:**

As previously stated these surgeries can be quite extensive, at times extending into the abdomen for further organ removal. Typically blood loss is small, however proximity to large vessels make significant bleeding a real possibility.

**Breast surgery**
These procedures range from simple lumpectomies, to bilateral mastectomies with abdominal flaps and major reconstruction (usually in conjunction with plastic surgery). The extent of the surgery dictates the duration and sequelae of the case.

Technique: usually general anesthesia. Simple lumpectomies can be performed under local/MAC. Regional anesthesia (paravertebral blocks) are an option but not commonly employed.

Monitors: standard. Truly extensive surgery in a patient with other indications may necessitate an arterial line. +/- urine output.

IV access: one IV should suffice.

Duration: anywhere from 30 min for simple cases to 8 hr affairs for major reconstruction.

Estimated blood loss: again, minimal to 500cc (most cases are on the low side).

Position: supine. Breast reconstruction cases may require the patient to be “sat up” at times to check alignment and symmetry of the breasts.

Special equipment: none.

Special considerations:

If position changes are anticipated (e.g., breast reduction), a more secure airway such as an ETT is indicated.

Even for long cases, there tends not to be large amounts of blood loss due to relatively avascular location of surgery.

If axillary lymph node dissection is planned, avoid placing IV lines or monitors on the side of the affected extremity.

**Other surgeries on the trunk/back**

These are usually lipoma or other soft tissue mass removal or biopsies. Anal exams/fistulotomies are also included. The surgeries are typically short with minimal dissection.

Technique: general or local/MAC. Hypobaric neuraxial techniques work well for anal surgery.

Monitors: standard.

IV access: one IV.
II. Intraabdominal surgery

There are three major categories of intraabdominal surgery—major open, minor open, and laproscopic.

Major open procedures

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 resections, including whipple procedures
- liver resections
- major bowel resections
- major abdominal explorations or lymph node dissections
- open gastric bypass
- splenectomy

Technique: general. Again, an epidural would be of benefit to these patients but we tend not to put them in due to surgical request.

Monitors: standard. Usually an arterial line. CVP can be helpful to guide fluid resuscitation.

IV access: at least two large IVs.

Duration: 3-6 hrs.

Estimated blood loss: 500 to over 2L.

Position: supine.

Special equipment: warmers for IV lines, forced-air warming blankets.

Special considerations:
Insensible fluid losses from exposed bowel can be extensive. The recommended rate of fluid replacement for these losses is at least 10cc/kg/hr. Note 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 dehydrated and volume depleted. Bear this in mind during induction and choose the appropriate induction agent. Electrolyte disturbances are also possible.

For certain cases, notably whipples, the surgeons have the mindset from the beginning that the patient will remain intubated at the end of the procedure and that the ETT will be removed later in the ICU. This can be discussed on a case-by-case basis with the surgeon. Routine postop intubation is not necessary.

Keep in mind there may be a possibility of a “peek and shriek” (the surgeons open the belly and find inoperable conditions, and quickly close). Don’t go overboard on NMBs at the beginning of the case.

**Minor open procedures**

Clearly there is some overlap between a minor and a major abdominal procedure. A simple bowel resection or colectomy tends to be a minor affair, but depending on surgical skill and particular patients 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
- colectomies, sigmoidectomies
- biopsies
- hernia repairs
- appendectomy

Technique: general anesthesia. Rarely, a neuraxial technique such as continuous spinal if there is a strong reason to avoid general anesthesia. This will not be pretty- manipulation of the bowel and peritoneal traction is notoriously hard to block with neuraxial techniques. The patients may not feel pain but will be extremely uncomfortable. Inguinal hernias may be repaired under a variety of techniques, including general, regional (paravertebral blocks), neuraxial and local/MAC.

Monitors: standard. Rarely an arterial line.

IV access: one large IV should suffice for the vast majority of cases.

Duration: 1- 4hrs.
Estimated blood loss: < 500cc.

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/robotic procedures**

Many traditionally open procedures are now being performed laparoscopically or minimally invasively. Robotic procedures are included in this section because they have the same anesthetic implications. Laparoscopic procedures often reduce the extent of the surgery, the exposure to the patient as well as fluid shifts and blood loss. However, depending on surgical skill and the procedure they 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. Examples of laparoscopic/robotic procedures include-

- cholecystectomy
- hernia repairs
- appendectomy
- gastric bypass, banding, or Nissen fundoplication
- bowel resection

Technique: general anesthesia.

Monitors: standard. Rarely an arterial line and urine output for longer procedures.

IV access: one IV.

Duration: 1 – 8 hrs depending on the procedure (use of a robot typically slows down a case).

Estimated blood loss: usually minimal.

Position: supine. Significant trendelenburg or reverse trendelenburg for long durations depending on the case.

Special equipment: if applicable, provided by the surgeon (bougies and esophagogoscopes).
Special considerations:

Steep T-burg or reverse T-burg for long periods of time can have significant impacts on patient physiology, including ventilation and impeding venous return. There have been many cases where a patient has been in steep T-burg for hours, and at the end of the case had to remain intubated due to significant facial swelling and potentially hazardous extubation.

Insufflation of the abdomen with CO2 creates unique issues for the anesthesiologist. In no particular order, they are- increased CO2 load, increased intraabdominal pressure and difficulty with ventilation, possibility of CO2 embolism, subcutaneous deposition of CO2, pneumothorax and pneumomediastinum. 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 CO2. In general, minute ventilation will have to be increased by 1/3 of baseline. Intravascular injection of CO2 is usually benign but the surgeon should be notified. Typically you will see a sharp rise in EtCO2 on the capnograph beginning with insufflation. Pneumothorax or mediastinum should be treated as needed. If left uncorrected, rising CO2 load can create profound acidosis with all of its associated problems.

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

Orthopedic procedures comprise a significant percentage of cases at all three hospitals. The patient population is very broad, comprising the elderly patient with many medical problems coming for a fractured hip, to a young otherwise healthy college student and everything in between. The orthopedic surgeons are likewise heterogeneous in terms of personality and ability. Our orthopedic surgeons run the gamut from friendly to acerbic, skilled to minimally competent. With time and experience you will discover who is who, and what to expect intraoperatively. This guide will hopefully ease the learning process. Typical scenarios for orthopedic cases range from scheduled, elective surgery to urgent or emergent repairs for open fractures. In fact, orthopedic procedures make up a large part of “add-on” or late night cases.

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 the residency and especially during the third year regional rotation. The goal of this guide is not to teach how to perform a regional block. Where applicable, however, the choice of regional anesthetic will be indicated.

Many orthopedic procedures are “outpatient” or same day surgeries, meaning the patients will typically go home soon after recovery from surgery and anesthesia. The anesthetic technique for these patients can be challenging and is geared towards quick wakeups, quick room turnover, and good control over pain and nausea, all components which may keep a patient in the PACU for extended periods of time. Be mindful of these short procedures and tailor the anesthetic to the patient’s needs and discharge goals.

Many orthopedic procedures involve routine use of a tourniquet. The tourniquet is intended to reduce blood flow and thus loss to the operative site and improve visualization. Many surgeons feel that “higher BPs” can increase flow past the tourniquet and bleeding. They will often ask for us to lower the patient’s blood pressure. The challenge for us becomes trying to maintain a good working environment with the surgeons and still doing what is in the best interests of the patient. Clearly hypotension, or even relative hypotension can be deleterious. There are many techniques to negotiate around this problem- one is to recognize the situation and perhaps give the surgeons a number they want to hear when they ask, “what’s the BP?” Risks of tourniquet use include pain (increases with duration of use) and hypotension following deflation due both to relief of pain and washout of metabolic waste products. These same waste products typically lead to a transient rise in CO2 levels, potassium and lactic acid. Tourniquet use on the lower extremity is associated with a higher incidence of DVT. Lastly, the ischemia produced by a tourniquet makes it relatively contraindicated in sickle cell patients.

DVT is a feared and not uncommon complication of orthopedic procedures, particularly on the knee and hip. Prophylactic anticoagulation is often given, and compression stockings are the rule. Subcutaneous, prophylaxis doses of heparin are not a
contraindication to regional anesthesia (see neuraxial blockade and anticoagulation section). Regional anesthesia may lower the risk of DVT by improving regional blood flow via vasodilation, and reduction of inflammatory mediators. However, the routine use of postoperative anticoagulation can make the placement, use and removal of an indwelling epidural catheter tricky. For knee replacements in particular, UCSD is moving towards peripheral nerve blocks which do not carry the same risks.

Procedures on fractured long bones carry with them the risk of emboli, particularly fat emboli. This will often be heralded with a sudden drop in EtCO2 and potentially a drop in blood pressure, cardiac output, and oxygen saturation. Other classic signs include mental status changes and petechiae which typically appear 1-3 days post op. Treatment is supportive.

Joint replacements involve the use of polymethylmetacrylate to cement the artificial joint in place. This cement can cause a variety of intraoperative problems, including hypotension, dysrhythmias, increased pulmonary vascular resistance, hypoxia, and exacerbation of debris embolization (the cement itself expands within bone). The cement itself liberates heat via an exothermic reaction which can burn tissues. Treatment is typically supportive.

I. 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-
- spinal tumor resections
- instrumented fusions, especially multilevel
- anterior and posterior procedures
- corpectomies, especially multilevel
- “redo” procedures

Spine procedures with typically little blood loss include-
- discectomies
- laminectomies (without instrumentation/fusion)
- single level procedures
- cervical spine procedures
- minimally invasive or percutaneous procedures

A consideration of virtually every spine procedure is the necessity of placing the patient in the prone position. This position demands special padding is placed to protect the chest, genitals, breasts, and especially the face. 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. The padding of the face and protection of the eyes is our primarily our responsibility. There is a small but finite incidence of postoperative blindness in patients undergoing a prone procedure. As this outcome is so devastating we do everything we
can to avoid it. The exact cause of postoperative blindness in prone patients is unknown but seems to be related to hypoperfusion (anemia, hypotension, low oxygen saturation) and direct pressure on the eyes. Therefore, we use special padded pillows for the face with spaces around the eyes and lips to avoid direct pressure. The Prone-view mask offers these benefits as well as having a mirrored faceplate to allow direct observation of the face. As a general rule we try to keep the patient’s BP within 20% of their baseline, their hematocrit above 30%, and their saturation as high as possible. Thus, an arterial line is very useful in prone spine cases and is almost always indicated.

The prone position also has implications for airway management and every one of our monitors and lines. The airway must be doubly secured in the prone position because if it is lost it would be next to impossible to resecure it in the prone position. One cardinal rule of prone cases is that the patient’s stretcher 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- it is next to impossible to set some things to right in the prone position. These efforts must be made while the patient is still supine, before the patient is turned.

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
- disconnect BP cuff, EKG leads +/- arterial line and hang up neatly for immediate reconnection
- disconnect and cap off all but one IV line
- ventilate with 100% O2, disconnect pulse oximeter and breathing circuit
- flip patient, reconnect circuit, confirm ventilation
- reattach monitors and IV lines

**Spine surgeries with significant blood loss**

Technique: general anesthesia

Monitors: standard, plus arterial line, strong possibility of central line. Urine output.

IV access: large access needed as significant fluid resuscitation/transfusion is likely. At the minimum, 1 large (16G plus) IV. Often one large peripheral IV plus a cordis for massive transfusion.

Duration: anywhere from 1- over 16 hrs depending on the surgeon and the case.
Estimated blood loss: several hundred cc’s to many liters.

Position: prone, supine for anterior cases.

Special equipment: prone face protection, fluid warmers for IV lines, forced-air warming blankets (potential for significant heat loss). One lung ventilation for anterior thoracic spine procedures.

Special considerations:

Blood loss and resuscitation is really the key factor here. Stay on top of blood loss and give fluids liberally to maintain normal blood pressures. Invasive monitoring, urine output and frequent ABGs can help guide therapy. Pay attention to all the potential negative sequelae of massive transfusion, which includes coagulopathy, hypothermia, reaction to blood products, and electrolyte disturbances.

Rarely, a surgeon might request an intraoperative “wakeup test” which is exactly what it sounds like. Typically, this is done in cases where there is strong possibility of neurologic damage such as tumor removal, and the surgeon wishes to perform a neurologic exam intraoperatively to confirm nothing untoward has happened. This must be established with the anesthesia provider beforehand, since we must tailor our anesthetic to allow for this. The patient should be told beforehand to expect this during the surgery to keep anxiety to a minimum. When the time for wakeup nears, the patient should not have neuromuscular blockade on board and the anesthetics should be discontinued. It may be helpful to employ short-acting agents if this is anticipated. Thankfully this test is rare.

More commonly, the surgeons will employ evoked potentials to monitor for impending neurologic damage. The choice of evokes (sensory, motor or both) influences the type and amount of anesthetic agent we can use. For example, if motor evoked potentials are to be employed we cannot completely paralyze the patient. Typically the evoked potential personnel (generally outside technicians) 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 spine is generally done by another surgical team (e.g., trauma or vascular). If the procedure has both anterior and posterior elements at the same time, 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 often need one-lung ventilation to allow the surgeons to have adequate exposure to the spine. The techniques of one lung ventilation will be discussed in the cardiothoracic anesthesia section. Anticipate if there will be a need for one-lung ventilation and choose an appropriate ETT from the outset of the case.

**Spine surgeries without significant blood loss**
Technique: general anesthesia; sometimes MAC for minimally invasive or percutaneous procedures

Monitors: standard, +/- arterial line

IV access: usually one IV

Duration: 1-8 hrs

Estimated blood loss: usually less than 500cc

Position: prone, supine for anterior cases.

Special equipment: prone face mask

Special considerations:

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 dependent on the duration of the case and patient comorbidities because the same issues of blindness exist.

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. Also, 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.

The orthopedic surgeons tend to do their minimally invasive or percutaneous procedures under MAC/local. Here, there is little for us to do other than standard monitors and to ensure the patient is comfortable. The awake patient can warn the surgeon if the procedure is too close to a vital structure such as a nerve root.

II. Orthopedic lower extremity procedures

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 pilon fractures). In these cases we do not place blocks at the request of the surgeons, to avoid compromising their ability to monitor for this syndrome. We routinely place peripheral nerve blocks in certain types of 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 itself.
**Hip surgery**

Technique: general or neuraxial (might need continuous technique given duration of surgery).


IV access: one large IV.

Duration: 3hrs for first time operation, potentially double for redo or complicated hip.

Estimated blood loss: usually < 1000 cc, although significant blood loss can go unnoticed within the joint and surrounding structures preoperatively. Redo operations can involve much more blood loss.

Position: lateral, operative side is up.

Special equipment: none.

Special considerations:

- 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.

- The regional team will often place lumbar plexus blocks on the operative side for postop pain control. These are not necessary for the surgery to proceed.

- A great deal of time often passes between induction of anesthesia, and actual positioning, preparation and incision. I personally have witnessed over 1 hr delays between “anesthesia ready” and “incision” times. Be aware of this, and take efforts to keep the patient warm. A large amount of body surface area becomes exposed prior to draping, and sometimes it seems like the patient is just sitting there, getting colder by the second.

- Risks of DVT and reaction to the cement.

**Total knee replacement or arthroplasty**

Technique: general is preferred. The surgeons mobilize the leg and make a lot of noise during the surgery which may bother the awake patient. Furthermore the procedure can be quite lengthy. Post op pain is significantly higher than for hip replacements. Peripheral nerve blocks are almost always placed preop for pain control and rehab- typically a single
shot sciatic block and a continuous femoral nerve catheter. These can significantly lower intraop anesthetic requirements.


IV access: one large IV.

Duration: 3-4 hrs, longer for redo or repeat procedures.

Estimated blood loss: < 500cc; a thigh tourniquet is often employed.

Position: supine.

Special equipment: none.

Special considerations:

   Risks of reaction to cement, use of a tourniquet and risk of DVT.

Other lower extremity procedures

Examples include-
   - knee arthroscopies
   - joint or fracture repair
   - amputations
   - 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 arthroscopies because they will soon go home, and would be unable to bear weight safely on a “blocked leg”. A tourniquet is often employed (see above under total knee arthroplasty).

Technique: general, neuraxial, or regional. Sciatic, popliteal, femoral or ankle blocks as applicable.

Monitors: standard.

IV access: one IV will generally suffice.

Duration: 1-4 hrs.

Estimated blood loss: < 200cc 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.

III. Orthopedic upper extremity procedures

Extensive shoulder surgery (joint replacement, reconstruction)

Technique: general anesthesia. Interscalene block often placed for post op pain control.

Monitors: standard.

IV access: one large IV.

Duration: 3-4 hrs.

Estimated blood loss: usually less than 500cc.

Position: beach chair (sitting), sometimes supine 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 to the table well, appropriate padding must be placed, and the airway should be doubly secured because access will be limited intraoperatively.

Clavicular surgery

Technique: general anesthesia. Blocks usually difficult due to highly proximal nature of surgery.

Monitors: standard.

IV access: one large IV.

Duration: 3-4 hrs.

Estimated blood loss: usually less than 500cc.
Position: supine.

Special equipment and considerations: none.

**other upper extremity procedures**

Examples include-
- humeral fractures
- elbow surgery
- radial and ulnar surgery
- nerve transposition
- hand surgery- from minor to extensive

Technique: general or regional- interscalene, infraclavicular, axillary or more distal blocks as applicable, +/- continuous catheter for longer post op pain control. 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 dupytren’s contracture release can often be done under a Bier block.

Monitors: standard.

Duration: 30min – 4 hrs.

Estimated blood loss: usually < 100 cc (often tourniquet).

Position: supine. The arm may be individually positioned.

Special equipment and considerations: tourniquet, possible outpatient status.

**I+D, skin graft procedures**

Incision and drainage procedures of previously infected sites are very common procedures. Often these cases will also employ wound vacuums or skin grafting from another site. In general they are minor procedures.

Technique: general or regional. Skin grafting from another site may make regional anesthesia a poor choice.

Monitors: standard.

Duration: 30min – 4 hrs.

Estimated blood loss: minimal to < 500cc.

Position: typically supine, unless I+D of the back, decubitus ulcers, etc.
Special equipment and considerations: possible tourniquet.
Anesthesia for urologic/gynecologic surgery

Urologic and gynecologic surgery comprises a significant percentage of cases performed at UCSD. Although they are two 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 surgery (e.g., laparoscopic/robotic procedures, major pelvic dissections) and for further information the general surgery section should also be reviewed.

One 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 alternatively 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 thigh can result in common peroneal nerve injury and foot drop. Flexion of the thigh can also produce sciatic, femoral and obturator nerve injuries. Although not specifically our domain, as the primary perioperative physician we are also responsible to ensure these areas are properly padded and supported.

The lithotomy position also creates increased intraabdominal pressure, leading to a loss of lung volumes and functional residual capacity. Many of these procedures also employ steep trendelenburg which further increases these problems, in extreme cases leading to difficulty with ventilation and oxygenation.

The intraoperative use of indigo carmine or methylene blue is common in many of these procedures. The dyes when given intravenously 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 these days can create artifactual decreases in saturation and even transient hyper or hypotension.

The TURP syndrome is a commonly asked question on the boards and something every anesthesiologist needs to be aware of. It classically is seen during TURPs (transurethral resection of the prostate) hence its name but in essence can occur any time large amounts of irrigating solutions are used. Overabsorption of hypotonic fluid leads to fluid overload, hyponatremia, hypoosmolarity and solute toxicity. The amount of absorption is determined by pressure of the irrigation fluid (the higher the solution is hung, the more pressure), opening of vascular structures (e.g., the venous sinuses of the prostate), and the length of the procedure (absorption reported to occur at ~ 20ml/min of the procedure). The problems described above can manifest themselves as confusion, agitation, hypotension, and arrhythmias all the way to dyspnea, coma and death. Clearly, most of these signs are masked by general anesthesia, and for this reason many of us feel neuraxial blockade is safer any time TURP syndrome is a possibility.
For conductivity reasons (surgical electrical instruments) electrolyte solutions cannot be used. Common hypoosmolar solutions include glycine, sorbitol and mannitol. 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.

The treatment of TURP syndrome is supportive. Usually volume restriction and diuresis are sufficient. Severe neurologic manifestations such as seizures or coma should be treated with hypertonic saline according to standard guidelines, with care not to correct hyponatremia too quickly. 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- release of kinases from bacteria within the prostate and urinary tract and in extreme cases from dilution by massive volume overload.

Lastly, sepsis is a possibility from any urologic procedure, due in large part to the release of gram-negative bacteria into the bloodstream.

Urologic/gynecologic procedures can be broken in minor, moderate and major categories.

**Minor procedures**

Examples of these include TURPs, cystoscopies, hysteroscopies, 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 to 2 hrs.

Estimated blood loss: < 100cc (exception is D+C, D+E, still probably < 500cc).

Position: lithotomy or supine, trendelenburg.

Special equipment: none.
Special considerations:

TURP syndrome was previously discussed. Often a spinal anesthetic is the best choice to allow for an awake patient and monitoring of changing neurologic status. ESWL’s employ high frequency acoustic shocks which can damage or reset pacemakers. Discuss with an attending/pacemaker representative on how to proceed.

Many of the 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. No special precautions are necessary but to be aware this can occur (the gynecologists will inform you if this happens). At that point additional interventions may become necessary. The gynecologists may sometimes ask for specific agents to aid in uterine contraction, e.g., carboprost. These agents and anesthetic implications for the parturient are discussed in the OB section.

**Moderate procedures**

Examples include hysterectomy +/- oophorectomy, vaginal vault reconstructions, ureteral slings, simple nephrectomy, and major laparoscopic/robotic procedures such as laparoscopic prostatectomy, laparoscopic oophorectomy.

Technique: almost always general anesthesia due to length and open nature of most procedures.

Monitors: standard. Arterial line for long cases.

IV access: typically one IV will suffice.

Duration: anywhere from 2-8 hrs. Robotic prostatectomies were notorious in the past for taking eight hrs (or longer) but the primary practitioners are improving.

Estimated blood loss: 100 – 500cc, rarely more.

Position: supine, lithotomy, steep trendelenburg.

Special equipment: warmers are often indicated due to length of the procedure.

Special considerations:

IV dye such as methylene blue may be asked for (see above).
Long laparoscopic or robotic procedures are also discussed in the general surgery section. Frequent checking of ABGs may necessitate an arterial line. The combination of steep Trendelenburg, insufflation of the belly, and long surgical times can create significant pulmonary and ventilation issues as discussed above. Furthermore, airway edema from this position may necessitate post op intubation.

Several of our laparoscopic surgeons are notorious for asking for the patients to be “run dry” and for IV fluids to be restricted. The rationale behind this is unclear— one overheard reason was to “keep urine at a minimum to improve visualization in the field”. This seems like poor reasoning, inducing oliguria or anuria to improve operative conditions. While it is true that blood and insensible losses in minimally invasive 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. When in doubt be political and defer to your attending, but keep in mind your duty is first and foremost to the patient. Several recent morbidity and mortality conferences have focused on this issue.

Major procedures

Examples include major pelvic dissection, radical nephrectomy, prostatectomy, or cystectomy, pelvic lymph node dissection, and typically any surgery for gynecologic or urologic cancer.

Technique: general anesthesia. An epidural may be indicated and asked for to manage postop pain in higher abdominal procedures.

Monitors: standard, arterial line, +/- CVP.

IV access: at least 2 large IV’s.

Duration: 4-12 hrs.

Estimated blood loss: 500 – 3L, possibly more.

Position: supine, back extended, or thoracoabdominal approach. Often Trendelenburg.

Special equipment: fluid and body warmers, transfusion likely.

Special considerations:

These cases should be treated like major abdominal cases as outlined in the general surgery section, with regards to blood loss, fluid management and physiologic consequences. Blood loss can be insidious and should be closely monitored.
Most of the positions have similar pulmonary consequences as the lithotomy position.

IV dye such as methylene blue may be asked for (see above).

Many of the same comments regarding fluid restriction made above are relevant to these cases (the same surgeons are often asking for the same thing). It is obviously even more important to ensure that these cases, with their major blood losses and fluid shifts, are resuscitated appropriately.

Renal tumors are often in close proximity to major vessels including the IVC. Compression or retraction can decrease venous return and cause hypotension. Rarely, a renal cancer may present with tumor extending into the IVC or even the heart. In these situations cardiopulmonary bypass and a joint procedure with the cardiothoracic surgeons is indicated. Clearly, the anesthetic management of these cases is profoundly different. See the CT anesthesia section for more information. These cases are rare and will be discussed beforehand in a multidisciplinary approach, so you should have time to prepare for them when they arise.
**Anesthesia for vascular surgery**

Vascular surgery patients are some of the most ill people we regularly take care of. There is often presence of other serious disorders which both cause the vascular disease and complicate the clinical picture. Examples of these diseases are diabetes, hypertension, renal failure and a long history of smoking and COPD to name a few. These comorbid conditions create serious conditions such as heart disease or cerebral-vascular disease. A careful and detailed preoperative workup is necessary. Often these patients are in their “best medical state” when they present for surgery.

Common intraoperative issues seen with vasculopaths include the tendency for very labile BPs. Preinduction they may be normotensive, upon induction they may become radically hypotensive, and after intubation or surgical stimulation profoundly hypertensive. These wild swings can be tough to manage and often occur despite our best efforts.

Another issue is difficulty in obtaining 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, local infection or the presence of a fistula which makes securing IV access even more difficult. Similarly, their arteries can be quite calcified or have had prior instrumentation and present a challenge in obtaining an arterial line. Unfortunately it is precisely this class of patient which most often needs an arterial line.

**Minor vascular procedures**

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

Technique: most AV fistulas and angiograms are done under local/MAC which is well-tolerated by patients although GA, neuraxial or regional blocks all are potential options.

Monitors: standard usually suffices.

IV access: one IV.

Duration: 1-2 hrs.

Estimated blood loss- minimal – 300cc.

Position: supine.

Special equipment: none.
Special considerations:

I+Ds and amputations are also discussed in the orthopedic section.

Patients in renal failure needing a fistula are typically predialysis, or have an indwelling dialysis catheter. Ensure medical optimization prior to surgery. That being 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 < 6 and no EKG changes. Recent trends in the patient’s potassium can be helpful in deciding how to manage a high potassium.

If the indwelling catheter is to be used, you must be aware that it contains concentrated heparin to avoid the catheter clotting off. The dialysis catheters should probably be avoided altogether because of risk of infection and compromising the line but sometimes this is not possible. If it is to be used, the heparin must first be withdrawn and discarded. Aspirating from the catheter until undiluted blood appears is the best way (over two times the dead space in the catheter). Similarly, when finished using the catheter must be refilled with heparin. Consultation with your attending, a nephrologist or the vascular surgeon can be helpful.

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 a completely responsive, awake patient is required. Our lines and monitors must also have significant length and be tangle-free to allow free motion of 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 procedures**

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

Technique: almost always general. Peripheral vascular 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 be done under MAC/local, but possibility of catastrophic rupture and need for emergency GA makes this a poor choice in the former, and our surgeons have very little experience with MAC/local for the latter (See more below).
Monitors: standard, usually an arterial line for bypasses (sometimes not necessary), always for CEAs and endovascular AAAs. Urine output. EEG for CEAs. Possibly a cordis or CVP for endovascular AAA repair (see below).

IV access: at least one large IV. Blood loss should be small for these procedures but has the possibility of developing rapidly.

Duration: 3-6 hrs.

Estimated blood loss: typically <100cc 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 Activated Clotting Times (ACT) due to frequent heparin administration. Warmers.

Special considerations:

In endovascular AAA repair the surgeon attempts to place a stent within the diseased portion of the aorta, shoring it up and preventing further growth/rupture of the aneurysm. This procedure employs angiography and the same table considerations apply as for angiograms (see above). 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 proceed rapidly to an open one in cause of a catastrophic event. To this end, some people also place a large central line (cordis) for use in case of rupture. This is usually not necessary. A ruptured or open AAA repair is a much different procedure (see below).

There are three basic goals in our anesthetic management of CEA- keeping the patient normotensive (or even supranormal), monitoring for neurologic ischemia, and tailoring our anesthetic for a quick wakeup to allow neurological checks. Maintaining the BP at baseline levels is important because the presence of carotid disease also implies disease within the cerebral vessels. Crossclamping of the operative carotid makes some of the areas of the brain dependent on collateral flow for perfusion. This flow is dependent on a sufficient pressurehead (in other words, the systemic BP). To this end, most anesthesiologists employ a phenylephrine infusion from the outset of the case to allow rapid titration and adjustment of BP. A nitroprusside infusion can be used to rapidly lower dangerously high BPs but is usually not necessary. Such minute-to-minute changes and adjustments necessitate an arterial line.

There are several ways to monitor for neurologic ischemia. The first and perhaps best way is to keep the patient awake, who can then act as his own monitor for neurologic insult. Unfortunately, our surgeons are not used to 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. In general, large reductions in electrical activity from a baseline, stable anesthetic, especially unilateral on the operative side, are worrisome for ischemia and the surgeon should be notified. At times the EEG is not employed (dependent on both the surgeon and the attending anesthesiologist). Other methods to maintain or monitor cerebral perfusion employed by the surgeon are creation of a temporary shunt to bypass the operative site and maintain flow, and measurement of “stump pressures” which is theoretically a measurement of collateral pressure and thus flow.

A quick, smooth wakeup allows serial neurologic checks to begin immediately, and reduces coughing and bucking which can put undue strain on a freshly-incised artery. To this end, many of us employ techniques which allow for this. One common method is to use a nitrous oxide-inhalational anesthetic (preferably desflurane)-fast opioid technique (often remifentanil, see the drug section for more information). All of these agents can be discontinued immediately before surgery ends with a predictably fast recovery. Remember that remifentanil offers no analgesia after it is discontinued, thus a longer-acting opioid should be given before the patient wakes up. Abrupt discontinuation of these agents with no transitional narcotic can result in an agitated emergence which is precisely what we try to avoid.

Monitoring of BP is continued in the post op period, with the goal being to avoid excessive hypertension (freshly-incised artery). Patients who have had CEA on both sides are prone to effects from denervated carotid bodies, namely hypertension (lack of baroreceptor reflex) and loss of hypoxic drive. The loss of hypoxic drive can be particularly devastating because many of these patients have COPD and baseline hypercapnia, and thus already have a blunted respiratory response to hypercapnia.

**Major vascular procedures**

Examples include open AAA repair, aortic, iliac or mesenteric bypasses. For the purposes of case logs the more peripheral bypass surgeries (e.g., fem-distal) are also considered “major vascular procedures”. I simply use the term and separate them here because the anesthetic management and implications can be very different.

Technique: general anesthesia. Thoracic epidural often employed for postoperative pain which can be severe.

Monitors: standard, arterial line, CVP, often a PA catheter. Urine output.

IV access: at least two large IVs.

Duration: 4-8 hrs.
Estimated blood loss: 500cc to many liters depending on duration and intraoperative outcomes.

Position: supine. Potentially thoracoabdominal approach for higher aneurysms.

Special equipment: warmers are mandatory, transfusion exceedingly likely. Equipment to check ABGs, Activated Clotting Times (ACT) due to frequent heparin administration. Rarely, a spinal drain to remove CSF (see below). Tube for one-lung ventilation may be necessary depending on location of the aneurysm.

Special considerations:

A large open AAA repair can be one of the most labor-intensive and difficult cases we do. There are many considerations, some of which are also covered in the cardiac anesthesia section. Consult a more 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 which is the “worst case scenario”.

Broadly, there are three types of AAAs- infrarenal, suprarenal and supraceliac, defined by location of the aneurysm. In that order, the operation becomes longer, more difficult, with more blood loss and more postoperative sequelae (gut ischemia, renal failure, coagulopathy to name a few). All AAA repairs have the potential for major blood loss, massive transfusion and the inevitable problems which result.

Crossclamping and unclamping of the aorta represent major physiologic changes to the patient and are key “moments” in the surgery. Crossclamping the aorta effectively “shuts off” blood flow to everything distal to the clamp. The patient’s whole blood volume is now distributed among a much smaller area, and the bypassed areas no longer contribute their normal capacitance or “pressure sink”. Thus, hypertension and volume overload can be profound. Therefore, a rapid, titratable vasodilator like nitroprusside is mandatory and should be started prior to crossclamp. Myocardial ischemia or CHF can result from increased ventricular workload and volume. Crossclamping by its nature produces ischemia distal to the clamp. Not surprisingly, renal failure and mesenteric ischemia are common when the clamp is placed above the respective arteries. Warmers below the level of the clamp should be turned off prior to clamping (e.g., lower body Bair hugger). Constant warming of ischemic extremities can literally cook that tissue.

Clamping of higher aneurysms may compromise spinal cord circulation from the anterior spinal artery (artery of Adamkewicz) with devastating neurologic injury and paraplegia. Often there is little that can be done other than to limit ischemic time, maintain perfusion/oxygenation and “hope for the best”. Rarely, a spinal catheter drain may be placed preoperatively to remove CSF. The rationale for this is that reduction of CSF, and thus spinal canal pressures may improve perfusion pressures. This is a rarely employed technique.
Unclamping of the aorta essentially entails the opposite physiology as when the clamp is placed. Total body capacitance is markedly increased and relative intravascular volume can drop precipitously. Compounding these issues is that reperfusion of previously ischemic limbs and organs washes out an enormous acid and potassium load into the central circulation (products of anaerobic metabolism and ischemia). All of these factors can result in profound hypotension, hyperkalemia, arrhythmias and even arrest. To prepare for this event we employ a quick-acting, titratable potent vasoconstrictor such as phenylephrine, and load the patient with volume as tolerated, titrating to vitals, CVP and wedge pressures from the PA catheter. Sodium bicarbonate and calcium are used as needed. Nitroprusside is discontinued for obvious reasons. Often, repair of an aneurysm involves anastomosis of graft to both iliac or femoral vessels, and in these cases the surgeons may unclamp one side at a time to allow less dramatic changes and for us to sequentially manage the situation. The clamp can also be partially unclamped to achieve this same effect.

The same intraoperative fluid shifts and insensible losses as with any large abdominal case apply (see the general surgery section).

If the aneurysm is proximal, the surgeon may need one-lung ventilation for effective exposure. This situation needs to be discussed from the outset. One of our surgeons is notorious for requesting a double-lumen tube and then not needing one-lung ventilation at all. Remember, it is easier to place a tube for one-lung ventilation after induction than intraoperatively.

The massive fluid shifts, blood loss and transfusion, and duration of the case may make post-op intubation and ventilation necessary.

A thoracic epidural can be invaluable in managing post-op pain and reducing pulmonary complications. The size and nature of the incision predisposes patients to splinting, atelectasis and ineffective ventilation if pain is not managed appropriately. These patients are also often the least tolerant of compromised pulmonary function. The thoracic epidural is generally placed preoperatively to confirm safe placement and functioning.

The basic mindset and approach to an open AAA repair should be the same as with any large case- that being, to stay on top of everything, and to keep everything normal as much as possible. Playing catch-up and performing the anesthetic in a reactionary, as opposed to proactive nature invariably results in falling behind and major physiologic perturbations.
Anesthesia for ENT and ophthalmic surgery

These surgeries are grouped together for one reason - they occur in close proximity to the airway, necessitating close communication between the surgeon and anesthesiologist. Few other types of surgeries combine the unique demands and challenges presented by these cases. It is absolutely essential to maintain a close relationship with the surgeon to ensure optimal patient care. Other unique issues are also discussed below.

Often times during these surgeries the surgeon is closer to the airway than the anesthesiologist. The head is usually away from us, making airway interventions both harder to detect when necessary and more difficult. Further compounding this issue is the airway is either in the surgical field, covered with drapes, or under constant manipulation by the surgeon. All of these contribute to the need for both communication with the surgeon, and establishing a doubly-secured airway from the outset to reduce potential future complications. A longer circuit is often necessary to reach from the anesthesia machine to the patient.

Many ENT surgeries involve cancer or other obstructive lesions near the airway which may make ventilation/intubation difficult or impossible. A thorough preop airway exam and plan is needed. Awake intubation or tracheostomy may be indicated. In potentially difficult situations the surgeon should be scrubbed and ready to perform an emergency tracheostomy if necessary.

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 reside in the trachea via the nose and is thus longer. It also has a preformed angle or “bend” in it which is designed to point towards the patient’s forehead. In this way the circuit can be placed away from the operative field. Nasal RAE’s 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. Generous lubrication is used. If placed under direct laryngoscopy, the RAE can be advanced past the vocal cords using McGill forceps, or by creating a preformed curve in the tube and guiding the tube towards the trachea. Trauma and 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 away from the operative field. The natural curve of the tube is meant to reside at the teeth and direct the tube towards the patient’s feet. Oral RAE’s can be placed in all the same ways as a standard ETT.

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 ET tubes.
Thus, an MLT tube is longer (will reach the trachea in full sized adults), is stiffer and has a high volume, low pressure cuff.

Armored or reinforced ETT's 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).

ENT surgeries routinely employ 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. Eye protection is mandatory for personnel and the patient (simply closing the eyes suffices). To prevent an airway fire, the following maneuvers are employed-

- FiO2 as low as the patient can tolerate, preferably 21%.
- Nitrous oxide can also support combustion and should be avoided.
- 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 rays, bronchoscopy, ABGs and lavage may be needed.

Surrounding structures are also at risk of fire, including the drapes around the patient’s head.

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. Attempting to placate the surgeon while still acting in the patient’s best interests can be extremely challenging and frustrating. There is no set answer for this problem and specific actions are usually done on a patient-to-patient basis. 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 from the surgeon, and repeated requests for deep extubations in patients with significant aspiration risk. Remember that our duty is to the patients, and that you can always defer to your attending.

I. Eye surgery

In general, most eye procedures are relatively non-invasive. Postoperative pain tends to be minimal. However, post-operative nausea and vomiting is a significant problem. Most patients should probably 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 decreased venous return, increased venous pressure
- increased systemic pressure (modest effect)
- hypoventilation
- succinylcholine (modest effect)
- topical anticholinergics, which decrease aqueous drainage from the eye, thereby increasing pressure. IV anticholinergics seem to have little to no effect.

Ophthalmologists sometimes place gas bubbles in the eye to assist with healing and immobility of intraocular structures (e.g., detached retina). These gas bubbles will avidly take up nitrous oxide, potentially leading to disastrous increases in intraocular pressure. Therefore, N2O is to be avoided before placement of a gas bubble (sulfur hexafluoride is most common) and 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 arrest 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
- anticholinergics as necessary
- deepening anesthesia or retrobulbar blocks if persistent

Minor eye surgery
Examples include cataract removal, vitrectomies, blepharoplasties

Technique: usually topical or local/MAC. Some patients may require general anesthesia. Retrobulbar or facial nerve blocks are also a possibility (usually performed by the ophthalmologist).

Monitors: standard. IV access: one IV. Duration: 30min – 2 hrs.

Estimated blood loss: minimal.

Position: supine, head away from anesthesiologist.

Special equipment: none.

Special considerations:

Oculocardiac reflex, often outpatient surgery, sharing the airway.

Major eye surgery

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

Technique: usually general anesthesia. These surgeries are more extensive, longer in duration and often require a completely still surgical field. Retrobulbar or facial nerve blocks are also a possibility (usually performed by the ophthalmologist).

Monitors: standard.

IV access: one IV.

Duration: 1-4 hrs.

Estimated blood loss: minimal to 100 cc.

Position: supine, head away from anesthesiologist.

Special equipment: none.

Special considerations:
Oculocardiac reflex, sharing the airway, PONV.

Ruptured globes are open-eye injuries and the goal is to reduce 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). Avoid PONV and its associated increases in intraocular pressure.

**ENT surgery**

ENT surgeries can be broadly classified as endoscopies, minor and major surgeries.

1. **endoscopies**

Examples include microdirect laryngoscopies, panendoscopies, or bronchoscopies.

**Technique:** general anesthesia is needed along with profound muscle relaxation. Unfortunately these procedures are often quick and done as outpatients, making quick emergence and turnover a priority.

**Monitors:** standard.

**IV access:** one IV.

**Duration:** 30min -1hr.

**Estimated blood loss:** minimal.

**Position:** supine, **head away from anesthesiologist.**

**Special equipment:** possibilities- laser tube, MLT tube, oral RAE, armored tube. Circuit extension for airway.

**Special considerations:**

- Use of a laser, sharing the airway, instrumentation near the endotracheal tube.
- Preoperative presence of potential difficulties with ventilation/intubation (e.g., polyps, cancer). A thorough airway exam and plan is needed. Postoperative airway issues from surgical instrumentation and secretions.
- Profound paralysis is often needed to ensure a motionless surgical field. Repeated small doses or an infusion of succinylcholine is an option, bearing in mind the possibility
of 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.

Wide swings in BP are common, due to the dramatic differences in levels of stimulation throughout the case.

II. Minor procedures

Examples include nasal surgery, ear surgery, oral surgery (including mandible ORIF), thyroid and parathyroid surgery. Tracheostomies are a relatively benign procedure but are often performed on patients with significant comorbidities.

Technique: general anesthesia.

Monitors: standard.

IV access: one IV.

Duration: 1-4 hrs.

Estimated blood loss: generally < 300cc.

Position: supine, head away from anesthesiologist.

Special equipment: possibilities- laser tube, oral RAE for nasal procedures, Nasal RAE for oral procedures, armored tube. Circuit extension for airway.

Special considerations:

- PONV, need for smooth emergence, sharing the airway. Preexisting airway issues which may make ventilation/intubation difficult (e.g., nasal obstruction from polyps, blood in oropharynx from mandibular fracture).

- ENT surgeons will often ask for hypotension to decrease intraoperative bleeding. Similar to fluid restriction as discussed in the uro/gyn section, this can be provided 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, and systemic effects of this (hypertension, tachycardia, arrhythmias) should be watched for.

- 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 aspiration. The stomach should be suctioned prior to emergence. Oropharyngeal packs are often
employed to soak up debris. These fluids are a primary reason while 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. Thus nitrous oxide is best avoided.

Patients with a mandibular fracture may also have a basilar skull fracture which is a contraindication to placing a nasal ETT. Recognize this possibility and confer with the surgeon as needed.

In contrast to general surgeons, ENT surgeons often perform thyroidectomies and parathyroidectomies with the head away from us.

The following is how a tracheostomy over an indwelling ETT is typically performed. Surgery proceeds until the surgeons are close to the trachea. At this point FiO2 is reduced to < 50% if possible to decrease the risk of fire. The cuff of the ETT is deflated to avoid puncture by the surgeons. From this point on, ventilation may be difficult or impossible due to large leaks. The trachea is entered and the ETT is pulled back just above the incision. The tracheostomy tube is placed, the circuit is connected to it, and ventilation is confirmed. The ETT can then be removed.

Depending on the speed of the surgeon and the patient’s status, significant desaturation can occur. If necessary the surgery can be stopped and the ETT moved distal to the incision and cuff reinflated for further ventilation.

III. Major ENT surgery

These cases include extensive maxillofacial reconstruction, tumor resection, laryngectomy, pharyngectomy, radical neck or face dissections, +/- free flap from the chest or other location. They are sometimes done in conjunction with plastic surgery.

Technique: general.

Monitors: standard, plus arterial line. Urine output. CVP may be useful.

IV access: at least one large IV, preferably more.

Duration: anywhere from 4-12 hrs.

Estimated blood loss: 500cc – 2L depending on extent of surgery. Most tend to be on the low side.

Position: supine.
Special equipment: facial nerve monitors often employed (see below). RAE or armored tubes possible. Warmers. Long circuit.

Special considerations:

Sharing 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 times because of proximity of disease/surgery to the airway the patients 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 ENT surgeons may monitor the facial nerve to avoid damage from dissection. They will provide this equipment, but in these situations muscle relaxation cannot be used.

Dissection around the carotid sinus can cause bradycardia and other arrhythmias as well as wild swings in BP. Treatment is supportive and cessation of surgical manipulation. Bilateral neck dissections in this area can result in denervated carotid bodies which results in loss of hypoxic drive and baroreceptor regulation of hypertension (common board question, also see carotid endarterectomy in the vascular section).
Anesthesia for organ transplantation

The most commonly performed organ transplantations at UCSD are kidney and liver transplants. Pancreatic transplants are uncommon but will be considered here. Anesthesia for organ donation will follow at the end of this section. Heart and lung transplants are done rarely and will be addressed in the cardiac anesthesia section.

The most important factor to consider in transplant surgery is 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, hypertension and anemia. Excretion 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 we take care of in anesthesia, and a liver transplantation is possibly the largest, most labor-intensive case we do. To begin, many of these patients have associated renal failure. Poor or non-existent liver function predisposes patients to coagulopathy, anemia, hypoalbuminemia, ascites and a peripherally dilated, high cardiac output state. Metabolism of drugs and synthetic function are deranged. Furthermore, the case itself involves significant blood loss, fluid shifts and major physiologic perturbations which will be considered in the specific section.

Associated disease, either the cause (e.g., diabetes, hepatitis C) of or a result of the organ failure 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 livers the window is generally 12 hrs and for kidney transplantations this is generally within 24 hrs (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 (excepting living-related kidney donations/transplant).

Living related kidney donation

Technique: general anesthesia.

Monitors: standard.

IV: one IV should suffice.

Duration: 2-4 hrs.
Estimated blood loss: less than 500 cc.

Position: supine or “airplaned” for laparoscopic harvests.

Special equipment: mannitol, heparin, protamine.

Special considerations:

Living related kidney donors are typically otherwise healthy patients and the harvesting 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 is being harvested just before the recipient is ready for implantation (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 will be reversed with protamine at the appropriate time. See below for more information.

Kidney recipient (living related or cadaveric)

Technique: general anesthesia.

Monitors: standard. Rarely, an arterial line is necessary. CVP placement was once routine but is now felt to not be necessary. Urine output.

IV: one large IV should suffice.

Duration: 3-4 hrs.

Estimated blood loss: less than 500cc.

Position: supine.

Special equipment: mannitol, lasix, solumedrol or other immunosuppressive (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 handling of volume. A CVP used to be routine but has been largely abandoned due to minimal utility.
Prior to anastomosis of the kidney to the iliac vessels heparin will be asked for. Depending on the duration of anticoagulation protamine may needed to be administered. Protamine must be given slowly (in small increments, 10-50mg per minute) to lessen the chance of a serious allergic reaction, hypotension or unacceptable pulmonary vasoconstriction. See the cardiac section for more information.

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. Heme in the new urine flow is common. Overt blood should be brought to the surgeons’ attention.

Solumedrol 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.

**Liver transplantation**

Technique: general anesthesia.

Monitors: standard, arterial line (often a femoral A-line as well, see below), CVP, PA catheter and/or transesophageal echo. Urine output.

IV access: as much as possible. Routine lines for a liver transplant include one peripheral IV, a cordis (9Fr central line), and a triple lumen Edwards catheter (central line with 3 large bore components). Rapid infusion catheters can also be used (essentially placing a large bore catheter in a peripheral vein using the Seldinger technique).

Duration: 6-10 hrs.

Estimated blood loss: **large.** There is no maximum. 100+ unit transfusions are not uncommon, while low blood loss/transfusion cases are rare.

Position: supine.

Special equipment: TEE, warming blankets and warm IV lines, thromboelastogram (see below), presence of a perfusionist to aid with massive transfusion.

Special considerations:

Liver patients should be regarded as “full stomachs” due to extensive abdominal distension/pressure and often recent GI bleeding, and thus should get a rapid-sequence induction with cricoid pressure.
Due to peripheral vasodilation throughout the case, caused by both the patient’s baseline disease and acidosis/hypocalcemia at critical portions, a radial arterial line may not accurately reflect the patient’s central BP’s. For this reason a femoral arterial line is often placed in addition 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 sooner 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.

A thromboelastogram or TEG is invaluable at helping us assess coagulopathy intraoperatively and guiding treatment. A TEG requires a special machine, is run by hematology and requires at least 1 hr notification prior to use. For this reason, it is typically employed by us only for liver transplantations. The TEG essentially functions in this way- a blood sample (3cc) is sent to the lab and placed in a special holder. A rod is then placed in the blood and set to motion. As the blood clots the viscosity, velocity and other characteristics change which translates to different motions of the rod, which are detected by the machine. Many variables are provided, but the ones we are most interested in are-

1. The R value- time to first clot formation, generally reflective of overall clotting function. Low R values are generally treated with FFP.
2. The maximum amplitude- reflective of platelet presence and function, and is treated by such.
3. the angle- low values reflect inadequacy in fibrinogen, and are generally treated with FFP or cryoprecipitate.

TEG values are given after 30 min and 1 hr of incubation. Therefore, they are reflective of a past patient state 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 help us with presenting a more linear, continuous picture.

The perfusionist is invaluable in assistance with meeting transfusion and volume requirements. The perfusion machine is capable of delivering very high flows of almost any solution (notable exception is platelets). Typically we provide the perfusionist with two of our large lines, generally the two smaller bore lines on the triple lumen catheter. In this way, the perfusionist can maintain high flows without preferential flow through one line. Working with the perfusionist requires a degree of communication and for the anesthesiologist to know “what he wants” when he talks to the perfusionist. They generally will not give anything we do not ask for. Common instructions to the perfusionist are to continuously give blood/blood products in a variety of ratios and flow rates, titrating to CVP or PA pressures, or simply at intermittent intervals based on patient needs. It is generally advisable to leave all blood/product infusion 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 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- the bag of cellsaver blood has air in it, and thus unlike a bag of PRBCs 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. A full discussion of CVVHD will not be addressed here, save for its implications for our anesthesia. The nephrology team will be present in the OR throughout CVVHD, and by necessity 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 diasylate used. The nephrology team can also run the patient hypo, hyper or euvoelemic 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, reperfusion of the transplanted organ and potentially concomitant renal failure. It should be aggressively managed and is most relevant during reperfusion of the new liver (see below), but deaths have occurred from hyperkalemia even hours after the neohepatic phase. Frequent monitoring is thus mandatory and is done via serial ABGs.

Hypocalcemia also results from several mechanisms. First, 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 impaired metabolism of citrate, worsening the situation. Frequent ABGs also help us monitor this situation, and massive calcium requirements are not uncommon (over 10 grams of calcium chloride). A continuous calcium infusion is often employed.

A liver transplant has three distinct stages-

1. Pre-anhepatic- the old liver dissected out, and remains connected only via the IVC, portal vein, hepatic artery, and common bile duct. Normalization of all abnormalities proceeds.
2. Anhepatic- the time after vessels are clamped, the diseased liver is explanted to reperfusion of the new liver. The IVC may be partially or completely clamped at this stage, reducing venous return and causing 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 may be needed at this stage 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 risk of air embolism. Lack of
liver function worsens the coagulopathy (no clearing of citrate), acidosis and electrolyte abnormalities. Aggressive resuscitation continues.

3. Neohepatic- the most dramatic stage 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 physiologic perturbations not unlike unclamping of the aorta (see the vascular surgery, AAA section). Washout and reperfusion of the new liver and previously ischemic organs introduces a high acid, high potassium (products of ischemia and anaerobic metabolism, as well as a component of the preservative) cold solution into the central circulation. The hemodynamic changes (specifically, hypotension) can be profound. Acidosis and metabolic waste products cause vasodilation and a decrease in cardiac contractility as well as predisposing to arrhythmias. Hyperkalemia exacerbates this situation and can cause arrest of its own accord. The potassium serum concentration generally rises 1-2 meq/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 aware of the dangers of reperfusion)-

- **everything is normalized as much as possible prior to unclamping.**
  
- 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 a precipitate (calcium carbonate, “chalk” will form).

- These ampules are often given immediately after unclamping, knowing that hyperkalemia and acidosis are inevitable. There is no time and we do not draw labs at this point, as the clinical situation changes on literally a second-to-second basis. Titration to BP and normalizing EKG changes (if any) are the goals.

- All anesthetics are d/c’d and the patient is ventilated with 100% O2.

- Slight hypercapnia is allowed during the anhepatic phase, with the goal being normalization of pH in the setting of slight respiratory acidosis. Hypocapnia is then achieved just before unclamping. This will help with combating acidosis, providing an extra cushion of alkalosis and drive excess potassium intracellularly.

- Some practitioners routinely start a dopamine or epinephrine infusion prior to unclamping for positive intropic/vasoconstictive effects.

After the new liver has been reperfused it will typically start metabolizing waste products, as well as synthesizing proteins and factors within the hour. Coagulopathy and hypocalcemia may be dramatically reduced when this happens. It is still important for us to maintain homeostasis and check labs frequently. A slight coagulopathy is typically tolerated at this point, with the goal being avoidance of thrombosis of vessels to 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 surgical decreases this risk.
As a rule due to the massive transfusions, fluid shifts and length of the case these patients are left intubated post op and allowed to recover in the ICU, although exceptions have happened with the rare, minimally traumatic case.

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 solumedrol is given after the new organ is implanted, and will also be requested by the surgeon.

**Pancreatic transplants**

Technique: general anesthesia.

Monitors: standard, plus arterial line (useful for both the length/physiologic perturbations of the case plus checking labs); rarely, a central line.

IV access: one large IV.

Duration: 4-8 hrs.

Estimated blood loss: generally less than 500cc.

Position: supine.

Special equipment: warmers.

Special considerations:

The indication of pancreatic transplant is almost always as a cure for Type I diabetes. These patients often have evidence of other sequelae of advanced diabetes, such as cardiac and renal disease. A combined kidney-pancreatic transplant is not uncommon.

The surgical technique, briefly, is removal of the old organ, implantation of the new pancreas in the abdomen, and connection of the relevant blood vessels/secretory pathways. A large abdominal incision is used and this case should be considered a “large open belly” case with all the usual associated insensible losses (see general surgery section). The arterial supply is from a graft direct from the aorta, the venous drainage (insulin) is to the IVC or portal circulation, and the exocrine drainage is either to small bowel or bladder (allows urine measurement of amylase to monitor for rejection).

Frequent blood glucose levels should be monitored, especially after the new pancreas is implanted.
Immunosuppressives will have to be given intraoperatively and will be asked for by the surgeons (see above).

**Anesthesia for organ donation**

Technique: general anesthesia.

Monitors: standard. These patients often have additional monitors placed preoperatively for management of their pre-mortal disease.

IV access: one IV.

Duration: 1-4 hrs. Our involvement ends with cross clamping of the aorta.

Estimated blood loss: n/a, but less than 500cc for our portion of the procedure.

Position: supine.

Special equipment: none.

Special considerations:

By definition these are ASA 6, brain-dead patients. Some or all available organs will be harvested. Our goals are to maintain perfusion/oxygenation to the organs, keeping the patients normotensive and well-oxygenated. Any hemodynamic drips the patient may be on preoperatively should be continued as the situation warrants. Spinal and accessory pain pathways may still be intact, producing hypertension upon surgical stimulation. This can be treated with opioid if necessary. Remember that the patient will not “feel pain” in the usual sense.

Our involvement generally ends with cross-clamping of the aorta. The surgeons will make an incision from sternal notch to the pubic symphysis, and after some inspection/dissection the aorta will be clamped. At this point the surgeons will notify us that we are no longer needed, the ventilator can be shut off and the circuit detached from the patient. We can leave at this point as the case is done for us.
Anesthesia for trauma and burn surgery

UCSD is both a level 1 trauma center and the only regional 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.

Anesthesia for trauma surgery, or the “OR resuscitation”

Many trauma patients require immediate surgery to have a 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. For a complete description of the trauma room setup, see the “code bag and emergency room setup section”.

The algorithm employed by UCSD’s trauma protocol that determines which patients need OR resuscitation and which do not is complex. Patients which are true candidates for an “OR resus” are generally brought straight from the field, bypassing the ED and any belabored admissions process. Typically, these are patients with known or suspected trauma in the field and unstable or no vital signs. Because resuscitation often begins in the field by the first responders (EMTs), these patients will often arrive intubated and/or with IV access already established. This will be addressed later, but suffice to say that the report from the field (e.g., properly placed ETT) is often quite different than 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 team, anesthesia 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 (or other relevant room). The EMTs continue with their report while the patient is transferred to the OR table. 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, **confirm proper placement** via EtCO2 or an esophageal bulb detector.  
- Intubating a trauma patient can present many challenges. These patients by definition are full stomachs with potential cervical spine injury. Thus, rapid sequence induction with manual inline 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. Briefly, check for a pulse, blood pressure, and heart rate. ACLS should be initiated whenever indicated. Definitive treatment of hemorrhage is to identify and stop bleeding, and thus 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 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 colloids, crystalloids and blood products. There is continual debate over the superiority of crystalloids/colloids with no good answer. Whatever fluid is chosen should be isotonic and warmed. Blood products should be given whenever indicated. Type O negative (“trauma blood”) can be given while definitive type and crossmatch is performed. Often there is a level 1 rapid infuser which can be used to rapidly deliver volume.  
- 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; peripheral IV’s suffice. Central access is also desirable but should not delay the case, and establishing a central line carries increased risk to the patient.  
- 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-

Anesthesia is often a secondary priority. It can be given as tolerated but the first goal is resuscitation of the patient. Trauma victims are often so unstable as to tolerate only a small amount of hypnotic (e.g., midazolam) and muscle relaxant.

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. Make sure to keep the patient warm, as hypothermia has myriad deleterious effects, including coagulopathy, acid-base disturbances, and left-shift of the Hb-O2 curve.

The more hands available the better. 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 (e.g., recognizing the need for ACLS and initiating therapy).

Finally, because it bears repeating- try and 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.

**Anesthesia for burn surgery**

As previously discussed UCSD is a major burn center and the only center within 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.

**Anesthesia for major burn surgery**

Prototypical examples include excisional debridement, skin grafting, tangential excision, and placement of wound vacs.

Technique: almost always 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, almost always an arterial line. CVP may prove useful to guide fluid and transfusion therapy. Urinary catheter.
IV access: large. May be difficult in extensively burned patients.

Duration: can be long, up to 8 hrs. Depends on the extent of the injury and complexity of the case.

Estimated blood loss: 100cc to several liters depending on the extent of injury.

Position: typically supine or prone.

Special equipment: transport monitors, needle electrodes for EKG, warm room, fluid warmers and warming blankets. See below.

Special considerations:

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, dust) can cause rapid, life threatening edema in these patients and loss of the airway which can be impossible to recover. 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 usually 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, but at times we may be called to assist with an acute burn that does not yet possess an airway. 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 (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 9’s” can be used to estimate the BSA affected. The arms, head and anterior and posterior portions of the legs, chest and abdomen each represent roughly 9% of TBSA. The perineum is the remaining 1%.

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 Hb-O2 dissociation curve and decrease in SaO2. Burns also cause an increase in capillary permeability throughout the entire body which predisposes to pulmonary edema and ARDS. Furthermore, long periods of an artificial airway and mechanical ventilation predispose these patients to pneumonia. Adequate ventilation and oxygenation can be quite difficult. Preoperative evaluation often reveals a patient that is difficult to ventilate, with high inspiratory pressures, hypoxemia and hypercarbia that persist throughout the intraoperative period. Lastly, as healing begins a profound hypermetabolic state arises, leading to increased O2 demand and increased CO2 production, both of which place additional demands on the respiratory system.
The increase in capillary permeability described 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 (see a text for more information). After 48 hrs capillary integrity begins to be restored, and colloid will remain in the intravascular space. For this reason our burn surgeons prefer us to use albumin (or blood products) 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 (see the general surgery section).

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 necessary measure is a major reason for the onus 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.

Excision of burned tissue causes major bleeding. While often overt, the magnitude of this 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 instability/critically ill nature of most of these patients, an arterial line is often 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 (e.g., femoral or dorsalis pedis). 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 usually present courtesy of the burn service).

Succinylcholine is contraindicated in burns older than 24 hrs. See the drug section for more information.

Because these patients return to the OR frequently, it is quite possible a recent anesthesia preoperative evaluation has been done on them. Always check for previous records in the patient’s charts which can save a tremendous amount of time and provide valuable information about prior anesthetics.

“Anesthesia transport” of these patients denotes when we must physically go and bring the patient from the BICU to the OR ourselves, with continual 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 or the nurse at the bedside. Mechanical ventilation means we must either bring a transport tank of O2 with a mapleson circuit, or have a respiratory therapist accompany the patient with a transport ventilator. The latter is usually preferable as it frees our hands up to attend to other patient needs. A transport monitor is mandatory, and can either be supplied by our workroom or the BICU. Be sure and bring resuscitation drugs, an IV bag with Y-tubing for rapid infusion if necessary, and equipment for possible reintubation. These patients often have multiple infusions, delivered through a towering assembly of infusion pumps and a bewildering tangle of lines. For all these reasons it is usually helpful to call the BICU nurse about 15 min before you anticipate arriving to pick up the patient. If requested, they are generally very helpful in detangling lines, disconnecting all non-essential infusions, hooking the patient up to a transport monitor, and summoning a respiratory therapist. There is nothing sweeter than arriving at an anesthesia transport patient and finding them completely “packaged” and ready to go.

**Anesthesia for 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, but generally not necessary.

Duration: 30 min to several hours.

IV access: one IV should suffice.

Estimated blood loss: less than 500cc.

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 in the Burn IMU (as opposed to the ICU) or outpatients returning for reconstructive surgery. Anesthesia transport is thus almost never a factor.
Obstetric Anesthesia

The obstetrical service at UCSD has a high volume of procedures and deliveries. In addition, a significant percentage of its patients are “high-risk”. Because of our 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 obstetrical 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.

Rotation Structure

Residents begin the rotation midway through their CA1 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 CA1 year, with the remaining people finishing the training during the CA2 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 a general anesthetic).

For the first week 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. Beginning the second week, the resident continues to work days with an attending who now is responsible for other locations. At the end of the second week overnight call begins, at which point the resident should have a firm grounding in OB anesthesia. For the remainder of the month the resident works with an attending similar to the second week, with a few overnight calls sprinkled in. There are generally 4-5 overnight calls in the month.

The usual day on OB (referred to as “OB day” on the schedule) begins at 6:40am with morning conference or 6:30 M+M on Wednesdays. 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 (discussed below). Other than scheduled procedures, the OB day resident is responsible for any labor epidurals and unscheduled procedures (e.g., urgent cesarean section). While on the actual OB rotation, the resident can expect to receive a daily lecture at some point during the day. The shift generally ends around 4-5pm, whenever the OB call resident for that night is done in preop clinic. 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 particularly ill or critical patient on the 4th floor.

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 and 11 and the code bags (see the emergency room 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 check
- ensure functioning suction
- 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 (see 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 impeding 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 anesthesia 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.

**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”.

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 raises closing volume and predisposes to atelectasis and shunting, especially when in the supine position. Because of increased oxygen consumption and reduced FRC, apneic parturients can 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.

V. 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 H2 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).
Summary table of physiologic changes of pregnancy

**CV system**

- CO: increased
- Blood volume: increased
- Plasma volume: increased (esp relative to RBC mass)
- Hematocrit: decreased
- BP, SVR, PVR: decreased

**Pulmonary system**

- Oxygen consumption: increased
- MV: increased (mostly TV, some RR)
- FRC: decreased
- Closing volume: increased
- Inhalational uptake/elim: increased
- Time to desaturation: decreased
- Danger of intubation: increased

**Neurologic system**

- MAC: decreased
- Sensitivity to LA: increased
- Dosing for SAB/epidural: decreased

**Hematologic system**

- 2,3-DPG levels: increased
- oxy-Hb curve: right-shifted
- Clotting factors: increased

**Other systems**

- GFR: increased
- Insulin needs: increased
- Gastric acidity: increased
- Intraabdominal pressure: increased
- LES tone: decreased
- Gastric emptying: ??

**Physiology of uterine blood flow**
The gravid uterus receives an enormous supply of blood, about 600ml/min or 10% of cardiac output. 90% of this flow 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 side, 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.

**The placenta and our drugs- which agents cross the placenta?**

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 bupivacaine and ropivacaine 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 ion and unable to “cross back” to the maternal circulation. Under these conditions potentially toxic buildup of local anesthetics in the fetal circulation is possible.

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 popular mnemonic “He Is Going Nowhere Soon”.

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 it is complete (10cm). The latent phase is typically from 0-4cm of dilation, where the cervix slowly becomes more effaced and dilated (typically over 8 hrs). 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 10-12 hrs for nulliparous patients and can be much quicker (4-8hrs) for multiparous ones. Pain during this stage of labor is visceral 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 is from full cervical dilation to delivery of the baby. It generally lasts 30min to 2hrs. Pain from this stage is via S2-4 (perineal and pudendal nerves) and is somatic in origin. These nerves are notoriously difficult to complete block with epidural anesthesia due to their rostral position and thickness of the nerve fibers. A pudendal (not paracervical) block is another anesthetic option and can be performed by the obstetrician.

The third stage of labor is 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.

Tips for epidural placement
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.

Briefly, the layers that an epidural needle will pass through on the way to the epidural space are- skin, subcutaneous tissue and fat, the supraspinous and interspinous ligaments, and the ligamentum flavum. Standard technique for placing a lumbar epidural is to locate the anterior superior iliac spines (“hip bones”); a horizontal line at this point correlates with the L4-5 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 cord injury and little risk of nerve root injury. The interspinous spaces are palpated and the epidural needle is advanced until the distinct resistance and “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 slowly advanced while gentle attempts at injection of the syringe are made. The ligamentum 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 is of paramount importance. Confirming proper positioning of the patient can often yield success in a difficult placement. Whenever I encounter difficulty in placing a epidural, nine times out of ten a recheck of position and adjustment of position will save the day. The patient’s shoulders should be level and the lower back arched or flexed so as to maximize the space between the interspinous processes.

If bone is encountered after fairly deep insertion this generally means that the underside of a spinous process is being encountered, and walking the needle caudad may help. Conversely, shallow contact with bone implies you are either over a spinous process or off midline.

At times a “pseudo loss-of-resistance” may be encountered. Here, there is a boggy and indistinct ability to inject saline, but it is unclear whether the epidural space has actually been reached or not. 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. 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 2-4cc of saline may help with placement of the catheter. A standard length Touhy needle is 9cm from tip to handle, and 11 cm 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 and 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 (e.g., opening a separate needle for CSE placement).

The obstetricians typically will consult us to place a labor epidural once labor is established, generally around 2-3 cm of cervical dilation. Epidurals placed after 3-4 cm 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).

**Tips for spinal 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 block is technically easier than an epidural as there is no need to find the epidural space. 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. 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 larger gauge needle may be needed for stiffness, a longer needle to be able 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 obstetric anesthesia spinals are generally reserved for surgical procedures such as cesarean section or tubal ligation, but rarely may be used in dilute concentrations as the sole anesthetic for labor (e.g., a nearly complete parturient in extreme discomfort where there may not be time to place an epidural).
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 the sometimes frantic requests from the obstetricians to proceed immediately with cesarean section with what is in the best interests of both mother and child. In general, the indications for cesarean section fall into one of several broad categories-

1. urgent or emergent c-section
   - bleeding
   - risk of infection (chorioamnionitis or herpes with ruptured membranes)
   - fetal distress
   - maternal death
   - umbilical cord prolapse

2. abnormal fetal presentation or failure of labor to progress

3. unsafe labor for fetus or mother
   - abruption
   - placenta previa, accreta, increta or percreta
   - previous uterine or vaginal surgery (including prior C-section)
   - multiple gestations

4. 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 and 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 five minutes of breathing 100% O2, should suffice. An assistant should be present to help with cricoid pressure and if the airway proves difficult. When the entire
team is ready, the patient is prepped and the obstetricians ready to make incision, general anesthesia is begun with a rapid sequence intubation and cricoid pressure. The obstetrical team should make incision as soon as the patient is unconscious. The goal is to delivery the fetus as quickly as possible from the time of induction. The remainder of the case can proceed as in a non-emergent cesarean section (see below).

**Anesthesia for the non-emergent C-section**

Technique: general, epidural or spinal.

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 to 1.5 hrs.

Estimated blood loss: 800cc to 1.2L.

Position: supine (left uterine displacement).

Special equipment: none.

Special considerations:

The general progression of a cesarean section is as follows-

- Regional anesthesia is induced and appropriate sensory level confirmed, patient prepped and draped (or patient prepped and draped in preparation for general anesthesia).
- Skin incision and surgery proceeds. Antibiotics should be withheld until surgical request as most OB’s prefer the patient not receive antibiotics until after delivery of the fetus.
- Uterine incision is made. Times above 3min from uterine incision to delivery have been shown to correlate with lower apgar scores and fetal acidosis.
- After the umbilical cord is clamped the OB’s typically ask for antibiotics and oxytocin. Oxytocin induces uterine contractions and helps maintain uterine tone after delivery. When given too quickly it can induce systemic hypotension due to smooth muscle relaxation. For this reason, it is often given diluted in the patient’s IV fluids and allowed to run in over the course of the entire IV bag. The typical starting dose is 20 units, and the obstetricians may ask for it to be increased. Other agents that are used to increase uterine contractions include methylergonovine and carboprost tromethamine. Methylergonovine (Methergine) is an ergot alkaloid which causes sustained uterine contraction. It is only given postpartum and in addition causes smooth muscle contraction throughout the body, potentially resulting in hypertension and bronchoconstriction. It is contraindicated in
asthmatics. The dose is 0.2mg IM. Carboprost (Hemabate) is a prostaglandin F2 analog which also causes uterine contractions. It is also given IM, 0.25mg at a time. Similar to methergine, it can cause bronchoconstriction and is contraindicated in asthmatics. Other side effects include diarrhea, nausea, and vomiting. Manual uterine massage can also help with uterine atony.

- The placenta is delivered and the uterus is “exteriorized” to aid with closure. If regional anesthesia is employed it is common at this stage for an uncomfortable sense of pressure to be transmitted via peritoneal traction and unblocked vagal afferents. A small amount of ketamine, nitrous oxide, or IV narcotic may be helpful. The uterus, fascial and skin layers are closed.

Regional anesthesia is often preferred due to less risk to the mother as described above, and because it allows patients to see the child immediately after 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 titratibility, inability to redose, and more profound hemodynamic changes.

Typical dose ranges for a SAB are 1.2-1.5ml of 0.75% bupivicaine. Most people often add 10-20 mcg of fentanyl and 0.2mg of morphine to the mixture. This can be drawn up sterilely beforehand and given to an assistant to inject into the spinal syringe prior to administration. The fentanyl is thought to improve quality of the block (although firm data is lacking) and the morphine is a good adjunct for post operative pain control. 200mcg of epinephrine is also used by some practitioners to prolong duration of the blockade. Although theoretically of benefit, many people (including myself) feel the data is inconclusive and that epinephrine only increases the side effect profile (e.g., nausea and vomiting) without increasing 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.

Epidural anesthesia is generally employed when the patient has a prior labor epidural and then progresses to need for a cesarean section. Less commonly, it is employed when the situation demands (e.g., the operation is expected to take a long time and redosing is to be expected, or when the titratability of an epidural vs. a SAB is preferred as in severe preeclampsia). Two options to speed the onset of the epidural include 3% chloroprocaine or 2% lidocaine alkalized with NaHCO₃ (1ml NaHCO₃ for every 9 ml of lidocaine). Usual doses are 15-20ml of either medication, titrated in increments. Epidural fentanyl can also be added (100mcg). Morphine for post-op pain control is typically reserved until after delivery of the fetus (4mg). Chloroprocaine needs to be redosed every 45min, lidocaine every 1hr and bupivicaine every 1.5hrs. At times an epidural block may be patchy or incomplete. Small doses of ketamine (10mg IV at a time) or narcotics can be of great assistance during these times but special care and vigilance must be maintained.
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 0.5% isoflurane is common in our institution. Often non-depolarizing 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 cesarean section in patients desiring sterility. It is performed after delivery of the fetus and typically adds 15 min to the procedure. A repeat cesarean section can take significantly longer than a “virgin” C-section due to scar tissue and adhesions from prior procedures.

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. These family members are often placed very near to the anesthesia provider, 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 duties. However, there are also times when they can be of great assistance, e.g. reassuring and calming a hysterical patient.

At times the obstetrician may ask for sublingual nitroglycerin to be administered to aid with uterine relaxation. This can be administered safely as long as attention is paid to maternal blood pressure.

**Anesthesia for placenta accreta/increta/percreta**

UCSD is privileged to be a center that performs many deliveries for patients with placenta abnormalities. Placenta accreta is when the placenta grows into the uterine endometrium, increta when it grows into the myometrium, and percreta 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. Typically these patients are taken to interventional radiology where uterine artery balloons are placed. These 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 preoperatively 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 intraabdominal case with the potential for massive blood loss.

Although it is common in this institution to do the cesarean portion under epidural anesthesia and the rest of the procedure under general, a strong argument can be made for doing the entire case under GA. 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 pitocin should be avoided at all costs. In this regard GA may be superior to neuraxial anesthesia in causing uterine relaxation.

Technique: general +/- regional anesthesia for portions of the procedure.

Monitors: standard, arterial line.

IV access: Large. Frequently a large central line is placed.

Duration: 2 – 4 hrs.

Estimated blood loss: 1L and up, potentially many liters.

Position: supine (left uterine displacement).

Special equipment: perfusionist available. Fluid warmers for volume resuscitation.

Special considerations: as above. Intraop induction of GA demands preparations be taken beforehand with regards to the airway (e.g., preparing a ramp for intubation, smaller ETTs, rapid sequence induction with cricoid pressure).

Anesthesia for other obstetrical 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.

Estimated blood loss: less than 100cc.

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 BTL. Similarly, intrathecal or epidural morphine is typically not given, as these patients tend to be discharged the same day and to avoid delayed respiratory depression.

**Anesthesia for non-obstetric surgery in the pregnant patient**

In general, all elective surgeries should be postponed until at least six weeks postpartum. Necessary surgery should proceed with the following items in mind:

- the 3rd to 10th weeks of pregnancy are when major organ formation occurs and when the fetus is most susceptible to teratogens.
- There is ongoing concern about the safety 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 than even earlier parturients 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. Typically the obstetrical 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, B-agonists or Mg²⁺ may be employed as tocolytics.
- 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.

**Special topics**

**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. PIH is defined as a systolic pressure above 140 or a diastolic pressure above 90 mm Hg. PIH is one end of a spectrum of disease states that include preclampsia, eclampsia, and the HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelets). Preclampsia is defined by hypertension, proteinuria (> 500mg/d) and upper body edema. Lower body edema is common in pregnancy and in and of itself does not qualify. Eclampsia is much more rare and includes seizures. Severe PIH 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).

PIH is more likely in nulliparous young women and those with a previous history of PIH. As a rule these women are prone to vasospasm, behave as if they were intravascularly “dry”, and are edematous.

Treatment of PIH includes bed rest, antihypertensive therapy (hydralazine or labetolol are common), and Mg2+ 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. 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 titrability 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.
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. Treatment includes resuscitation, immediate laprotomy 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.

Abruption

Abruption occurs when there is abnormal separation of the placenta, with bleeding into the space between the placenta and uterine wall. Abruption 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.

Amniotic fluid embolism

This is a rare (1:20000) condition in which amniotic fluid enters the maternal circulation. It has a very high mortality (over 50%). Most AFE’s 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. In many respects an AFE mimics a pulmonary embolism. 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 their to be any chance of effective maternal and fetal resuscitation.

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 one minute score correlates with survival, while the five minute score correlates with neurologic outcome. The five components of the apgar score are as follows:

<table>
<thead>
<tr>
<th>Component</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color</td>
<td>blue</td>
<td>body pink, blue extremities</td>
<td>completely pink</td>
</tr>
<tr>
<td>Heart rate</td>
<td>none</td>
<td>&lt; 100</td>
<td>&gt; 100</td>
</tr>
<tr>
<td>Resp</td>
<td>none</td>
<td>irregular</td>
<td>vigorous</td>
</tr>
<tr>
<td>Reflex</td>
<td>none</td>
<td>grimace</td>
<td>cries</td>
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<tr>
<td>Irritability</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tone</td>
<td>flaccid</td>
<td>flexion</td>
<td>active movement</td>
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The cardiac anesthesia rotation and cardiovascular physiology

The cardiac rotation at UCSD is an intensive two-month exposure to cardiothoracic physiology and procedures. Beginning in the middle of the first year, one resident at a time begins the rotation each month. Thus, by the middle of the second year an entire class will have completed their cardiac rotation. The first month is spent primarily at Thornton hospital, while the second is at the VA. During the first week of the first month, you will be paired one-on-one with a cardiac anesthesia attending who will cover the most critical information and basics of cardiac anesthesia. For the remainder of the month the cases and responsibilities will remain the same, but your attending may be covering multiple locations. At the VA your attending will always be covering another room, consummate with your increased experience.

The cases encountered at Thornton will generally be Pulmonary Thrombo-Endarterectomies (PTEs) or CABGs, with or without cardiopulmonary bypass (“pump”). Procedures for valvular disease or thoracic cases may also be encountered. PTEs are not performed at the VA, but CABGs and valvular procedures are common. Occasionally, you may be assigned to Hillcrest if there is a heart procedure there. In general, the heart resident does any cardiothoracic case available; if there are none scheduled for the day you may be assigned a general OR case. If both Hillcrest and Thornton have cardiac cases, the CA1 heart person is generally assigned to Thornton.

Call during the two months is discussed in greater detail in the section on call responsibilities. During the first month you will be assigned “CA1 heart call” (whether or not you are a CA1 or 2) every day of the week except Saturdays. This is pager call, and you are responsible for returning to the hospital for emergency cases (e.g., takebacks for bleeding). On weekends you will be called in for the same or for the occasional case which gets booked electively. All of these are uncommon, for all practical purposes making being on call virtually the entire first month not particularly onerous. There is always a senior resident who is on-call for “heart transplant” as well. While the heart transplant person is theoretically on call only for cardiothoracic transplants, the attendings may choose to either call the CA1 heart call or the heart transplant person in for a case. At times as the CA1 heart call you may be asked to finish an ongoing case at Hillcrest, even if you have just finished a day at Thornton. Again, this is at the discretion of the attendings. You should 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 “first right” to any cardiothoracic case available. While at the VA you are not part of the Thornton/Hillcrest heart call pool (since a new resident will be starting their first month of cardiac).

It is expected that you will preop all your own patients during the heart months. Occasionally, especially at the VA, these patients will have come into our preop clinic already. Obviously during those times there is no need to repeat the preop, but typically
at Thornton or Hillcrest the cardiac patients are admitted the day before and have no anesthesia preop. Thus, a typical day during the first month of cardiac ends not when the case is done, but when the patient for the next day has been seen. See the preop section for more information.

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 things (e.g., making all the drips). Also, the vast majority of these patients need adequate IV access and an arterial line established before induction which you also need to allow time for. Compounding all of this is that heart cases typically start earlier- 6:30am is the norm at Thornton. What all this means is that you may find yourself having to come into the hospital at 5 am in the beginning. 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 will be covered in more detail later and are also addressed in the departmental cardiac rotation syllabus.

Cardiac surgeons are notorious throughout the country as being “difficult to work with”. As with any stereotype there are some truths and some falsehoods to this statement. Some of our surgeons do seem to fit into the irascible category. However, there are also some that are very professional and just fine to work with. What makes this situation complex is that from the surgeons’ standpoint they see a new, green resident come on the rotation each month who has to be broken in. Also, cardiac surgeons tend to understand the patient’s basic physiology better than any other type of surgeon (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 probably 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 should 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.

**Coronary anatomy and circulation**

Coronary blood flow is supplied by the right and left coronary arteries. The right coronary supplies the right atrium, 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 R dominant circulation. The remaining 15% of the time the PDA arises from the L coronary- a left dominant circulation. The **SA node** is supplied by the RCA the majority of the time (40%
of the time by the LAD). The AV node is almost always supplied by a branch from the RCA.

The L main supplies the LA and 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, while the circumflex supplies the lateral wall. Both the left and right circulations further divide into septal, diagonal and marginal branches.

Most coronary venous return is through the coronary sinus and anterior cardiac veins into the RA, although a small amount drains directly into the ventricles through the thebesian veins (this represents a portion of physiologic shunt).

While the RV is perfused during both systole and diastole, LV perfusion occurs almost entirely during diastole. Perfusion pressure is determined by the difference between aortic and ventricular pressures, and most meaningful flow occurs which the LV is relaxed and pressure is low (diastole). The endocardium is the area most sensitive to ischemia, because the highest transmural and thus 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, and 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., 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.

Determinants of supply-

1. Coronary perfusion pressure
   - increasing arterial pressure, especially diastolic pressure, and reducing LV end-diastolic pressure will increase CPP.
   - The converse of the above also holds.

2. 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.
3. **Arterial oxygen content**
   - determined by the PaO2 and hemoglobin content of the blood. Increases or reductions in either are directly linked to oxygen supply.

4. **Heart rate**
   - slower heart rates feature disproportionally increased diastolic time with relatively increased flow.

Determinants of demand-

1. **Heart rate**
   - faster heart rates increase myocardial oxygen consumption and vice versa.

2. **wall tension**
   - the primary determinants of wall tension are preload and afterload. Excesses of either increase wall tension and thereby oxygen demand.

3. **contractility**
   - increases in contractility necessitate increases in oxygen consumption.

**Valvular disease**

A significant portion of our cardiac patients present with cardiac valvular disease. The disease can be independent or concomitant with other pathology such as CAD. Similarly, the patients may be coming for valvular replacement, revascularization, or both. Each specific type of valvular dysfunction entails **specific anesthetic requirements**.

**Aortic stenosis**

AS is usually due to congenital defects (e.g., bicuspid valve), rheumatic disease, or calcific degeneration. These processes lead to gradual impairment of LV outflow. In compensation, the LV develops concentric hypertrophy which serves to both increase the transvalvular gradient or flow (more “squeeze” to force blood through a stenotic valve) and decrease LV wall stress. As the disease worsens, these patients are not able to increase cardiac output in response to demand (fixed obstruction to flow). Myocardial oxygen consumption is increased due to increased work of the hypertrophied myocardium. Supply is diminished from a fixed CO and thickened myocardium which impedes endocardial blood flow. The thickened, hypertrophied LV results in a “stiff” LV and a decrease in LA LV diastolic pressure gradient, resulting in decreased LV filling. This makes these patients very dependent on coordinated atrial contraction (the atrial kick) for diastolic filling.

The classic presenting signs of AS are exertional syncope, angina, and CHF. Angina can be independent of true CAD and results from functional ischemia and
inability to perfuse the hypertrophied myocardium. CHF has the worst prognosis of the three. Normal aortic valve area is > 2.5cm², while < 0.6cm² is considered critical AS. Typical surgical intervention involves replacement of the valve, with percutaneous valvuloplasty a rare option. The anesthetic goals for a patient with AS are listed below.

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 (due to inability to increase flow across a tight stenosis, CO becomes relatively HR dependent).
- Sinus rhythm **must be maintained**. As previously discussed these patients are very dependent on their atrial kick for LV filling. Intraop afib 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. Coronary perfusion is dependent on maintaining perfusion pressure. As explained above the AS patient already has compromised myocardial oxygenation, increased demand, and decreased supply. 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. As an example, for a systolic pressure of 120mm Hg the LV may still have to overcome pressures in excess of 200mm Hg to produce flow across the stenotic valve.
- Hypotension can set off a spiral of decreased coronary perfusion, myocardial ischemia, and 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.

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’s sx, cystic medial necrosis, and other connective tissue diseases.
The pathophysiology involves creating LV volume overload as a portion of systolic volume is ejected backwards into the LV during diastole. This also 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. Acutely, the LV is unable to dilate and the increased volume/pressure is 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/rhythm-
- high normal rates are preferable. Increases in diastolic time allow for increased regurgitation, thus 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. Similar, 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 general mnemonic for management of regurgitant lesions is FFF- “Fast, Forward, and Full”.

**Mitral stenosis**

MS is almost always due to rheumatic fever and thus is increasingly rare in this country. The valve leaflets and chordae tendinae 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 afib. 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 ultimately RV failure.

Heart rate/rhythm-
- maintenance of sinus rhythm is necessary to improve LV filling. Preexisting afib and/or clot may make cardioversion unwise.
- Avoid tachycardia so as to allow sufficient diastolic time and filling.
Preload-
   - adequate filling pressures must be maintained; however, excessive volume is readily transmitted back to the pulmonary circulation. Keep these patients euvolemic.

Afterload-
   - afterload should be maintained near-normal.

CO-
   - avoid increases in CO as the LV is poorly able to compensate (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 are unable to compensate in this way and primarily present with pulmonary edema. Anesthetic management includes-

Heart rate/rhythm-
   - keep HR at high-normal ranges. Slow heart rates, and thus 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.
   - Severe excesses in preload may increase dilation of the LV and mitral annulus and worsen regurgitation.

Afterload-
   - reducing afterload improves forward flow.

Again, the mnemonic for regurgitant lesions can be helpful- FFF- “Fast, Forward, and Full”.

**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 may be associated 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 mitral valve may be present. In this phenomenon the mitral leaflet is drawn by a 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 B blockers, Ca channel blockers and surgical myomectomy if severe. 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 can worsen obstruction. Thus, avoidance of excessive drops in preload and afterload and increased sympathetic output are the keys.

**One lung ventilation**

Single-lung ventilation can be one of the most challenging maneuvers we encounter in anesthesia. 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., bronchoplueral fistula, tracheobronchial disruption, bronchoalveolar lavage)

Other relative indications are mostly related to the surgical procedure or technique-
- pneumonectomy, lobectomy, thoracoscopy
- anterior thoracic spine procedures
- thoracic or high abdominal aortic aneurysms
- single-lung transplant
- severe unilateral lung dz with hypoxia
- esophageal surgery

Several types of ET tubes and tools are available to create lung separation and one-lung ventilation and include double lumen ETTs, univent tubes, and the use 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.

1) DL tube
Advantages include-
- can suction either lung
- can ventilate either lung

Disadvantages include-
- larger and potentially more difficult to place
- cannot be left in long term- must be changed for a regular ETT at the end of the case if post op ventilation is needed
- no appropriate pediatric size

2) Univent tube
Advantages-
- may be technically easier to place
- can be left in after the conclusion of a case

Disadvantages-
- cannot easily switch ventilation or suction between lungs
- no appropriate small pediatric size
- lung deflation is often slow and incomplete due to small efflux lumen

3) Bronchial blocker or Fogarty catheter through a standard ETT
Advantages-
- many sizes, can be used in peds
- can be used to quickly tamponnade bleeding

Disadvantages-
- cannot suction either lung
- cannot switch ventilation easily
- lung deflation is often slow and incomplete due to 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. Similarly, after the patient is positioned for surgery both position and lung separation should be rechecked (e.g., many procedures requiring one-lung ventilation are performed in the lateral position). Any movement or repositioning of the patient can result in tube migration and 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 thorough and probably should not be deviated from.

Before starting, have all the appropriate equipment available. This includes the correct DLT size (see the chapter in Miller for more info), a firm clamp, suction catheters, a working fiberoptic scope and stethoscope.

1. Place DLT at estimated appropriate depth, inflate both bronchial and tracheal cuffs
2. Listen for bilateral breath sounds
3. Clamp one lumen at a time and listen for absence of breath sounds on clamped side and continued presence on the contralateral side
4. Place fiberoptic bronchoscope down tracheal lumen, confirm proper placement of tube and bronchial cuff (see below), adjust as necessary
5. Secure tube
6. After final positioning, run through the checklist again

**Confirming correct tube placement via FOB**

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 R and L mainstem bronchi occurs at the carina. The right mainstem can be further identified by noticing the straighter, more caudal takeoff (responsible for most aspirations going down the right side) and the fairly rapid takeoff of the RUL. The L mainstem by contrast is much longer and the first division encountered is usually that of the upper/lingual segments and the lower lobe.

DLTs by contrast can be shaped for either L mainstem or R mainstem placement of the bronchial orifice and cuff (“L sided vs. R sided DLTs”). In practice, most people almost always use a L sided DLT as they are technically easier to place, with less complications and still allow separation of either lung. The main disadvantage of R sided DLTs is that the right upper lobe bronchus takes off of the right mainstem bronchus quickly, and is easy to block with the tube. Such placement would result in ventilation of only the R middle and lower lobes during one lung ventilation. With correct L 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 distal (bronchial) end should pass down the L mainstem. When inflated the blue bronchial cuff should be visible just past the carina
- the R mainstem should be easily entered, and the takeoff for the RUL should be easily observed
- going down the bronchial lumen, one should see the subdivisions of the L lung (first division encountered is usually that of the upper/lingual segments and the lower lobe)
- consult the appropriate text for a fuller discussion of bronchial anatomy and divisions

One lung ventilation physiology

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 poorly 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 (one lung ventilation), the non-dependent lung continues to receive some amount of pulmonary blood flow. This blood never becomes oxygenated and represents a shunt which can lead to hypoxemia. Fortunately, hypoxic pulmonary vasoconstriction limits flow to the now non-ventilated lung. Most perfusion and all ventilation is delivered to the dependent, non-operative lung. Factors which can decrease HPV and increase shunt include hypocapnia, vasodilators and inhaled anesthetics.

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 (PEEP, autoPEEP from inadequate expiratory times), low FiO2 (causes 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 and faster respiratory rates are usually employed during single lung ventilation. Hyperventilation and hypocapnia are avoided as they inhibit HPV.

Correction of hypoxemia during one-lung ventilation

Hypoxemia and desaturation during one lung ventilation must be corrected. 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 92% as their cutoff, although clearly this varies according to the patient and their disease state. The following steps can be employed to correct hypoxemia:

1. ventilate with 100% O2. Most anesthesiologists do this automatically for the duration of one lung ventilation.
2. applying 5-10cm H2O of CPAP to the non-dependent lung. This can cause reinflation and interference with surgery
3. PEEP to the dependent lung (may worsen hypoxemia if decreases perfusion)
4. Periodic dual lung ventilation (also interferes with surgery)
5. in extreme cases, the surgeon can clamp the pulmonary artery to the non-ventilated lung, essentially eliminating all shunt from that source

**Intra-aortic balloon pump counterpulsation**

An IABP is a device designed to improve contractility and ease the work of a failing 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 descending aorta. Inflation of the balloon is timed just after the dicrotic notch, and the balloon then rapidly deflates (ideally just before LV ejection). This combination of events reduces afterload, increases coronary perfusion and raises end-diastolic pressures.

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 (1:1) or every other beat (1:2). The latter allows the heart to work every other beat on its own and 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 beyond several hours post op, anticoagulation becomes necessary. Complications of the IABP include limb or distal (e.g., mesenteric) ischemia, aortic dissection or rupture, infection, coagulopathies (especially thrombocytopenia), gas emboli, renal failure, and decreased perfusion to the spinal cord resulting in paraplegia. Contraindications to IABP placement include AR, sepsis, and severe vascular disease (potential problems placing the balloon, increased 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 of CPB when all other measures have failed. VADs may be temporary, intended for a few days’ use, or longer term (months) as a bridge to transplantation. Essentially, they serve as pumps to augment ventricular flow. As they are foreign bodies patients with a VAD present require systemic anticoagulation.

CO in patients with a VAD becomes very preload dependent. To a certain extent, the CO can be set by adjusting the rate the VAD will pump blood at. However, if inadequate filling exists, the pump will be ineffective no matter the rate it is set at. Thus, an increase in inotropy with a VAD should be achieved with volume administration first.

**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. Administration of FFP or ATIII concentrate will reverse this state, allowing heparin anticoagulation 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 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 agtroban. Plasmapheresis may be necessary to remove significant amounts of antibodies.
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 good general “game plan” for the CT cases encountered here at UCSD.

Cardiopulmonary bypass

In general, any case involving CPB can be broken down into three broad stages-the prebypass, on bypass and post bypass period. 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, 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. This blood is oxygenated, CO2 is removed, and the blood is then returned 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, and many different solutions can be added depending on surgical preference or patient needs (e.g., mannitol, albumin, aprotinin). Venous blood drains from 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, potentially with 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 or centrifugal pumps. The former uses rollers to squeeze blood through large tubing to the patient, 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 un-physiologic type of flow may be partially responsible for decreased organ perfusion during CPB. Centrifugal pumps spin and use centrifugal force to propel blood out 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. Suction lines return blood from the field and the heart (the heart continues to receive a small amount of drainage from the bronchial and thebesian circulation and must be periodically drained). A separate cardioplegia pump is used to deliver cardioplegia solution to the heart (see below). Additionally, inhaled anesthetic can be directly added via the oxygenator.

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 cell damage.

1. Cardioplegia

Potassium cardioplegia is most commonly used. Infusion of this solution via the coronary arteries causes cardiac arrest and cessation of electrical and mechanical activity, reducing oxygen requirements dramatically. Cardioplegia is also often delivered retrograde via the coronary sinus to ensure all myocardium is reached (since CAD by definition may limit flow via the arteries). This solution must be reinfused periodically, and washed out prior to coming off bypass.

Distention of the heart and electrical activity both increase myocardial oxygen demand. Fibrillation is especially detrimental. Satisfactory conditions are not met until the heart is both empty and asystolic.

2. Hypothermia

Systemic hypothermia reduces metabolic oxygen requirements, about ½ 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.

3. Other factors

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 (shown to attenuate reperfusion injury). Unfortunately anesthesiologists have little control over most of these components.

Prebypass period

This time period is from induction of anesthesia to the 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 PA catheter are placed. Relevant labs such as the ACT, baseline ABG and cardiac indices should be obtained. The TEE probe is placed and an exam performed. If aprotinin is to be used, a test dose should be administered at this time. Aprotinin is a serine protease inhibitor which has been shown to decrease bleeding and transfusion requirements during cardiac surgery. There is a small but definite incidence of allergic reactions to aprotinin, the incidence of which increases with repeated exposure to the drug. A typical test dose in 1 cc IV. After a negative test dose, 2,000,000 kallikrien units are loaded over 30 min, and then a constant infusion of 500,000 ku/hr is continued throughout the case. Alternatively, aminocaproic or transexamic acid can be used. They may be less effective than aprotinin but are less likely to cause allergic reactions.

CPB requires systemic heparinization to prevent catastrophic clotting within the pump, which would probably be fatal. As surgery proceeds, the perfusionist will calculate the dose of heparin needed based on body weight and the ACT. The patient is allowed to cool in preparation for systemic hypothermia with CPB (in other words, not actively warmed). Similarly, a small amount of hemodilution is beneficial. The surgeons attempt to do as much dissection as possible prebypass 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 min later to ensure proper anticoagulation. An ACT > 400s is generally considered safe. The aortic cannula is typically placed first. During this time, SBP should be 100mm Hg 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 or using a short-acting vasodilator such as nitroprusside. Afterwards the venous cannulae are placed and venous flow to the reservoir is confirmed. When good return is established the arterial side is unclamped and CPB is initiated. Flow is gradually increased as proper cannula placement, venous return and arterial pressures are confirmed. Soon after cardioplegia and cooling are begun.

**Bypass period**

Physiologic management of the patient is largely turned over to the perfusionist at this point. The ventilator is stopped, and if needed, vasoactive drugs are administered by the perfusionist. Anesthesia is maintained by inhaled agent by the perfusionist, who has a vaporizer on the CPB machine. Additional muscle relaxant may need to be administered which can be given to the perfusionist. The PA catheter typically migrates distally during CPB and should be withdrawn 2-3 cm upon initiation of CPB.
The surgeons may pass off a separate line to the anesthesiologist to infuse a constant stream of cold irrigation. These cold bags will be provided in the room and should be continued for as long as the surgeon may desire. Urine output should be monitored and reported to the perfusionist. The perfusionist may also request syringes of drug to manage the patient’s physiologic parameters (e.g., nitroprusside).

As the surgery concludes, the surgeons will call for the patient to be rewarmed. The patient must be rewarmed prior to termination of CPB, but if rewarmed too soon the protective effects of hypothermia are negated. Vasodilation can improve pump flow and speed the warming process, but overly rapid rewarming reduces the solubility of gases and can lead to the formation of bubbles (and thus gas emboli). At this stage, light anesthesia is common and most practitioners administer additional muscle relaxant and amnestic agents such as a propofol infusion, 50mcg/kg/min or a benzodiazepine. The perfusionist will also provide the anesthesiologist with a syringe of protamine for eventual heparin reversal. Protamine must not be administered while the patient is on CPB, for catastrophic clotting and death are the likely results. Set the syringe clearly labeled far away from the IV lines.

Prior to separation from CPB, the patient should be warm, acid/base status and hematocrit normalized, a stable cardiac rhythm and rate obtained (generally 80-100 bpm, pacing may be needed), and ventilation resumed. This is a critical time which the attending should be present for. The venous return lines are clamped, and the heart fills and begins to eject blood. The cardiac surgeons usually call for an inotrope at this point (typically 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 brief TEE exam may be called for to further assess cardiac or valvular function. 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 well and truly terminated and the cannulas removed the surgeons will call for protamine. Prior to administration, you should 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 every one is on the same page (e.g., the perfusionist will shut the CPB suction off). Protamine should be administered slowly (in 3-5ml increments over 5-10min). The side effects of protamine include hypotension from vasodilation, pulmonary hypertension, and myocardial depression, all of which are exacerbated by fast administration. Allergic reactions are also known to occur, which may be more common in diabetics who previously received insulin containing protamine.

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 (see cardiac physiology section) or rarely a Left or Right Ventricular Assist Device (LVAD or RVAD). Possible causes for poor myocardial performance include poor myocardial protection during CPB leading to ischemic injury, long CPB time, myocardial stunning, ongoing ischemia (e.g., air bubbles in the coronaries), continued valvular dysfunction, and poor baseline cardiac.
performance. 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.

**Post bypass period**

This period consists of surgical hemostasis, placing chest tubes, closing the chest, and transporting the patient to the ICU. An ACT should be checked 3 min after protamine administration to confirm reversal of heparin. Systolic blood pressures around 100mm Hg are generally desired to minimize bleeding and myocardial work. 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 post-operative bleeding which may necessitate a trip back to the operating room.

The perfusionist will usually be able to spin down a significant amount of cell saver blood (processed RBCs recovered from suction and washed) which 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 PA catheter 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 readied to bring during 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 to 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. Similarly, full monitoring and ventilation during transport is mandatory. Once in the ICU care may be relinquished to the ICU nurse per standard protocol.

### I. On-pump cases

Prototypical cases include CABGs, valvular repair/replacements and PTEs.

**Technique:** general anesthesia.

**Monitors:** standard, arterial line, CVP and PA catheter, TEE, plus some form of neurologic monitoring such as the BIS or EEG. Urine output. +/- femoral arterial line.

**IV access:** one large IV to begin, with central access established during the case.

**Duration:** 6-10 hrs.

**Estimated blood loss:** difficult to quantify secondary to hemodilution and CPB salvage. 500cc- 2L not uncommon.
Position: supine.

Special equipment: in addition to special monitors, multiple pumps for infusions during the case, cooling jacket for the head in PTEs.

Special considerations:

The basic steps for surgery involving CPB are described above. Additionally, valvular pathology and goals for anesthesia are described in the cardiovascular physiology section.

Almost every cardiac patient should have an arterial line placed awake, prior to induction of anesthesia. Many centers also place a central line and PA catheter pre-induction. Here at UCSD the CVP and PA catheter are generally placed after induction, although this may vary with specific patient requirements. Premedication should be used judiciously. Pain, anxiety and an increased sympathetic state are undesirable, as are hypoventilation, hypoxia, and hypercarbia.

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 inotrope (e.g., dopamine) and vasodilator (e.g., nitroprusside) ready. It may be useful to prepare a nitroglycerin infusion for patients with CAD. These infusions are generally attached to the PA catheter infusion port after placement.

The term “cardiac induction” is meant 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 chest wall 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% O2 in the short term is negligible, while the potential benefits in a class of patient especially intolerant of hypoxia are substantial. Inhalational anesthesia, additional narcotic, IV agents and muscle relaxant can all be used for maintenance. Unfortunately, recall under anesthesia is more common in cardiac cases, as well as obstetrical and emergency surgeries. Due to the increased potential for hemodynamic instability and the use of CPB, adequate anesthesia in the cardiac patient
cannot be guaranteed (although every effort is of course still made). Patients should specifically be informed of this rare possibility during the preop visit.

Other intensely stimulating points during the surgery include skin incision, sternal splitting, and dissection/manipulation around the aorta. 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 all the attendant potential for scar tissue and adhesions of critical structures to the chest wall (e.g., L internal mammary artery, ventricular wall!). In contrast to sternotomy in a virgin chest which can be quite fast, redo median 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. In spite of this the potential for surgical mishap is still high. Therefore, redo sternotomy patients should have blood immediately available (e.g., in the OR and checked in) for immediate administration should the need arise.

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. Long standing 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 creates 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 SVR only, without the low pressure LA to eject blood into.

The PTE

UCSD is one of the few centers in the world to perform pulmonary thromboendarterectomies regularly. In fact, the majority of cases you will encounter during the first cardiac month 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 instruction.

As a class most PTE patients tend to be younger and have fewer comorbidities than other cardiac patients. The basic pathophysiology of the disease involves chronic formation of clot and intimal hyperplasia in the pulmonary arteries, leading to pulmonary hypertension and right heart overload, with hypertrophy, dilation and eventual failure. Regardless of the cause of the pulmonary thrombus, elevated PA and R heart pressures are a hallmark of these patients. Indeed, it is not uncommon to see a PTE patient with RV pressures equal to or above systemic pressures!
The decisive surgical maneuver involves opening the main pulmonary arteries so that clot can be extracted. This necessitates total circulatory arrest, which in turn is the reason for most of the major anesthetic differences between a PTE and a standard CPB case. Circulatory arrest must be performed under deep hypothermic conditions for cellular (particularly neurologic) protection. As the name implies, the CPB machine is stopped and all flow to the patient ceases during deep hypothermic circulatory arrest (DHCA).

Premedication is generally avoided in PTE patients to avoid hypercarbia and hypoxia, both of which acutely exacerbate pulmonary hypertension. Placement of the PA catheter may prove difficult due to elevated pulmonary artery pressures, and risk of rupture from balloon inflation is increased. Indeed, many practitioners do not wedge the balloon at all in these patients, preferring to avoid this risk and do without the information provided by the pulmonary capillary wedge pressure. A femoral arterial line is also placed post-induction, which may provide more accurate pressures than the radial line after profound 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 basal metabolic oxygen requirements of the brain. Upon initiation of CPB the patient is cooled to < 20°C. The perfusionist will ask for 500mg of pentothal, to be administered prior to DHCA. Temperature is monitored in multiple redundant locations, including blood (PA cath and CPB machine), tympanic (probably best reflection of brain temperature), bladder (reflects core temperature) and rectum. The latter two are generally slow to reflect changes in temperature, particularly the rectum due to an insulating effect of the feces.

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 expulses any last vestiges of 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 an important safety mechanism, the condescending way some of the CT surgeons use it can be a source of frustration. For example, some insist on the checklist being repeated verbatim, and will question if you deviate from it by even one word. That being said, it is important to know when to pick your battles and remain political. It is probably best to just play along and not get upset over a trivial matter, while continuing to act in the best interests of the patient.

Once DHCA is initiated the surgeons have 20-30 min to complete the thromboendarterectomy. As time is of the essence distractions and delays are unwelcome.
The surgeons expect us to be especially attentive at this stage so surgery can proceed expeditiously. If both pulmonary arteries are to be worked on the surgeons will typically go back on CPB between each artery to provide perfusion and lessen total time spent in DHCA. Before resuming DHCA the entire above checklist will have to be repeated.

After the thromboendarterectomy is complete DHCA is terminated and the patient must be rewarmed. Because of the profound hypothermic conditions this stage can be quite lengthy, sometimes approaching 2 hrs in obese patients. Pulmonary reperfusion injury is an uncommon but potentially devastating complication in these patients, and may be signaled by worsening hypercarbia, hypoxia, pulmonary edema or frank hemorrhage.

II. Off pump CABGs

Off pump CABGs present unique challenges to the anesthesiologist. While previously reserved for select patients with 1-2 vessel disease and good target vessels for anastomosis, these days patients with more profound CAD are increasingly being done off pump. This technique demands a fair amount of surgical skill, to work around and on a still-beating heart. CPB and the perfusionist are placed on standby in case of emergent need or inability to perform the procedure off-pump.

Because the patient never goes on CPB, physiologic management remains in anesthetic hands for the entire procedure. Manipulation of the heart has the potential to produce large hemodynamic swings and arrhythmias which must be dealt with. Suspension or lifting of the heart to allow access to posterior vessels causes especially profound changes which the surgeons should be notified of if they occur. When the heart is lifted out of the chest cavity interpretation of the EKG becomes difficult if not impossible. TEE in this situation is similarly rendered useless.

The advantages of off pump surgery are (generally) faster surgical times, 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 warm from the beginning of the case via standard mechanisms (e.g., fluid warmers, warming blankets). In general, anesthetic management of off-pump cases, including induction, maintenance and hemodynamic stability are the same as for CPB cases.

Technique: general anesthesia.

Monitors: same as for CPB cases.

IV access: same as for CPB cases.

Duration: 4-6 hrs.
Estimated blood loss: < 500cc.

Position: supine.

Special equipment: see above.

Special considerations: see above.

III. Thoracic aorta surgery

Examples include elective or urgent repair of aortic dissection, aneurysm or trauma, and repair of coarctation.

Technique: general anesthesia.

Monitors: standard, arterial line, CVP, PA catheter, urine output. +/- TEE, EEG.

IV access: multiple large-bore catheters including central access needed.

Duration: 4-6 hrs.

Estimated blood loss: 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, equipment for one lung ventilation (e.g., DLT and FOB), +/- neuro monitoring.

Special considerations:

Surgery for thoracic aortic lesions is rare but invariably complex. They may combine 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 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. For more details see the vascular (AAA) and liver transplant sections. One lung ventilation is covered in the previous section on cardiovascular physiology.

IV. Thoracic cases involving the lung

Examples include thoracoscopy, thoracotomies for lobectomy/pneumonectomy, repair of bronchial trauma, bronchial-alveolar lavage and lung transplants.
Technique: general anesthesia.

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: 2-12 hrs depending on the case.

Estimated blood loss: 100cc – 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 physiology section.

Thoracoscopies, tissue biopsies, and pluerodesis 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 beat to beat monitoring, but further invasive monitors such as the PA catheter 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 postop FEV1 < 0.8L or 40% of predicted, FEV/FVC < 50%, and room air PaCO2 > 45 or PaO2 < 50 mm Hg are at high risk for post operative 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 anesthesia consult.

Bronchial-alveolar 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.

Lung transplants are a unique type of case. The window to transplant a lung before organ compromise is significantly shorter than for other solid organs (e.g., kidney). Therefore, these cases must be performed expeditiously, with close coordination between the transplant teams. Single lung transplantations are usually performed without
CPB. A DLT is necessary to allow ventilation to the other lung when the operative side is being resected. If used, the PA catheter may need to be withdrawn if it floats to the same side as the operative pulmonary artery. Double lung transplants are either done sequentially, with a DLT and no CPB, or at the same time which necessitates CPB. The same considerations as CPB for heart disease are present. FOB is used after the anastomosis is created to check the integrity of suture lines. Fluid restriction and minimizing peak inspiratory pressures are necessary goals to prevent pulmonary edema and trauma to the transplanted lung.

Excessive fluid administration and pulmonary pressures may lead to pulmonary edema and respiratory failure in lung resection cases. In the case of a pneumonectomy, all cardiac output that previously went to both lungs now must be received by the one remaining pulmonary artery. Transplanted lungs lose their lymphatic drainage which predisposes to pulmonary edema. In this context it is easy to see that excessive volumes may be detrimental. Goals for fluid administration for lung resection cases should be to provide what is physiologically necessary but little more. Similarly, efforts to reduce pulmonary artery pressures should be undertaken, and include avoiding hypoxia and hypercarbia.

If patients are to remain intubated postoperatively (common in major resection cases), a DLT must be changed to conventional ETT at the end of the case. In this regard a Univent tube or bronchial blocker through a standard ETT may be advantageous, as either may be left in at the end of the case.

Thoracic incisions are associated with a high degree of postoperative pain. Furthermore, the unavoidable chest movement during breathing exacerbates this pain. This causes patients to take shallow, ineffective breaths (“splinting”) which may compromise effective ventilation. Occurrence of these events in the setting of lung resection is clearly deleterious. To combat this problem aggressive pain control is instituted, often in the form of a thoracic epidural. An epidural, with a combination of dilute local anesthetic and narcotic, is perhaps the most effective measure at attenuating post op pain. In addition, sedating side effects from parenteral opioids can be avoided. The epidural is generally placed preoperatively, as patient cooperation may be poor in the post operative period. It should be considered for all but the most minor thoracic cases.

V. Cardiac tamponade and constrictive pericarditis

Cardiac tamponade can be managed surgically by a subxiphoid window, thoracotomy or complete median sternotomy. Procedures to relieve constrictive pericarditis include pericardial stripping or window.

Technique: general anesthesia.

Monitors: standard, plus arterial line. Further monitors may be useful but should not delay the case if possible.
IV access: at least one large IV.

Duration: 1-4 hrs.

Estimated blood loss: usually < 100cc, not taking into account evacuation of any existing blood.

Position: supine.

Special equipment: +/- TEE.

Special considerations:

Cardiac tamponnade is a physiologic state in which filling of the heart is constricted by the inappropriate presence of blood or other fluid around the heart. It may be seen in CT patients postoperatively due to continued bleeding. Other diseases which may cause tamponnade include pericardial effusions (e.g., from cancer), infections, trauma, MI, and autoimmune disorders. Constrictive pericarditis occurs when the pericardium becomes stiff and fibrotic, leading to impaired ventricular relaxation and filling. Clinically the two states can be quite similar.

Signs of tamponnade are frequently tested and include an equalization of diastolic pressures throughout the heart (rarely seen), hypotension, tachycardia, tachypnea, JVD, pulsus paradoxus (decrease of SBP more than 10mm Hg on inspiration) and an inability to lie flat. EKG findings include decreased voltages in all leads and diffuse ST segment elevation.

Anesthetic management of tamponnade is critically important and depends on the severity of the disease. Relatively stable patients with little to no tamponnade physiology are on one end of the spectrum, with a patient in extremis on the other end. Severe tamponnade should be alleviated with a pericardiocentesis or subxiphoid window prior to induction of anesthesia. Depending on the size and location of the offending fluid a subxiphoid window may be therapeutic or a larger incision may need to be made. The goals of anesthesia are 1) maintain CO and sympathetic tone, and 2) avoid positive pressure ventilation. Maintenance of CO and inotropy is an absolute. As most of our induction agents depress CO and sympathetic tone, they are poorly suited for a patient with symptomatic tamponnade. Etomidate or ketamine are the agents of choice, as both maintain CO and spontaneous ventilation. Bear in mind that ketamine is a negative inotrope in vivo; a patient with severe tamponnade, who is already maximally sympathetically driven, may display unmasked negative inotropic effects when ketamine is administered. After obtaining a deep state of anesthesia, intubation is done without muscle relaxant to maintain the ability to spontaneously ventilate. Fluid loading is usually advisable to aid diastolic filling. An arterial line is mandatory for all but the most stable of patients, and should be placed prior to the induction of anesthesia. Other monitors such as CVP, a PA catheter and the TEE may all be useful in providing further information.
and assisting with patient management, but unfortunately are time consuming and should not delay the case.
The Neuroanesthesia rotation and Neurophysiology

The neuroanesthesia rotation at UCSD is a two month, intense exposure to neurosurgical cases and the specific anesthetic demands they entail. It is done at some time during the CA-2 year. Throughout the 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 exposure to 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. 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 other 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 neuro resident, the daily schedule assignment is made specifically with the rotation in mind. The resident will be assigned the most complex or interesting neuro case of the day. In some cases this might simply be a large spine surgery with the use of motor/sensory evoked potentials. Many of these patients will be inpatients and thus require a preop eval the night before (see the preop section for more information). Furthermore, the expectation is that the neuro resident will finish his own case, especially since emergence is a critical portion of most neuroanesthesia procedures. Thus, 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. Spine surgeries are covered in the orthopedic section.

Neurophysiology and Anesthesia

Most if not all anesthetics have profound effects on neurophysiology. Delivering a rationale 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 within the field of neuroanesthesia. Due to our faculty, we enjoy an atypically 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 continuing 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 section. For more details on the effects of specific agents, see the drug section.

Cerebral blood flow and autoregulation

CBF is usually ~ 50ml/100g/min and is higher in grey matter, lower in white matter. Flow rates lower than 25ml/100g/min are associated with EEG slowing and neurologic impairment, below 20ml/100g/min with an isoelectric EEG, and below 10ml/100g/min with irreversible neurologic damage. At typical physiologic MAPs, cerebral blood flow is tightly autoregulated. Classic teachings describe fairly tight control between MAPs of 50-150 mm Hg. Within this range autoregulatory mechanisms keep CBF constant, despite potentially wide swings in blood pressure. Below or above this range CBF becomes pressure (or MAP) dependent, rising or falling with similar changes in blood pressure. Chronic hypertension shifts this curve to the right, with correspondingly higher limits for autoregulation.
CBF is directly linked to PaCO₂. Each 1mm Hg decrease in PaCO₂ corresponds with a 1.5ml/100g/min reduction in CBF. Conversely, increasing PaCO₂ corresponds with increasing CBF. This effect is due to CO₂ tension within the CSF and is not seen with acute changes in HCO₃ (which cannot cross the blood brain barrier). Clinically, most practitioners aim for a PaCO₂ between 25-30mm Hg to achieve favorable reductions in CBF. Prolonged changes in CSF CO₂ tension result in a change in CSF HCO₃ 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 CMRO₂ (see below), while hyperthermia has the opposite effects. Each 10°C decrease in temperature reduces CMRO₂ by 50%, and each 10°C increase doubles CMRO₂. Hypothermia produces an isoelectric EEG at around 20°C and is the most effective strategy for neuroprotection (see below), making it useful in situations of decreased or absent CBF (see deep hypothermic cardiac arrest in the cardiac anesthesia section).

**CMRO₂**

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. CMRO₂ 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 min of the insult.

Reduced metabolic needs of the brain correlate with reduced CMRO₂. In turn, reduced CMRO₂ entails less need for and a reduction in CBF. Reductions in CMRO₂ are thus favorable in reducing CBF requirements.

**Intracranial pressure**

The brain can be thought of as being enclosed within a rigid space (the skull). This space is occupied by brain tissue, interstitial fluid, blood, and CSF. An increase in any of the components that occupy the space (volume) must be offset by a decrease in another component, or there will be a necessary rise in intracranial pressure. Examples of increased occupancy include tumor, bleeding, or obstruction to CSF outflow.

Small increases in volume are generally compensated for quite well, with little to no increase in ICP. Compensatory mechanisms include shifting CSF to the spinal space, increased resorption and 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 (noncompliant space). Large increases in ICP can lead to brain ischemia and catastrophic herniation of brain tissue.

Cerebral perfusion pressure is MAP – CVP or ICP, which ever is higher. Typically, ICP is less than 10 mm Hg. It should be obvious that large increases in ICP have a deleterious effect on CPP.

Thus, in general reductions in CBF = reductions in CBV = decrease in ICP. Similarly, increases in CBF increase CBV and potentially ICP. Decreases in CMRO₂ cause a decrease in CBF and vice versa. Neuroanesthetic strategies aim to reduce CMRO₂ and CBF, reduce ICP and maintain MAP and CPP.

Effect of anesthetic agents on CBF, CMRO₂ and ICP

1. Inhalational anesthetics

Inhalational anesthetics alter the normal coupling of CBF and autoregulation in a dose dependent fashion. When used, inhalational anesthetics thus create a tendency for CBF to parallel MAP. Of the inhalational agents, halothane appears to have the greatest uncoupling effect. 1 MAC or less of desflurane, sevoflurane and isoflurane generally has little effect on CBF-autoregulation coupling.

CBF tends to increase with inhalational anesthetics at doses higher than 1 MAC, while CMRO₂ decreases. At doses < 1 MAC, both CBF and CMRO₂ decrease in a dose-dependent fashion. Above 1 MAC, CBF tends to increase, with no further reduction in CMRO₂.

Inhalational anesthetics are routinely used in neuroanesthetic cases. If ICP becomes a critical issue, it may be prudent to d/c all inhalational anesthetics to eliminate any possibility of autoregulation-uncoupling or unwanted increases in CBF.

Nitrous oxide typically has minimal effects. It may increase CBF on its own, however this effect is minimal and may even show a decrease when combined with other agents (especially IV agents). When not contraindicated, nitrous oxide is routinely employed in neuroanesthesia due to its rapid onset/offset, allowing for quick emergence and early neurologic testing.

2. Opioids

Opioids decrease CMRO₂ and CBF to a small extent. They are primarily used to blunt sympathetic responses to noxious stimuli, e.g. incision and laryngoscopy. Short acting opioids are preferable to allow early neurologic examination and avoid prolonged emergence. They are a mainstay of neuroanesthesia.

3. Barbiturates
Barbiturates are ideally suited agents for neuroanesthesia. They produce profound decreases in CBF and CMRO₂, with relatively more reduction in CMRO₂. Thus, supply tends to exceed demand. Furthermore, they have antiepileptic properties and can used to produce an isoelectric EEG for neuroprotection. Lastly, barbiturates cause an increase in CSF resorption.

4. Propofol and etomidate

These agents also reduce CMRO₂ and CBF and are good agents for neuroanesthesia. Propofol has anticonvulsant properties, while etomidate may activate seizure foci. Propofol has a short elimination half-life, enabling rapid neurologic assessment in the post op period.

5. Benzodiazepines

These agents reduce CBF and CMRO₂ but only to a small extent. They have significant anticonvulsant properties. Benzodiazepines may prolong emergence, especially when used as an infusion, and thus are best used as adjuncts or avoided entirely in neuroanesthesia.

6. Ketamine

Classically, ketamine is felt to increase CBF and thus potentially ICP. A few recent papers have challenged this thinking when ketamine is used as part of a balanced anesthetic. Nevertheless, dogma persists that ketamine increases ICP, and thus it should be avoided when ICP is an issue.

7. Succinylcholine

Succinylcholine can cause transient increases in ICP. A typical neuroanesthetic dilemma often presented is whether or not to use succinylcholine in a situation of increased ICP. Bear in mind this increase is usually small and far outweighed by increases due to inability to ventilate or difficult laryngoscopy and easily attenuated with anesthetic agents. Thus, in situations where the advantages of succinylcholine are called for (e.g., facilitating intubation), it remains an appropriate drug.

Neuroprotective techniques

1. Hypothermia

- even slight hypothermia 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, arrhythmia)
2. Maintenance of CPP
   - MAP should be maintained so as to ensure adequate CPP
   - Reduction of ICP as below

3. Reduction in CMRO₂
   - reduction of metabolic demand is beneficial
   - many anesthetic agents above can produce an isoelectric EEG and drastically reduced metabolic demand. However, there is still a basal metabolic demand which cannot be eliminated (unlike with extreme hypothermia)
   - see a text for “barbiturate coma” for more information

4. avoid hyper or hypoglycemia

5. avoid hypoxia

Techniques to reduce ICP

These techniques all center around reducing the volume of one of the three components of the intracranial space mentioned above- brain matter, interstitial fluid, blood and CSF. Some techniques may be impractical while others are under direct surgical, as opposed to anesthetic control.

1. Reduction in brain volume
   - Examples include removal of tumor or offending mass by the surgeon. Typically this is not under our control and obviously a non factor for reducing ICP prior to surgery. Rarely, a craniectomy may be performed to create “more space” for remaining tissue and relieve ICP.

2. Reduction in interstitial fluid volume
   - osmotic diuretics- mannitol is the prototypical example. It increases serum osmolality, which draws intracellular water into the intravascular space and off brain tissue, decreasing brain volume. Typically there is first a transient increase in blood volume which may worsen ICP, followed by hypotension from vasodilating properties and reduced intravascular volume from diuresis. Mannitol works quickly 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. By decreasing brain volume, there is more “room” for an aneurysm, AVM or hematoma to expand, theoretically increasing the risk of bleeding.
   - Loop diuretics- these are slower in onset than mannitol but are useful adjuncts as they have a synergistic effect and reduce CSF production.
3. Reduction in blood volume

- choice of anesthetic agent to reduce CBF and CMRO₂
- hyperventilation
- avoidance of hypoxemia
- avoidance of hypertension (since autoregulation is typically impaired under general anesthesia, and because extremes of hypertension increase CBF); hypotension should also be avoided as it implies decrease in CPP
- facilitating cerebral blood drainage- head up, avoiding increases in jugular venous pressure (circumferential ETT ties, extreme flexion or rotation of the head, IJ catheters, PEEP)

4. Reduction in CSF

- loop diuretics
- choice of anesthetic agent (e.g., barbiturates)
- ventriculostomy or other drains by the surgeon
- other agents to reduce production (acetazolamide, steroids)

Anesthetics and evoked potentials

Evoked potentials are a form of electrophysiologic monitoring used to test the integrity of nerves that may be compromised by the surgical procedure. Brainstem auditory or visual responses or sensory +/- motor evoked potentials can all be tested/monitored. Typically, the surgeons employ an outside technician to monitor the evoked potentials. In the past several members of our own anesthesia monitoring crew also handled this duty. In general, somatosensory evoked potentials (SSEP) are employed during surgeries that could compromise the spinal cord (e.g., spine surgery, aortic surgery), visual evokes (VER) are used to monitor the upper brainstem and optic nerve, and brainstem auditory evokes (BAER) are used for surgeries around the 8th cranial nerve and posterior fossa.

Very generally, evoked responses rely on stimulating the particular nerve in question and monitoring for cortical response. A “good” or unchanging response implies an intact neural pathway, while changes in the response could by a signal of impending nerve damage. Taking the BAER 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.

Similarly, SSEP rely on stimulating mixed (motor and sensory) nerves and monitoring efferent or afferent responses. In the case of motor responses stimulus is applied above the area of concern (e.g., the spinal cord) and the efferent response is measured.
The evoked responses are classified according to their latency (time from stimulus to response) and amplitude (height of the response). Changes in either parameter may be transient and expected (e.g., irrigation of the area near the nerve) or more sustained and worrisome, as with surgical irritation or damage. Most anesthetics affect the characteristics of the evoked response, and must be adjusted or omitted entirely. Other than interventions we can make in response to a worrisome evoked signal (e.g., correct acidosis, notify surgeon), much of our responsibility during the use of evoked potentials will be to utilize an anesthetic that has little effect on the monitoring and signals. Generally, the technicians also understand the necessary anesthetic limitations, and preop discussion with them can help you plan the anesthetic accordingly and avoid later hassles.

Briefly, all inhalational agents decrease amplitude and increase latency. This effect is usually minimal at ½ MAC or less 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. VER are most affected by our anesthetic agents, then SSEPs, then BER (BER are the most robust and least likely to be affected). Thus, a typical “evoked anesthetic” may be similar to a TIVA- opioid and IV sedative infusions, +/- a small amount of inhalational agent. If motor evokes are to be employed, NMBAs must be omitted from the anesthetic for obvious reasons. Generally, the specific anesthetic plan should be discussed with your attending and the monitoring technician to make sure everyone is on the same page.
Anesthesia for neurosurgery

The following are brief descriptions of the typical neurosurgical procedures encountered at UCSD, as well as the anesthetic implications. Spine surgeries are covered under the orthopedic section. Basic neurophysiology and anesthesia is addressed in the appropriate section.

Neuroanesthesia for vascular malformations

Examples include aneurysm clippings and resection of arterio-venous malformations.

Technique: general anesthesia.

Monitors: standard, plus arterial line. CVP may be a useful adjunct to guide fluid therapy and if mannitol is to be used. Urinary catheter.

IV access: at least one large IV. Catastrophic bleeding, although unlikely, is a possibility.

Duration: 4-6 hrs, potentially more for complicated cases.

Estimated blood loss: 100cc, much more if there is unanticipated/uncontrollable bleeding.

Position: supine, lateral or semilateral; depends on location of the aneurysm. Head away from anesthesiologist.

Special equipment: precordial Doppler if increased risk of venous air embolism (see posterior fossa craniotomy), as well as CVP for same. IV infusions to rapidly control BP. At times a ventriculostomy or bolt may be employed by the neurosurgeons to monitor ICP (see below).

Special considerations:

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 ENT surgery and emergency craniotomy room setup, and include limited access to the airway, increased risk of airway dislodgement due to close surgical proximity, and hazards with turning the OR table 180 degrees.

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 potentially 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 a CVP is not as clear cut, and many of our craniotomies are performed without one. Advantages of a CVP include ability to monitor central pressures, having large central access, and the ability to aspirate air in situations of venous air embolism. The disadvantages of placing a CVP are myriad and include infection, bleeding, and pneumothorax. Use of a CVP 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, see more below). The potential for large blood loss in AVM or aneurysm surgery demands large IV, but not necessarily central access. Similarly, blood products should be available prior to surgery.

Current neurosurgical management involves early (within 72 hrs) clipping of ruptured aneurysms/SAH or late (after 2 weeks). Between these time periods is the window for cerebral vasospasm (generally 4-14 days post bleed). Vasospasm is thought to be a response to blood clot around cerebral vessels, and can lead to brain ischemia and further cerebral damage. Treatment of vasospasm involves the use of the calcium channel blocker nimodipine, as well as “triple H therapy”- Hypertension, Hyperdynamic circulation, and Hemodilution. Triple H therapy is effected by volume loading with NS and the use of vasopressors such as dopamine. Because triple H therapy may increase the likelihood of rebleeding, most neurosurgeons attempt to clip 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 reduce the transmural pressure around the site of rupture, increasing the relative “driving pressure” and the chance of rebleeding. 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 camino bolt or ventriculostomy. The ventriculostomy has the added advantage of being able to drain CSF directly, allowing for reductions in ICP. These monitors are generally placed pre or intraoperatively by the neurosurgeons, but in the OR are monitored by us. Close communication with the surgeons is necessary for specific management goals (e.g., target 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, placement of “pins” (surgical headframe) by the surgeon, 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. 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 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, cranial and dural opening. Having quick, titratable IV agents inline is mandatory, e.g., a nitroprusside and phenylephrine infusion to lower or raise the BP, respectively.

Additional anesthetic goals of a standard craniotomy are avoidance of hypercarbia and hypoxia, avoiding increases in ICP/minimizing brain volume to create an optimal surgical field, avoiding spontaneous patient movement, a smooth emergence (coughing and straining are especially deleterious with fresh neurosurgical incisions), and a “quick wakeup” to allow early neurologic assessment. Clearly, there are times where some of these goals may be difficult to achieve, and sometimes one goal is in direct conflict with another (e.g., smooth wakeup vs. quick wakeup). Strategies to achieve these goals are discussed below.

Measures and conditions that increase/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 less than 0.5 MAC of inhaled agent, coupled with N2O (if not contraindicated), propofol and opioid infusions with muscle relaxation. This combination allows relatively quick offset of anesthesia, ensures paralysis, and should not increase ICP. Propofol in particular is favored by our neurosurgeons for its beneficial effects on ICP, CBF and CMRO2. You will often find yourself being 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” (Total IV Anesthetic) with propofol, opioid and muscle relaxant. A significant opioid base should allow for a smooth emergence, as well as blunting hemodynamic responses to stimulation (see above). Depending on the context-sensitive half life of the opioid chosen (see the drug section), it may be necessary to d/c 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 and avoiding stimulation of the patient during critical portions (e.g., stage II). Unfortunately, a heavy dose of opioid may slow that same emergence. Tailoring the anesthetic to allow for both requires considerable experience, but in general propofol infusions should be d/c’d about 30min before the end of the case and fentanyl infusions about 1 hr 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 (e.g., severely depressed mental status at baseline, severely ill patient, see next section).

**Emergent craniotomy for the head injured/trauma/raised ICP patient**

A significant portion of emergent craniotomies involve situations with acutely raised ICP, usually due to intracerebral 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 “anesthesia for the trauma patient” section. Typical cases include: acute subdural or epidural hematoma evacuation (burr holes or formal craniotomy), interparenchymal hematoma evacuation, chronic subdural evacuation, potentially within the setting of trauma.

Technique: General anesthesia.

Monitors: standard, plus arterial line. CVP may be necessary/desirable. Urinary catheter.

IV access: large.

Duration: 1 – 4 hrs.

Estimated blood loss: 200cc to potentially much more depending on the injury.

Position: typically supine, **head away from the anesthesiologist**.

Special equipment: potential ICP monitor placed by neurosurgeons.

Special considerations:

Several considerations unique to the trauma patient include: aspiration risk or full stomach, potential for cervical spine instability, an obtunded or combative patient, and hypovolemia or under- resuscitation. 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 inline stabilization and cricoid pressure is used. Control of the airway must proceed with attenuation of profound increases and decreases in BP (and thus CPP and ICP). Arterial and venous access must also be achieved as soon as possible. Clearly this is a multiperson task that often involves several members of the anesthesia team. Do not be afraid to call for help if needed.

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 itself- that is, the surgery should not be delayed for want of better IV access, an arterial line, etc. The first priority is to allow the surgery to proceed. Other necessary measures such as fluid resuscitation and obtaining arterial access can proceed concurrently while surgery is underway. Note- this is much different
than in an elective craniotomy where 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 and 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 “just” an intracranial bleed (e.g., chronic subdural hematoma) may be extubatable after the surgery is concluded, which can be discussed on a case by case basis with the neurosurgeons.

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.

**Craniotomies for mass lesions**

Examples include removal of tumor or other mass (e.g., infectious), including pituitary tumors (e.g., transsphenoidal resection of sellar mass).

Technique: General anesthesia.

Monitors: standard, plus arterial line. Urinary catheter. CVP may be useful but is not necessary.

IV access: one large IV should suffice.

Duration: 2-6hrs.

Estimated blood loss: 100-500cc. Typically less than for vascular malformations and intracerebral hemorrhages, and potential for catastrophic bleeding is less (but still present).

Position: generally supine or lateral, +/- head turned to side. **Head away from anesthesiologist** (exception: 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 hypercarbia and hypoxia, tight control of blood pressure, smooth induction and emergence, and early neurological examination. Depending on the growth or expansion of the intracranial
lesion, the patient may have signs or symptoms of increased ICP which should be treated accordingly.

The transsphenoidal approach to pituitary (sellar) tumors 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. We are lucky here at UCSD to have generally fine neurosurgeons with good technique. As such, this operation is typically 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 are often 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 of craniotomies still apply, such as potential measures to “shrink the brain” (e.g., hyperventilation, propofol) and 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 potential perioperative conditions for transsphenoidal resection of pituitary mass center around the mass itself. The most common hypersecretory pituitary mass is a prolactinoma, but masses secreting ACTH, TSH, GH and others are possible. 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 thus necessary and frequently helpful. Lastly, transaction of the pituitary stalk can lead to panhypopituitarism.

Anesthesia for posterior fossa procedures

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 5th cranial nerve “Jenetta procedure”).

Technique: general anesthesia.

Monitors: standard, plus arterial line. CVP generally useful, especially in case of venous air embolus. Urinary catheter. Precordial Doppler.
IV access: at least one large IV.

Duration: 4-6 hrs.

Estimated blood loss: usually less than 300cc; there is potentially for large hemorrhage.

Position: lateral, semilateral, prone, or sitting, head away from 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. Essentially, a venous air embolism 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 thus facilitate entrainment of air. Any time the involved sinuses are above the level of the heart the risk of air entrainment is increased. Thus, the sitting position is most likely to encounter an air embolism. It is employed by the surgeons to facilitate exposure (rare in this institution). However, every posterior fossa craniotomy involves some elevation of the head above the heart.

Signs of venous air embolism include hypotension, hypoxia, increased dead space ventilation, circulatory arrest, and paradoxical embolism if a patent foramen ovale or R-L circulatory connection 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 Et nitrogen 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, “rapier-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).

Treatment of VAE consists of 1) notifying the surgeon who can pack the field with gauze and flood with saline, 2) raising cerebral venous pressure with IV fluids, lowering the head and jugular compression, 3) evacuation of the air by aspiration via a central venous catheter, 4) supportive measures if needed (CPR, pressors), 5) d/c nitrous oxide and increase FiO2 to 100%. Turning the patient to the left lateral decubitus position may keep the air in the right atrium and decrease passage to the RV and pulmonary circulation.
Other potential complications specific to positioning for posterior fossa surgeries (especially the sitting position) include swelling of the tongue and pharyngeal structures, quadriplegia, and pneumocephalus. In regards to the first two complications, excessive neck flexion is thought to play a role. Ensure that the neck is not completely flexed in these patients prior to the start of surgery (generally at least two finger breadths 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 as is common in many craniotomies. N2O 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 N2O 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 venous pooling in the lower extremities and possible 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 atropine in addition to removal of the offending surgical stimuli.

Injury to cranial nerves arising from the brainstem is also a potential complication of these surgeries. The post operative deficit depends on the nerve involved. One feared complication is damage to the 9th cranial nerve which may result in inability to maintain a patent airway. Evoked potentials can be employed to monitor for nerve damage (e.g., auditory evoked potentials for acoustic neuroma resection). If motor potentials or electromyography is used (e.g., facial nerve dissection) neuromuscular blockade may need to be omitted.

**Anesthesia for minor neurosurgical procedures, including stereotactic surgery**

Examples include placement, revision or removal of a ventriculoperitoneal shunt or lumboperitoneal shunt and stereotactic surgery, e.g., placement of a deep brain stimulator.

**Technique:** general or MAC/local for stereotactic surgery.

**Monitors:** standard.

**IV access:** one IV.
Duration: around 1 hr for shunts, 4-6 hrs for DBS.

Estimated blood loss: < 100cc.

Position: Supine or lateral for shunts, bed 90 or 180 degrees away from the anesthesiologist. Stereotactic surgery is typically done with the patient sitting up, head fixed in a rigid frame and away from the anesthesiologist (see more below).

Special equipment: none.

Special considerations:

VP or LP shunts are minor procedures. Patients coming for placement of a shunt generally have chronic hydrocephalus, for which placement of the shunt will drain intracranial fluid off and reduce ICP. These patients may have signs and symptoms of raised ICP, which include nausea/vomiting, confusion, ataxia or in the acute phase even papilledema. Shunt revisions are typically performed on previously functioning shunts which have now become obstructed or ceased to function. 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 are generally not required. Of course, it is always desirable to avoid overt hypercapnia or hypoxia.

Stereotactic surgery is performed for diagnostic purposes, intractable epilepsy, or for dyskinetic disorders such as Parkinson’s disease. They are performed under MAC/local, with the goal being an awake patient that can respond or interact with the surgeon during critical portions of the procedure. As the brain itself is insensate, the only anesthesia that is required is during skin incision to opening of the dura. These portions are carried out with generous local anesthesia by the surgeon and IV sedation by us. Once the painful portions of the procedure are concluded the sedation is turned off to allow an awake, interactive patient. Sedation and further local anesthesia is also generally employed for closure.

Because any sort of movement could be catastrophic, all patients have their heads fixed in a stereotactic frame for these procedures. These frames hold the head rigidly in place and also serve as guides for image-guided portions of the procedure. Because of this frame, and because the patients are positioned sitting, head away from the anesthesiologist, airway misadventures can be disastrous. Intense monitoring and judicious use of medications during the sedation phases of the procedure are mandatory to ensure a patent airway.

Stereotactic procedures are typified by long periods of boredom (since the patient is awake, generally stable and the surgery is minimally invasive) with bursts of stress while titrating in sedation and monitoring the patient for airway issues.
The SICU rotation

The SICU experience consists of two one-month blocks taken during the CA1 and 2 years. During this time you are part of the “critical care team” in the SICU (not to be confused with trauma, neuro or any other host of teams with patients in the SICU) that consists of 1-2 anesthesia residents, 1-2 surgery residents, medical students, a trauma fellow, and an attending. Both surgery and anesthesia attendings cover the ICU. While on the SICU rotation, you are not part of the MOR call pool and generally not responsible for typical “anesthesia” 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. Thus, with the exception of OB/GYN, cardiac, ENT and ortho patients, the SICU team does not function as the primary care team but rather as consultants. All non-primary patients are rounded on, plans are made and recommendations communicated to the primary team, but actual order writing and administration of care is the responsibility of the primary team. Midnight calls from the nurses are directed to the primary, not the SICU team. Clearly for those patients who are actually part of the SICU primary service this does not hold. Therefore, the UCSD ICU experience can be less onerous than at other institutions simply by virtue of the fact that we don’t have to care for as many 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. Rounds are then generally made either with the fellow, the attending, or both (usually 8:30am but differs according to the attending). Notes and recommendations are written, and any necessary orders or management of primary patients are carried out. There is usually a lecture around 11am – noon, often with a member of the team presenting on a topic. At this point typically the non-call members sign out to the person on call. Thus, non-call days are generally very light, ending around 1pm.

Other responsibilities of the rotation are to receive consults from other teams and assist with placement of lines 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 anesthesia residents we have unique responsibilities while on this rotation that the surgical members do not have. They include:

- carrying the code pager and responding to it
- doing OR resuscitation cases
- making sure the code bags and emergency rooms are set up
- fulfilling whatever request our (anesthesia) attendings may have for us
More information on the code pager, emergency room setup 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 anesthesia attending *du jour* has the discretion to decide who does the case—be it a MOR resident, the SICU resident, or other. Expect to be involved or even to be the primary resident in any trauma resuscitation case. 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 ICU rotation you are still an anesthesia resident, part of our department, and thus subject to its whims. If a conflict arises between anesthesia and SICU duties it can be discussed amongst the appropriate attendings.

Residents from the Naval Hospital at Balboa rotate with us periodically. The primary purpose of their rotation is to gain experience with emergency intubations, resuscitation cases, and burn cases. They will spend a fair amount of time in the operating rooms doing cases as a regular resident. When they take call their responsibility is to hold the code pager, set up the emergency rooms, and respond to any airway emergencies or trauma cases. Thus, if the Navy resident is on call a large part of the SICU resident’s responsibilities are passed on.

The SICU experience represents a good learning opportunity in a fairly relaxed environment. The majority of the attendings are very pleasant, easy to work with, and good teachers. Many people find the lack 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 2 am pages. Of course, the downside to this is that perhaps the learning experience is not as rigorous as it would be in a completely closed unit. That being the case, make the most of the opportunity to learn from rounds and enjoy the frequent early days.
Pain rotation

The pain rotation consists of one mandatory month during the CA1 or CA2 year. It is also possible to take pain during the CA3 year as an elective. During the month you will see patients in pain clinic, observe and perform therapeutic procedures, and take pager call. These duties are usually shared between the resident rotating through pain, the pain fellows, and if present the CA3 taking the elective. The week generally consists of 2.5 days of clinic, and 2.5 days of procedures. Most of the experience is at Thornton hospital, although occasionally on certain days you may be assigned to either the clinic or procedures at the VA. 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 done, with the obvious emphasis being on the patient’s pain history. After these patients are seen they should be presenting to the attending du jour, who will then complete the interview and decide on the best course of treatment. Returning patients are generally less demanding, and are usually in the clinic for follow up after a procedure, a medication refill, or for general maintenance. In these cases you can consult the old notes in the computer system to get a quick overview of the patient. These cases should also be presented to the attending after you see them, but the visit is usually much quicker. While in clinic you are also responsible for dictating the note when the visit is complete- the procedure for this will be shown to you when you start the 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 will let you perform some of the injections. Depending on your performance and the attending you may eventually get to perform virtually every procedure that comes through the door. The various blocks performed will be explained during the rotation.

Pain call is pager call, meaning you are not required to be in house overnight. Patients will call the operator occasionally to speak to the pain physician on call, who will then forward the number to you. There is a dedicated cellular phone that will be given to you during the rotation so you can call the patients back, without having to use a number you don’t want them to track. Unfortunately, a small percentage of the pain patients have the potential to harass you if they learn your home phone number, even after you finish the rotation. Thus, it is advisable to use the “pain phone”. It is the policy of the pain department that 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. If someone’s pain is intractable they simply must go to an ER for evaluation, as they cannot be completely evaluated over the phone. Of course, true medical emergencies must be asked about, and if present the patient should again 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.
While on call, all inpatient consults will be directed to you. Consults received before 5pm should be seen that day. What this means is when you are on call the days may be long, as you generally don’t finish before 5pm, and then may have to see consults at both Hillcrest and Thornton. After evaluating the patient, the attending on call can be consulted for a definitive plan. The attending is also there to answer any questions you may have. Consults received after 5pm can be seen the next day. All inpatient consults being followed should be added to the pain rounds list (this will be explained), and the other members of the team should be notified so they know who to round on in the morning (also to be explained when the rotation starts).
The pediatric experience at UCSD

Authors Note: The following information was correct as of the 2007-8 academic year. The structure of the rotation may have changed significantly since that time. Explicit instructions and information will be provided to you at the start of your rotation. Obviously, defer to this information in case of discrepancies.

The vast majority of our experience with pediatric anesthesia takes place at Children’s Hospital of San Diego. While there are periodic pediatric cases at Hillcrest (and these children tend to be quite ill), CHSD provides intensive, daily exposure during the rotation. A one month, mandatory rotation is done during the second year, with an option to come back during the CA3 year as an elective rotation. Additionally, several months can be spent at Children’s Hospital in LA as a CA3 elective. Pediatric patients differ markedly in both physiology and the typical type of case encountered from adults, and that information will not be addressed here. What follows are the expectations and requirements during the CHSD experience.

The rotation at CHSD is quite different than almost every other experience during residency. Each room is staffed by an attending who expects to be doing the case himself, and there are no formal room assignments for the residents. Residents are generally free to choose which room and 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 us 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 rooms to start the other case. Another day you may feel like doing many outpatient cases, and be able to wander over to the same day surgery area. On still another day you may feel like doing a pediatric heart case. 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, well… 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. By and large most of the attendings at CHSD like to work with residents, are more than willing to teach, and are flexible with letting us jump from room to room. In addition, several of “our” UCSD attendings also work at CHSD.

The downside of this freedom is that the learning may be quite unstructured. Often, you have no idea what you’ll be doing the next day (unless you check the schedule the night before and start thinking about which room you want to be in) and thus can’t tailor your reading prior to a case. Most of the attendings prefer the residents not participate in the preop eval of the patients, as we slow the process down. You will quickly see that CHSD 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 it quickly becomes apparent who won’t want to work with you. CHSD is definitely 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.

The “diamond” or call resident is an exception to all of the above. Call is generally q4. The resident assigned to call usually cannot pick from all the rooms available but rather must be in one of the first four rooms which face the main OR board. This is to allow the attending they are with to become the floor person and run the board. This attending will field all the phone calls and make the necessary assignment changes at the board while the resident sits in the room. The proximity of the first four ORs to the board allows the attending to run back to the room in case of an emergency. As the diamond resident you may at times feel used in the truest sense of the word. It is not uncommon to be abandoned in a room for hours at a time while the attending runs the floor. In general the diamond resident is expected to stay until 5pm if q3, 7pm if q4, and be available by pager overnight. Typically the attendings will not call you back in for a late night case, and some will often let you leave early. When on call during the weekend, the usual expectation is that you will show up on Saturday mornings to do cases (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 have to come in on Saturday, but not on Sunday.

There are other notable impacts of the “maximizing productivity” stance of CHSD. Almost every case is extubated in the PACU by the PACU nurses and not the MDs. A typical case will end with the patient breathing spontaneously, still intubated, and brought to PACU. 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. However, while most of the PACU nurses are excellent, there have been several near disasters when a patient was inappropriately managed with an MD 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 CHSD defines the order in which the attendings may leave, with highest call having to stay latest. This is a rotational system which is generally balanced. The upside of being a high call is that the attendings get first choice on rooms, and can bump any lower call attending during the day. This is important because the attendings get paid proportionally to what they bill. Thus, high call attendings typically start the day in the rapid turnover, good insurance rooms, and as the day progresses may randomly jump to another room if a more lucrative opportunity presents itself. The impact on the residents is that you may work with several attendings during the day even if you stay in the same room. Furthermore, if you want to work with the same attending you may have to follow him 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 CHSD 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 are to 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. I won’t comment on this procedure except to say that the attendings typically 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 and figuring out who the attending assigned to that room is to ask if you can work with him. During my months at CHSD once I decided on a room I would usually try to set up in minimal fashion, perhaps by getting a tube ready or drawing up some drugs. As previously stated most of the attendings don’t even expect that much from the residents. This can be doubly frustrating because from the resident point of view you are doing new cases, on a scary class of patient with little to no experience. One way to alleviate these concerns is to be completely prepared and set up beforehand, but this is almost antithetical to the way most cases are done at Children’s. It definitely was annoying to show up thirty minutes early, draw up all the necessary drugs and be completely prepared, and then have the attending question why I had to draw up “all those drugs”. Bottom line- do what you have to do to feel comfortable, and remember that being over prepared is always better than the converse.

In summary, CHSD can be a great experience, but it takes initiative on the part of the resident. I personally would recommend as many months as possible, simply because we don’t do many pediatric cases outside of Children’s. A certain comfort level and expertise can only be obtained with repeated, daily exposure and Children’s does give us that.