INTRODUCTION TO THE CARDIAC ROOM

Introduction and Overview

Welcome to the Division of Cardiac Anesthesia at UCSD. This outline will serve as a framework for which you can use as a starting point for your preparation for the rotation. It will hopefully serve as a day to day clinical guide. It is by no means comprehensive and does not substitute your reading of the suggested textbooks and review of the recommended literature.

We hope you will come to appreciate the complexity of your patients’ disease processes and how an understanding of their physiology affects anesthetic management. From the preoperative evaluation, to the induction and maintenance of anesthesia, and commencement and separation from bypass, care of the cardiac patient requires you to utilize all of your skills as a well-rounded physician to specifically tailor your anesthetic to each patient. This rotation will require the highest level of preparation, commitment, and work ethic. Please take the extra time to come to the heart room prepared. You will only get the best experience having fully evaluated your patient personally and developed your own anesthetic plan prior to bringing your patient to the heart room. It is critical that you play an active role in your education. In return, we hope to provide you with a very rewarding and educational experience during your time with us.

It must be stressed that the care of the cardiac patient deserves your highest respect. This means conducting yourself professionally at all times and interacting with the patients and their families with the highest degree of courtesy. Remember, you are representing not only your department and division of cardiac anesthesia; you are an essential member of the cardiovascular institute, which serves as a major referral center nationwide for complex surgical procedures. You undoubtedly are among the best recruits of all the residency programs and represent the upper echelon of all residents in the country. We are proud that you are the face of the cardiac team.

The heart room also requires your highest respect. In the heart room, effective and clear communication with all members of the team is essential. Your anesthetic management is often dependent on knowledge of critical steps of the surgical procedure. Conversely, the surgical procedure is often dependent on our anesthetic care. Accordingly, clear and effective communication must always be exercised and repeating all requests in a closed loop fashion is mandatory! This means clearly repeating a request so that ALL members of the team (including surgeon, perfusionist, and nursing staff) can hear and respond. For example, if the surgeon requests that the table be raised, you close the loop by simply stating, “raising the table” prior to the actually elevating the bed. By repeating key information in a clear manner, we will minimize any errors related to a breakdown in communication. Again, ongoing communication with all members of the team is critical in the heart room.

During your time with us, you will be given a written and oral examination (oral board format) provided by one of the faculty. This will be very informal and will serve as an objective means to identify areas of strengths and weaknesses regarding your knowledge base, clinical management, and judgment. It will also help us improve the educational curriculum in order for you to get the best experience possible. Your residency director and the division chief will be given feedback on your experience.

We hope you enjoy your time with us. Please do not hesitate to ask questions regarding ANY matter or concern. The heart room is not the place to be afraid to ask questions. In addition to your attending anesthesiologist and surgeon, there is often a cardiac anesthesia/surgery fellow in addition to an echo resident present to help you. You will also find that the nursing, perfusion and anesthesia tech staff is also very knowledgeable. If you don’t know something ask!!!!!
Preoperative Evaluation of the Cardiac Patient Presenting for Surgery

It is STRONGLY recommended that you conduct your own evaluation prior to calling your staff the evening prior to surgery. This establishes you as the cardiac anesthesiologist taking care of the patient in the heart room. You will gain experience in relating a patient’s preoperative condition to the anesthetic and surgery. By knowing your patient better than any other member of the heart team, you will have earned the respect from all members of the OR. But more importantly, you will be a better and more complete perioperative physician. It is MANDATORY that you initiate the communication with your staff to discuss your anesthetic evaluation and plan.

In addition to your complete history and physical examination. Please afford specific attention to the following details as they may directly affect your anesthetic management. A review of systems eliciting the below history may prove very important perioperatively.

1. History of bleeding or coagulopathy. Please investigate all coagulation studies, platelet counts, marked hematocrit trends, history of transfusion, history of anemia, liver dysfunction, history of taking platelet inhibitors or anticoagulants, and congenital or acquired clotting disorders. For example, a history of heparin induced thrombocytopenia, Factor V leiden, antithrombin III deficiency, sickle cell anemia, hemophilia, or history of taking clopidogrel (plavix) or tirofiban (Aggrastat) are important preoperative factors that will directly affect your management.

2. History of esophageal pathology or symptoms, dysphagia, difficulty swallowing food or liquids, history of gastric or esophageal surgery, history of chest radiation, history of esophageal strictures, masses, or diverticuli. A history of difficulty passing the TEE probe is also important. Please notify your staff anesthesiologist regarding any contraindications to transesophageal echocardiography as above.

3. Evaluate for the presence of co-existing cerebrovascular disease, including auscultation for carotid bruits and review of the carotid ultrasound studies.

4. Review pulmonary function in all patients including history of COPD/Asthma and make sure any coexisting pulmonary disease is medically optimized. Please review all studies including X-ray, CT scans, pulmonary function tests etc.

5. Review all pertinent laboratory data and other tests including ECG, cardiac catheterization data, echocardiogram, and stress tests. The ECG may show evidence of a Left bundle branch block which may predispose you patient to cardiac arrest when introducing a PA catheter. Pay attention to coagulation studies, elevated creatinine data, potassium levels, LFTs, and other hematology and chemistries. Coexisting valvular abnormalities on echocardiography may mandate alter the induction and maintenance of anesthesia and the surgical procedure. For patients undergoing valve procedures, please review any evidence for infections, particularly urinary tract infections and their appropriate antibiotic coverage as this may delay surgery or mandate a change in antibiotic therapy.
6. Cardiovascular evaluation. It is a good habit to evaluate both radial pulses and obtain blood pressure readings in both extremities as some patients may have different readings due to arterial stenosis. For example, some patients may even lack a radial artery due to prior harvesting for CABG. This may require us to cannulate the femoral artery for blood pressure measurements. If the left arm has a lower pressure, this may affect the surgical harvest of the left internal mammary artery for bypass grafting.

7. Investigate all studies relating to baseline renal function and note any history of renal failure or insufficiency or any conditions predisposing your patient to renal dysfunction (i.e. renal artery stenosis, diabetes, hx of hemodialysis, NSAIDs, ACE inhibitors etc.). Determine baseline chemistries and BUN/Creatinine.

8. Please review all medications taken as an outpatient and highlight any changes in the medication list while inpatient. Pay special attention to drugs affecting coagulopathy (i.e. heparin infusion, clopidogrel, ASA, enoxaparin, etc.) Highlight any allergies and the specific reaction.

9. Right Heart catheterization. It is important to note the right and left sided filling pressures


11. Cardiac catheterization and anatomy.

Preoperative Holding Area

The patient should arrive in the holding area by between 530-600am. You should get into the habit of re-identifying your patient’s name and procedure. In addition, please pay attention to the following:

1. Vital Signs
2. Any problems or changes that may have arisen overnight (i.e. changes in medications, or acceleration of angina, worsening of pulmonary function etc.)
3. Identify any ongoing medications that the patient is currently receiving (i.e. heparin infusion, vasoactive infusions, prostaglandin therapy etc.). For instance, patients with critical coronary stenosis left main or left main equivalent should remain on the heparin infusion.
4. Perform a quick physical examination to note any changes from the night before. Assess level of consciousness, changes in cardiac or respiratory status, and note intravenous access.

After briefly assessing for any changes. Place an intravenous catheter for induction of anesthesia. If an IV has already been placed in situ, be careful to CONFIRM its proper function. If you are not satisfied with the in situ IV, place another access line. After completing IV access, place an arterial line as necessary. Be very mindful that certain patients may not tolerate any degree of sedation for your access lines. More specifically, it is generally NOT RECOMMENDED that you administer sedatives to patients presenting for pulmonary thromboendarterectomy until the induction of anesthesia. Similarly, unmonitored changes in preload/afterload or blocking the sympathetic response with opiates in patients with severe valvular disease (i.e. aortic or mitral stenosis), severe heart failure, or tamponade may cause cardiac compromise. Keep in mind that even the most subtle change in your patient’s respiratory pattern or hemodynamics may cause cardiovascular instability. On the other hand, patients undergoing CABG surgery may benefit from sedation as the stress and anxiety provoked during line placement may precipitate myocardial ischemia. You are responsible for understanding your patient’s physiological condition and any sedation administered requires your bedside vigilance. If you have any doubt about providing appropriate sedation, please ASK your attending. Also keep in mind that family members may
be present during your procedures. It is STRONGLY RECOMMENDED that you escort them to the waiting area and close the drapes to insure patient privacy. This will also ease the time pressure of your morning routine and prevent family members from interrupting your procedures. After completion, please allow the family members to finally greet their loved one prior to heading to the OR. We realize that there is considerable time pressure in the morning but the goal is to bring the patient back to the OR no later than 630 a.m.

If you are receiving a patient in the ICU, make sure to provide a transport monitor to provide the same level of monitoring that is achieved in the ICU. Make sure you have adequate intravenous access and personally check that all necessary medications be continued during the transport. You should have all necessary drugs to resuscitate the patient in the event of cardiac collapse or decompensation. We are assuming the complete care of the cardiac patient from the second you leave the patient’s ICU bed. Transporting a patient requires an even heightened level of vigilance being that there may be several “distracting” or uncontrollable variables that may adversely affect you as you transport the patient.

INDUCTION AND MAINTENANCE OF ANESTHESIA

Once you arrive in the operating room, make sure you safely transport your patient to the OR bed. Some patients may not tolerate the physical activity of moving themselves and require the team to physically transport them over on a board (i.e. patient in active myocardial ischemia, critical aortic stenosis, decompensated PTE patient). After safely transporting the patient, place all of your monitors in a timely manner. There will be an anesthesia technician to help you with this. In the meantime, it is a good idea to begin envisioning your induction sequence (i.e. hemodynamic goals, airway management, drug dosing, etc.) during the preoxgenation period. Prior to the induction of anesthesia, it is a good idea to review your baseline hemodynamics, vital signs, heart rate, rhythm, ST segments, and any other data (i.e. preoperative PVR, cardiac output, pulmonary pressures, etc.) that may come into play. Based on your assessment, make any necessary adjustments prior to induction. For instance, if preload maximization or heart rate optimization is necessary prior to induction, optimize your patient’s physiology prior to inducing.

The following describes the TRADITIONAL approach to the induction and maintenance of anesthesia in patients presenting for MYOCARDIAL REVASCULARIZATION. This traditional approach to induction of anesthesia with high dose narcotics utilizes a graded stimulus sequence with careful and slow titration of usually fentanyl, sufentanil, or morphine. The main goal of the high dose narcotic technique is prevent myocardial oxygen supply and demand mismatch by achieving stable hemodynamics throughout the induction and intraoperative period. Narcotics in adequate amounts may have little or no effect on myocardial contractility and cause a decrease in systemic vascular resistance and heart rate only through a decrease in sympathetic output. For a high dose narcotic induction sequence, typically 10-25 mcg/kg of fentanyl or 1-1.5 mcg/kg of sufentanil is titrated over several minutes during a series of graded stimuli prior to tracheal intubation. The graded induction sequence is as follows. Slow titration of fentanyl is administered until the respiratory pattern changes or apnea occurs. Assessment of ventilation and unconsciousness is achieved by observing the chest rise, auscultation, placing a hand on the bag, capnography and direct feedback from the patient (i.e. eyelid closure, response to questioning etc.). After titrating the narcotic dose to loss of consciousness or apnea, the hemodynamic response to insertion of an oral airway is tested to assess the need for additional narcotic. This is followed by increasing the stimulus by insertion of the foley catheter, followed by insertion of the laryngoscope. At any point, if a change in hemodynamics is observed, additional narcotic is titrated. After achieving stable hemodynamics through narcotic administration, addition of a muscle relaxant is then given to facilitate tracheal intubation.
It should be noted that it may be prudent to administer a small dose of muscle relaxant (i.e. 1 mg of vecuronium or rocuronium) prior to the titration of high dose narcotics. This “priming dose” is often required help prevent the onset of severe chest wall rigidity which will dramatically impair your ability to ventilate. If this occurs, the administration of succinylcholine will promptly break the rigidity and improve ventilation. The mechanism behind this phenomenon is unknown but may be mediated through a central dopaminergic and/or GABAergic pathway in the higher brain centers. It has also been hypothesized that narcotic induced paradoxical closure of the glottis may also contribute to difficulty in ventilation during high dose narcotic based inductions.

The above technique describes a typical traditional high dose narcotic based induction using a method of graded stimuli to gauge the depth of anesthesia. Although this traditional approach was extremely effective in achieving hemodynamic stability in patients at risk for myocardial ischemia, it was burdened with high incidence of intraoperative recall, extended extubation and ICU periods (due to such high doses of narcotics) and did not exploit the later discovered myocardial protective effects of inhalational anesthetics. Accordingly, the traditional induction has been replaced by a more balanced approach combining lower doses of narcotics (i.e. 5-15 mcg/kg fentanyl) with other anesthetics (i.e. inhalational agents, propofol, benzodiazepines etc.). This more modern technique exploits the important concept of blunting the sympathetic response to surgical stimulation with opiates while taking advantage of the important ischemic preconditioning properties of inhalational anesthetics (discussed later) and the potent amnestic properties of benzodiazepines. This more balanced approach has also been applied to safely anesthetize a wider range of cardiac surgical patients (i.e. valvular disease, congenital, etc). This is now known as the fast-track program designed to improve postoperative morbidity and mortality by minimizing prolonged ventilator and ICU time, improving myocardial revascularization success rates, and reducing overall cost of hospital stay. The mainstay of fast-tracking a patient is the concept of extubating the patient in less than 8 hours (i.e. same day or morning of POD #1).

The following is an example of a fast track protocol utilizing a balanced anesthetic, with the goals of safely achieving hemodynamic stability, ensuring general anesthesia (amnesia, analgesia, unconsciousness), and early extubation. It should be highlighted that the below is merely an example of an induction sequence and the clinician should tailor his protocol to each patient with knowledge of the individual’s specific physiology. Please also remember that each attending also has a preferred sequence and a specific rationale for each. If you have any questions, please communicate with your attending for the day.

- Premedication with midazolam 1-2 mg IV preoperatively (caution with PTE, aortic stenosis, patients, see below)
- Fentanyl (5-15 mcg/kg) and Etomidate (0.15-0.25mg/kg) in combination for induction of Anesthesia
- Rocuronium (0.1 mg/kg), Vecuronium (0.1 mg/kg), or Pancuronium(0.1 mg/kg), or Succinylcholine as indicated for muscle relaxation for intubation and duration of case
- Maintenance of Anesthesia with isoflurane (0.5% -1.0%) or Sevoflurane (1.5% -2.0%).
- Inhalational anesthetic during Cardiopulmonary bypass
- Midazolam at the onset of the rewarming process (a period of high incidence Of recall, see below)
- Inhalational agent upon separation of CPB and thereafter
- Propofol infusion (20-50mcg/kg/min) for transport to ICU

There are several other advanced and specialized techniques described in the literature for the delivery of anesthetics in the cardiac patient at various centers. Such techniques include total intrathecal anesthesia,
The use of high dose remifentanyl with nitrous oxide, and propofol or dexmedetomidine based anesthetics. These techniques are beyond the scope of this manual. The interested reader is encouraged to consult the literature and listed references.

INTRAOPERATIVE MANAGEMENT OF THE PREBYPASS PERIOD

After induction of anesthesia and tracheal intubation, confirm proper positioning of the endotracheal tube and secure it. Tape the eyes and empty the stomach with an orogastric tube. Then prepare to place central venous line (usually cordis catheter or also referred to as introducer) and pulmonary artery catheter. The echo resident or cardiac fellow will insert the TEE probe while you steriley scrub and don your sterile gown. A brief overview of central vein cannulation will be described here. For an in depth discussion on this topic, the clinician is encouraged to refer to the provided references on MONITORING. The right internal jugular vein is usually the site of choice for insertion of a central venous catheter. The right internal jugular vein and the right carotid artery are often in juxtaposition and have the following relationship. The carotid artery USUALLY is positioned medial to the internal jugular vein from a cephalad position (i.e. high in the neck) and as it courses caudad (lower in the neck), it tends to assume a more lateral position. In simple terms, this means that the carotid is often medial to the internal jugular vein high in the neck (i.e. above thyroid cartilage). In regions lower in the neck (i.e. below thyroid cartilage), the carotid may be superimposed (directly below or behind the internal jugular). In other words, if you attempt to cannulate the internal jugular vein low in the neck, the risk of carotid puncture may be higher since the practitioner may puncture the internal jugular vein and advance the needle into the carotid artery which may lie just behind the vein. It is crucial that you understand this anatomy. Ultrasound guided cannulation provides direct visualization of the anatomy and defines the relationship of the carotid and internal jugular veins. IT is therefore strongly encouraged that you at the VERY LEAST confirm the anatomy prior to cannulation. Ultrasound guided “real time” or live cannulation has been shown reduce complications, improve efficiency and help in identifying abnormal anatomy. We strongly encourage you to start with ultrasound guided venous cannulation. There are several methods of cannulating the internal jugular using landmarks. One method is to identify the origin of the sternal and clavicular heads of the sternocleidomastoid muscle. By following their paths to the point of intersection (top of triangle), the internal jugular often lies underneath this region. Another popular technique describes palpation of the carotid pulse at the level of the cricoid cartilage and insertion of the finder needle lateral to the carotid artery. The landmark for this method is the midpoint between an imaginary line drawn from the mastoid process and sternal notch. Usually this is at the level of the cricoid cartilage. This midpoint often marks the location of the internal jugular, lateral to the carotid.

Whatever method the clinician utilizes, confirmation of the correct vessel must be confirmed prior to vessel dilation and insertion of the large bore catheter. This can be done with ultrasound confirmation, venous and arterial blood gas analysis, and visualization of the wire in the right atrium by TEE or measurement of the CVP pressure with pressure tubing and/or confirmation of a venous pressure tracing by transducing a waveform. After confirming venous cannulation, the clinician should make a small scalpel incision at the skin followed by dilating the vessel with the dilator/cordis device. Then the cordis catheter is advanced over the wire into the vessel while pulling the dilator device out. You should ask your staff if you have any questions. One important recommendation is that insertion of the cordis catheter or dilator is easier if the clinician applies pressure at the more proximal end of the catheter when advancing (i.e. near the insertion site as opposed to the distal end furthest away from the entry site). This prevents the catheter from bending or kinking.

After successful introduction of the cordis introducer, the pulmonary artery catheter will be floated into the chambers of the heart. The PA catheter is packaged in such a way to facilitate proper advancement into the chambers and finally the pulmonary artery. When you initially take the catheter of its package, maintain its
“natural curve” prior to insertion. Before insertion, flush the catheter’s proximal and distal ports (i.e. CVP, Right atrial/Ventricle, and PA). Confirm proper balloon inflation and deflation. Also confirm that the temperature thermistor is connected properly (i.e. you see a temperature reading on the monitor). After confirming proper function of the PA, note the demarcations on the catheter. Twenty centimeters is the approximate distance from the skin the CVP or right atrium. Thirty centimeters is the appropriate distance to the right ventricle and 40-50cm is the approximate distance to the pulmonary artery. These distances are guidelines, your patient’s anatomy and physiology will determine the exact proper distance and position to the PA. More importantly, the distinct waveforms as the catheter tip traverses each chamber is the endpoint used to float the PA catheter. Please review the waveform morphologies and familiarize yourself with these tracings. Briefly, the right atrial or CVP tracing is described by your classic A, C, V pressure waves and X and Y descents during the cardiac cycle. The right ventricular tracing is dominated by a sudden rise in systolic pressure with very or negligible change in diastolic pressure. The final PA tracing is achieved when a sudden rise in diastolic pressure (diastolic jump) is suddenly observed and often appearance of a notch on the waveform similar to a radial artery tracing. The PA and RV pressure tracings also can be distinguished by the downward or upward slope of the tracing prior to systole, respectively. After entering the PA, minimal advancement may result in a pulmonary artery occlusion pressure waveform (PAOP). This is often indicated by a loss in the systolic component of the PA tracing. After “wedging” the catheter, the balloon should be DEFLATED and withdrawn back 2-3cm to prevent pulmonary artery related injury due to distal migration or constant occlusion of a distal branch. It should be noted that wedging the catheter is not necessarily indicated every time you place a PA catheter. Please review the appropriate references for placement of PA catheters and invasive monitors. Also review the complications and indications/contraindications related to these procedures.

Once the PA catheter is properly placed, it is a good time to obtain baseline hemodynamic parameters such as cardiac output/index, CVP, PA pressures, MAP, systemic and pulmonary vascular resistance, etc. It is also a good time to send a baseline blood gas and ACT (activated clotting time). In addition, it is also a good time to connect your intravenous lines, fluid warmers, infusion ports etc. prior to antibiotic administration, and preparing for incision and sternotomy. During this period, you will be tempted to review the TEE examination and not focus on the patient. Please remember that you are the primary physician for your patient. Your priorities are to keep a broad overall perspective on your patient at all times. It is appropriate to review the TEE findings after you are in complete control of the case and have a strong facility of all aspects of anesthetic management of the cardiac patient. Formal instruction in TEE should be reserved for the later parts of your rotation (i.e. 3-4th week at Thornton or 2nd month at the VA) and during your advanced cardiac month. We realize that there are often many members of the cardiac team in the heart room and it can get crowded. But your vigilance is expected at all times even when it gets so “chaotic.”

Before incision, it is worthwhile to check the following.

1. Confirm adequate ABCs(i.e. oxygenation and ventilation, hemodynamic stability, cardiac output, perfusion pressures).
2. Assess for depth of anesthesia (need for more opiates/benzos or increasing inspired inhalational agents for incision and sternotomy).
3. Assess for need for more muscle relaxant as movement during critical portions of the procedure may be catastrophic (i.e. patient moves during sternotomy and surgeon punctures right ventricle).
4. Timely administration of appropriate antibiotics (within 30mins of incision), Usually Cefuroxime or Vancomycin.
5. Initiation of antifibrinolytics (i.e. 2.5 gram load followed by 1 gram/hr of aminocaproic acid for every pump case, with exception of PTE).
6. Assessment of baseline hemodynamics and cardiac function.
7. Confirmation of function of all lines and infusions (i.e. drips into the RA/PIP or RV port, warmed volume line into side port of cordis).
8. Baseline ACT/ABG

INTRAOPERATIVE MANAGEMENT
During the prebypass period, be cognizant of the following stages of heightened surgical stimulation when delivering your anesthetic: skin incision, sternotomy, pericardiotomy, and aortic cannulation. The pericardium and aorta are deeply innervated structures and are very sensitive to surgical stimulation. As such, these steps in the surgery may warrant titration of your anesthetic to prevent harmful hemodynamic perturbations due to an inadequate plane of anesthesia. The clinician should turn off the ventilator and pop-off valve prior to sternotomy (usually one will hear the saw being turned on) to prevent lung laceration. It is always prudent to confirm to the surgical team by clearly stating the “lungs are down” prior to sawing of the sternum. Please also confirm re-institution of ventilation by saying “lungs are up” when sternotomy is completed. After sternotomy, dissection of the chest may require reduced lung volumes to facilitate surgical exposure. Consider lower tidal volumes (i.e. 4-6cc/kg) or hand ventilating so that surgical exposure is maximized. The next stage primarily consists of dissection of the chest and preparation for great vessel cannulation. During the chest dissection, be aware of the pericardiotomy as this may be very stimulating. In the case of CABG surgery, there may be a long period of reduced stimulation when the saphenous veins or left internal mammary artery is harvested for the bypass grafts. Following the dissection period, purse strings are usually then placed on the aorta in preparation for aortic cannulation. Prior to aortic cannulation, adequate anticoagulation MUST be confirmed. This is usually achieved with a heparin dose of 3-4mg/kg (or 300-400 units/kg, 1 mg=100units) yielding an ACT greater than 450 seconds (approximately 3-4X baseline ACT). It is absolutely essential that you communicate with everyone in the room about the amount and time of heparin administration. Adequate heparinisation is an absolute PREREQUISITE for instituting cardiopulmonary bypass. Inadequate anticoagulation is catastrophic and life threatening as clots may form on the bypass circuit. Check an ACT 3 minutes after heparin administration. If possible, given heparin through a central line, and confirm by drawing back blood through the injection port before injecting. This ensures that the injection port is venous. After heparin is given, draw back again and confirm blood return. It is also a good idea to given heparin over ~60seconds to minimize hypotension from histamine release that is often seen with rapid boluses. There is no room for error when it comes to the timing and adequacy of anticoagulation and its reversal with protamine (discussed later). If you have any questions please ask. Please review the pharmacology of heparin in the suggested readings. Pay special attention to mechanism of heparin action, heparin induced thrombocytopenia, and heparin resistance.

AORTIC AND GREAT VENOUS CANNULATION
For cardiopulmonary bypass to commence, aortic and venous cannulation must be established and anticoagulation must be adequate. Sites of aortic access include ascending aorta (most common for a virgin chest), femoral artery, descending thoracic aorta, or axillary artery. Aortic cannulation is usually first established and is facilitated by reducing the systolic blood pressure to approximately less than 100mmHg and minimizing tachycardia to reduce the chance of intimal tearing and aortic dissection. This can be achieved in a number of ways (i.e. inhalational agents, narcotics, propofol, nitrates, nicardipine, reverse trendelenberg etc.). Be cognizant of patients with critical aortic stenosis or left main coronary disease who rely on adequate perfusion pressure as hypotension may precipitate hemodynamic collapse. Following aortic cannulation, the pressure can be allowed to rise to baseline in anticipation for venous cannulation. Venous cannulation is usually achieved through the right atrium or separately through cannulae in both the IVC and SVC. Other sites of venous access include femoral vein, left atrium and pulmonary vein. It should be
mentioned that cannulation of the great vessels can lead to rapid blood loss and resultant hypotension. Be aware of this and prepare accordingly. However, having an aortic cannula provides a rapid means of transfusion in the event of rapid blood loss during venous cannulation. If this occurs, simply asking the perfusionist to transfuse volume through the aortic cannula can easily restore circulating blood volume during rapid blood loss.

CARDIOPULMONARY BYPASS PERIOD

After confirming adequate heparinization and great vessel cannulation, commencement of bypass is safe. Please check the following prior to initiation of bypass.

1. Ensure adequate muscle relaxation. Movement during critical parts of the surgery (i.e. diaphragm movement during circulatory arrest and pulmonary endarterectomy is unacceptable). Shivering during systemic hypothermia may increase oxygen consumption and promote muscle breakdown and cause potential renal failure.

2. Venous return: make sure that the venous line is draining appropriately to the reservoir. The rate and adequacy of venous drainage depends on gravity and the height difference between the right atrium and the reservoir. Inadequate drainage can also be affected by improper positioning of the venous cannula. Inadequate drainage may lead to engorgement of the cerebral vessels and severe cerebral edema. Check for bulging sclera and swelling of the face and head. A sudden rise in CVP may also be an indication of adequate central venous drainage. If in doubt ask the perfusionist. The extracorporeal bypass circuit is in series, so any loss of volume in the circuit will be reflected by inadequate venous return to the reservoir.

3. Arterial flow: return of oxygenated blood from the oxygenator is returned to the patient via the arterial cannula. At UCSD, CPB flow (Liters/min) is nonpulsatile and is controlled by the perfusionist. The presence of pulsatile flow indicates that the LV is filling with volume and is still ejecting due to a variety of mainly 2 reasons. First, the aortic valve may be incompetent and flow from the aortic cannula may leak back across the valve and into the aorta. Second, blood flow is inappropriately conducted antegrade from the RV to the PA to the LA and finally to the LV without being captured by the venous cannula. A mean arterial pressure of 50-70 mmHg is considered adequate provided pump flows are sufficient to meet oxygen consumption.

4. Communicate with the perfusionist if the patient required inotropes or vasodilators during the prebypass period. The perfusionist may ask for a nitroprusside or nitroglycerin syringe to control systemic vascular resistance.

5. Make sure the inhalational agent on the perfusionist’s circuit is initiated.

6. Make sure the PA catheter is withdrawn 2-3cm so that migration during the bypass period does not cause pulmonary artery injury.

7. Discontinue the ventilator when adequate cardiopulmonary bypass is confirmed.

After the initiation of CPB, an aortic cross clamp will be applied on the ascending aorta proximal to the aortic cannula. This separates the systemic circulation from the cardiopulmonary system. This time represents the period in which there is no flow in the coronary arteries and thus is referred to ischemic time. During this ischemic period, notice that the heart is arrested by cardioplegia and hypothermic from direct application of cold ice or chilled by topical saline solution. During the bypass period, you may be required to provide a solution of chilled saline for the surgeons to directly apply to the myocardium to maintain
topical hypothermia. Be sure to replace the saline bag (3 liter) as needed. Topical hypothermia is a very important method for myocardial protection (see below).

During the cardiopulmonary bypass period, pay attention to the patient’s temperature during the cooling period. Most cases will require systemic mild to moderate hypothermia (i.e. cooling to 26-35 degrees) as a means for the purpose of organ protection during CPB. The metabolic rate dramatically decreases in proportion to the degree of hypothermia. In cases of deep hypothermic arrest (i.e. for PTE), cooling to 17-20 degrees lowers the basal metabolic to such a low level allowing the perfusionist to arrest circulation for up to 20 minutes without significant end organ damage. During valvular or on pump CABG, the surgeons often topically apply an ice jacket to the heart thereby reducing the metabolic rate of the myocardium to an almost negligible amount. The continuous application of ice and cold saline is also utilized by the surgeon to protect the heart during periods of ischemia. Topical and systemic hypothermia have been the mainstay of reducing basal metabolic rate for decades and is still the principle method of myocardial protection at UCSD.

The perfusionist controls both the fraction of inspired oxygen and rate of oxygen delivery through the circuit, thereby controlling the patient’s arterial oxygen and carbon dioxide levels by adjusting a knob. The perfusionist will analyze blood gas samples (arterial oxygen, carbon dioxide, glucose, electrolytes, hematocrit, pH, base excess) to ensure that oxygen delivery is adequate. The hematocrit is usually checked on an hourly basis and due to a variety of factors (i.e. hypothermia, reduction in metabolic rate, nonpulsatile flow, decreased viscosity from isovolemic hemodilution, improved rheology etc.); a hematocrit above ~22 is usually adequate for the bypass period. The physics that govern ideal blood flow and oxygen delivery are beyond the scope of this syllabus. Pertinent references on this interesting topic are provided for those who are interested.

REWARMING PERIOD
The rewarming period generally is accomplished over through a heat exchanger and usually is achieved over 30-60 minutes depending on the degree and length of hypothermia. Body surface area may also affect the rate of rewarming. As with cooling, adequate core (tympanic) and shell (bladder or rectal) equilibration to a temperature of 36.0 to 37.0°C is optimal. It is recommended that you administer additional muscle relaxant and midazolam to prevent unnecessary shivering and prevent intraoperative recall. The rewarming period represents a time of increased risk of recall. This process is usually gradual since rapid rewarming may lead to overshoot which has been associated with increased incidence of postoperative neurological dysfunction. On the other hand, inadequate or insufficient time for rewarming may lead to undershooting which may reduce myocardial performance, delay metabolism, cause vasoconstriction, and affect coagulation. During the rewarming period, notice that the peripheral (referred to as shell temperature) and central compartments (referred to as core temperature) increase in temperature at different rates, reflecting a difference in blood flow. That is, major organs such as the heart and brain receive more flow that peripheral compartments such as muscle or fat layers. When the gradient between the core and shell are equal, the temperature gradient has reached equilibrium and the process is complete.

PREPARING FOR SEPARATION FROM CARDIOPULMONARY BYPASS
The rewarming period is a good time to prepare for separation from bypass. Prepare any necessary inotropes, vasodilators, pressors, protamine, calcium chloride, etc prior to coming off bypass. The inotrope of choice at UCSD is dopamine. Most on pump cases will employ a low dose dopamine infusion prior to separation from CPB. Depending on your patient’s physiology, the use of epinephrine, dobutamine, phenylephrine, milronone, vasopressin etc. may be necessary for separation of bypass. Make sure you discuss your plan with your attending and communicate with your surgeon the plan prior to coming off. Also make sure that you start your inotrope/vasopressor well before (i.e. 5-7 minutes) separation from bypass to insure adequate
circulation and effect time. Make sure that you communicate to the surgical team your inotrope of choice prior to coming off. The following considerations may help guide you in determining if inotropy is necessary and if so, which agent(s) may be most effective in facilitating separation from bypass.

1. Preoperative RV/LV function and baseline filling pressures (i.e. CVP, PA pressures, RVEDP, LVEDP). In general, preoperative systolic function usually does not immediately improve. Thus, if a patient has poor ventricular function at the onset, inotropy is usually required to separate from bypass. Exceptions to these guidelines are patients undergoing PTE where immediate restoration of right ventricular function is often observed following removal of pulmonary artery clot burden.
2. Aortic crossclamp time (ischemic time) and bypass time. The longer the ischemic time, the more likely there is a greater degree of reperfusion injury, myocardial stunning, and need for inotropy, especially when myocardial protection is suboptimal (i.e. severe LVH, incompetent aortic valve, inadequate cooling, inability to administer cardioplegia, etc).
3. Hemodynamic parameters immediately prior to separation (i.e. on 1 liter/min of flow). When venous return to the right side of heart is only partially captured and the LV is allowed to eject, examine the hemodynamics and assess adequate CO, MAP, and SVR prior to separation.
4. Age greater than 70 years and female gender may be factors that have been correlated with increased requirement for inotropy.
5. Base your decision on the pharmacological properties and the mechanism of action (Beta vs. alpha effects, calcium sensitization, etc.) of the inotrope/vasopressor and the desired hemodynamic effects that are necessary given the patient’s physiology. For instance, if HR and CO are adequate, increasing the SVR with an alpha agonist may be all that is needed to augment mean arterial pressure.
6. Experience of the anesthesiologist and surgeon.
7. Institutional preference.

DEAIRING DRILL
Following operations in which the chambers are exposed (i.e. valvular or PTE surgery), it is crucial to remove intracardiac air prior to coming off bypass to prevent systemic embolization (i.e. coronary emboli, cerebral emboli). This is done by placing the head of the patient in the trendelenberg position as air usually rises to the nondependent regions. Make sure ventilation through the lungs are adequate to help move air from the pulmonary veins. Tilting the table side to side may also mobilize air. During ventilation, an empty chamber may be allowed to fill with blood flow (i.e. venous capture discontinued) and a deairing needle in the aorta is often used to aspirate residual air. Alternatively, a vent line (usually in the PA) may be used to aspirate air. Tranesophogal echocardiography is very sensitive in detecting residual intracardiac air. Close inspection of the LV apex, LA, LA appendage, and pulmonary veins are common areas of retained air. Communication with the surgeon and the perfusionist during this deairing process is important in preventing systemic embolization after aortic declamping.

AORTIC DECLAMPING AND DEFIBRILLATION
During separation from bypass, the heart may be very arrhythmogenic due to prolonged ischemia, reperfusion injury, inadequate myocardial protection, residual cardioplegia, inadequate rewarming, and air emboli. It is not uncommon to defibrillate the heart during removal of the crossclamp and re-establishment of myocardial blood flow. This is usually achieved with 10-30 joules of power given direct to the myocardium. There are several points to keep in mind during the defibrillation process. Hypo/hyperkalemia (i.e. due residual cardioplegia), prolonged crossclamp (ischemic time) time, inadequate rewarming, patients with valvular disease, inadequate revascularization and myocardial protection, and coronary air emboli have all been shown to contribute to refractory defibrillation. Accordingly, to optimize the conditions for successful defibrillation, ensure adequate coronary perfusion pressures (MAP above 60 or higher),
normothermia (i.e. 36-37°C core and shell), normokalemia, complete deairing of cardiac chambers, and utilize sufficient energy (i.e. 10, 15, 20 joules with internal paddles). Administration of lidocaine (1mg/kg) and magnesium (2-4grams) has also been shown to reduce the number of countershocks following aortic declamping. Typically, 2grams of magnesium sulfate and 100mg of lidocaine is given just prior releasing the aortic crossclamp. At UCSD, this is often given by the perfusionist.

**SEPARATION FROM CARDIOPULMONARY BYPASS**

It is critical that you have an organized and systematic approach to the termination of CPB. This will ensure optimal care and consistent results. The following is an example of a checklist of items to consider prior to separation.

1. **A and B. Airway and Breathing.** Check endotracheal tube placement. Begin ventilation of lungs by hand using lower tidal volumes followed by gradual recruitment breaths to eliminate atelectasis. Pay attention to airway compliance. Observe that the lungs are expanding in the field during this process. Note symmetry of expansion (i.e. rule out iatrogenic pneumothorax or mainstem intubation) of lungs. Place patient on ventilator and titrate inhalational anesthetic as tolerated.

2. **C. Circulation.** Check to confirm the rate, rhythm, contractility, preload and afterload is adequate to maintain cardiac output and mean arterial pressure.

   **Rate.** If the heart rate is inadequate to maintain cardiac output, consider atrial, ventricular, or atrioventricular pacing at a rate of 80-100 (depending on patient’s physiology) to augment cardiac output.

   **Rhythm.** If an unstable arhythmia is present such as SVT, VTACH, atrial fibrillation, consider cardioversion.

   **Contractility.** Confirm that the predetermined pressors or inotropes are being infused properly to ensure adequate contractility and tone. Ensure adequate mean arterial pressure (i.e. greater than 60mmHg or higher) with appropriate flow.

   **Preload.** To construct the Frank-starling curve (i.e. cardiac output as function of LVEDV), examine the filling pressures of the heart and TEE(transgastric short axis view of LV, “doughnut view”) to determine response to volume infusion.

   **Afterload.** Examine the mean arterial pressure and obtain cardiac output data from the PA catheter to determine systemic vascular resistance. Remember the mean arterial pressure, CO and SVR are related by the formula: SVR=MAP/CO X 80. Or MAP = CO X SVR/80. In other words, CO and SVR are inversely related. If the MAP is low, but the CO is high, it follows that SVR must be low. In this case, tone is needed to increase SVR and MAP. You should become familiar with this important hemodynamic relationship.

3. **D. Dilution.** During the start of CPB, 1-2Liters of a crystalloid solution is used to “prime” the pump and thus dilutes the hematocrit. This isovolemic hemodilution process is reversed with diuresis and thus reconcentrating the hematocrit before separation from CPB. However, due to blood loss and loss of volume during CPB, the hematocrit may be too low for separation. Make sure that the HCT is above 20% to maximize arterial oxygen content for separation. Transfusion of RBCs may be necessary to accomplish this. Be aware that dilution of coagulation factors and platelets may also contribute to coagulopathy (discussed below).

4. **D. Deairing Maneuver as Above.**
5. **E. Electrolytes.** As mentioned earlier, hypokalemia/hyperkalemia may impair maintenance of an organized rhythm. Calcium, magnesium, sodium, and glucose homeostasis may all affect cardiac function during the post-bypass period. Send an ABG to make sure that your chemistries are within normal limits. Correct any abnormalities.

6. **G. Glucose.** Hyperglycemia as a result of a multitude of factors is common after separation from bypass. In general, aggressive management with insulin is usually not warranted unless the patient has a history of diabetes and/OR the glucose level is above 250. This is a controversial and debated topic. **Please discuss this with your attending and fellow** prior to the pharmacological correction of hyperglycemia.

Once you have performed a systemic check of the above factors, CPB may be terminated. This is done usually gradually by clamping of the venous line so that capture of the venous return (from the RA) to the reservoir is slowly discontinued. You will often hear the perfusionist say “taking the venous” to indicate that venous return has been discontinued by the perfusionist. At the same time, arterial flow (from the pump to the patient) is decreased.

**HEMOSTASIS AND COAGULATION**

After completion of volume transfusion from the venous reservoir through the aortic cannula by the perfusionist and confirmation that there are no major issues that would prompt reinstitution of cardiopulmonary bypass (i.e. poor cardiac function, leaking valve, etc.), anticoagulation is reversed with protamine. In general, protamine is dosed based on a heparin assay performed by the perfusionist (a standard curve extrapolated from serial ACTs are conducted by the perfusionist to determine dose). The dose ratio of protamine to heparin is usually 1.5-1.0 (protamine in mg) to 1mg (heparin in mg) when attempting to reverse full heparinization. Prior to the administration of protamine, please make sure to directly observe the field as the surgeons are often suturing the aortotomy incision and confirm that it is an appropriate time to give heparin. Please make sure to directly communicate with the surgeons AND perfusionist prior to starting protamine and make sure everyone CLOSES the loop. That is, make sure you get verbal confirmation from both the surgeon and perfusionist that it is appropriate prior to starting protamine. Errors in protamine administration are often CATASTROPHIC. Once you confirm that it is an appropriate time, administer protamine over 5-7 minutes, as a protamine reaction through a thromboxane mediated mechanism may precipitate severe and abrupt pulmonary hypertension and hemodynamic collapse. A histamine mediated mechanism causing hypotension is also believed to play a role in hemodynamic collapse. In this event, be prepared to support the hemodynamics with inotropy and vasopressor therapy (epinephrine) and possibly reinstitute pulmonary bypass with full heparinization.

Protamine's action in reversing heparinization is thought to be due to its polycationic arginine residues (positively charged) that bind to the negatively charged sulfur groups of heparin. The ionic interactions facilitates the formation of a heparin-protamine complex that is likely excreted through the reticuloendothelial system. It is important to note that the half life of protamine is slightly shorter than heparin which may lead to residual effects from unbound heparin causing bleeding from this "heparin rebound" effect. In this event, an additional dose (~50mg) of protamine is often given. Protamine is isolated from salmon sperm and is often a mixture of various fish containing antigens that may lead to allergic reactions in fish allergic individuals.
Coagulopathy and resultant bleeding after bypass is complex and multifactorial. One of the most important mechanisms leading to postbypass coagulopathy is the qualitative and quantitative dysfunction of platelets following exposure to the CPB circuitry. Platelet dysfunction is thought to be a result of both platelet disruption and consumption direct contact from the circuit as well as hemodilution of the platelet concentration on bypass. The perioperative use of aspirin in combination with clopidigrel or glycoprotein IIb/IIIa inhibitors have also been suggested to contribute to platelet dysfunction postbypass.

The hemodilution of both platelets and coagulation factors also plays a role in postbypass bleeding. Prior to the commencement of bypass, the perfusionist often "primes" the CPB circuit with crystalloid solution. This usually results in a ~15% decrease in circulating coagulation factors. This generally does not cause coagulopathy unless a critical level of 30% reduction (from normal levels) in coagulation factors is reached.

The actual management of coagulopathy is very complex and a brief outline is presented here. Please refer to the suggested and required readings for reference. In the presence of bleeding NOT from direct surgical bleeding (i.e. aortic bleeding from cannula sites), the general approach is to obtain point of care testing as soon as clinically feasible in the form of Thromboelastograph (TEG available at the VA and new CV center) and coagulation testing (Platelet count, PT/PTT and INR). This will guide therapy and serve as an endpoint to measure subsequent management. In the absence of timely coagulation data, platelet therapy may be the first prudent step as platelet function may be the most important and common etiology for postbypass bleeding (described above). Ascertaining that adequate protamine has been administered is an important first step as insufficient protamine dosing or heparin rebound is often a common cause of coagulopathy. If coagulopathy continues at this point, both the TEG and/or coagulation studies may provide insight into a coagulation factor/fibrinogen deficiency and/or platelet function related etiology.

CHEST CLOSURE AND TRANSPORT

After confirmation of adequate hemostasis and hemodynamic stability, the surgeons will attempt to pack and close the chest with sternal wires. Be vigilant during this time period as abrupt hemodynamic changes often occur as chest closure in combination with positive pressure ventilation may markedly decrease preload and overall cardiac pump function. It may also be prudent to begin preparing the patient for transport at this point as the surgical end time approaches. During this time, it may be prudent to decrease your volatile anesthetic and convert over to intravenous anesthetics such as a low dose propofol infusion or titration of midazolam. Beware of oversedating your patient with enthusiastic doses of narcotic at this point as a sympathectomy may cause abrupt hypotension. Your priority is always that patient at this time, but actions to prepare the patient for transport (such as discontinuing the fluid warmer, inserting an oro-gastric tube, capping all IV lines appropriately etc.) may make the transition to the ICU smoother.

It is extremely important to mention that transfer of the patient from the bed to the ICU transport bed may cause sudden perturbations in hemodynamics. Air embolus from open chamber surgery, lack of stimulation, a sudden redistribution of fluid, oversedation, and accidental discontinuation of inotropes or pressors have all been implicated in hemodynamic collapse after transfer from bed to bed. Please be VIGILANT.

During transport of your patient to the ICU, it is often a time of "relaxation" as many of the staff are finished. This is not a time for the anesthesiologist to lose sight of the countless hours of detail that went into taking care of your patient. The transport is often a time where hemodynamic instability occurs due to a variety of reasons including but not limited to inadequate oxygenation and ventilation, accidental discontinuation of inotropes, inadequate vigilance and monitoring, etc. The bottom line is the vigilance and care that you so carefully provide in the OR should be extended to the ICU transport. BE VIGILANT. IT IS YOUR MOST IMPORTANT MONITOR.
REQUIRED READING

1. UCSD DEPARTMENT OF ANESTHESIOLOGY, CARDIOTHORACIC ANESTHESIA GOALS AND OBJECTIVES, CA-1 YEAR.
2. POLICIES AND PROCEDURES GUIDE
3. INTRODUCTION TO THE CARDIAC ROOM
4. Essentials of Cardiac Anesthesia by Joel Kaplan. Chapters below

Required Chapters in Essentials of Cardiac Anesthesia
Chapter 1. Assessment of Cardiac Risk
Chapter 2. The cardiac catheterization laboratory
Chapter 3. Cardiac Physiology
Chapter 4. Coronary Physiology
Chapter 7 and 8. Cardiovascular Pharmacology
Chapter 9. Monitoring.
Chapter 13. Anesthesia for Myocardial Revascularization
Chapter 14. Valvular Disease
Chapter 20. Transplantation
Chapter 22. Cardiopulmonary Bypass and the Anesthesiologist
Chapter 24. Discontinuing cardiopulmonary bypass

Miller's Anesthesia
Chapter 49. Anesthesia for Thoracic Surgery
Chapter 50. Anesthesia for Cardiac surgery procedures.

Suggested Reading

Clinical Anesthesia by Morgan and Mikhail. Corresponding chapters on Thoracic anesthesia and Cardiovascular physiology and cardiac surgery.

Clinical Anesthesia by Paul Barash. Corresponding chapters on Thoracic anesthesia and Cardiovascular physiology and cardiac surgery.