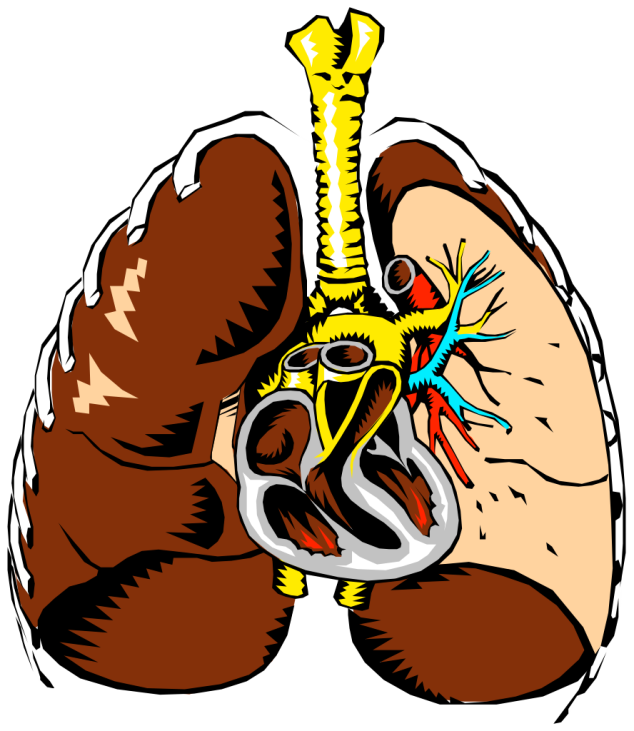
Handbook of Cardiothoracic Critical Care Surgery

Advent Health Orlando

Advanced Cardiac Surgical Unit



Version 1.0

2021

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STAFF PHONE BOOK AND KEY NUMBERS

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**Staffing:**

The ACSU works as a team comprised of intensivists, cardiac surgeons, advanced practice providers(APP’s), ECMO specialist, pharmacists, and nursing staff. Rounds are conducted daily in the morning and if needed in the late afternoon. There will be a charge physician daily to oversee all activities. Everyone should be communicating with each other regarding patient issues, test results, treatment plan, and changes in patient condition.

## Orientation and Training:

All new Advanced Practice Providers joining the group will have a 3-month orientation, with an extension to 6 months if needed. The APP will be mentored directly by the groups staff and the clinical staff that the APP will be rotating with. During this Orientation time the APP will not be taking on any direct individual patient care. Orientation will be focused on learning the essential workflow of the unit, by direct shadowing of patient care, didactic education on the essentials of cardiothoracic critical care via presentations, power points, literature, and rotations with different clinical practices. Rotations with practices will include CV surgery, Echocardiography, Advanced Heart Failure and Transplant team, Lung Transplant Team, Pulmonary Hypertension, ECMO training via simulation lab (will need to complete ELSO course within the first 6months of hire), and Fundamentals of Critical Care.

The APP will be required to be present for CV Rounds, listen in on the weekly critical care talks per teams’ meetings and other educational series that will be planned. An end of the week recap with the APP’s mentor will be done to look at pathway progress and discuss how the orientation is progressing for the APP.

We reserve the right to change the orientation and rotation schedule at any point in time to in order to meet the individual needs of each APP joining the group.

**Morning & Evening Sign out:**

Morning report/handover is held daily on WT 9th floor in the CCM office at 5am and 5pm. Everyone is welcome to participate in the sign-out and voice opinions or concerns as time allows. The ICU patients are to be presented by the patients covering provider. Patients that have planned procedures, new surgeries, patients that will be arriving to the unit will all be discussed on sign out.

Morning and Evening Sign out lasts ~45minutes based on acuity of patients

**CCM & CT Surgery Attending Rounds:**

Unless otherwise specified morning rounds start at 7:30am, 8:00 on weekends (except Dr Silvestry at 8:30am). The charge physician to call surgical staff to check availability before starting rounds without the surgeon. Notes are to be prepared by the attending, APP’s, and fellows and must include:

Morning Rounds:

1. Name, Age, Post-op day, and reason for critical care.
2. Overnight events
3. Vital signs
4. Inputs & outputs, current weight in kilograms, pre-operative weight
5. Inotrope and vasopressor infusion doses
6. ECMO/Mechanical Circulatory Support (MCS) settings
7. Ventilator settings
8. Physical Exam
9. Laboratory data
10. Results of Films/Cultures/OR findings
11. Plan of care

Evening Rounds

Time to be announced by intensivist (usually 17:00)

Essential data to prepare for evening round should include:

1. Plan of care for the evening and above data
2. Results of studies performed that day.
3. Determine patients to be discharged/transferred in the morning.
4. Discuss consult recommendations.

**Daily Responsibilities:**

* Patient daily progress notes to be done by covering provider.
* Review all laboratory data
* Evaluate orders placed overnight.
* Evaluate patient infusions: discontinue orders if no longer on infusion.
* Evaluate medications and discontinue those that are no longer indicated.
* Order lab work for following day: Morning labs @ 04:00
* Review necessity for invasive lines, Foley catheters, insulin infusion, DVT Prophylaxis
* Examine MCS (i.e., examine ECMO oxygenator, check distal perfusion catheter)
* Alert intensivist when case will arrive in ICU
* ICU care team to be present ay bedside for OR sign-out from anesthesia
* Prior to transfer to the telemetry floor, transition of care orders is to be placed in EPIC. This includes discontinuing ICU orders and reviewing all medications/orders.
* ICU NP/PA is will verbally sign out patient to the telemetry PA/NP or covering MD prior to leaving ICU.
* The ICU team is responsible for calling consults discussed during rounds prior to patient transfer.
* Talk with consultants regarding recommendation and discuss with intensivist prior to implementing.
* Labs are to be ordered for 2am collect, ECMO/TAH: require biomarkers.

**Important Observations:**

1. Check sternal stability daily. Serosanguinous drainage from a sternotomy incision is a dehiscence until proven otherwise. Do not open, probe, or express these wounds. Notify the intensivist/attending surgeon of your assessment. Please notify CT Surgeon and PA on rounds of findings
2. Do not open any incision unless directed to do so, please notify intensivist/attending and CT Surgeon on call
3. Post-operative patients with leukocytosis and fever should be cultured and procalcitonin should be drawn before antibiotics are given. Cultures are best drawn from peripheral sticks; line cultures are not recommended. Do not delay antibiotics for cultures; if nursing is unable to draw blood, any ICU team member should attempt the draw. Have a low threshold to consult ID and discuss with intensivist regarding need for antibiotics.
4. Monitor current inotrope and vasopressor doses.
5. If the nurses are concerned, you should be concerned.
6. All the patients should be assumed to be critical.

**Immediate Notification of Intensivist:**

1. ANY CONCERN. It is better to notify than not to notify. Ensure you have all pertinent information available prior to calling attending.
2. Changes in patient condition: acute change in status, arrhythmias, hemodynamic instability (i.e.: increasing pressor requirements), bleeding, MCS alarms.
3. Addition or significant dose changes of vaso-active drugs.
4. New significant base deficit, anion gap or new lactic acidosis
5. Unexplained oliguria or anuria
6. Significant unexplained lab findings
7. Chest tube output of >200cc/hr.
8. Changes in hemodynamics with unexplained cause.
9. Need for invasive test or procedure.
10. Urgent intubations/re-intubations.
11. Acute mental status changes.

**Post op Admissions (General Procedural Overview):**

* Notify Attending intensivist upon first call of the arriving case so they are available for admission.
* At time of first call, review post-op orders placed by surgical PA
* Make sure antibiotics x 48 hours only: Ancef (or vancomycin) if closed chest or open chest with closed skin. Broad spectrum antibiotics should be started for all open chests (Vanco/Zosyn).
* Write CVICU admit note (ECMO, CABG, LVAD, Tx)
* Follow up all post-op labs and data.
* Evaluate EKG: Check for ischemia, any AV block or bundle branch block and compare to pre-op EKG to note any significant changes.
* Test epicardial pacing wires.
* Evaluate ABG: Correct acidosis and make necessary ventilator changes.
* Evaluate CXR – ETT, Swan Ganz catheter, NG/OG tube, IABP tip, and line placement, rule out pneumothorax or hemothorax. Nursing should call radiology to do stat CXR when patient arrives.
* Compare SvO2 to thermodilution cardiac output
* Patients to be placed on SIMV if plan is for extubating within 6 hr. window. Goal Extubation within 6hr of arriving to CVICU.
* The patient is sedated with Precedex out of the OR unless open chest or documented reason.
* Monitor Chest tube output- Notify Attending if > 200 per hour.
* Monitor filling pressures, cardiac index and urine output.
* Blood sugar control- Portland protocol. Blood sugar goal < 200 at 6 am.

INITIAL POSTOPERATIVE ASSESSMENT

Significant cardiopulmonary destabilization may occur during transport from the OR to the ICU. Thus, upon arrival to the ICU, observe vital signs carefully as the anesthesiologist describes the patient's underlying conditions, the operation performed, and special instructions for management. Have a very clear plan for: (1) desired mean arterial blood pressure, (2) when to begin ventilator weaning, (3) threshold for blood transfusion and preferred fluids for volume resuscitation, (4) the plan for starting, increasing, or weaning inotropic agents. While performing the physical examination continue to observe patient and monitor for stable blood pressure, heart rate and rhythm, and oxygenation.

Immediate postoperative hypotension:

* All post-op patients require a working arterial line upon arrival to the CVICU.
* DO NOT assume that hypotension is due to a poorly functioning arterial line until adequate BP is confirmed by cuff.
* If BP REMAINS INADEQUATE, then quickly perform the following assessment:
* Confirm rhythm. A Fib may be obscured by AV sequential pacing, pacemaker may not be capturing, bradycardia may be physiologically significant.
* “Cordis Interruptus” Confirm that inotropic agents previously infusing are still infusing. Follow the drug from bag (concentration and volume, clamps open) to patient (Swan is inserted correctly, stopcocks are correctly positioned?).
* Examine chest tubes: is bleeding profuse or are clots occluding tubes? Is a pneumothorax present?
* Exclude hypoxia. Listen for breath sounds and consider using the bag valve if you have any doubt.
* Treat emergently with volume infusion (PRBC or Albumin, 500 ml) and Calcium Chloride (500 mg, IV push) to regain physiologic blood pressure until definitive therapy can be initiated. Consult Cardiac Output section and make intensivist aware for selection of next treatment.

A. Physical Examination.

1. Airway.

a. Check endotracheal tube position and cuff pressure.

b. Listen for air leaks around the cuff.

c. Check the integrity of the ventilator tubing.

d. Confirm End tidal CO2 (responsibility of anesthesia).

2. Breathing.

a. Auscultate laterally to confirm bilateral breath sounds and absence of wheezing.

b. Examine the chest for symmetric respiratory motion.

c. Review initial ventilator settings with respiratory therapist.

d. Tachypnea is one of the first signs of critical illness. Work it up every time. “Sedating the patient for tachypnea” should be done after all other causes ruled out.

e. Evaluate airway pressures. Peak Airway pressure, peak inspiratory pressure, Plateau pressure,

3. Circulation.

a. Palpate bilateral femoral, DP and PT pulses.

b. IF PEDAL PULSES ARE NOT PALPABLE, Use Doppler. (ESPECIALLY IF IABP or ECMO PRESENT).

c. Assess capillary refill time in the feet.

d. If no signals are present warm the extremities, reevaluate, and consider consulting vascular surgery.

d. Auscultate for murmurs, rubs, or arrhythmias. Rubs are common and not significant.

4. Neurologic.

a. On arrival to the ICU, patients remain under the influence of narcotics and paralytics. Initial assessment may be limited to pupillary responses.

b. Ensure a response to commands and movement of all extremities prior to any additional sedatives or pain medications.

c. Always evaluate the agitated patient.

5. Examine the abdomen for gastric/abdominal distention.

B. Monitoring.

1. Note vital signs, including HR, BP, PAD, CVP, temperature, and respiratory rate.

2. Note dosages of inotropic, antiarrhythmic and vasoactive drugs.

3. Check the IABP, if used, for proper timing and function. Also note BP with the IABP turned off temporarily.

C. Chest suction devices.

1. Note the volume of fluid in the Pleura-Evac. Examine the chest tubes for quality and quantity of drainage.

2. Confirm that suction tubing is attached, and suction level is appropriate. Normally set to -40 per our protocol.

3. Note the presence, volume, and pattern (i.e., continuous or intermittent varying with the respiratory cycle) of air leaks. Exclude a loose connection or leak at the chest wall.

D. External Pacemakers and Temporary Pacing Wires.

1. Ventricular pacing wires always exit from the left chest and atrial wires always exit from the right chest. There are two wires for each chamber. Connect to temporary pacer.

2. Assess the ventricular and atrial pacing capture thresholds. See section on pacemakers. Notify the Intensivist or Attending surgeon if thresholds exceed 10 mA.

E. Diagnostic information.

1. Blood work: CBC, CHEM 7, ABG, Co-Ox panel, PT/PTT.

2. ALWAYS OBTAIN AND EVALUATE EKG. Compare to previous EKG.

3. Portable CXR to be done on all patients post operatively.

F. Review of Patient History/Review of Brief Op Note.

1. Cardiac.

a. Previous myocardial infarctions, anginal class and pattern, Congestive Heart Failure Class.

b. Cardiac catheterization, MUGA, ECHO data: ejection fraction, valve function, septal defects.

c. Previous cardiac therapies such as CABG, PTCA, thrombolytic therapy.

d. Preoperative cardiac rhythm, Hx of atrial fibrillation, ventricular fibrillation, ectopy,

2. Pulmonary: tobacco use, asthma, COPD, previous pulmonary surgery, recent URI, or pneumonia.

3. Neurologic: previous CVA/TIA and residual effects.

4. Hematologic: history of bleeding disorders, history of anticoagulant (Coumadin, Xa, direct thrombin inhibitor), antiplatelet (ASA, Plavix), drug use, history of heparin induced thrombocytopenia.

5. Peripheral vascular disease.

a. History of claudication, previous vascular procedures.

b. History of previous DVT, Vein removal for CABG or vein stripping.

6. Urologic.

a. Preoperative renal function, recent changes in renal function.

b. Symptoms of ongoing UTI.

c. Symptoms of bladder outlet obstruction, especially in men.

d. Difficult Foley placement in operating room? Traumatic foley placement, discuss with Intensivist if POD #1 still planned removal. Consider Flomax.

7. Gastrointestinal.

a. History of peptic ulcer disease, cirrhosis, or hepatitis.

b. Previous abdominal surgery.

8. Endocrine.

a. Diabetes mellitus: what was the pre-op Hemoglobin A1c, is an insulin infusion needed (see Portland Protocol below).

b. Thyroid dysfunction, on Thyroid replacement.

c. Steroid use/dependency. Does this person need stress dose steroids?

d. Post-operative hypotension: consider adrenal insufficiency and get a cortisol level.

**Post-Operative Pathway (General – for CABG, Valves)**

1. POD 0

* Perform Initial assessment on arrival to ICU as stated above.
* Monitor volume status and resuscitate with 5% Albumin (up to 1 L) and blood products; avoid blood products in transplant and VAD patients unless necessary
* Wean Inotropic agents by following Fick CI and PA Swan catheter (if both correlating), know the plan for weaning inotropes with CHF patients with VAD’s
* Wean vasopressors as appropriate for Map goal 65-90, set the goal for nursing
* Follow up Post-op CXR- evaluate ETT, OGT, and Swan placement, and labs, replete electrolytes as appropriate
* Monitor CT output, notify surgeon if >200/hr., order blood products as appropriate based on labs; avoid blood products in transplant and VAD patients unless necessary
* Evaluate EKG, test pacing wires and verify underlying rhythm if patient is being paced
* Wean sedation, work with respiratory for PSV trial once awake and following commands, check ABG and extubate within 6 hours of arrival to CVICU.
* Verify with critical PA if epicardial wires are to be pulled or cut, and confirm with label on wires

1. POD 1 – CABG/Valve pathway
   * Start beta blocker if not on vasopressors or inotropes. Start low dose and titrate up if BP tolerates (MAP Goal>65). Metoprolol for normal ejection fraction (EF>40%) and carvedilol if the EF < 40%
     1. Risk of Atrial Fibrillation post-cardiac surgery is 33%. Adding a Beta blocker post-op decreases the risk of Atrial fibrillation post heart surgery by 50%.
     2. Documentation should be done if beta blocker contraindicated.
     3. Consider Amiodarone for atrial fibrillation prophylaxis if BB cannot be started.
        1. Amiodarone 1mg/min x6hrs, followed by 0.5 mg/min for 18 hours, and transition to 400mg PO TID x5 days, then 200mg PO qday. (IV loading dose required of 150 mg/ 15minutes)
     4. If patient is still requiring vasopressors POD#1, order a cortisol level for morning labs.
        1. Cortisol <15, start hydrocortisone 100mg IV q8hr x5 doses
        2. Cortisol >15, start Midodrine 5-15mg PO q8hr
   * Start Aspirin 325mg on all patients. Place patient on 81mg if dual therapy or requested by attending. All heart transplants (OHT) are 81mg unless otherwise directed. All MCS should receive 325mg.
   * Statin: start home statin if LFTs normal range. Lipitor 80mg if NSTEMI. Pravastatin if on Amiodarone. No statin needs in OHT.
   * Diuresis: Compare Pre-operative and post-operative weights (kg). Evaluate fluid status on CXR, I&O balance and physical exam daily and evaluate the need for loop diuretics. If placed on standing diuretics, consider starting standing Potassium supplements if appropriate.
   * DVT prophylaxis: compression boots plus sq Lovenox; heparin subq if creatinine >2 or presence of renal failure.
   * Restart necessary home medications if appropriate.
   * Bowel regimen: standing Colace, MiraLAX, senna unless loose stool.
   * Pain control: refer to pain protocol. Transition Dilaudid 0.25 & 0.5mg IV to Oxycodone IR 5 & 10mg PO q4hr PRN mod/severe pain if pathway on POD#1 morning.
   * Gastric dilatation- Always place NG to decompress stomach. If patient refuses make patient NPO AND DOCUMENT REFUSAL. F/u abdominal x-ray.
   * Diet- advance as tolerated. Consider TF if intubated greater than 2 days
   * Diabetes: Gluccomander IV Insulin protocol for postoperative stress hyperglycemia. Look to transition to Sub Q Sliding Scale once insulin infusion <1unit/hr. and tolerating at least P0 ordered diet.
   * Chest Tubes: Place to water seal POD#1 if minimal output, no air leak present, no PTX, closed chest, and no plan for return to OR. Discontinue Chest tubes once output <100ml over 8hrs.
   * Epicardial Pacer wires: Discontinue on POD#2, unless AVR/MVR discontinue on POD#3. Check if the wires are to be pulled or cut as listed in the initial post-op admission note to the CVICU as well as verify on the labels of each wire. Check platelets, INR, PTT (if available) prior to pulling wires. On weekends, contact surgeon on-call prior to removing pacer wires.
   * Foley: discontinue by POD 2 @ 5am if off pressors and hemodynamically stable. Follow up void check in 8hrs.
   * Swan: Discontinue POD#1 am if off inotropes and hemodynamically stable. Exception: Discontinue swan on POD#2 s/p MVR.
   * Perioperative antibiotics: Should be discontinued within 48 hours. If continued document reason in chart. Ancef x5 doses unless contraindicated, use Vancomycin. If open chest with closed skin, continue with Ancef x 48hrs protocol. If open chest with open skin, continue Vanco and Zosyn until chest closure.
   * Chest pulmonary toilet, incentive spirometer, wean Fio2, order appropriate nebulizer treatment if needed. Add home pulmonary medications. Consider need for Metanebs.
   * Physical therapy and occupational therapy orders to be placed
   * Transfer when stable. Be sure to specify where the patient is going in transfer order.
     1. CHF, Heart Transplant, VAD, TAVR patients go to ?
     2. CABG, Valves, dissections, etc. to ?.
2. POD 2 and 3
   * Increase BB as tolerated. (MAP Goal >65)
   * Start ACE-I, especially if HTN, poor EF, or DM. Unless contraindication (elevated creatinine or allergy)
   * Blood sugar control- start home DM meds if tolerating full diet.
   * Evaluate Diuresis
   * Evaluate removal of chest tubes and pacing wires
   * Evaluate need for starting anticoagulation and follow-up with surgeon regarding AC plan (VAD, valve, a fib etc.).
   * Evaluate need for Plavix if ACS.
   * Evaluate need for critical care.

**Weekend Protocol for APPs covering the CVICU:**

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     1. Epicardial wire removal

a. Epicardial wires may only be pulled on the weekend while a surgeon is in house (same per weekly protocol). During the weekends, the on-call surgeon usually likes to be notified FIRST THING in the morning so that they can plan rounds and hospital time around any wires that will need to be removed.

b. Please keep the following information in mind prior to texting the surgeon regarding removal of epicardial wires: CUT vs PULLING of wires (listed in post-op note), heparin infusions (should be held 4-6 hours prior to wire removal), INR (double check with surgeon if INR is greater than 1.8), Platelet count (recommended for PLTs to be greater than 100,000), post op heart blocks (double check clinical status of patient as well as cardiology’s note for recommendations).

2.     Scheduling OR cases on the weekend

a. Non-Emergent OR cases:

b. Urgent/Emergent OR cases: Clarify with the surgeon the time that the patient will be taken to OR to allow the bedside nursing team to get the patient ready to transport on call to OR. All delays should be evaluated by our team.

6. ECMO Consults

a. Generally, all ECMO consults should be called through the transfer center and to the attending surgeon or the charge intensivist in the ASCU. However, if a consult for ECMO is called directly to the ASCU charge intensivist cell phone, re-direct to transfer center. Be sure to get the patient’s information and the direct number for the call back attending physician who is currently caring for the patient.

**Extracorporeal Membrane Oxygenation (ECMO)**

ECMO is a mechanical circulatory device for severe refractory cardiogenic shock and/or respiratory failure. The Advent Health ACSU Adult ECMO program is well known and affiliated with multiple area hospitals for ECMO evaluation, placement, management, and weaning. The ECMO program is intensivist driven in the ASCU and incorporates the operating room for vascular repairs and decannulation.

* Evaluation
  + Consults are made directly to surgeons or ICU attending. Direct all consults called to transfer center.

**ECMO Candidate Algorithm**

1. **Does the patient have 1 or more ABSOLUTE contraindications for ECMO?**

[ ] multi-organ failure

[ ] brain injury

[ ] aortic dissection

[ ] bacteremia or sepsis

[ ] severe aortic regurgitation

[ ] received TPA

[ ] progressive & non-recoverable pulmonary disease

[ ] unwitnessed cardiac arrest

[ ] chronic severe pulmonary HTN

[ ] obesity (>120kg?)

[ ] no bridge to device, recovery, or transplant

If YES, the patient is NOT a candidate for ECMO.

If NO, please see question 2.

1. **Is the patient between the ages of 18-30years old?**

If YES, please see bullet a)

If NO, please see question 3.

1. *Does the patient (age 18-30yrs) have 4 or more RELATIVE contraindications for ECMO?*

[ ] fails to meet selection criteria for ECMO

[ ] contraindication for anticoagulation

[ ] recent surgery or trauma

[ ] CPR >20 minutes

[ ] bleeding

[ ] organ failure – AKI, Shock liver, etc.

[ ] > 7days mechanical ventilation?

[ ] malignancy

[ ] immunosuppression (HIV, transplant Pt, rheum. Pt, etc.)

If YES, the patient is NOT eligible for ECMO.

If NO, the patient should be further evaluated for possible ECMO cannulation.

1. **Is the patient between the ages of 31-50 years old?**

If YES, please see bullet b)

If NO, please see question 4.

1. *Does the patient (age 18-30yrs) have 3 or more RELATIVE contraindications for ECMO?*

[ ] fails to meet selection criteria for ECMO

[ ] contraindication for anticoagulation

[ ] recent surgery or trauma

[ ] CPR >20 minutes

[ ] bleeding

[ ] organ failure – AKI, Shock liver, etc.

[ ] > 7days mechanical ventilation

[ ] malignancy

[ ] immunosuppression (HIV, transplant Pt, rheum. Pt, etc.)

If YES, the patient is NOT eligible for ECMO.

If NO, the patient should be further evaluated for possible ECMO cannulation.

1. **Is the patient between the ages of 51-65 years old?**

If YES, please see bullet c)

If NO, please see question 5.

1. *Does the patient (age 51-65yrs) have 2 or more RELATIVE contraindications for ECMO?*

[ ] fails to meet selection criteria for ECMO

[ ] contraindication for anticoagulation

[ ] recent surgery or trauma

[ ] CPR >20 minutes

[ ] bleeding

[ ] organ failure – AKI, Shock liver, etc.

[ ] > 7days mechanical ventilation

[ ] malignancy

[ ] immunosuppression (HIV, transplant Pt, rheum. Pt, etc.)

If YES, the patient is NOT eligible for ECMO.

If NO, the patient should be further evaluated for possible ECMO cannulation.

1. **Is the patient between the ages of 66-75 years old?**

If YES, please see bullet d)

1. *Does the patient (age 66-71yrs) have 1 or more RELATIVE contraindications for ECMO?*

[ ] fails to meet selection criteria for ECMO

[ ] contraindication for anticoagulation

[ ] recent surgery or trauma

[ ] CPR >20 minutes

[ ] bleeding

[ ] organ failure – AKI, Shock liver, etc.

[ ] > 7days mechanical ventilation

[ ] malignancy

[ ] immunosuppression (HIV, transplant Pt, rheum. Pt, etc.)

If YES, the patient is NOT eligible for ECMO.

If NO, the patient should be further evaluated for possible ECMO cannulation.

* + If likely requiring ECMO make sure patient has an upper extremity Arterial line and at least one upper extremity triple lumen CVC if VA-ECMO or lower extremity triple lumen CVC if VV-ECMO.
  + Notify collaborating teams of urgent/emergency ECMO cannulation being done in 9900
    - on-call perfusionist
    - on-call cardiac surgeon
    - CVICU Charge RN
    - Radiology Tech for fluoroscopy if VV ECMO
      * Page 22-2912 to access the radiology technician
      * See section on Portable Fluoroscopy Equipment for further details
  + STAT ECHO
* VA ECMO
  + - Mostly performed percutaneously at bedside during CODE BLUE event.
    - Also performed in OR when planned as a subclavian approach or as central cannulation when unable to come off cardiopulmonary bypass
* Veno-venous (VV) ECMO
  + Placed under fluoroscopy at bedside with C-arm
  + Provides Oxygenation and ventilation.
  + No Hemodynamic support
  + via right internal jugular vein with Avalon or subclavian with Crescent catheter, which is a bi-caval.
  + Requires cerebral saturation monitors on all patients
* Veno-arterial (VA) ECMO
  + Indication: cardiogenic shock.
  + Provides complete cardiovascular support
  + Require a distal perfusion catheter to SFA on arterial cannula side
  + Requires cerebral and lower extremity saturation monitors on all patients.
  + Requires upper extremity arterial line.
* Hemodynamic Transesophageal weaning
  + Refer to instructions regarding HTEE weaning process.
  + Read the TJUH JTCVS Journal article and see Table 2.
    - “Weaning of extracorporeal membrane oxygenation using continuous hemodynamic transesophageal echocardiography”
* Bridging off ECMO
  + VV ECMO is bridge to recovery, evaluation for lung Tx or terminal care.
  + VA ECMO support can be bridged to recovery, MCS, PCI/Bypass, TAH, OHT, transplant.
  + According to UNOS heart listing criteria, patients on VA ECMO are listed as Status 1
* Anticoagulation/Antiplatelet
  + 325mg Aspirin
  + Avoid irreversible agents such as Plavix, Dabigatran, Efferent, etc.
  + Heparin infusion
    - PTT goal 45-55 unless thrombosis
    - Consider appropriate antithrombin dosing if AT3 deficient.
* Contra-indications
  + Sepsis
  + Irreversible disease process
  + Severe neurological injury
  + Inability to anticoagulated
  + Significant calcification in aorta or femoral vessels.
  + Bridge to nothing

**Meduri Protocol for VV ECMO**

Meduri Protocol per JAMA 1998;280(2):159-65...  
  
Methylprednisolone IVP LD 2 mg/kg once, followed by  
2 mg/kg/day on Days 1-14  
1 mg/kg/day on Days 15-21  
0.5 mg/kg/day on Days 22-28  
0.25 mg/kg/day on Days 29-30  
0.125 mg/kg/day on Days 31-32  
  
HOWEVER, we use a modified 14 day schedule:  
Methylprednisolone IVP LD 2 mg/kg once, followed by  
2 mg/kg/day divided into q6h dosing x 12 doses (3 days)  
1 mg/kg/day divided into q6h dosing x12 doses (3 days)  
0.5 mg/kg/day divided into q6h dosing x12 doses (3 days)  
0.25 mg/kg/day divided into q12h dosing x 6 doses (3 days)  
0.125 mg/kg/day q24h x2 doses (2 days)  
  
For example, a 100 kg patient would get a 200 mg load once followed 6 hours later by  
50 mg ivp q6h\* x 12 doses, then  
25 mg ivp q6h\* x 12 doses, then  
12.5 mg ivp q6h\* x 12 doses, then  
12.5 mg ivp q12h\* x 6 doses, then  
12.5 mg ivp q24h\* x 2 doses, stop

FUNDAMENTALS OF ACUTE CARE: HEMODYNAMICS

**I. HEMODYNAMICS**

**A. Goals**

The goal of hemodynamic monitoring is to ensure optimum oxygen delivery to the organ tissues. Hemodynamic monitoring on the CVICU consists of:

1. Vital signs.

a. MAP >65 mmHg

b. Normal Sinus Rhythm, 60-100 bpm

c. Afebrile

d. Respiratory rate.

2. Urine output: 0.5cc/kg/hr.

3. Physical examination.

a. Baseline Mentation

b. Warm

c. Peripheral Pulses

d. Diaphoretic?

Alterations in these signs or observations indicate an appropriate work up!

Remember: “ALL COMPENSATIONS FAIL”

**B. Building Cardiac Output**

Cardiac output is the total volume output of the heart and in the absence of septal defects is the same for the right ventricle and the left ventricle.

Cardiac output = Heart rate \* Stroke Volume

Cardiac output = Heart rate \* (ejection fraction \* end diastolic volume)

The determinants that may be manipulated to control cardiac output are therefore:

1. Heart rate.

2. Rhythm.

3. Preload.

4. Afterload.

5. Contractility.

Patients suffering from inadequate output (cardiac index less than 2.2 L/min/m2) should have each of these parameters sequentially evaluated and treated in the order listed above.

The normal ranges, physiologically acceptable perturbations, and interventions to control these factors are outlined below.

**1. Cardiac Output: Heart rate: Normal 60-100.**

Tachycardia (HR > 100)

a. Tachycardia may diminish the length of diastole with concomitant impairment of diastolic activities:

1) Coronary perfusion

2) Ventricular filling.

b. Increased heart rate is indicative of either compensation for impaired ventricular filling or a response to fever, chronotropic medications, or excessive pain.

c. Tachycardia can be physiologically beneficial in adults with impaired ventricular function and dilated ventricles, and elderly adults. A relative tachycardia is also beneficial in mild to moderate aortic insufficiency to limit regurgitant fraction.

d. We may consider pacing a patient or using a chronotropic medication to maintain adequate cardiac output.

e. Supraventricular dysrhythmias resulting in tachycardia must have ventricular rate controlled by termination of the rhythm or limiting the ventricular response. See Dysrhythmias below.

Treatment of Tachycardia

Determine significance and etiology of tachycardia and treat accordingly:

a. Physiologic tachycardia in dilated ventricles may be tolerated up to 100 BPM without impaired ventricular filling.

b. Optimize preload (is the patient dry?)

c. Eliminate fevers

d. Eliminate dysrhythmias or take steps to control ventricular response (see Dysrhythmias below).

e. Consider decreasing chronotropic agents (dobutamine, epinephrine) if cardiac output is adequate.

f. Are the epicardial leads attached? Does the pacemaker need to be interrogated?

Bradycardia (HR<60)

a. Bradyarrhythmia’s may lead to cardiogenic shock when compensation by increased stroke volume is inadequate to maintain adequate cardiac output (CO = SV x HR).

b. Bradycardia may be the result of heart block, excessive beta blockade, amiodarone, or digoxin toxicity.

c. Myocardial edema within the first 48 and hypothermia initially post-op are common causes of bradycardia.

c. Pacemaker failure.

Treatment of Bradycardia

Determine the physiologic significance of bradycardia (is hypoperfusion present?)

a. Bradycardia with HR>50 if blood pressure is adequate in most patients.

b. Initially, the open-heart patient may not tolerate a relative bradycardia.

c. Initial therapy is to begin atrial pacing.

d. If atrioventricular (AV) conduction is not consistent, initiate AV sequential pacing (go to Pacemaker section).

e. Patients without pacing wires may be treated immediately with epinephrine or transcutaneous pacing if unstable. In less emergent situations, consult EP and/or treat with chronotropic agent infusion (i.e.: Isoproterenol). These interventions require ICU admission if not already in ICU

f. Stop beta blocker.

**2. Cardiac Output: Rhythm**

a. Normal sinus conduction provides optimal left ventricular filling and optimal output. This is particularly important in the ventricle impaired by dilation or concentric hypertrophy.

b. Atrial pacing alters cardiac dynamics little.

c. AV sequential pacing is inferior to atrial pacing with normal conduction but is essential if AV conduction is not consistent or dependable.

d. Atrial pacing is of no value in atrial fibrillation although rapid atrial pacing may be used to “recapture” the atria and return the heart to a normal sinus rhythm (Usually Atrial Flutter).

e. Dysrhythmias must be aggressively treated. See Dysrhythmia and Pacemaker sections.

f. If patient is chronic atrial fibrillation or paroxysmal, they will likely not maintain sinus rhythm following open heart surgery.

g. Goal: sinus rhythm or normal atrioventricular activation sequence.

**3. Cardiac Output: Preload**

|  |  |
| --- | --- |
| Hemodynamic Parameters | Normal Values |
| Central Venous Pressure (CVP) | 0-8 mmHg |
| Right Ventricular Pressure | 15-30/0-8 mmHg |
| Pulmonary Artery Pressure (PAP) | 15-30/3-12 mmHg |
| Pulmonary Capillary Wedge Pressure (PCWP)  PAPI ( | 2-12 mmHg |
| Left Atrial Pressure (LAP) | 2-8 mmHg |

a. Preload is the length of the myofibrils at end-diastole. A series of approximations allow estimation of relative preload.

1) Left ventricular end-diastolic volume (EDV) approximates myofibril length.

2) Left ventricular end-diastolic pressure (EDP) approximates EDV but is related to EDV in a curvilinear manner.

3) LV EDP is successively approximated by the pulmonary artery capillary wedge pressure (PACW), pulmonary artery diastolic pressure (PAD), and central venous or right atrial pressure (CVP).

4) Do not wedge the PA catheter without attending physician present.

5) The anesthesiology team will provide optimal filling pressures from the operating room, which is documented in post-op note and handoff for future post-op management.

* + 1. The optimal pressures in the OR are not always the correct filling pressures. The correct filling pressure for a patient is one that results in the best hemodynamics. This may differ in the ICU.
    2. \*DO NOT ASSUME THAT ALL PATIENTS NEED THE SAME LEVEL OF PRELOAD\*
    3. Patients with aortic stenosis have concentric hypertrophy and require greater filling pressures to appropriately stretch the ventricle.
    4. Patients with long standing mitral regurgitation have thin, dilated ventricles and require lower filling pressures to achieve appropriate stretch.

b. Low filling pressure is acceptable if it results in an adequate cardiac output.

\*DO NOT TREAT THE NUMBERS, ASSESS THE PATIENT'S VENTRICULAR FUNCTION\*

c. The effective left ventricular preload is impaired by impaired systemic venous return:

1) cardiac tamponade,

2) positive end expiratory pressure (PEEP)

3) cardiac herniation s/p pneumonectomy (rare at tjuh)

4) tension pneumothorax

5) excessive pulmonary hypertension

d. Pulmonary artery pressures will not accurately reflect preload the filling of the LEFT VENTRICLE under conditions of:

1) Mitral stenosis or other pulmonary venous obstruction.

2) Pulmonary hypertension.

3) Severe aortic regurgitation (high mean aortic gradient)

4) Cardiac tamponade from any cause.

e. Excessive intravascular volume causes pathologic ventricular dilation which may in turn result in myocardial ischemia. Pulmonary edema can result from pulmonary capillary wedge pressures once they begin exceeding 25-30 mmHg.

f. Elevated filling pressures:

1) impaired ventricular function: i.e., myocardial ischemia or cardiomyopathy,

2) excessive volume infusion, valvular dysfunction, or septal defects.

**Treatment of Hypovolemia**

a. Determine the physiologic cause of low filling pressures.

b. Volume replacement is usually colloid based. Consider your needs when resuscitating with volume.

c. The initial volume resuscitation for the open-heart patient is usually 5% human albumin. Total administration for POD0 may result in 1-1.5 L. If more volume resuscitation is necessary, notify Surgeon and consider blood products if appropriate or 0.9% NS.

d. Consider fresh frozen plasma or platelets if coagulopathy present.

e. Determine the patient's hematocrit, state of blood loss, and need for additional oxygen carrying capacity (i.e., elderly, poor pulmonary function, diabetic, steroid dependent, impaired renal function, hypoxemia).

f. If volume infusion is needed and additional O2 carrying is appropriate choose PRBC.

g. If volume infusion is needed but PRBC are not desired, choose FFP if ongoing bleeding, otherwise Albumin is appropriate.

f. Crystalloid is not generally used for volume expansion; the effects are generally not as long lasting, and more volume is required.

h. Crystalloid may be required to keep up with volume balance in patients who have prodigious urine output (i.e., >500-1000 ml/hour).

I. Differentiate between the patient's intravascular status and total body fluid status (use wedge pressure).

k. Consult the attending if you have used over 1L 5% Albumin or requiring unexpected blood products.

l. Carefully choose who you transfuse, consider co-morbidities, patient wishes, and transplant status. Follow up with surgeon before administering blood products.

**4. Cardiac Output: Afterload**

Afterload is the resistance against which the left ventricle must eject blood and is approximated

by the mean arterial pressure or systemic vascular resistance (calculated).

a. Normal MAP is 65-90 mmHg.

b. Normal SVR is 800-1200 dynessec-cm-5.

c. MAP should be maintained at 65 mmHg after cardiac surgery. Hearts which have had an ischemic insult are better able to generate an adequate cardiac output if afterload is maintained at the lower end of the normal range.

d. Maintaining a low MAP (60-70) is critically important when any aortic surgery has been performed (aortic dissection, aortic transection, aortic aneurysm repair) to prevent damage to suture lines and risk of rupture/bleeding.

e. The MAP is maintained at a slightly higher level (75-80) in patients who are elderly, chronically hypertensive, impaired renal function, have concentric ventricular hypertrophy (generally after aortic valve surgery for aortic stenosis), or have known carotid or renal artery stenosis.

Treatment of Hypotension

a. Assess the physiologic cause of hypotension.

I. tamponade

ii. tension pneumothorax

iii. Hemorrhage

b. Optimize heart rate to 90-100 min if pacing wires are available.

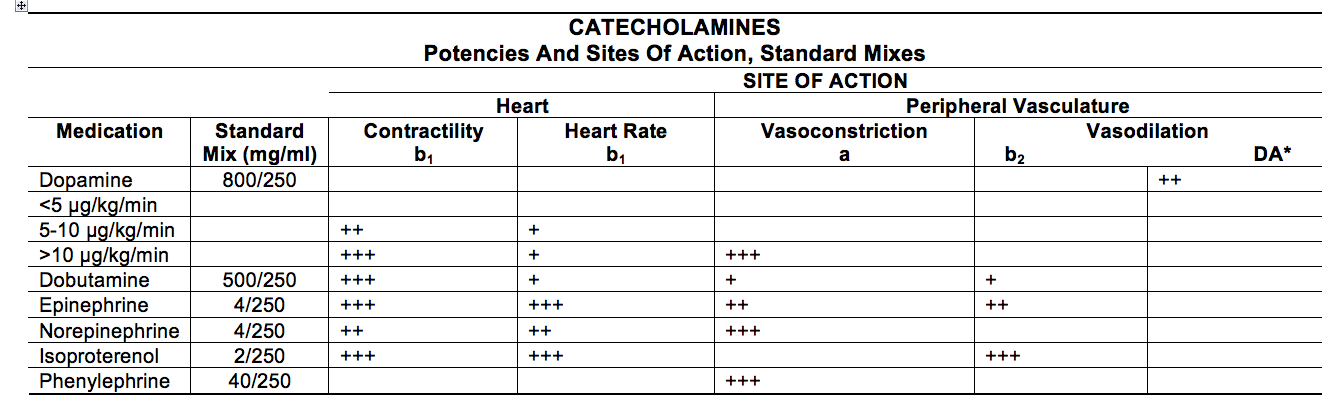
c. Stop afterload reducer. Beta Blocker, Ace inhibitor, etc.

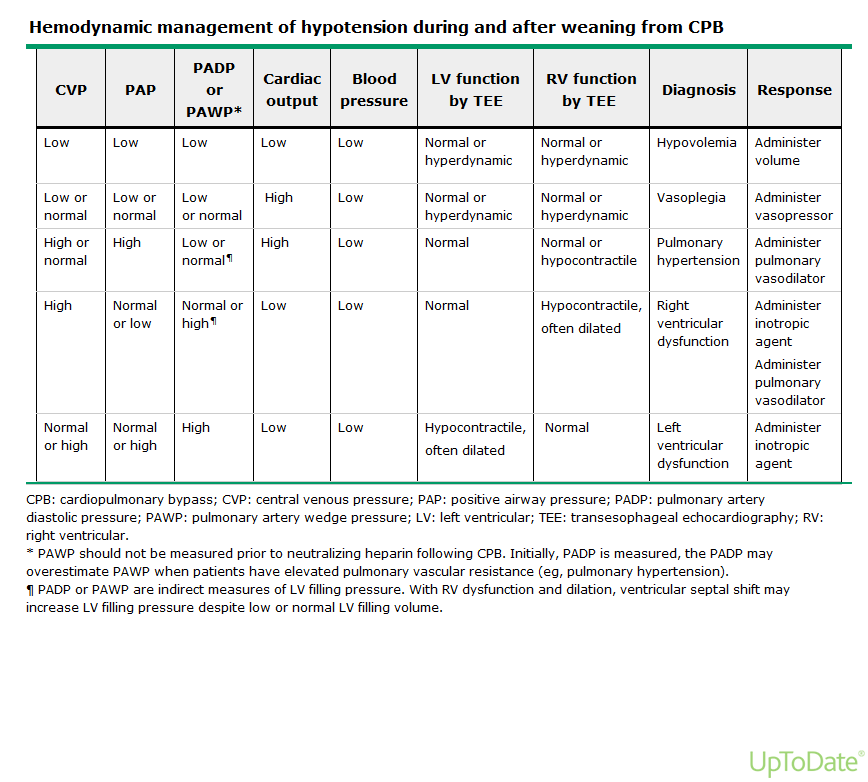
e. Optimize preload: volume infusion of colloid

f. Hypotension (systolic blood pressure <50) requires rapid intervention. Administer calcium chloride (calcium chloride is a salt which is available immediately after administration, calcium gluconate is a sugar which liberates calcium only after metabolism in the liver) 500-1000 mg (1000 mg/10 ml) or epinephrine (10mcg).

g. If BP does not respond to increased heart rate or volume challenge, then phenylephrine or vasopressin infusions are the preferred vasopressor.

h. Remember that increasing the central arterial pressure with catecholamines may not improve perfusion of specific organ beds (flow restriction by vasoconstriction). If the distal extremities are well perfused (warm feet) then the rest of the body is likely well perfused.





**Vasoplegia Guidelines and Clinical Protocol**

**1. Development:**

Vasoplegic syndrome is defined as end organ hypoperfusion despite normal or high cardiac output. The incidence of Vasoplegia in the setting of cardiac surgery is up to 30% and is associated with increased ICU and hospital length of stay, renal failure and mortality. Mortality associated with Vasoplegia approaches 50%. Risk factors include cardiopulmonary bypass (CPB), duration of CPB, mechanical circulatory support, heart transplantation, reduced ejection fraction, preoperative ACEI or ARB, and blood transfusion. Data to support definitive treatment for Vasoplegic syndrome are lacking. Recent research regarding the management of Vasoplegic syndrome involves the use of agents that target nitric oxide (NO) production, such as methylene blue and supplements such as ascorbic acid and cyanocobalamin. Some data may be extrapolated from treatment for septic shock, such as corticosteroids and supplements.

The purpose of this guideline is to provide education to a multidisciplinary group caring for cardiac surgery patients to increase awareness of vasoplegic syndrome, promote early recognition, and provide guidance of available management to be applied to each individual case.

**2. Criteria for use:**

Patients undergoing cardiac surgery, both intra-operative and post-operative in the cardiovascular intensive care unit.

**Vasoplegia criteria could include:**

* Mean arterial pressure (MAP) < 60 mmHg
* Systemic vascular resistance (SVR) < 800 dynes/sec/cm5
* **Adequate volume resuscitation**
* Cardiac index (CI) > 2.2 L/m/m2
* Vasopressor requirement (any two of the following)
  + Vasopressin ≥ 0.04 units/min
  + Phenylephrine ≥ 2 mcg/kg/min
  + Norepinephrine ≥ 0.2 mcg/kg/min
  + Epinephrine ≥ 0.08 mcg/kg/min

**3. Content**

Therapeutic Management:

* Vasopressors
  + Target multiple receptors (alpha, beta, vasopressin)
  + Preferentially, add vasopressin when moderate doses of alpha, beta agonists are required
* Optimize acid base status
* Target increase in blood viscosity
  + Increased blood viscosity increases hemodymanic hinderance, thereby improving SVR (Pouseuille’s Law)
  + Target hemoglobin 10 g/dL
    - Resume standard hemoglobin goal once patient is clinically improving
    - When ordering blood in Epic, use “symptomatic anemia” indication and add a comment that “treatment based on vasoplegia guideline.”
  + Target fibrinogen ≥ 200 mg/dL
* Adjunct medication bundle
  + Methylene Blue
    - **Use as an early adjuvant**
    - 2 mg/kg bolus followed by 0.5 mg/kg/hr for 12 hours
    - Administration: via central line; no drug compatibility information available, administer in dedicated line.
    - **Contraindication:** G6PD deficiency; methylene blue can cause serious or fatal serotonergic syndrome in patients who are receiving serotonergic medications: serotonin reuptake inhibitors (SSRIs), serotonin and norepinephrine reuptake inhibitors (SNRIs), and monoamine oxidase inhibitors (MOAIs).) Additional agents that can contribute to serotonin syndrome include fentanyl, tramadol. Serotonergic drugs should not be used within 72 hours of methylene blue administration. If a patient is currently on a serotonergic medication, weigh the risk and benefit of administering methylene blue.
  + Ascorbic acid (Vitamin C)
    - 1500 mg IVPB q6h x 4 days
    - Limited side effect profile
    - **Caution** in renal failure without renal replacement therapy due to risk of nephropathy
  + Hydrocortisone
    - 50 mg IVP q6h x 4 days
  + Thiamine
    - 200 mg IVPB q12h x 4 days
* Antihistimines IF patient is receiving vancomycin
  + Diphenhydramine 50 IVP q8h
  + Famotidine 20 mg IVP q12h
* **Use Hydroxocobalamin (B12) ONLY if methylene blue is contraindicated and patient is in critical condition AND other medications fail**

Hydroxocobalamin (Cyanokit)

* + 5 g IVPB over 15 min (may repeat dose once)
    - Evidence for use is preliminary: **Use only if methylene blue is contraindicated** (i.e. G6PD deficiency, presence of multiple serotonergic agents)
      * **Only use if other medications fail**
    - May cause lab inaccuracies:
      * Hemoglobin (12-16 h)
      * Creatinine, glucose, alkaline phosphatase, LDH (1 day)
      * Bilirubin (4 days)
      * PT, PTT, INR (1-2 days)
    - Causes red colored urine, mucus membranes, skin
    - Only stable for 6 hours after reconstituted

Appendix A:

|  |  |
| --- | --- |
| **Vasoplegic Syndrome** | |
| **Criteria** | * MAP <60 mmHg * SVR < 800 dynes/sec/cm5 * Adequate volume recuscitation * CI > 2.2 L/m/m2 * Vasopressor requirement (any 2 of the following)   + Vasopressin ≥ 0.04 units/min   + Phenylephrine ≥ 2 mcg/kg/min   + Norepinephrine ≥ 0.2 mcg/kg/min   + Epinephrine ≥ 0.08 mcg/kg/min |
| **Management** | * Vasopressors- target multiple receptor sites, include vasopressin * Optimize acid base status * Optimize blood viscosity * Hgb ≥ 10 g/dL * Fibrinogen ≥ 200 mg/dL |
| **Medication**  **Bundle** | * Methylene Blue (MB) \*rule out contraindications * 2 mg/kg bolus followed by 0.5 mg/kg/hr for 12 hours * Ascorbic Acid (Vitamin C) 1500 mg IVPB q6h x 4 days * Hydrocortisone 50 mg IVP q6h x 4 days * Thiamine 200 mg IVPB q12h x 4 days |
| **Consider** | * Diphenhydramine 50 mg q8h AND Famotidine 20 mg IVP q12h IF receiving vancomycin * Hydroxycobalamin (Cyanokit) \*if MB contraindicated, last line therapy * 5 g IVPB over 15 min (may repeat dose once) |

**Midodrine Guidelines**

*Initial dosing of midodrine:*

Post-operative patients requiring pressors on POD 2, cortisol normal:

* If low-medium dose of single vasopressor, start midodrine 10 mg q8hr (not TID)
* If high dose vasopressor OR more than one vasopressor, start midodrine 15 mg q8hr (not TID)

Chronically critically ill patients requiring pressors (non-septic):

* Start 15 mg q8h (not TID)

*Weaning of Midodrine:*

For normotensive patients: when off pressors for 24 hours AND MAP ≥70 mmHg or SBP ≥120 mmHg, wean by 5 mg/dose once a day to 5 mg q8h (not TID) then stop.

\*If patient is hypertensive, consider discontinuing agent\*

**Definitions:**  
***Low dose pressor:* phenylephrine ≤ 0.9 mcg/kg/min, vasopressin 0.01 units/hr, norepinephrine ≤0.5 mcg/kg/min**

***Moderate dose pressor:* phenylephrine 1-2 mcg/kg/min, vasopressin 0.02-0.03 units/hr, norepinephrine 0.6-0.9 mcg/kg/min**

***High dose pressor:* phenylephrine >2 mcg/kg/min, vasopressin ≥ 0.04 units/hr, norepinephrine >1 mcg/kg/min**

**STOP!**

**Wean patient off prior to discharge.**

**If transferring to long term care facility, provide instructions to wean.**

**Administration of Peripheral Norepinephrine in the ICU**

* Norepinephrine 8mg/250ml NSS (32mcg/ml) can be administered peripherally or centrally.  If administering peripherally, the **MAXIMUM RUN TIME is 24 hours from the time it was initiated.**If administration is required longer than 24 hours regardless of the number of times the infusion was stopped, it MUST be administered CENTRALLY (Changing the peripheral site does not restart the clock)**.**
* Peripheral administration of Norepinephrine must be through a dedicated large vein with at least a 20 Gauge catheter. **HAND OR FOOT IVs MAY NOT BE USED.**
* Norepinephrine 32mg/250ml NSS (128mcg/ml) **MUST be administered CENTRALLY**.
* In patients who require peripheral vasopressors intra-op, a plan for access must be discussed with the ICU attending in the event vasopressors are needed in the post-operative period.
* For treatment of extravasation, Nitroglycerin 2% ointment has been placed in all Critical Care PYXIS on override for immediate use.  Phentolamine is available from Pharmacy upon order entry into EPIC.

**Treatment of Hypertension**

* 1. Assess the physiologic significance of hypertension. Treat any MAP >100
  2. Cardene IV is a channel blocker that is also rapidly effective, easily titratable, and does not inhibit the AV node.
  3. Sodium nitroprusside (SNP) has a rapid onset with extremely short half-life.
     1. Always give this in a central line. (Avoid phlebitis)

c. Hydralazine (IV, PO) is also an effective, rapid acting anti-hypertensive agent.

e. ACE inhibitors are effective afterload reducers. ACE inhibitors should not be used in the immediate postoperative period.

e. The young hyperdynamic patient with difficult to control hypertension usually responds well to labetalol.

f. Adjust afterload only after preload has be optimized.

**5. Cardiac Output: Contractility**

Contractility is defined as the slope of the stroke work and end-diastolic volume relationship. This represents the ventricle's ability to do work with the available preload. In other words, Squeeze!

a. Adequacy of contractility is assessed after the preceding 4 factors are optimized (i.e.: rate, rhythm, preload, and afterload.)

b. Very few patients will require limitation or reduction of contractility. This important set of patients includes those with:

1) Myocardial ischemia.

2) Aortic surgery (dissection, transection, AVR, aortic root replacement).

Assessment of Contractile Function

a. Optimize heart rate and cardiac rhythm.

b. Optimize preload then optimize afterload.

c. If systemic perfusion remains poor (low CO, low SvO2) then consider:

1) Tamponade.

2) Tension pneumothorax.

3) Re-zero transducers, recheck SV02 calibration, recheck CO constant and volume.

4) Exclude metabolic or respiratory acidosis.

d. If perfusion is still poor, then consider inotropic medications.

Treatment of Contractile Dysfunction

a. Goal: 2.2 liters minute-1meter-2 and/or adequate systemic perfusion.

b. The preferred Inotropes are epinephrine, dobutamine, or milrinone.

c. Discussion should be held with intensivist whenever inotropes are required.

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| PACWP  Or  PAD | MAP | Cardiac Index (L-min-m-2) | | |
| (mmHg) | (mmHg) | <2.2 | 2.2-3.0 | >3.0 |
| <7 | <65 | Volume | Volume | Volume |
|  | >65 | Volume | Observe | Observe |
|  | <65 | Volume | Volume/Pressor | Volume/Pressor |
| 8-14 | 65-90 | Volume/Inotrope | OBS | OBS |
|  | >90 | Volume/  Afterload reduction | Afterload reduction | Afterload reduction |
|  | <60 | Inotrope  vasopressor | vasopressor | vasopressor |
| 15-19 | 65-90 | Consider Volume  Inotrope | Observe | Observe |
|  | >90 | Afterload Reduction  Inotrope | Afterload Reduction | Afterload Reduction |
|  | <65 | Inotrope  Vasopressor | Vasopressor | Vasopressor |
| >20 | 65-90 | Inotrope | Observe  Consider Diuresis | Observe  Consider Diuresis |
|  | >90 | Vasodilator | Vasodilator  Consider Diuresis | Vasodilator  Consider Diuresis |

This Algorithm is fundamental for the typical open heart surgery patient during initial presentation. Consider the patients appropriate filling pressures and current vaso after medications.

**6. Cardiac Output: Additional Therapy**

If adequate systemic perfusion does not result from optimization of cardiac output factors and inotropes are ineffective, consider:

a. Assess and correct metabolic acidosis (serum total C02, lactate).

b. Exclude myocardial ischemia, valvular dysfunction, or ventricular septal defect: ECHO.

c. Pericardial effusion or Cardiac Tamponade.

d. Tension Pneumothorax.

e. Ventilator malfunction, pulmonary insufficiency, or hypoxia.

f. Pulmonary embolism (unusual in immediate postoperative setting after CPB).

g. Aortic dissection (are pulses bilaterally symmetric?).

h. Ventricular assist devices:

1) ECMO.

2) Intraaortic balloon pump.

3) Left ventricular Assist Device (LVAD).

**A. Mixed Venous Oxygen Saturation (SvO2)**

1. SvO2 is an estimate of the oxygen extracted by the systemic circulation and is used as an index of perfusion; the SvO2 in a normal individual is 65%

2. SvO2 can be drawn as either a central mixed venous from a CVP port on an upper extremity CVC or from a PA port from an SGC.

3. The oximetric monitor is the optimal method to assess the effect of interventions intended to improve oxygen delivery.

4. Assess the monitor before instituting therapy for low SvO2.

a. Confirm that hemoglobin is stable since last calibration.

b. Confirm that the light monitor is within the appropriate range.

c. Confirm that the catheter is in the pulmonary artery.

d. Confirm that the catheter is connected to the Oximetric machine.

5. A decreased SvO2 following cardiac surgery with stable cardiac output is most likely shivering.

Table 1: Possible causes of changes in SvO2.

|  |  |  |
| --- | --- | --- |
|  | Oxygen Delivery | Oxygen Consumption |
| Decreased SvO2 | diminished | stable |
|  | stable | increased |
| Increased SvO2 | improved | stable |
|  | stable | diminished |

6. Etiology of alterations in oxygen consumption or delivery.

a. Increased Oxygen consumption.

1. Shivering.

2. Double clutching the ventilator

3. Fever.

4. Dialysis.

b. Decreased Oxygen consumption.

1. Sepsis.

2. Resolution of adverse conditions.

c. Increased Oxygen delivery.

1. Improved cardiac output.

2. Improved hemoglobin concentration.

3. Increased hemoglobin oxygen saturation.

d. Decreased Oxygen delivery.

1. Impaired cardiac output.

2. Anemia (bleeding?).

3. Decreased hemoglobin oxygen saturation.

a) Pulmonary edema.

b) Ventilator malfunction

c) Pneumothorax.

d) ARDS, pneumonia.

e) Excessive secretions.

f) Malposition ET tube.

**D. Cardiac Tamponade**

1. Tamponade occurs when fluid accumulation in the pericardial space leads to increased intracardiac pressure or direct impingement on the cardiac chambers. This fluid impairs diastolic filling of the ventricles. There may be atrial compression or ventricular compression.

2. Tamponade may occur postoperatively in any cardiac patient regardless of the state of the pericardium.

**Tamponade may occur DESPITE a widely open pericardium** **and draining chest tubes.**

3. The patient with tamponade may have elevated, normal, or equalized intracardiac pressures by PA catheter.

4. Compensatory mechanisms include:

a. increased left ventricular ejection fraction

b. reflex tachycardia

c. increased systemic vascular resistance

d. renal conservation of fluid (i.e., oliguria) to maintain intravascular volume.

5. The diagnosis is a clinical decision. Requires a high degree of suspicion and should be considered in any patient with unexplained hypotension, shock, cardiac arrest or EMD. Consider tamponade when:

a. Ventricular function is less than expected for a particular patient.

b. Volume infusion improves ventricular output but has only a transient effect.

c. Inotropic agents are required to support the circulation when none were needed post-op.

d. Bleeding has slowed dramatically and/or dumping of 200-300 ml occurs sporadically.

6. Differential Diagnosis.

a. Tension pneumothorax.

b. Hypovolemic shock.

c. Myocardial infarction.

d. Pulmonary embolism.

e. Right heart failure including coronary air embolism.

f. Congestive heart failure.

g. Constrictive pericarditis.

7. The attending surgeon should be aware if tamponade is suspected.

Diagnosis of Tamponade

1. Symptoms: dyspnea, fatigue, agitation, air hunger

2. Signs.

a. Beck's Triad: hypotension, elevated central venous pressure, and muffled heart sounds.

b. Pulsus paradoxus: greater than 10-15 mmHg decrease in systolic BP on inspiration.

3. Diagnostic Studies.

a. CXR: enlarged cardiac silhouette, exclude pneumothorax, diaphragmatic rupture.

b. EKG: decreased voltages, nonspecific ST wave changes, electrical alternans.

c. Echocardiography.

1) Most accurate diagnostic test. However, the diagnosis is still clinical.

2) Evaluate for ventricular function, pericardial fluid collection, and examine heart for:

a) Abnormal respiratory variation in trans-valvular flow velocities.

b) Bulging of intraventricular septum into the LV.

c) Diastolic collapse of the RV.

d) NB: Tamponade physiology can be present in the post-operative patient even in the absence of ECHO findings as a posterior clot can prevent adequate filling of the right ventricle.

Treatment of Tamponade

1. \*\*If tamponade is suspected on clinical grounds, and the patient is hemodynamically unstable, do not delay treatment waiting for an echocardiogram.

2. Intravascular volume administration to increase CVP and attempt to overcome pericardial pressure.

3. Pericardiocentesis should be considered in any non-operative patient who is hemodynamically unstable and in whom the diagnosis of tamponade is suspected. (May need subxiphoid pericardial window to follow)

4. A peri-operative patient in cardiogenic shock, with the diagnosis of tamponade, and responsive to no other measures needs his sternotomy opened NOW.

**II. MECHANICAL VENTILATION**

**A. Indications for Ventilatory Support**

1. Conditions which result in need for ventilatory support:

a. Level of consciousness compromises ventilation or inadequate to protect airway.

b. Inadequate capacity to clear secretions.

c. Inadequate muscle strength.

d. Excessive work of breathing.

e. Upper airway obstruction.

f. Inadequate oxygenation despite increased FiO2 and adjunctive measures.

g. Atelectasis or consolidation unresponsive to more conservative therapy.

2. Specific indications for intubation:

a. Any patient visibly working to breathe needs consideration for intubation.

b. Hypoxemia unresponsive to increased FiO2 PO2 < 60 on FiO2> 80-100%.

c. Hypercarbia where pCO2 >55 or CO2 narcosis has ensued.

d. Respiratory rate >35-40.

e. Adjunctive measures have failed: oral airway, positioning, reversal of sedation.

3. Specific situations where intubation is to be considered:

a. Bag-mask-valve ventilation is inadequate.

b. Prolonged ventilatory support is required.

c. Patients with compromised airways.

d. Maxillofacial fractures.

e. massive hemoptysis.

f. Supraglottic obstruction or laryngospasm or laryngeal edema.

g. Airway protection when high risk for aspiration:

1) Excessive secretions.

2) Massive upper gastrointestinal bleeding.

4. Perform intubation electively under controlled circumstances.

5. Do not wait for respiratory failure requiring emergent intubation.

|  |
| --- |
| Mandatory equipment at bedside for intubation |
| Laryngoscope  Endotracheal tube (7.5-8 single lumen tube is preferred)  Oxygen with bag,valve,or mask  Suction apparatus with Yankauer tip  Intravenous access  Medications: epinephrine, lidocaine, atropine, sedative  Monitoring: blood pressure, EKG  Magill forceps  Stethoscope |

**B. Ventilator Modes**

1. Intermittent Mandatory Ventilation (IMV), Synchronized IMV (SIMV):

* Machine delivered rate and tidal volume are set.
* Air flow is provided so that patient may breathe spontaneously, although unassisted, between breaths. Pressure support is usually added in this mode to assist patient’s spontaneous breaths.
* Rate settings may be adjusted to provide tapering support as a weaning mode.
* SIMV allows ventilator breaths to be synchronized with patient-initiated breaths to improve patient comfort.
* In absence of patient initiation, IMV/SIMV provides the same ventilation as CMV.

3. Assist/Control ventilation (AC):

* Ventilator delivers preset tidal volume when triggered by patient's negative inspiratory pressure.
* Full breath delivered each cycle, no partial breaths possible.
* A minimum mandatory rate may be set.
* Anxious or hyperpneic patients at risk for hyperventilation.
* Triggering sensitivity must be set at critical level.
* Avoid respiratory alkalosis (too sensitive).
* Wasted inspiratory effort (too insensitive).
* Respiratory muscles will atrophy in this mode.

4. Pressure support (PS):

* Ventilator provides a constant pressure during patient-triggered inspiration.
* Augments patient's own inspiratory effort.
* May be used alone or in conjunction with other ventilator modes.
* Requires spontaneous ventilatory effort.
* Does not guarantee a minimum minute-ventilation.
* May be weaned independently from IMV rate.
* Preserves respiratory muscle coordination.
* Pressure support of 6-8 will augment for the resistance created by the endotracheal tube.
* The most comfortable mode in the awake patient.

4. Airway Pressure Release Ventilation (APRV):

* In standard, volume control ventilation, air is infused into the lungs under pressure for each breath and the passively allowed to escape. The “baseline” is end expiration.
* In pressure release ventilation, the patient is maintained at full inspiration, and, with each breath, the air is allowed to escape. The “baseline” is full inspiration.
* Ventilation occurs during the movement of air in either mode, so the tidal volume remains important to allow for CO2 removal.
* As a higher percentage of time is spent during inspiration, alveoli are stented open, and recruitment is maintained.
* Lower peak airway pressures can often be maintained although ventilation is often more difficult.
* The “augmented” part of the name refers to the ability for patients to “over breathe” the ventilator setting.
* This is an advanced mode of ventilation in that it infrequently used and not familiar to everyone involved in the patient’s care.

**C. Standard Ventilator Settings**

|  |  |  |
| --- | --- | --- |
| Initial Ventilator Settings | | |
| Rate | 10-15 | (Min-1) |
| Tidal Volume | 6-8 | (ml/kg/IBW) |
| Fi02 | 50 | (%) |
| Pressure Support | 10 | (Cm H20) |
| PEEP | 5 | (Cm H20) |

1. Rate:

a. Frequency of respirator cycling, usually set at 10-15 breaths/minute.

b. Increase rate or tidal volume to increase alveolar ventilation.

2. Tidal volume (Vt):

a. Volume cycled ventilators. set directly, start at 6-8 ml/kg.

b. Decrease if Peak Inspiratory Pressure (PIP) exceeds 35 cmH2O.

c. Pressure cycled ventilators: set via inspiratory pressure, adjust for desired Vt.

3. Inspiratory flow rate (IFIR):

a. Usually set at 50-80 L/min.

b. Higher IFR decreases inspiratory time and lengthens time for exhalation.

c. Normal inspiratory time: expiratory time (I:E) ratio is 1:2 to 1:3.

d. When compliance is diminished, high IFR results in excessive Peak inspiratory Pressures.

e. Inverse ratio ventilation (I:E ratio > 1:1) is useful for not compliant lungs and for patients with interstitial lung disease who have a low PaO2.

- Increasing the inspiratory time increases the mean pressure of the airways without increasing the peak airway pressure as more time is spent during the inspiratory phase

- Care should be taken using this approach as “auto-peep,” a sequential stacking of unexpired air, can result.

4. Peak Inspiratory Pressure (PIP):

a. Pressure cycled ventilator: airway pressure at which breath will be terminated.

b. If adequate PIP is delivered:

1) Good breath sounds bilaterally.

2) Visible chest movement.

3) Vt delivered is sufficient to maintain a normal PaCO2.

c. Volume cycled ventilator: overpressure alarm.

5. Fi02:

a. Fraction of oxygen in inspired air (room air is 0.21).

b. Most postoperative patients can start at 50%.

c. Adjust FiO2 to provide for PaO2 of 60-90 mmHg and SaO2 >90%.

d. FiO2 may be directly and safely reduced using PaO2 :FiO2 ratio.

Desired PaO2

Desired FiO2 =x Actual FiO2

Actual PaO2

e. FiO2 in excess of 50% subject’s patient to risk of oxygen toxicity after 48 hours.

f. Wean Fi02 aggressively to limit re-absorption atelectasis.

g. Do not extubate the post-op open heart until oxygenation is adequate with 50%.

6. Positive End Expiratory Pressure (PEEP):

a. Purpose: recruit alveolar units, improve functional residual capacity (FRC), and reduce FiO2 below toxic levels.

b. Diffusion is improved by thinning the air: blood interface.

c. Major benefit accrues from an increase in functional residual capacity.

1) Improves small airway patency at end-expiration.

2) Counterbalance’s airway closing forces.

3) Recruits alveolar units.

d. PEEP of 5 cm H2O is comparable to physiologic PEEP provided by glottic closure

e. Right ventricular (RV) performance is limited by PEEP.

1) Airway pressure increases pulmonary vascular resistance.

2) Capacitance volume shifts out of the thorax limiting preload to the right ventricle.

3) The RV must eject against a higher afterload with a limited preload.

4) Left ventricular performance is also reduced by preload limitation.

5) This functional hypovolemia may be ameliorated by increasing intravascular volume.

f. If PEEP levels are above 10 cm H2O:

1) Monitor intravascular volume: CVP monitoring is mandatory. Swan Ganz catheter is considered.

2) Optimize by maximizing compliance without limiting oxygen delivery (DO2).

3) High levels of PEEP narrow the delivered pressure amplitude, limiting Vt.

4) PEEP redistributes extravascular lung water and increases total lung water.

5) Ventilator disconnects are limited to brief periods or not at all.

6)

8) Increases risk of complications of mechanical ventilation.

9) May preclude transport.

10) PEEP levels above 15 cm H2O you risk pneumothorax. Consider Chest tube for prophylaxis.

7. Pressure support:

a. Physiologic setting of 5-10 cm H2O overcomes resistance of ventilator circuit and endotracheal tube.

b. Allows inspiratory support of patient-initiated breaths.

c. Stepwise reduction of PS allows gradual strengthening of respiratory muscles in de-conditioned patients.

8. Continuous positive airway pressure (CPAP):

a. Physiologically equivalent to PEEP and PS.

b. Maintains small airway patency by improving FRC.

c. Administer CPAP postoperatively to hasten return of FRC to preoperative levels.

d. If patient cannot clear secretions, CPAP is contraindicated.

e. If the patient is still requiring CPAP over 1 hour and is still dyspneic intubation is required.

f. CPAP may bridge the patient in pulmonary edema some requiring intubation while waiting for diuretic response.

**D. Ventilator Troubleshooting and Alarms**

|  |  |  |
| --- | --- | --- |
| Problem | Causes | Solutions |
| Hypercarbia | Ventilator malfunction  Ventilator disconnection  Low Vt because of circuit capacitance or air leak | Reconnect patient to ventilator  Review ventilator and alarm settings  Eliminate air leaks |
| Hypercarbia: Inadequate Vt | Bronchospasm  Mucus plugging  Atelectasis  Tension pneumothorax  Incorrect ventilator settings  Inadequate compensation for dead space | Treat bronchospasm and mucus plugging  Increase Vt or PEEP to improve compliance and recruit alveoli  Exclude pneumothorax  Consider pressure-cycled mode |
| Increased physiologic dead space | Pulmonary embolism  Diminished cardiac output with inadequate perfusion | Exclude or treat pulmonary embolism  Improve cardiovascular dynamics  Eliminate extra circuit tubing |
| Excessive CO2 production | Shivering  Rewarming  Hypermetabolic state  Sepsis  Excessive carbohydrate: fat caloric ratio  Fever | Treat and correct underlying problem  Consider muscle paralysis to control shivering until postoperative rewarming is complete |

**E. Hypoxia and Sudden Respiratory Decompensation**

1. EVALUATION:

a. Disconnect patient from the ventilator and hand ventilate with 100% FiO2.

b. Check for CO2 (ensure ET tube is still in trachea).

c. Perform physical examination.

d. Evaluate for adequate bilateral ventilation.

e. Evaluate hemodynamic status.

f. Laboratory evaluation.

1) Obtain ABG and mixed venous blood gas.

2) Obtain CXR.

* Check endotracheal tube position.
* Exclude pneumothorax.
* Assess lung fields for edema, consolidation, or Westermark's sign.

2. TREATMENT OF HYPOXIA AND SUDDEN RESPIRATORY DECOMPENSATION:

Evaluate inspiratory pressures:

a. High resistance to manual ventilation.

1) Exclude kinked tube or patient biting on tube.

2) Obstructed endotracheal tube: tumor, thrombus, or secretions.

a) Attempt deep endotracheal suctioning.

b) Include lavage with 5-10 ml normal saline.

3) If no obstruction, consider malposition tube.

a) Examine for bilateral breath sounds.

b) Confirm tube position on CXR.

4) Consider bronchodilators.

5) Consider diuresis if cardiac function is adequate.

6) Perform bronchoscopy for bronchoalveolar lavage for culture and sensitivity.

7) The presence of ARDS indicates that lung injury has already occurred. Goal for treatment is supportive care of circulatory, respiratory, renal systems until the lungs heal. Excessive FiO2 and inspiratory pressures cause ongoing lung injury and retard healing.

b. Low or normal inspiratory pressures.

1) Hemodynamically stable.

a) Exclude ventilator malfunction, review settings on panel NOW.

b) Consider PE, subjective dyspnea, shortness of breath.

c) Correct subjective dyspnea by increasing Vt.

d) Exclude pathologic processes before increasing sedation.

2) Hemodynamically unstable.

a) Exclude pneumothorax.

b) Consider PE, sepsis, relative hypovolemia, bleeding.

c) Assess cardiac function and consider Swan Ganz catheter.

**F. Weaning from Mechanical Ventilation and Extubation**

1. Methodology:

a. Reduce or stop sedation to optimize respiratory drive.

b. Progressively decrease the ventilator rate until extubating parameters are reached.

c. Weaning of IMV may continue if pCO2 remains < 35-48 mmHg.

2. Criteria for extubating:

|  |  |  |
| --- | --- | --- |
| Parameter | Units | Threshold |
| NIFM  Vt  Respiratory rate  F:Vt ratio  Oxygenation:PaO2  Ventilator settings:  rate  pressure support  FiO2 | cm H2O  ml-kg-1  breaths-min-1  breaths-min-1-liter-1  mmHg  min-1  cm H2O  % | <-20  >4  8-18  <105  >60  <6  <10  <0.50 |
| F = respiratory rate (min01); Vt = tidal volume (liters).  NIFM: Negative inspiratory force maximum. | | |

3. Assessment of Readiness for extubating:

=> Rapid, shallow breathing is an adaptation to compensate for inadequate muscular strength or endurance.

a. Adequate strength and endurance.

* Pressure support of 8 for 30 minutes without signs of fatigue.
* Muscular paralysis reversed.
* Sustained head lift (>5 sec).
* Tidal volume of 35 ml/kg indicates adequate strength to sustain ventilation.

b. Adequate oxygenation.

* PO2 > 60-80 mmHg with FiO2 <0.50.

c. Patient alertness.

* Responsive to commands.
* Must be able to lift head (strap muscles of the neck are the last muscle group to recover from paralysis).
* Bilateral hand grip.
* Ability to cough (intact gag reflex).
* Quantity and quality of secretions can be controlled by patient.

4. Extubation Procedure:

a. Preparation.

1) Review respiratory mechanics, last ABG, and physical examination to confirm that Extubation is appropriate.

2) Explain process to patient to minimize anxiety.

3) Be sure stomach is decompressed by a.m. CXR (use O-G if necessary).

b. Removal of endotracheal tube.

1) All equipment for re-intubation must be immediately available.

2) Perform endotracheal suctioning and oral suctioning. Deflate balloon.

3) If patient can ventilate around ET tube, laryngeal edema is insignificant.

4) Give a large hand ventilated breath and remove ET tube on exhalation.

5) Encourage patient to cough.

6) Apply supplemental O2 via nasal cannula: 0.05 to 0.10 above FiO2 on the ventilator.

7) The majority of cardiac patients should be weaned to 40% FiO2 on the ventilator and extubated to 4 Liters nasal cannula O2. Nasal cannula is more comfortable, less claustrophobic, allows coughing and expectoration, and facilitates communication with the patient.

8) Monitor mental status, respiratory rate, pulse oximetry, and hemodynamics for signs of decompensation

**III. DYSRHYTHMIAS**

**A. Tachydysrhythmias**

1. Evaluation.

a. Laboratory data: hematocrit, electrolytes (K+, Mg++), ABG.

b. Chest radiograph: exclude pneumothorax, consolidation, significant atelectasis.

c. 12 lead EKG with rhythm strip including leads II, V1.

d. Review history (in the chart and the monitor)

e. Initial assessment identifies 1 of 4 types (A fib/flutter, narrow-complex tachycardia, stable wide complex tachycardia: unknown type, and stable monomorphic and/or polymorphic.)

1. Most of the narrow complex tachycardia is atrial fibrillation.

* If the diagnosis is not secure after the above evaluation, consider atrial cardiogram. Record a rhythm strip and/or atrial electrocardiogram during administration.

3. Prevention.

a. Prompt treatment of hypokalemia and hypomagnesemia.

b. Maintain arterial 02 saturation >90%.

c. Prophylactic beta blockade or Amiodarone on POD 1.

d. Avoid high catecholamine states.

4. Treatment of atrial fibrillation.

Goals: first to control ventricular response and then conversion to sinus rhythm if possible.

Select treatment based on current ventricular response.

a. Ventricular response <100.

1) No control of ventricular response needed.

2) consider conversion to sinus rhythm with amiodarone or PO beta blocker.

b. Ventricular response 100-150.

1) If not hemodynamically stable then DC cardioversion.

2) If stable and EF >40%, treat first with IV metoprolol 5mg IVP x3 dose. If no conversion, then move to IV amiodarone. However, if hypotension a concern, start treatment with IV amiodarone (see section below).

If stable and EF <40%, treat with Amiodarone 150mg IV bolus. Avoid BB (see section below).

3) If no central line considers diltiazem IV.

4) Be aware of patient’s ventricular function (EF) prior to using IV Betablockers.

5) Be aware that nurses on 5 NE and 5 West are only able to push IV metoprolol with provider bedside. If no provider at bedside, order IVPB.

c. Ventricular response >150.

1) If not hemodynamically stable then DC cardioversion.

2) Call attending and go through above progression

3) Consider central line access.

**B. For Stable Patient (no serious signs or symptoms).**

1. Atrial fibrillation (not with Wolff-Parkinson-White (WPW)).

a. Heart function preserved (EF>40%) use 1 of the following

1) Metoprolol 5 mg IV push (may be repeated q 5 to 15 minutes for a total of 15 mg.

2) Amiodarone bolus (150mg) followed by 6-hour infusion of 360mg, can re-bolus x 2 if need be but check with attending first.

3) Diltiazem 10 mg IV push (may be repeated q 5 to 15 minutes for a total of 30 mg. May need qHp).

b. Poor EF (<40%) (nearly all our patients). Avoid BB.

1) Amiodarone bolus (150mg) followed by 6-hour infusion of 360mg, can re-bolus x 2 if need be but check with attending first

2) Digoxin 0.5 mg IV, followed by .25 mg IV q 2 hrs. x 2 doses (total loading dose 1 mg; expect 20 to 40 minutes between first dose and substantial ventricular response).

3) May need to add a third agent, (metoprolol or diltiazem) but check with attending first.

2. Atrial fibrillation with WPW - check with attending. Note: Adenosine, blockers, calcium channel blockers, and digoxin may all be harmful in these patients and therefore become acutely unstable.

3. Atrial Flutter.

a. Diagnosis: regular saw-toothed atrial pattern on 12 lead or atrial EKG with a regular ventricular response to every 2nd, 3rd, or 4th beat. Typically presents with ventricular rate of 150 bpm.

b. Treatment Goal: terminate rhythm, control ventricular response.

c. ASSESSMENT AND TREATMENT IS IDENTICAL TO ATRIAL FIBRILLATION. ONCE MEDICATIONS ARE BEGUN, then Rapid Atrial Pacing can be accomplished. RAP benefits hemodynamics by converting to SR or A fib, either of which have more easily controlled ventricular response rate. The attending must be made aware before RAP is initiated.

d. Two techniques are used. Be sure that the rapid atrial pacemaker is connected to the ATRIAL wires!!! (FIRST DETERMINE ATRIAL RATE BY THE ATRIAL ELECTROCARDIOGRAM.)

1) Ramp technique: Begin atrial pacing (20 ma current) at 10 BPM > the flutter rate, then gradually increase the rate. When the atrial complex in lead II becomes positive, either abruptly discontinue pacing or gradually slow pacing to the desired rate.

2) Constant rate technique: atrial pace (20 ma current) at 10 BPM faster than the flutter rate for 30 seconds, then abruptly stop pacing. Usually not effective.

4. Narrow-complex tachycardias. Avoid vagal maneuvers in patients with known carotid disease or in whom a formal carotid duplex was not performed preoperatively. Determine junctional paroxysmal supraventricular, or ectopic/multifocal atrial tachycardia.

a. Junctional tachycardia.

1) EF >40%.

I) No DC cardioversion.

ii) Amiodarone.

iii) beta blocker.

iv) Calcium channel blocker.

2) EF < 40%, CHF.

I) No DC cardioversion.

ii) Amiodarone.

b. PSVT.

1) EF>40%.

a) Priority: I. diltiazem

ii. metoprolol

iii. digoxin

iv. DC cardioversion

v. Discuss with electrophysiology fellow

2) EF <40%.

a) Priority: I. No DC cardioversion

ii. digoxin

iii. Amiodarone

iv. Diltiazem

c. Ectopic or multifocal atrial tachycardia.

1) EF>40%.

a) Priority: I. No DC cardioversion

ii. Diltiazem

iii. Metoprolol

iv. Amiodarone

2) EF<40%.

a) Priority: 1. No DC cardioversion

2. Amiodarone

3. Diltiazem

**C. Ventricular Dysrhythmias**

**1. Diagnosis and significance:**

a. If PVCs are frequent (>6 per minute), multifocal, or occur in runs (>3 PVCs), TREAT.

b. Premature ventricular contractions (PVC) originate distal to the bundle of His and are wide (>0.12 sec), abnormal QRS complexes which are not preceded by a P wave.

c. Findings which favor VT over SVT with aberrancy include: QRS duration >0.14 seconds, AV dissociation, fusion beats, left axis deviation, right bundle branch block with mono or biphasic QRS in lead V1.

2. Evaluation: obtain same laboratory work and test data as for atrial fibrillation.

Correct precipitating factors: consider a competing temporary pacemaker, myocardial ischemia, or PA catheter.

a. Unifocal PVC's raise the possibility of endocardial stimulation by a misplaced PA catheter tip. Treatment is relocation of the catheter; lidocaine is ineffective in suppressing mechanically induced PVC's. If PA catheter induced PVCs are excluded, then treat:

1) For preserved EF: amiodarone, lidocaine, sotalol, procainamide (check with attending).

2) For poor EF: amiodarone or lidocaine (check with attending).

b. Multifocal (polymorphic VT) PVCs are more likely due to global metabolic insult, and should prompt a thorough search for electrolyte imbalances, hypoxia, or myocardial ischemia. For normal baseline QT interval, treat ischemia and correct electrolytes. Medications such as metoprolol, lidocaine, amiodarone, procainamide, or sotalol may also be indicated, check with attending. For prolonged baseline QT interval (suggests torsade’s) give 2 gm magnesium and correct any remaining electrolyte abnormalities. Additional magnesium, overdrive pacing, amiodarone or lidocaine may be indicated; attending should be aware. Seek to discontinue arrhythmogenic medications (epi, dop, milrinone) if pressure tolerates.

IV. PACEMAKER USE

A. Indications Emergency Cardiac Pacing:

|  |
| --- |
| INDICATIONS FOR EMERGENT PACING |
| Hemodynamically unstable bradyarrhythmia’s (systolic BP<80 mm Hg) with HR<60.  Bradycardia with malignant escape rhythms.  Mobitz type II or complete heart block in the setting of acute MI.  Overdrive pacing of refractory unstable tachyarrhythmias. |

B. Indications For Cardiac Pacing:

|  |  |
| --- | --- |
| TEMPORARY | PERMANENT |
| Symptomatic bradycardia after acute MI  Bi or trifasicular block after acute anterior MI  Bridge to permanent pacing  Following cardiac surgery  Low cardiac output syndrome | Sick sinus syndrome  Mobitz II AV block  3rd degree heart block  Low cardiac output syndrome  Bifasicular block after acute MI  Symptomatic bilateral bundle branch block |

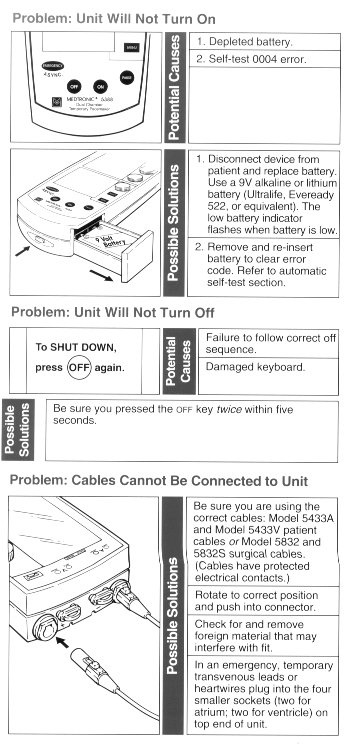
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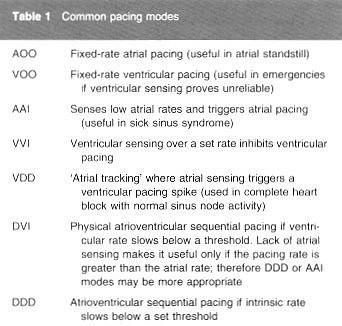
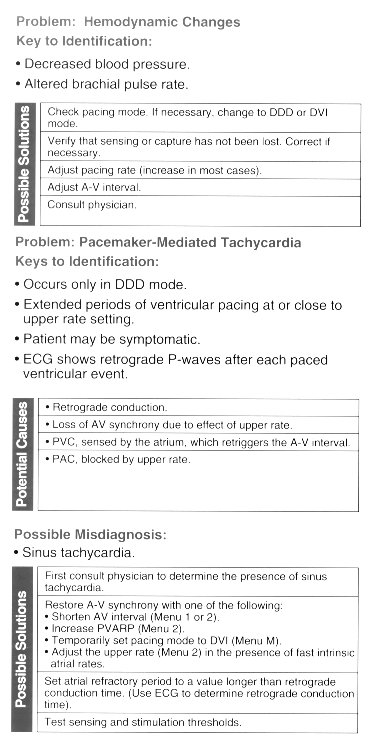
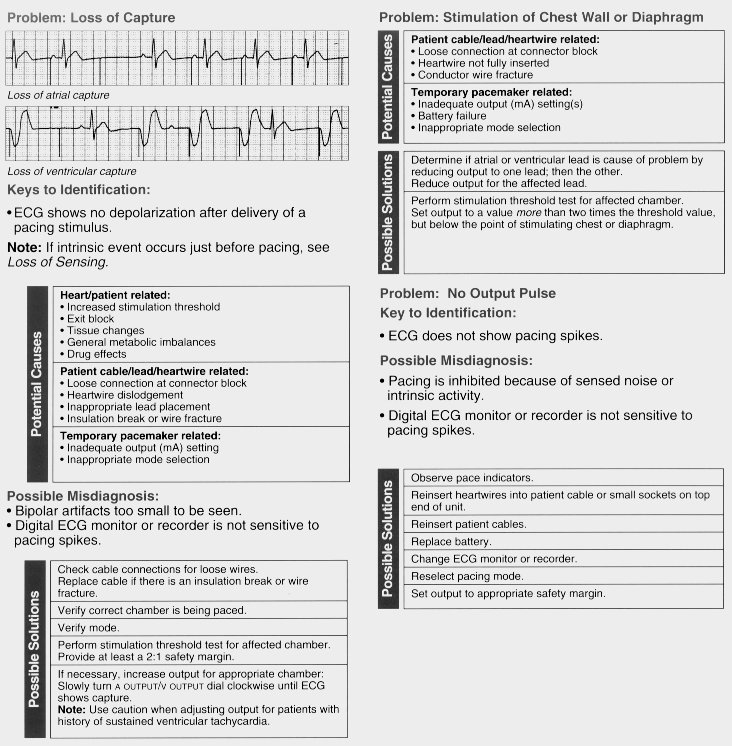
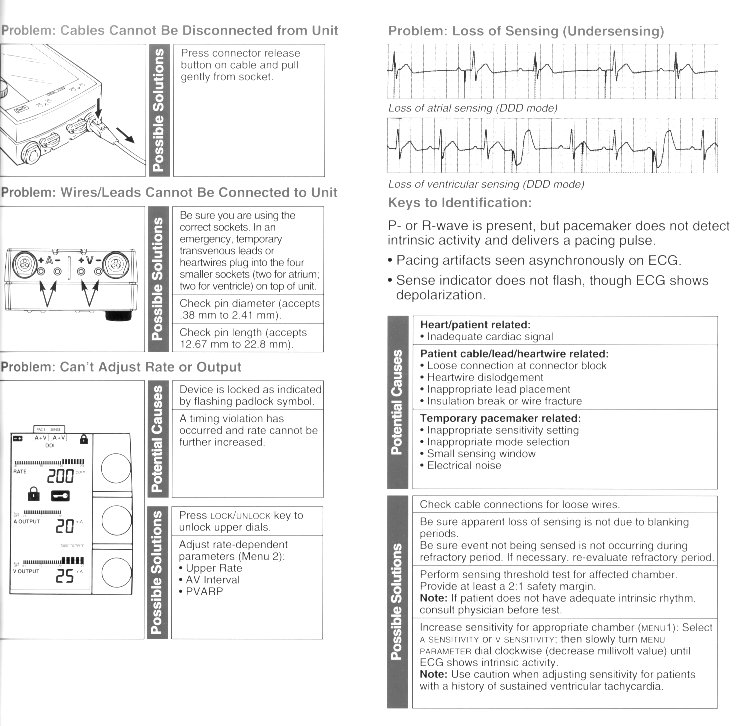
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| --- | --- | --- |
| Chamber Paced | Chamber Sensed | Mode of Response Once the Signal is Sensed |
| A=Atrium | A=Atrium  V=Ventricle | I=Inhibit pacing if an electrical signal is sensed |
| V=Ventricle | D=Dual (both chambers) | T=Trigger an electrical response if no electrical signal is sensed. |
| D=Dual (both chambers) | 0=No chamber is sensed: asynchronous mode | D=Dual response where the atrium is triggered, and the ventricle inhibited. |

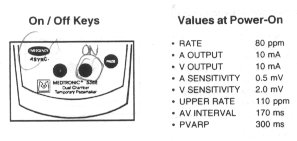
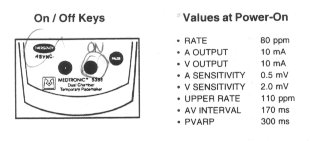
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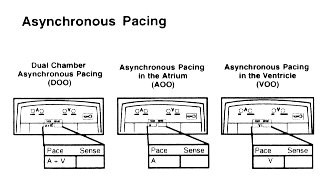
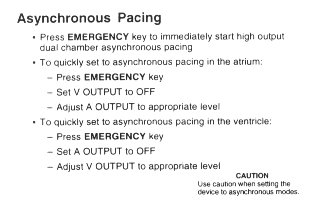
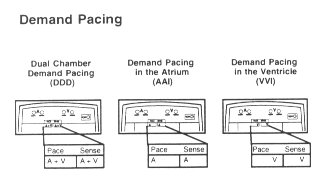
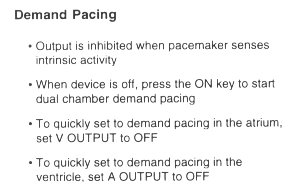
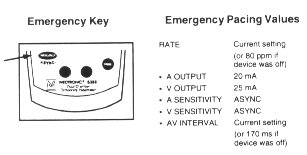


Please Note: **Currently using the upgraded pacemaker monitor, however still similar concept and information.**









The temporary pacemakers available on this service are shown.

1. Atrial output: milliamperes of power delivered to pace the atrium. 0.1 is equivalent to OFF.

2. Vent output: milliamperes of current delivered to pace the ventricle. 0.1 is equivalent to OFF.

3. AV interval: mSec of time between Atrial spike and Ventricular spike. Set at 150-165 mSec

4. Vent sensitivity: sets threshold at which a signal on the ventricular wires is interpreted as a QRS complex. Set this to 1.0 mV. NEVER, NEVER set to asynchronous when pacing the ventricle.

5. Vent rate: rate at which the pacer will fire atrial and ventricular output.

6. The threshold is the minimum current needed to capture a specific chamber. Obtain this value by slowly decreasing the mA until capture is lost. Repeat for each chamber paced. Do not perform this function on pacer dependent patients without an Intensivist or surgical Attending present.

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| PACING MODES AND SETTINGS FOR THE  MEDTRONIC 5330 PACEMAKER  USED ON THE THORACIC SERVICE | | | | | |
| Atrial Output | Vent Output | AV Interval | Vent Sensitivity | Vent Rate | Mode |
| 150% of THC | 0 | N/A | N/A | 80--105 | A00: A-paced |
| 150% of THC | 150% of THC | 165 mSec | 1.0 mV | 80--105 | DVI: AV sequential |
| 0 | 150% of THC | N/A | 1.0 mV | 60 | VVI: V backup |
| 0 | 150% of THC | N/A | 1.0 mV | 80--105 | VVI: V-paced |

THC threshold current

V. TREATMENT OF BLEEDING

A. Hemorrhage following cardiopulmonary bypass procedures must be aggressively treated.

B. Causes.

1. Coagulopathy.

2. Intraoperative technical error. Hemorrhage should not be ascribed to mechanical causes until any underlying coagulopathy is corrected.

C. Evaluation of bleeding.

1. Always consider whether the patient may be better served by a return to the operating room. Always strive to have the patient warm and replete with platelets and coagulation factors prior to return to the O.R.

2. Guide decisions to return to the operating room by the 1000 RULE:

a. Bleeding of 1000 ml in 1 hour.

b. Bleeding of 500 ml/hr. for 2 hours.

c. Bleeding of 300 ml/hr. for 3 hours.

d. Bleeding of 250 ml/hr. for 4 hours.

3. Any patient who is hemodynamically unstable because of bleeding, whether the volume of blood loss is in excess of the capacity to transfuse or tamponade is imminent, needs to go to the operating room immediately. The pt. should be re-explored in the O.R. if possible.

4. Consider timing. It is safer for the patient to go back to the OR earlier in the evening then at midnight. Don’t wait until the patient is unstable to make the decision if you can help it. Contact the surgeon as soon as you have concern.

D. Treatment of Bleeding. [You need bricks (PRBC) and mortar (clotting factors) to form clot.]

1. What is the last hematocrit? What is total blood loss? Over the last hour? Over the last 15 min? Is a blood transfusion needed NOW? In the bleeding patient, aggressively maintain a hematocrit of 30% (bricks). Remember Hb/Hct isn’t a sensitive marker for acute bleeding.

2. What is the state of the coagulation system?

a. Patient's body temperature: start bear hugger if T <36.0°C.

b. Coagulation factors: evaluate prothrombin time (PT) and fibrinogen level. Correct elevated PT (INR >1.5) with FFP (fresh frozen plasma). Fibrinogen level <180 mmolL1 is treated with cryoprecipitate. Give factors only if requested/approved by surgeon.

c. Heparin level: obtain PTT. Treat abnormal PTT with 25 or 50 mg of protamine. The risks of protamine induced anticoagulation are minuscule compared to the benefit of reversing heparin. Consider heparin rebound in:

1) obese patients and

2) patients on preoperative heparin infusions.

d. Platelet level: none of the platelets after CPB are functional, regardless of the platelet count. In the bleeding cardiac patient, transfuse platelets as quickly as possible. A platelet count of <80,000/mm3 in a bleeding patient is an absolute indication for platelet transfusion.

e. **Remember to check and replace calcium**. Blood products contain citrate, a calcium chelator to prevent clot formation while in storage.

3. Chemical adjuncts to control of bleeding.

a. Amicar: epsilon aminocaproic acid is begun in the operating room to inhibit fibrinolysis.

b. DDAVP (desmopressin): This compound causes release of von Willebrand factor from platelets.

1) Randomized studies demonstrate slightly reduced blood loss and transfusion requirements in surgical patients.

4. Mechanical adjuncts to control bleeding.

a. PEEP: not proven to be of benefit in randomized trials, an individual patient may benefit from an increase of PEEP to as high as 10 cmH20. Monitor cardiac output and filling pressures carefully, however. See PEEP in mechanical ventilation section, above.

b. Elevation of the head of bed 45% may be helpful in decreasing venous pressure in the thorax and controlling persistent venous oozing.

CARE AFTER SPECIFIC CARDIAC PROCEDURES

I. MYOCARDIAL REVASCULARIZATION

A. Coronary Artery Bypass Grafting.

1. ECG: the ECG must be carefully monitored postoperatively. Any significant changes noted on the monitor must be documented by a formal 12 lead ECG. ST segments elevation or depression >1.0 mV must be evaluated by 12 lead ECG. ST depression may represent graft dysfunction (spasm), coronary air embolism, coronary dissection, or atheroembolism. Persistent ST depression, particularly if associated with hemodynamic compromise, warrants consideration of cardiac catheterization. ST elevation signals evolving infarction and warrants immediate diagnostic maneuvers.

a. Graft Occlusion: sudden graft occlusion within 24 hours of surgery is rare. Signs of graft occlusion include sudden hypotension, abrupt loss of contractility not explainable by other mechanisms, and typical EKG changes (ST segment elevation). Therapy consists of immediate reoperation to replace the occluded graft.

b. Arterial graft spasm: this results in ischemia and ventricular dysfunction. Consider nitroglycerine. Early treatment results in resolution of anterior ischemia and hemodynamic instability. Delayed treatment or spasm refractory to nifedipine, NTG, or SNP with progressive EKG changes or continued hemodynamic instability is treated by reoperation and addition of a vein graft to the LAD. Right sided infarcts may require increased filling pressures in order to push blood right to left.

2. Beta adrenergic antagonists are started in the morning of POD 1. Beta blockers reduce the incidence of postoperative atrial fibrillation from 30% to 50%. (This is especially true if started early postop day 1.)

3. Aspirin is administered to all patients after CABG. POD 0 6 hours after the operation the patient receives the first dose. POD 1 starts daily ASA. ASA has important beneficial effects on reducing anastomotic fibroplasia. Consider Plavix if off-pump bypass or if a stent is present.

II. VALVULAR SURGERY

A. General comments for valve patients.

1. Anticoagulation: Anticoagulation is usually delayed 48 hours postoperatively to ensure hemostasis and until chest tube output has decreased. Mitral valve repairs and bioprosthetic valves may on occasion be treated with 3-4 months of warfarin.

|  |  |  |  |
| --- | --- | --- | --- |
| Valve or Condition | Aspirin | Target  PT-INR | Heparin Prior to  Adequate PT-INR |
| Atrial fibrillation or atrial thrombus | CAD: 81 mg  No CAD: +/- | 3.0-3.5 | YES |
| St. Jude Aortic | 81 mg | 2.0-2.3 | NO |
| St. Jude Mitral | 81 mg | 2.5-3.5 | YES - if A-fib or thrombus |
| Bio prosthesis or Homograft | 325 mg | ------- | YES - if A-fib or thrombus |
| Mitral Valve Repair | 325 mg | ------- | YES - if A-fib or thrombus |

1. Pacing wires may be taken out on POD #3 unless otherwise directed by attending surgeon.
   * 1. The wires are to be cut if platelets are less than <100 or INR >1.5.

4. Arrhythmias: MVR or AVR may result in injury to the cardiac conducting system and result in AV conduction delays or complete AV dissociation (complete heart block [CHB]). The injury may be apparent immediately postoperatively or appear 24 to 48 hours later due to progressive inflammation and/or myocardial edema. Do not treat these patients with prophylactic digoxin.

5 Elevated systemic blood pressures: close vigilance is required to maintain mean blood pressure near 70-80 mmHg, since even transient hypertensive episodes may disrupt an aortotomy incision or dislodge a newly placed prosthetic aortic or mitral valve.

6. All patients undergoing valve procedures are at higher risk for neurologic complications. A careful preoperative neurologic examination (documented in chart) is followed by careful postoperative examinations.

B. Aortic Stenosis (AS).

1. Pathophysiologic principles.

a. thickening, calcification, or fusion of aortic leaflets leading to LV outflow obstruction

b. young adults= bicuspid valve, elderly = degeneration changes

c. Inability of cusps to open causes pressure overload, LVH, and decreased LV compliance

d. Concentric left ventricular hypertrophy (LVH) results in a thickened LV wall, small LV cavity, and stiff, noncompliant ventricular chamber. An elevated LVEDP is required to achieve adequate LVEDV.

e. Atrioventricular synchrony is important. Atrial contraction is critical in achieving adequate LV EDV.

f. small changes in coronary perfusion may result in ischemia (causes include A-Fib, hypotension, ventricular dysrhythmias).

g. Treat tachycardia, even when of sinus origin.

h. Remember, fixed CO secondary to valve stenosis, therefore syncope or sudden death if peripheral vasodilatation occurs.

I. normal Aortic valve area (AVA) 2.5-3.5cm2, AVA=>1.5 is mild AS, 1-1.5 is moderate AS, severe AS is AVA=<1.0, critical AS is AVA<0.5

2. Post-operative management.

a. The pathologic loading of these ventricles has been removed. They are expected to do well.

b. Maintain AV synchrony (30% of stroke volume)

c. Low threshold for cardioversion if A fib occurs secondary to risk of profound hemodynamic deterioration.

d. Keep heart rate 60-85. Allow slightly increased MAP (75-80) to ensure adequate myocardial perfusion.

e. These patients may become hyperdynamic postoperatively secondary to excellent systolic function. Do not treat a hyperdynamic state by withholding preload. **THE VENTRICLE IS PRELOAD DEPENDENT!**

f. Hypertension usually occurs hours after termination of bypass and must be controlled to decrease overall myocardial oxygen demand and aortic suture line.

g. ACE inhibitors will help to reduce LVH postoperatively.

h. Heart block that persists for more than 1 week is likely to be permanent and require a pacemaker. Make sure EP is on board early.

I. Inadequate temporary pacing wires (i.e., thresholds greater that 15 mA) must be treated by transfer to the ICU, positioning of a temporary pacing wire, and expedient placement of a permanent pacemaker.

C. Aortic Insufficiency (AI).

1. Pathophysiologic principles.

a. Acute AI secondary to Aortic Dissection/endocarditis causes acute LV failure/pulmonary edema due to inability of LV to acutely dilate to handle the volume overload

a. Ventricle is dilated and hypertrophied (eccentric hypertrophy) as a result of volume overload.

b. Allow higher heart rate when uncorrected Al is present. Diastole and thus regurgitant time are decreased.

c. These ventricles will also require high levels of preload but are much less sensitive to small changes in volume than in concentric LVH (aortic stenosis).

d. PAD will exhibit much smaller changes in response to volume challenge because of large compliant ventricle.

e. Increased afterload and impaired contraction will eventually lead to fall in EF (chronic AI pts)

2. Indications for Surgery

a. Acute AI with CHF

b. Endocarditis with compromise, sepsis/persistent bacteremia, abscess formation, conduction abnormalities.

3. Postoperative management.

a. Dilated ventricles benefit from low to normal afterload to reduce ventricular wall tension.

b. AI patients usually vasodilated after surgery and require alpha adrenergic agent for hypotension (phenylephrine or norepinephrine)

c. Preoperatively these patients are total body volume overloaded and will require postoperative diuresis beginning POD 1.

c. ACE inhibitors are beneficial.

d. See comments above concerning heart block.

D. Mitral Stenosis (MS).

1. Pathophysiologic principles. (Normal MV area is 4-6cm2)

a. Almost always a cause secondary to rheumatic fever

b. Causes thickening and fusion of the valve leaflets along with shortening of the chordae

a. These ventricles have been exposed to limited preload as a result of stenosis of the mitral valve. Although functionally ‘untrained’, LV contractility and stiffness are normal.

b. The left atrium is markedly enlarged, and pulmonary pressures are elevated. Note whether thrombus is present in the left atrium preoperatively.

c. These patients are diuretic dependent preoperatively.

d. Most valves require replacement rather than repair.

e. Untreated can cause the development of pulmonary hypertension leading to right sided heart failure

2. Postoperative management.

a. Inotropic support for 1-2 days is usually required (RV rather than LV dysfunction).

b. Aggressive diuresis is required to treat total body water excess. Expect to discharge patients on their preoperative regimen of diuretics at a minimum.

c. Consider overnight intubation and mechanical ventilation if pulmonary edema and severe LV dysfunction are present postoperatively. These patients may not have sufficient cardiac reserve to tolerate earlier Extubation.

d. Patients in chronic atrial fibrillation for more than 3-6 months preoperatively are unlikely to remain in sinus rhythm postoperatively. If patients return from the operating room in sinus rhythm, amiodarone infusion may help maintain sinus rhythm for at least the first 24-48 hours postoperatively.

e. Extremes of afterload are to be avoided.

E. Mitral Regurgitation (MR).

1. Pathophysiologic principles.

a. Causes: abnormalities from the annulus (dilatation) valve leaflets (myxomatous changes with

prolapse) destruction (endocarditis) chordate tendineae (rupture/elongation) or papillary muscle

(Rupture, ischemic causes)

b. Acute MR usually results from MI with papillary muscle rupture causing LV volume overload

c. LV volume overload secondary to reduction of forward output and flow reversal into small

noncompliant left atrium causing cardiogenic shock and acute pulmonary edema

d. Chronic MR allows for progressive increase in compliance of LV and LA followed by progressive

dilatation of LV. The increase in preload causes an overall increase in overall stroke volume

therefore, maintaining cardiac output.

e. Ventricular function may be impaired with an overall EF secondary to the decrease in afterload

due to the ventricle unloading into the left atrium.

d. Ventricular function is worsened in the immediate postoperative period by valve repair/replacement. The ventricle remains dilated but no longer can eject retrograde into the low-pressure pulmonary veins.

e. Left atrial enlargement and preexisting chronic atrial fibrillation with or without atrial thrombus are present as in mitral stenosis.

2. Indications for Surgery

a. Acute MR with CHF or cardiogenic shock

b. Endocarditis with compromise, sepsis/persistent bacteremia, abscess formation, conduction abnormalities.

c. NYHA class II-IV with severe (3-4 MR) independent of EF (increased risk with decreased EF)

3. Postoperative management.

a. Ventricular failure: inotropic agents are often required to support ventricular function for 1-3 days postoperatively. Diligent control of afterload is critical in avoiding atrioventricular dehiscence, maintaining adequate cardiac output, and avoiding myocardial ischemia from ventricular dilatation. IABP may be required to further unload the ventricle in patients with limited LV performance

b. Pulmonary hypertension may not resolve immediately after valve repair/replacement. Milrinone, dobutamine, nitric oxide or inhaled epoprostenol may be required in the immediate postoperative period.

c. Ventricular enlargement predisposes to ventricular dysrhythmias, particularly if LV filling pressures become excessive. Aggressive attention to electrolyte replacement and treatment of dysrhythmias is critical. These patients rarely recover from cardiac arrest.

d. Preexisting atrial thrombus is treated with early anticoagulation with heparin until Coumadin adequately prolongs the PTINR.

e. Preoperative diuretics are increased or continued in the postoperative period.

F. RV dysfunction:

Not uncommon after MS or MR especially with pts with preexisting pulmonary hypertension.

1. Pathophysiologic principles.

a. Causes: poor myocardial protection,

b. Factors increasing RV afterload

1. positive pressure ventilation

2. increased extravascular lung water

3. blood components

4. blood gas and acid base abnormalities

5. systemic inflammatory response to surgery and bypass

6. reversible vascular spasm

2. Isolated RV dysfunction seen with high CVP, variable PA pressures, hypovolemic LV and low CO

***\*\*remember functional TR will render thermodilution cardiac outputs unreliable\*\****

3. Management:

a. fluid administration to optimize preload (stop if CVP >/= 20mmHg without achieving decent CO

secondary to risk of RV dilation on RV myocardial oxygen flow and LV function.

***\*\*remember, when RV dilates it shifts the interventricular septum towards the LV impairing LV***

***diastole and dysfunction, this dysfunction reduces systemic perfusion pressure, causing RV***

***ischemia, elevation of PA pressure and RV afterload\*\****

b. Inotropic drugs to support both RV and LV (milrinone, dobutamine)

c. Pulmonary vasodilators to improve RV function and decreasing RV afterload

a. Nitric Oxide

b. inhaled epoprostenol

PREVENTION OF BACTERIAL ENDOCARDITIS

This prophylactic treatment should include all patients with prosthetic cardiac valves or who have undergone valve repair procedures.

1. **Indications for subacute bacterial endocarditis prophylaxis (AHA 2007 current guidelines)**

1. Prosthetic cardiac valve or prosthetic material used in valve repair
2. Previous endocarditis
3. Congenital heart disease
   * + 1. Unrepaired cyanotic congenital heart disease, including palliative shunts and conduits
       2. Completely repaired congenital heart defect with prosthetic material or device during 1st 6 months after procedure
       3. Repaired congenital heart defect with residual defects at the site or adjacent to the site of a prosthetic patch or device
4. Cardiac transplantation recipients with cardiac valvular disease

**Antimicrobial Prophylaxis for the Prevention of Bacterial Endocarditis**

|  |  |  |
| --- | --- | --- |
| **Situation** | **Drug** | **Dosage Regimen- all single dose only, within 30 to 60 minutes prior to procedure (children: not to exceed adult dosing)** |
| Oral therapy | Amoxicillin | 2gm PO (children 50mg/kg) |
| Unable to take oral medications | Ampicillin | 2gm IM or IV (children 50mg/kg IM or IV) |
| **OR** cefazolin | 1 gm IM or IV (children 50mg/kg IM or IV) |
| Allergic to penicillin or ampicillin- oral dosing | cephalexin2 OR | 2gm PO (children 50mg/kg) |
| clindamycin OR | 600mg PO (children 20mg/kg) |
| azithromycin or clarithromycin | 500mg PO (children 15mg/kg) |
| Allergic to penicillin or ampicillin and unable to take oral medications | cefazolin2 OR | 1gm IM or IV |
| Clindamycin | 600mg IV |
| Surgery on infected soft tissues | cefazolin OR vancomycin, depending on antimicrobial susceptibility | see above for dosage |
| Cystoscopy when UTI eradication not possible | anti-enterococcal therapy (amoxicillin or vancomycin), depending on antimicrobial susceptibility, if available. If not available, consult with infectious diseases. | see above for dosage |
| Recent prior or concurrent antimicrobial therapy | consult with infectious diseases, see full text of AHA guidelines |  |

III. Aortic Dissections

General Information: Aortic dissection occurs when an intimal tear occurs that allow blood to pass into the media creating a false channel. The channel (hopefully) is contained by the outer medial and adventitial layers of the aorta. (If not, you now have an aortic rupture which is highly probable for immediate demise).

1. Stanford Type A dissection or DeBakey type I-II:

a. tear involves the ascending aorta (somewhere between the aortic root and left subclavian artery)

b. acute type A occurs within the last 2 weeks, chronic type A after 2-week mark.

c. emergent surgery is indicated for acute type A patients unless hemorrhagic or massive ischemic CVA

d. chronic type A dissections can be done on a more urgent/elective basis based on symptoms

d. depending on location of tear and extent of dissection complications can include:

- cardiac tamponade including hemopericardium (most common cause of death)

- aortic regurgitation

- myocardial infarction

- stroke

- compromised blood flow to mesenteric, femoral, iliac, renal arteries causing ischemia

e. TYPE A DISSECTION CALLS REQUIRE IMMEDIATE NOTIFICATION THE ON-CALL ATTENDING SURGEON. CALL THE SURGEON,THEN RAPIDLY EVALUATE THE PATIENT. UPDATE THE SURGEON WHILE EVALUATING THE PATIENT.

f. Always make sure the OR charge nurse is made aware and the cardiac team is called in as an emergency.

g. Call the On-call fellow and PA if the case is going. Consider transfer directly to the OR holding area.

h. Get as much of a history from the patient as possible.

I. Stat CT scan of the head if neurological status in question.

2. Stanford Type B dissection or DeBakey type III

a. tear occurs just distal to the left subclavian artery and does not involve any of the ascending or arch

b. usually treated medically with surgery reserved for complicated dissections and vascular compromise

c. type B dissections are to be followed by cardiac surgery and at type admitted to our service for evaluation.

IV. Intra-arterial Balloon Pump (IABP)

General Information: IABP counter pulsation helps with hemodynamic support of the ischemic or failing heart by decreasing myocardial oxygen demand and improving coronary perfusion.

1. Indications:

a. ongoing ischemia refractory to medical therapy or hemodynamic deterioration

b. prophylactic placement prior to surgery in pts with left main coronary artery disease or poor EF.

c. unloading of cardiogenic shock, MI, (acute MR or ventricular septal rupture secondary to MI)

d. post-operative myocardial ischemia

e. low cardiac output refractory to moderate to high doses of inotropic medications

f. inability to wean from cardiopulmonary bypass or temporary support as bridge to device/transplant

2. Absolute Contraindications

a. Aortic Insufficiency

b. Aortic Dissection

3. Pathophysiology

a. unloading of heart by rapid deflation just prior to ventricular systole (decreasing afterload)

b. increasing diastolic coronary perfusion pressure by rapid inflation just after aortic valve closure

4. Complications:

a. Balloon rupture: (look for blood in the balloon tubing which is indicative of a ruptured balloon)

\*\*Make attending aware and remove balloon immediately\*\*

b. Aortic dissection or rupture of the iliac artery or aorta: extremely rare occurrence

c. if tip of balloon above left subclavian can cause stroke

d. if tip of balloon below the level of the diaphragm can cause renal or mesenteric ischemia

5. Weaning of the IABP

a. immediately post-operative/post insertion the IABP should be to a 1:1 configuration.

b. once you start weaning down and off the inotropic medications, you can start weaning off the IABP

c. decrease the inflation ratio from 1:1 down to 1:2 for about 1-2 hours and then 1:3 for another 1-2 hours

6. Removal of IABP

1. Check coagulation (aPTT, INR) and platelets (>50) before removal to prevent hematoma/aneurysm
2. Baseline limb
3. Turn down the augmentation to 1:3
4. Put balloon on assist-standby mode: observe trace to make sure there is no augmentation
5. Cut off sutures
6. Turn off the balloon pump
7. Pull balloon out; wait 3 seconds to flush out clot (hold pressure distally to insertion area for 3 seconds, then proximally for 3 seconds to allow for proximal and distal flushing of the vessel.
8. Apply pressure for 45-60 minutes at site of arterial puncture followed by pressure dressing over the area.
9. Keep in bed for 6 hours to monitor for distal limb ischemia (temperature, capillary refill, pulses)
10. Monitor next 24/h for hematoma formation and distal limb ischemia.

CARE AFTER AORTIC SURGERY

I. CARDIOPULMONARY RESUSCITATION

A. Basics.

1. Call a "CODE" when respiratory or cardiac arrest has occurred. I.E., you need more people to help you now.

2. Have the attending surgeon notified. It is important to have attending surgeon present as soon as possible during a code they have a superior knowledge of postoperative complications, a superior ability to obtain central IV access, and superior ability to perform any invasive therapeutic procedure. Consider the patient for ECMO support. Consider emergent chest opening.

3. Assess the nature of the arrest and act quickly.

4. Ventricular fibrillation or pulseless ventricular tachycardia: SHOCK NOW.

B. Process circumstances of arrest while initiating treatment.

1. Airway/Breathing.

a. Begin ventilation with bag valve mask. Provide maximally available Fi02.

b. When you call an RRT (rapid response) anesthesia will arrive for intubation

c. Accomplish intubation if no spontaneous ventilation or ventilation is inadequate.

d. The most experienced provider does the intubation (if anesthesia not available, highly unlikely).

2. Circulation.

a. If no pulse is present, and the patient does not have a non-pulsatile flow, begin chest compressions. Defibrillation is absolute first therapy for V. Fib.

b. Place backboard under patient.

c. Compress the chest to left of sternum at 5-6th interspace at a rate of 100 compressions/min.

d. Assess rhythm and administer therapy. THINK:

1) Ventricular fibrillation, pulseless ventricular tachycardia.

* Defibrillation.
* Epinephrine.
* Amiodarone.
* Magnesium.
* Lidocaine.

2) Bradycardia.

* Use existing temporary pacing wires.
* Epinephrine.
* Transcutaneous pacing (Zoll pacer).
* Transvenous pacemaker.

e. Establish dependable IV access, preferably central circulation i.e., subclavian or IJ line.

f. Plan for transfer to intensive care unit.

g. Remember medications that are effective given via endotracheal tube.

* Atropine.
* Lidocaine.
* Isoproterenol.
* Epinephrine.
* Naloxone.

3. Examine patient, obtain lytes, renal, glucose, ABG, CBC, Mg, ionized calcium, PO4, ECG, portable CXR all CODE-STATUS.

4. Institute treatment for tamponade, hypoxia, pneumothorax, hemothorax, anemia as needed.

5. Give sedation when blood pressure will tolerate effect.

6. Consider etiologies of arrest.

a. Ventricular tachycardia.

b. Ventricular fibrillation.

d. Electromechanical dissociation (EMD).

e. Supraventricular tachycardia, may have degenerated into ventricular dysrhythmia.

f. Acute respiratory failure.

g. Hyperkalemia.

h. Severe acidosis.

I. Tension pneumothorax.

j. Cardiac tamponade.

k. Complete heart block or loss of pacer capture.

C. Monitoring.

1. Frequent physical examination for evaluation of blood pressure and heart rate as well as evidence of congestive heart failure or new murmurs.

2. ECG monitor.

a. 12 lead ECG.

b. Continuous ECG monitoring for identification of arrhythmias or heart block.

3. Central venous or pulmonary artery catheters: if hemodynamic instability is present, intravascular volume status is unknown, or centrally administered agents are required for support of circulation.

**Medication Protocols: Postoperative Pain Management**

**Surgical Cardiovascular ICU Pain Management Algorithm**

*If patient is receiving opioid analgesics pre-operatively,* ***recommend APMS consult***

***Pre-Op:*** pregabalin 150 mg PO x 1 dose

*To be given 1.5 hours prior to anesthesia induction*

***Monitoring: Reassess pain 30 minutes after IV pain medications***

***and 1 hour after PO pain medications***

**POD 0**:

*Continue dexmedetomidine until 1-hour post-Extubation*

* acetaminophen (APAP) 1000 mg NG/OG q6h
* pregabalin 75 mg PO q12h x 6 doses
* hydromorphone PRN OR PCA
  + - Moderate Pain (NRS 4-6): hydromorphone 0.25 mg IV q3h PRN
    - Severe Pain (NRS >6): hydromorphone 0.5 mg IV q3h PRN

**POD 1-5**

Bowel Regimen: docusate 100 mg PO BID

senna 2 tabs PO daily

polyethylene glycol 3350 17g PO daily

bisacodyl 10 mg pr prn no BM in 48 h

***If tolerating PO (d/c IV hydromorphone):***

Mild Pain Moderate Pain Severe Pain

NRS 1-3 NRS 4-6 NRS 7-10

APAP 1000 mg PO q6h x 72 h (standing) oxycodone 5 mg PO q4h PRN oxycodone 10 mg q4h PRN

*If pain not controlled with PO oxycodone, if pain not controlled with PO oxycodone,*

*consider 1x dose of hydromorphone 0.25 mg IV consider 1x dose of hydromorphone 0.5 mg IV*

***If not tolerating PO:***

Mild Pain Moderate Pain Severe Pain

NRS 1-3 NRS 4-6 NRS 7-10

Continue acetaminophen 1000 mg hydromorphone 0.25 mg IV q3h prn hydromorphone 0.5 mg IV q3h

NG q6h standing prn

Other PRN pain medication options:

|  |  |
| --- | --- |
| Medication | When to use |
| tramadol | * >65 years old and/or patient doesn’t tolerate oxycodone |
| hydromorphone PCA | * Severe pain medications (as listed above) are not adequate for pain control   + Pain score upon reassessment remains >6 after receiving pain medication |
| ketorolac | * Patient doesn’t tolerate oxycodone and/or pain treatment inadequate   *Check with surgeon prior to ordering* |
| oxycodone | * Increase to 10 mg for moderate pain, 15 mg for severe pain |

III. INITIATING/CONTINUING ANTICOAGULATION

A. Choices of anticoagulants.

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  |  |  | Monitoring | |
| Medication | Dominant Effect | Starting Dose | Test | Frequency |
| Aspirin | Irreversible platelet inhibition | Valves: 81 mg/day  CABG: 325 mg/day  Acute ischemia: 325 mg | None or Ivey  Bleeding time | Preoperatively if Bleeding tendency |
| Heparin | Combines with antithrombin III to inhibit serine protease coagulant factors | Bolus: 5000 units  Infusion: 800-1500 units/hour | aPTT:  Prolong to 2-3 times control (60-80 seconds) | 2 hours after bolus  6 hours after starting infusion  Then q 12-24 hours  Q day PLT count |
| Warfarin  (Coumadin) | Inhibits hepatic production of Vitamin K dependent factors: II, VII, IX, X, and proteins C and S | 5 mg po q HS | PT-INR  AVR: 2.0-2.2  MVR: 2.5-3.5  A-fib: 2.5-3.5  CHF: 2.5-3.5 | Q am until PT-INR prolonged then 1-2/week then 1-2/mon then q 1-2/mon for life |

B. Initiating anticoagulation.

1. Postoperative patients: patients who are less than 5 days out from an operation where cardiopulmonary bypass was used should not be bolused with heparin. If heparin is needed in these patients start an infusion at 1000 units/hr. and check a PTT in 68 hours.

2. Inhibiting thrombosis: patients with an ongoing thrombotic complication that requires immediate anticoagulation are treated with heparin. Large starting doses are occasionally required. Minimum starting dose is 5000 units, consider higher dosing (i.e., 1015,000 units) when DVT or PE are present. Check a PTT in 12 hours to insure adequate anticoagulation.

C. Continuing anticoagulation.

1. In patients who are on heparin while waiting for Coumadin to have an effect, heparin may be discontinued when the INR exceeds 2.0.

2. Confirm that the patients understands that the PT/INR must be checked either at HMC or by his local MD and that these arrangements have been made prior to discharge.

3. Points on Coumadin.

a. This medication interacts with a broad spectrum of other medications. Confirm these interactions whenever prescribing other medications to patients on Coumadin or prescribing Coumadin.

b. The effect of Coumadin on the coagulation system is closely linked to the amount of Vitamin K in the body and the status of the liver enzyme system. It is acceptable for patients to have 13 drinks/week and to eat leafy green vegetables. What is important is that the patient's diet remain constant from week to week, keeping the relative amounts of various foods in stable proportion. This is particularly important for ethanol.

D. Complications of anticoagulation.

1. Bleeding: excessive anticoagulation may result in a bleeding diathesis. Bleeding may occur from the GI tract, urinary tract, or in wounds. Treat by discontinuing heparin, consider giving protamine to reverse heparin. If the patient is anticoagulated with Coumadin, then treatment of bleeding entails Vitamin K administration and fresh frozen plasma to replete coagulation factors. Bleeding in the presence of a mechanical prosthetic valve must be aggressively investigated and the source eliminated, or the valve replaced with a bio prosthesis.

2. Clotting: "Patients die from clotting."

a. Patients initiated on Coumadin without a recent procedure on CPB should be anticoagulated until the PTINR becomes prolonged. Protein C and S are natural anticoagulants which are also affected by warfarin and have extremely short half-lives. These patients are at risk for a hypercoagulable state during the first 2472 hours of warfarin administration.

b. The platelet count must be followed daily in patients on heparin. Thrombocytopenia on heparin may represent HITT (heparin induced thrombocytopenia and thrombosis) with the devastating consequences of intravascular thrombosis or uncontrollable bleeding. If profound thrombocytopenia develops on heparin, then heparin must be discontinued and alternatives such as warfarin should be considered. Diagnose HITT by obtaining a heparin induced platelet aggregation test and a serotonin release assay. If this test is positive, the patient should receive no exposure to heparin, including heparin bonded circuits and catheters, low molecular weight heparin, or heparinoids. HITT antibodies circulate for three to six months.

**Nurse-driven parenteral anticoagulant management**

Following cardiac surgery, anticoagulation or excessive anticoagulation is associated with a higher incidence of large pericardial effusions or tamponade.1 For this reason, intensivists in a cardiothoracic ICU will target a low intensity heparin utilization when patients require anticoagulation following cardiac surgery (i.e., indications such as atrial fibrillation and prosthetic valve). Additionally, new ECMO circuits allow for lower intensity anticoagulation with heparin to balance the risk of clotting and bleeding events in patients on ECMO.

In the Surgical Cardiac Care Unit, there is not currently a standardized practice to initiating and modifying heparin or argatroban doses. This protocol will standardize the practice with the intention to provide better care and reach goal aPTT levels more quickly and efficiently. Nurse-driven protocols have been successful in the past. The most compelling example is the implementation of the Portland Insulin Infusion Protocol which improved SCIP adherence to the blood glucose endpoint from 87% to 97% in the first 9 months.

**Process:**

1. Prescriber (physician, physician’s assistant, nurse practitioner) will order unfractionated heparin infusion and dosing protocol (specifying which nomogram and PTT goal) with or without a bolus dose, and initial rate as per the protocol. Order set will include nomogram protocol, initial rate ± initial bolus.

2. Pharmacist reviews and verifies order.

3. Nurse reviews and verifies order.

4. Nurse verifies baseline PTT, platelet level, and weight (use actual body weight for calculations).

5. Nurse enters weight, initial rate into Baxter® pump, (if initial bolus of heparin is ordered, will obtain from pharmacy, as bolus dosing from Baxter® pump of anticoagulant agents are not permissible at this time).

6. Nurse enters initial rate ± initial bolus and time of initiation into MAR flowsheet.

7. Second nurse will verify and cosign initial rate ± initial bolus.

8. PTT will be drawn 6 hours after heparin infusion begins. Nurse will order PTT under P&T protocol, and sign the order “Protocol, User.”

9. PTT results are run STAT and will be available to the nurses electronically within 1 hour.

10. Nurse reviews PTT result and adjusts rate per protocol.

11. Nurse updates rate in MAR flowsheet and time of rate change.

12. Second nurse will cosign the adjusted rate.

13. After two consecutive aPTT within goal range, obtain next PTT with am labs and daily thereafter.

14. Prescribers will discontinue the anticoagulation orders, when necessary, i.e., no longer requiring anticoagulation or if necessary to be held for a procedure.

**Nomograms:**

***Surgical CVICU:***

**Heparin: Low Intensity (indicated for most patient’s s/p CT surgery or ECMO)**

*Initial dose:* 10 units/kg/hr. for ECMO and VAD; 12 units/kg/hr. for post-op A Fib, or for bridging for valve replacement**. No bolus recommended.** Max starting dose: 1000 units/hr.

Note: normal PTT is 28-38 seconds.

*\*\*round to the nearest 50 units when calculating dose and dose modifications*

|  |  |
| --- | --- |
| *Dose Modification:* PTT result (sec) | ACTION |
| ≤35 | ↑ by 2 units/kg/hr. |
| 36-44 | ↑ by 1 unit/kg/hr. |
| **45-55** | **At goal; no change** |
| 56-64 | ↓ by 1 unit/kg/hr. |
| 65-74 | ↓ by 2 units/kg/hr. |
| 75-84 | Hold x 1 hour and ↓ by 3 units/kg/hr. |
| >85 | Hold x 1 hour and ↓ by 4 units/kg/hr. \*\*notify prescriber\*\* |

|  |  |
| --- | --- |
| PTT result (sec) | ACTION |
| ≤40 | ↑ by 2 units/kg/hr. |
| 41-49 | ↑ by 1 unit/kg/hr. |
| **50-65** | **At goal; no change** |
| 66-74 | ↓ by 1 unit/kg/hr. |
| 75-84 | ↓ by 2 units/kg/hr. |
| 85-94 | ↓ by 3 units/kg/hr. |
| ≥ 95 | Hold x 1 hour and ↓ by 4 units/kg/hr. \*\*notify prescriber\*\* |

|  |  |
| --- | --- |
| PTT result (sec) | ACTION |
| ≤40 | ↑ by 3 units/kg/hr. |
| 41-50 | ↑ by 2 units/kg/hr. |
| 51-59 | ↑ by 1 unit/kg/hr. |
| **60-75** | **At goal; no change** |
| 76-84 | ↓ by 1 unit/kg/hr. |
| 85-94 | ↓ by 2 units/kg/hr. |
| ≥ 20 sec higher | ↓ by 3 units/kg/hr. |
| ≥ 30 sec higher | Hold x 1 hour and ↓ by 4 units/kg/hr. \*\*notify prescriber\*\* |

**PRN Electrolyte Riders Updated 2016**

For Post-Cardiac Surgery Order Set

Potassium Chloride

•40 meq (20 meq/100 ml x 2 doses) prn if serum K+ 3.5-3.9 mmol/L

IF CRCL<30ML/MIN,CHECK W/ MD BEF ADMINIS

•80 meq (20 meq/100 ml x 4 doses) prn if serum K+ <3.5 mmol/L

IF CRCL<30ML/MIN,CHECK W/ MD BEF ADMINIS

obtain chem 7 order for 2hr infusions

Magnesium Sulfate

•2 gm IV over 2 hours if serum Mg 1.8-2 meq/L

•4 gm (2x 2gm) IV over 4 hours if serum Mg <1.8 meq/L

|  |  |
| --- | --- |
| Default Cerner goal  **Adjust hepain based on aptt: 84-111**  aPTT 76 sec or less: Rebolus with 80 units/kg and increased dose by 4 units/kg/hr  aPTT 77-83 sec: Rebolus with 40 untits/kg and increase dose by 2 units/kg/hr  aPTT 84-111 sec: Continue current dose  aPTT 112-118 sec: Decrease dose by 1 units/kg/hr  aPTT 119-125 sec: Decrease dose by 2 units/kg/hr  aPTT 126 sec or greater: Hold herparin for 1 hr and decrease dose by 3 units/kg/hr  Only use 1,000 unit/ml heparin for boluses | Most standar goal we use on the floor  **Adjust hepain based on aptt: 50-70**  aPTT 37 sec or less: Rebolus with 80 units/kg and increased dose by 4 units/kg/hr  aPTT 38 -49 sec: Rebolus with 40 untits/kg and increase dose by 2 units/kg/hr  aPTT 50-70sec: Continue current dose  aPTT 71-82 sec: Decrease dose by 1 units/kg/hr  aPTT 83-95 sec: Decrease dose by 2 units/kg/hr  aPTT 96 sec or greater: Hold herparin for 1 hr and decrease dose by 3 units/kg/hr  Only use 1,000 unit/ml heparin for boluses |
| Worried about bleeding(GI, etc) lower goal  **Adjust hepain based on aptt: 45-60**  aPTT 38 sec or less: Rebolus with 80 units/kg and increased dose by 4 units/kg/hr  aPTT 39 -44 sec: Rebolus with 40 untits/kg and increase dose by 2 units/kg/hr  aPTT 45-60 sec: Continue current dose  aPTT 61-66 sec: Decrease dose by 1 units/kg/hr  aPTT 67-74 sec: Decrease dose by 2 units/kg/hr  aPTT 75 sec or greater: Hold herparin for 1 hr and decrease dose by 3 units/kg/hr  Only use 1,000 unit/ml heparin for boluses | Worried about clot formation, higher goal  **Adjust hepain based on aptt: 70-90**  aPTT 56 sec or less: Rebolus with 80 units/kg and increased dose by 4 units/kg/hr  aPTT 57sec -69 sec: Rebolus with 40 untits/kg and increase dose by 2 units/kg/hr  aPTT 70-90 sec: Continue current dose  aPTT 91-103 sec: Decrease dose by 1 units/kg/hr  aPTT 104-116 sec: Decrease dose by 2 units/kg/hr  aPTT 117 sec or greater: Hold herparin for 1 hr and decrease dose by 3 units/kg/hr  Only use 1,000 unit/ml heparin for boluses |

IV. CHEST TUBE MANAGEMENT

A. Goals of tube thoracostomy.

1. Effect drainage of fluid and air from the pleural space.

2. Allow complete expansion of the lung and eliminate dead space within the hemithorax.

3. Hasten pleural apposition to allow healing and sealing of air leaks.

4. Remove tubes when drainage has ceased or risk of leak (i.e., esophageal resection ) is past.

B. "Care map" for postoperative chest tubes.

1. Cardiac Surgery.

a. All patients will have mediastinal tubes (sizes vary between surgeons) and usually 1 or 2 pleural tubes.

b. If no air leak exists, chest tubes can be safely removed on postoperative day 1-2 after the patient has sat up and dumped pooled fluid from the chest. Drainage must be less than 100 ml per 8 hours prior to removal.

c. If an air leak exists, isolate and place that specific tube to a separate Pleura-Evac.

d. If mediastinal drainage persists for more than 350 ml/24 hours, leave chest tubes in until drainage ceases. If drainage persists past POD 2 then separate tubes to mediastinal and pleural space Pleura-Evacs, then remove the tubes which have acceptably low drainage.

V. CONTROLLING BLOOD GLUCOSE

**

**











VI. CONGESTIVE HEART FAILURE I PULMONARY EDEMA

A. Congestive Heart Failure (CHF).

1. Definition/Pathophysiology.

a. CHF is the result of cardiac function that is insufficient to maintain systemic perfusion adequate to meet the metabolic needs of the organs.

b. CHF may be a result of ischemic heart disease, cardiomyopathy, or chronic valvular heart disease. Both systolic and diastolic dysfunction may be involved.

2. Diagnosis.

a. Symptoms: may be acute or chronic.

1) Congestive symptoms may involve systemic and pulmonary systems: pedal or peripheral edema, orthopnea, paroxysmal nocturnal dyspnea, dyspnea on exertion, nocturia.

2) Diminished cardiac output: fatigue, weakness, poor exercise tolerance, confusion.

b. Physical findings.

1) Tachycardia, hypotension.

2) Pulmonary congestion: rales, expiratory wheezes, tachypnea, pleural effusion.

3) Vasoconstriction: peripheral cyanosis, cool extremities, diminished peripheral pulses.

4) Jugular venous distention, hepatomegaly, hepatojugular reflux, ascites, pedal edema.

5) S3 gallop.

c. Laboratory data.

1) Hypoxemia.

2) Metabolic acidosis.

3) Hyponatremia.

d. CXR.

1) Cardiomegaly.

2) See signs of pulmonary edema, below.

e. Assessment of ventricular function: echocardiography, MUGA, Swan Ganz catheter.

1) Elevated filling pressures.

2) Diminished cardiac output.

3) Diminished SvO2.

4) Pulmonary edema may result from diastolic dysfunction despite normal systolic function.

f. Differential diagnosis.

1) Pulmonary signs and symptoms

a) Pneumonia.

b) COPD or asthma flare.

c) ARDS or volume overload.

2) Cardiomegaly: pericardial effusion.

3) Hypotension.

a) Volume depletion.

b) Pulmonary embolism.

c) Sepsis.

3. Etiology.

a. Ischemia.

b. Volume overload, particularly 24 days postoperatively when third space losses are given up to the vascular space and renal function/diuretic dosing is inadequate to eliminate fluid.

c. Cardiomyopathy or acute valvular decompensation (valve dehiscence, repair failure).

d. Post infarction VSD.

4. Treatment in the ICU.

a. Supportive.

1) Bed rest.

2) Supplemental oxygen to keep SaO2 > 90%.

3) Consider CPAP or intubation if work of breathing is excessive.

4) Treat complicating derangements (such as infection, anemia, DM) which may have led to decompensation of previously stable CHF.

b. Afterload reduction.

1) Angiotensin converting enzyme (ACE) inhibitors (captopril, enalapril, benazepril)

2) Hydralazine.

3) Sodium nitroprusside.

4) Calcium channel blockers.

c. Improve myocardial contractility.

1) Treat low cardiac output as discussed in Cardiac Output Section, above.

2) If conservative treatment is not effective or hemodynamic instability is present then proceed to invasive monitoring (Foley, arterial pressure line, PA catheter).

3) Treat low cardiac output with inotropic agents. Start with Dopamine. Other effective agents include dobutamine and milrinone.

d. Correction of overloaded total body water.

1) Diuresis, DIURESIS, DIURESIS. Drive BUN/Cr ratio to 2030.

2) Fluid restriction (<2000 ml/day).

3) Dietary sodium restriction (<2 gm/day).

4) Nitrates may help acutely to shift capacitance volume to venous system.

e. Nutrition.

1) Provide adequate calories and protein, preferably enterally. Tube feed early.

2) Consider TPN if bowel function inadequate .

NEW YORK HEART ASSOCIATION FUNCTIONAL CLASSIFICATION

|  |  |
| --- | --- |
| Class | Description |
| I | Patients with cardiac disease but without resulting limitations of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain. |
| II | Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain. |
| III | Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain. |
| IV | Patient with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased. |

B. Pulmonary Edema.

1. Pathophysiology.

a. Pulmonary edema is the result of excess extravascular lung water. Water accumulates in the alveoli and interstitial space which decreases pulmonary compliance, oxygen diffusion, and increases work of breathing.

b. Source may be either cardiac or extracardiac (usually inflammatory).

1) Cardiogenic pulmonary edema: excess hydrostatic capillary pressure.

2) Noncardiogenic pulmonary edema.

a) Altered permeability of the alveolar capillary membrane.

b) Low plasma oncotic pressure will not result in pulmonary edema but predisposes to transudation with less severe noxious insult.

2. Diagnosis.

a. Symptoms: Dyspnea, orthopnea, cough, fatigue, altered mental status, agitation.

b. Signs: Tachypnea, tachycardia, rales, jugular venous distention, peripheral edema.

c. Laboratory data: hypoxemia, uremia.

d. Chest radiograph: vascular cephalization, enlarged hilar vessels, interstitial edema, pleural effusion.

3. Treatment.

a. Hypoxia: Supplemental oxygen, intubation.

b. Reduce pulmonary blood volume: increase venous pooling with nitrates, morphine.

c. Diuresis: if renal function is failing may need greater diuretic dosing or even dialysis.

d. Correct low cardiac output consider dopamine early, see Building Cardiac Output.

e. Monitoring: ECG, urine output, daily weight, consider Aline if hypoxia, consider CVP.

f. Identify and treat underlying or precipitating factors (e.g., tachyarrhythmias, malignant hypertension, valvular disease, fluid overload, acute MI, allergic reaction, sepsis).

VI. MANAGEMENT OF HYPERKALEMIA

A. Definition.

1. Hyperkalemia is important at levels greater than 5 mmolL1.

2. Signs and symptoms.

a. Signs: lengthened PR, loss of R wave voltage, widened QRS, Peaked T waves.

b. Symptoms: tingling, malaise, weakness, paresthesia’s, paralysis.

B. Treatment.

Decide on the physiologic significance of the hyperkalemia based on likely cause, renal function, and potassium level. Treatment consists of discontinuing any supplementation, eliminating potassium from the body, and if needed rapid decrease in serum value by forcing

K+ into cells. Important points:

1. Acidosis is an important precipitating factor in hyperkalemia. A decrease of 0.1 pH unit = 0.6 meq/L increase in K+.

2. Patients on epinephrine infusions requiring insulin infusion will have diminished serum K+ as it is all being driven into the cells. Accept K+ level of 4.04.2 meq/L in these patients, or massive hyperkalemia will result when the insulin infusion is terminated.

3. Ascertain the causes of hyperkalemia: over administration, impaired renal function, acidosis, gastrointestinal bleeding, hemolysis, excessive PRBC transfusions.

|  |  |  |
| --- | --- | --- |
| Treatment Modalities for Hyperkalemia | | |
| Potassium Level | ECG | Treatment |
| K+ >5.0 meq/L | no T-wave changes | 1. stop all supplements  2. repeat lab test in 6 hours |
| K+ >5.8 meq/L | no T-wave changes | 1. stop all supplements  2. repeat lab test stat  3. administer Lasix + kayexalate |
| K+ >6.3 meq/L | regardless of ECG | 1. stop all supplements  2. repeat lab test stat  3. administer Lasix, kayexalate  4. call fellow |
| K+ >6.3 meq/L | ECG: peaked T-waves | 1. stop all supplements  2. repeat lab test stat  3. administer Lasix, kayexalate, insulin, glucose, consider calcium  4. call fellow |

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Drug | Rationale | Dose | Onset | Duration |
| Calcium gluconate | Direct K+ agonist | 1000 mg IV over 2-5 min | Minutes | 60 minutes |
| Glucose and Insulin | Redistribute K+ intracellularly | 25 gm Glucose, 10 units Insulin IV | 15-30 minutes | 2-4 hours |
| Sodium Bicarbonate | Redistribute K+ intracellularly | 50-100 meq IV | 15-30 minutes | 1-2 hours |
| Kayexalate (SPS)1 | Anion exchange resin | 50-100 gm enema  30-60 gm po | 60 minutes | 4 hours |
| Furosemide | Increase renal excretion | 20-80 mg IV | 15-30 minutes | 4 hours |
| Hemodialysis | Direct removal | ------ | 20-30 meq K+/hr. |  |
| Peritoneal dialysis | Direct removal | ------ | 10-15 meq K+/hr. |  |

1Removes 1 meq K+/gm of SPS given PO and 0.5 meq K+/gm of SPS given rectally.



PROCEDURES

I. CENTRAL VENOUS CATHETERIZATION

A. General approach.

For internal jugular vein and subclavian vein cannulation, the patient should be supine. Turn the patient's head away from the desired side of cannulation; prepare the skin with povidone iodine solution; drape the field with sterile towels. Sterile gown, gloves, and mask are mandatory.

B. General technique.

1. Access the vein: The Seldinger (guidewire) technique is used. Infiltrate the desired area with lidocaine. Puncture the skin with the 18g needle mounted on a 10ml syringe, maintaining constant negative pressure; advance slowly until blood appears. Upon entering the vein, disengage the syringe and cover the needle with a sterile gloved finger in order to minimize bleeding and to prevent air embolism. Insert the wire through the needle and advance slowly into the vein. The wire should pass with minimal resistance over its entire length. Remove the needle over the guidewire. Enlarge the puncture site to 34 mm with a sterile blade. Pass a dilator over the guidewire into the vein, always retaining control of the guidewire in order to prevent embolization of the wire; remove the dilator over the guidewire.

2. Place the catheter: In order to pass a single, double, or triple lumen catheter, thread the catheter over the guidewire and into the vein to the measured depth, taking particular care not to contaminate any part of the catheter. Remove the wire through the catheter and cap all ports. In order to insert an introducer and sheath, pass the introducer and sheath together over the wire. Slow, gentle pressure will usually safely advance the introducer into the vein.

3. Secure positioning: when certain of correct placement, remove the introducer and wire through the sheath, and immediately cap the sheath with its previously flushed connector. Aspirate and flush all ports of the catheter or sheath to ascertain intraluminal placement. Secure the catheter to the skin with a suture. Dress the venipuncture site a clear polyurethane adhesive dressing (Op-site).

4. Verify the location of the catheter obtain a chest film immediately after placement. Catheters should descend toward the heart, and the tip should be found in the superior vena cava or at the Cavo atrial junction. The film should also be inspected for the presence of pneumothorax.

C. Specific sites: internal jugular vein.

Central approach: Identify the triangle formed by the sternal and clavicular heads of the sternocleidomastoid muscle. Palpate the adjacent carotid artery pulse and retract the carotid medially with gentle digital pressure. Insert a 22g "seeker" needle to locate the internal jugular vein prior to inserting the 18g needle. Use the provided Angio catheter instead of the needle only method. The needles are inserted at the apex of the triangle of the sternocleidomastoid, directing the needle inferiorly and laterally along the medial aspect of the clavicular head, toward the ipsilateral nipple. If the vein is not entered on the first pass (34 cm), maintain negative pressure in the syringe while slowly withdrawing the needle. Once the seeker needle locates the vein, follow the same trajectory with the Angio catheter. When venous blood is encountered, remove the needle and thread the catheter. Follow the Seldinger technique as described above. If venous blood is not encountered, reassess the landmarks and make a second pass. If arterial blood is encountered, immediately remove the needle and maintain pressure over the artery for 10 minutes before resuming.

D. Specific sites: subclavian vein.

Infraclavicular approach: the ideal target is the junction of the internal jugular vein and the axillary vein subclavian vein which is immediately posterior to the junction of the lateral head of the sternocleidomastoid muscle with the clavicle. Follow the deltopectoral groove laterally from this point laterally and inferiorly for 24 cm. Direct the needle superiorly and medially, just deep to the clavicle and over the first rib, toward the above target. If the vein is not entered on the first pass (5 cm), maintain negative pressure in the syringe while slowly withdrawing the needle. Then, remove the syringe and follow the Seldinger technique as described above. If venous blood is not encountered, reassess the landmarks and make a second pass. If arterial blood is encountered, remove the needle immediately.

E. Special considerations.

1. Success rates for internal jugular vein cannulation range from 6080%; for subclavian vein cannulation, 8595%. The complication rate for internal jugular vein cannulation is 520%, and for subclavian vein cannulation it is 610%.

2. If air is encountered during an unsuccessful attempt at central venous catheterization, obtain a chest film prior to attempting to cannulate a contralateral site, in order to avoid undetected bilateral pneumothorax.

3. Regarding air embolism, a 4 mm Hg gradient across a 20g catheter permits air may flow at a rate of 90 ml/sec, emphasizing the importance of correct patient positioning and other precautions against air embolism. The treatment of suspected air embolism: place the patient in the left lateral decubitus position, administer 100% oxygen, and attempt to aspirate the air through the catheter.

4. Central venous catheters should be changed over a wire (if the site is not infected) or replaced every 5 days, to minimize infectious and thromboembolic complications. In cases of suspected catheter borne infections, the catheter is removed, and its tip submitted under sterile condition for culture.

5. The internal jugular vein is the preferred site in patients with bleeding disorders, as coagulopathy or thrombocytopenia may increase the incidence of hemorrhagic complications and are therefore relative contraindications to cannulation of the subclavian vein.

6. The subclavian vein is the preferred site for long-term central venous access, providing greater patient comfort and facilitating catheter care.

II. PULMONARY ARTERY CATHETERIZATION

A. General approach.

Patient preparation: the patient is monitored continuously with electrocardiography and should have a functioning intravenous catheter in place for treatment of cardiac dysrhythmias.

B. Technique.

1. Access the central circulation: the internal jugular vein or the subclavian vein, using the Seldinger technique as described above, and insert the catheter sheath. After securing the sheath, ensure that the pulmonary artery catheter has been properly flushed and that the balloon is functional. Thread the catheter through its sterile protective sleeve.

2. Insert the PA catheter: with the balloon deflated, insert the catheter through the sheath to a depth of 20 cm, at which point the tip will be beyond the end of the sheath and in the right atrium. Inflate the balloon and advance the catheter slowly, while inspecting the electrocardiogram for dysrhythmias and the pressure tracing for the succession of characteristic waveforms. Once the proper position has been reached (pulmonary capillary wedge), the balloon is deflated. At this point, the tracing should return to that of the pulmonary artery. Inflate with only .5 cc of air. If the wedge tracing reappears, the tip is in too far. Deflate and pull back the catheter 1-2 cm and repeat. If .5 cc of air doesn’t wedge the balloon, add another 5 cc. Again, if the wedge tracing returns with only 10 cc of air, the tip is still advanced too far distally. Deflate the balloon and withdraw the catheter 1-2 cm. The catheter should wedge only after the full 1.5 cc of air is slowly injected and not before then or the tip of the catheter has been advanced too far distally. After the catheter has been successfully positioned, draw the protective sleeve over it and secure the connector.

3. Verify position: the disappearance of the characteristic pulmonary artery waveform when the balloon is inflated and its prompt return when the balloon is deflated is mandatory. Obtain a chest film to ascertain that the tip of the catheter is in a main branch of the pulmonary artery. The film should also be inspected for the presence of pneumothorax.

4. Fluoroscopic guidance may facilitate the passage of the catheter into the pulmonary artery in difficult cases.

III. ARTERIAL CATHETERIZATION

A. General approach.

1. Although the catheter over needle technique is acceptable, particularly for the radial artery, the Seldinger technique is preferred for femoral and axillary arterial cannulation. An introducer needle may be used, or the guidewire may be passed through the catheter, prior to advancing the catheter entirely into the artery.

2. If arterial blood is not encountered, reassess the landmarks and make a second pass. If arterial blood is encountered but the catheter cannot be passed, immediately remove the needle and maintain pressure over the artery for 10 minutes before resuming. If the artery cannot be cannulated in three attempts, discontinue the procedure and choose another site.

3. Once the catheter has been successfully passed, remove the needle and attach the hub of the catheter to the pressure tubing. Secure the catheter to the skin with a suture or tape. Apply a clear adhesive sterile dressing (Op-site).

B. Radial artery.

1. Assess collateral circulation by performing the Allen test. Occlude the radial and ulnar arteries digitally until blanching of the hand is noted. Release the ulnar artery. Ulnar collateral circulation is considered inadequate if more than 5 seconds elapse before blushing of the hand occurs, in which case another site is selected.

2. Placement: support the patient's hand in dorsiflexion with a roll of gauze under the wrist. Secure the palm and lower arm to the board. Use sterile precautions, as previously described. Palpate the radial artery just proximal to the head of the radius. Inject the proposed cannulation site with .1-.2 cc of 2% lidocaine. Not only will this increase patient comfort but will decrease arterial vasospasm. Insert the 20g needle with the bevel down. Advance the catheter and needle through the artery. Withdraw the needle and pull back catheter slowly until rush of blood. Thread wire through catheter and advance over wire.

C. Femoral artery.

1. Equipment: 18g (4inch) catheter over needle; other supplies as listed above.

2. Anatomy: the femoral artery is found at the midpoint of a line between the anterior superior iliac spine and the symphysis pubis. Lateral to medial in the femoral canal are the nerve, artery, vein. Scrub and clip the groin area and prepare the skin with povidone iodine solution. Drape the field with sterile towels. Infiltrate the skin with lidocaine.

3. Placement: enter the skin at a 45-degree angle, with the catheter over needle or the needle alone (Seldinger technique). If the Seldinger technique is chosen, the wire must pass without any resistance, indicating that it is intraluminal and that it has not dissected. If the artery is not entered on the first pass (34 cm), maintain negative pressure in the syringe while slowly withdrawing the needle. If arterial blood is encountered, remove the syringe and follow the Seldinger technique as previously described.

D. Axillary artery.

1. The arm is hyper abducted, externally rotated, and immobilized. Shave the axilla and prepare the skin with povidone iodine solution. Prepare a sterile field with towels. Infiltrate the skin with lidocaine.

2. Placement: enter the skin as high as possible in the axilla, with the catheter over-needle or needle alone (Seldinger technique). If the Seldinger technique is chosen, the wire must pass without any resistance, indicating that it is in the lumen, and that it has not dissected. If the artery is not entered on the first pass (5 cm), maintain negative pressure in the syringe while slowly withdrawing the needle.

E. Special considerations.

1. Arterial catheters should be changed over a wire (if the site is not infected) or replaced every 35 days to minimize infectious and ischemic complications.

2. Remove a catheter immediately when signs of distal ischemia appear.

3. Brachial artery cannulation is not recommended, owing to its inadequate collateral circulation and unacceptably high incidence of complications.

4. Risk factors for ischemic complications include female sex; low cardiac output state; use of vasoconstricting agents; multiple failed attempts to cannulate; peripheral vascular disease; and prolonged duration of cannulation.

IV. THORACENTESIS: Not performed by CT Surgery🡪 consult CVIR

A. General approach

If possible, the patient is placed in the sitting position, with the arms supported on a bedside table. Alternatively, the patient may be supine, with the head of the bed elevated to 90°. The fluid level is located by percussion. Thoracentesis is performed two interspaces below the percussed fluid level, but not lower than the eighth intercostal space. Prepare the skin with povidone iodine or chlorhexidine solution and drape the field.

B. Technique.

1. Infiltrate the skin with lidocaine, using a 25g needle. Anesthetize the deeper tissues with a 20g needle, including the periosteum of the superior edge of the rib below the chosen intercostal space. While aspirating the syringe to avoid intravascular injection of lidocaine, enter the pleural cavity over the superior aspect of the rib, thus avoiding the intercostal neurovascular bundle. Lidocaine is administered into the intercostal tissue just shallow to the pleura, and subsequent aspiration should confirm the presence of pleural fluid.

2. Remove the needle and reenter the pleural space with the catheter over needle attached to a syringe. Remove the needle, covering the hub of the catheter to prevent pneumothorax. Mount the stopcock and the 20ml syringe to the catheter.

3. For diagnostic purposes, fluid may be aspirated directly.

4. For drainage of a large effusion, the extension tubing is connected to the stopcock, so that the pleural fluid can be withdrawn into the syringe and ejected through the tubing into a collecting container. Upon completion of the procedure, a small sterile dressing is placed.

5. Obtain a chest film in order to document the removal of the pleural fluid and to exclude pneumothorax.

C. Diagnosis.

The pleural fluid is collected under sterile conditions and analyzed for: differential cell count; gram stain and bacterial culture; fungal and mycobacterial culture; cytology; protein and glucose; amylase; LDH; and pH.

V. TUBE THORACOSTOMY🡪 Performed by Intensivist, Surgeon or Fellow

A. Indications.

Pneumothorax (greater than 20% in magnitude); hemothorax, hydrothorax, or chylothorax; prophylaxis for high-risk patients prior to positive pressure ventilation; prophylaxis after penetrating thoracic injury.

B. General approach.

The patient is placed in the supine position. The preferred site for tube thoracostomy is the fifth intercostal space, in the anterior axillary line. Prepare the skin with povidone-iodine solution.

C. Technique.

1. Infiltrate the skin with lidocaine, using a 25g needle. Anesthetize the deeper tissues using a 20g needle, including the periosteum of the ribs above and below the chosen intercostal space. While aspirating the syringe to avoid intravascular injection of lidocaine, enter the pleural cavity over the superior aspect of the rib, thus avoiding the intercostal neurovascular bundle. Lidocaine is administered through the pleura, and subsequent aspiration should confirm the presence of pleural air or fluid.

2. Remove the needle and incise the skin one interspace below the desired site of insertion. Create a subcutaneous tunnel with blunt dissection using the Kelly clamp. Your tunnel should be made in a superior-posterior direction (this is the direction the tube will travel once in the pleural space). Failure to do so will invariably result in placement of the chest tube into the fissure. Alternatively, the tract can be dissected sharply (usually this is less painful). Enter the pleural space with the tip of the clamp, just over the superior margin of the rib; open the clamp, spreading the pleura gently.

3. With a gloved finger, confirm penetration into the chest by palpating the lung, sweeping away pleural adhesions, if present. Grasp the tip of the thoracostomy tube with the Kelly clamp, and insert both into the pleural space. Direct the tube posteriorly and superiorly for drainage of hemothorax or hydrothorax, or anteriorly for pneumothorax.

4. Ensure that the last hole in the tube is within the thoracic cavity.

5. It is more important to have the tube adequately placed than it is to have the incision look small. Be sure the incision is appropriately sized so that palpation of the pleural space is possible.

5. Secure the tube to the chest wall with a suture. Cover the wound with a sterile dressing, consisting of petrolatum-soaked gauze and sponges.

6. Inspect and secure all connections.

7. Obtain a chest film to assess the position of the catheter and the evacuation of air or fluid from the thoracic cavity.

D. Complications.

Hemorrhage; laceration of the lung; infection; cardiac injury; subcutaneous placement.

reexpansion pulmonary edema; and intraperitoneal placement. If tube insertion into the heart or lung parenchymal or pulmonary artery is suspected because of massive blood return, CLAMP THE TUBE. THE TUBE WILL NEED TO BE REMOVED IN THE OR.

VI. Nasogastric tube Insertion (see Hospital Policies and Procedures for detailed insertion guidelines)

1. Dobhoff tube
   1. Must use Cortrak Monitor for all Dobhoff insertions
      1. Must complete TJUH Cortrak Competency Checklist
      2. Must be signed off on 5 supervised prior to performing procedure independently.
   2. Follow up with Abdominal X-ray prior to use
   3. Once placement confirmed, place “may use access” order in EPIC.
   4. Bridal Dobhoff if prone positioning
2. OGT/NGT
   1. Auscultate to confirm position first, then Abdominal X-ray prior to use
   2. Once placement confirmed, place “may use access” order in EPIC.

**How to Activate OR for Emergent surgery plan from ICU**

1. ICU team identifies need for emergent surgery requiring emergent return to OR.
2. ICU Advanced Practice Provider (APP) notifies primary surgeon or on-call surgeon if primary surgeon is not available.
   1. Surgeon confirms the need of emergency surgery. Is it urgent vs emergent?
      1. Emergent: immediate threat to life or health
      2. Urgent: no immediate threat to life or health, but if not taken care in a given [period](http://www.differencebetween.net/science/health/difference-between-implantation-bleeding-and-period/) of time, then the situation may turn into an emergency situation.
   2. Surgeon notifies ICU APP if cardiac OR team or regular OR team is needed.
   3. Surgeon notifies ICU APP if OR needs to prepare special equipment.
      1. Cardiac tray, exploration tray, etc.
3. ICU APP places order “Case request operating room” in EPIC.
4. ICU APP calls the OR charge nurse (215-783-1169 7am-3pm, 215-783-2679 after 3pm)
   1. Provide Patient’s name, MR#, Surgeon, Type of surgery
   2. Request emergent surgery and the need for cardiac OR team or regular OR team.
   3. Request OR Room 22 if available (if not OR Room 24 okay)
   4. Request special equipment if needed
5. OR charge nurse makes the following calls:
   1. Anesthesia on-call.
   2. OR nursing staff
   3. Perfusion on-call (1-800-521-9757)
   4. Bedside ICU RN to give anesthesia contact information🡪 ICU RN calls report to anesthesia.
6. ICU APP makes the following calls:
   1. CT Surgery fellow on-call.
   2. OR PA/RNFA on-call.
   3. EP Fellow on-call to turn off AICD if applicable
   4. CVICU respiratory therapist if patient transporting to OR from CVICU: 267-586-0938
7. ICU APP places order for MSBOS in Epic, then call blood bank to ensure they see order and prepare blood x5-6356
8. Fellow or Attending Surgeon
   1. Obtains consent if time allows.
   2. Responsible for delegating transport time
      1. Will determine who will accompany an unstable patient to OR.
9. Anesthesia resident takes consent if time allows.
10. Anesthesia (either attending or resident), respiratory therapist, bedside nurse, and perfusion if pt. is on VAD/ECMO will transport the patient to OR directly (not to the holding area).
11. Surgeon checks the patient in the OR.

**How to Activate OR for Emergent Consult CT Surgery Case**

1. Advanced Practice Provider (APP) assesses patient and situation after consult received
2. Speak with primary team
3. Relay information to on-call surgeon.
   1. Surgeon confirms the need of emergency surgery.
   2. Surgeon notifies APP if cardiac OR team or regular OR team is needed.
   3. Surgeon notifies APP if OR needs to prepare special equipment.
      1. Cardiac tray, exploration tray, etc.
4. APP places order “Case request operating room” in EPIC.
5. APP calls the OR charge nurse (215-783-1169 7am-3pm, 215-783-2679 after 3pm)
   1. Provide Patient’s name, MR#, Surgeon, Type of surgery
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6. OR charge nurse makes the following calls:
   1. Anesthesia on-call.
   2. OR nursing staff
   3. Perfusion on-call (1-800-521-9757)
   4. Bedside ICU RN to give anesthesia contact information🡪 ICU RN calls report to anesthesia.
7. CT Surgery APP makes the following calls:
   1. CT Surgery fellow on-call.
   2. OR PA/RNFA on-call.
   3. EP Fellow on-call to turn off AICD if applicable
8. APP places order for MSBOS in Epic, then call blood bank to ensure they see order and prepare blood x5-6356
9. Enter pre-op order set in EPIC
   1. IP J CTS CABG Pre-op
      1. Full set of labs, type & screen, D/C heparin orders, NPO, chlorhexidine skin prep wipes, UA, STAT portable CXR if time allows, Bactroban nasal ointment x10doses, review current med list.
10. Fellow or Attending Surgeon
    1. Obtains consent if time allows.
    2. Responsible for delegating transport time.
       1. Will determine who will accompany an unstable patient to OR.

**Pre-Op To-Do List:**

Ordered By CT Surg APPs 1-2 days prior to surgery

🡪CABG/VAD pts

1. Diagnostic Cath
2. TTE
3. PA/LAT CXR
4. Carotid U/S 🡪 c/s Vascular surg if > 75% occlusion or symptomatic
5. Radial U/S – IF < 60 yrs., non-smoker, non-DM, or requested by attending
6. Full set of labs – CBC, Chem 7, LFTs, PT/INR, PTT
7. Hgb A1c
8. UA/Culture
9. Type & Screen
10. MSBOS – blood order for surgery (2 PRBC for all Routine cases)
11. NPO after 2359 (may take meds)
12. Bactroban ointment. to BL Nares x10doses
13. Lyrica 150mg x1 dose @6am the day of surgery
14. Chlorhexidine shower (night before) + wipes (morning of)
15. Call EP to turn off AICD in the holding area if applicable
16. D/C existing PICC/Central lines – if unable, discuss with surgeon

🡪Valve pts – all the above, but add…

1. CT Mandible with contrast
2. OMFS c/s

**\*Strict Sternal Precautions\***

**Applies to: All sternectomy patients, all open chest patients, all pec/Lat/omental flap patients.**

Follow for 2 months and then follow up with plastic/cardiothoracic surgery.

1. No lifting, pushing, pulling > 5lbs.
2. No shoulder flexion >90 degrees (Do not lift arms above shoulder level)
3. No shoulder horizontal abduction > midline. (Do not reach out to side)
4. No shoulder extension > midline (Do not reach back)
5. No pushing with hands to stand.
6. Must complete log roll to get OOB
7. No chest compressions.

**How to Activate Portable Fluoroscopy for Bedside VV ECMO Cannulation**

1. Anesthesia resident takes consent if time allows.
2. Surgeon checks the patient in the OR.

1. Page # 2912 to access the radiology technician
2. If unable to reach anyone by the OR pager, page the OR supervisor at #3709
3. Pull the C arm, bed and aprons from the large SPD closet to the bedside
4. Transfer the patient onto the fluoroscopy bed and wait for technician to operate fluoroscopy-anyone in the room during the procedure must wear an apron
5. Each staff member that needs to be in the room during the fluoro use must wear a control badge at the collar area outside of the apron. The badges can be found in the long side medication room. They must be signed out on the form and once they are signed out, they belong to that particular staff member until the end of the quarter
6. After you are finished with the bed wipe it down and place it in the unit hallway. Call the Patient Flow Management Center 34BED Option 4 for a “STAT clean of a Fluoroscopy Table”-The bed should remain in the hallway until cleaned by EVS (turnaround time is 30 minutes)
7. Be sure to wipe down any apron that was used
8. After the bed is cleaned by EVS, be sure to return all equipment to the large SPD closet
9. Be sure to plug the fluoroscopy bed into the wall outlet

To be utilized for patients requiring:

* Veno-Venous ECMO therapy cannulation and cannula repositioning
* Difficult Veno-Arterial ECMO cannulation
* Difficult SGC placement
* Insertion of temporary pacemaker wire

Portable Fluoroscopy Equipment