UCSD Department of Anesthesiology

Primer on Anesthesia

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2nd Edition

Edited by:

Preetham Suresh, MD
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Welcome to the UCSD Anesthesia Department!! We, the current members of the department, are very excited to have you join us. As you transition from all prior stages of medical training to this, your anesthesia residency, keep in mind that this final stage is by far, the most crucial. The practices you establish and knowledge you gain will be far more relevant to your future careers then anything else you’ve learned up until this point. Think about how hard you’ve worked to get to this point and now is the time when it matters most so pour your heart into it! You no longer are learning tidbits of information in preparation for an exam or in order to get a good grade. You are learning the practice of anesthesia so that you can safely deliver anesthetics and take the best possible care of your patients.

Keep in mind you only have three years to see and do as much as you possibly can with the advantage of having the continuous backup of another anesthesiologist. You will never again be able to get the opinion of a faculty member on each aspect of each and every case that you do. Take every opportunity to understand the rationale behind the decisions and techniques you use in the OR. Understanding the ‘why’ is crucial to being able to apply what you learn to the new and often unpredictable situations that you are sure to encounter in the future.

Initially, you will be overwhelmed by the routine of delivering a basic anesthetic and it will seem like there is so much to do and remember. It will get easier with time. Early on you will feel like you are just responding to things that happen in the OR. With experience, you will anticipate what might happen and will take steps to ensure that it never does. Anesthesiologists are not measured by how well they manage a crisis; it is by how well they prevent it from ever happening. You would never say of a racecar driver that they are so skilled because every time they hit another car, they recover really well. This ability to anticipate and be prepared doesn’t come by accident. It comes from learning about your patients and the surgeries they will be having. You need to know the implications of each of your patient’s comorbidities and the medications they are on. The department buys you several books to assist you in this process but they are only useful if you read them. Make a commitment to read at least 5 minutes every night. Some nights you will have more time and energy and will be able to get an hour of reading done. Other times you will be exhausted and will only be able to get through the highpoints for your cases the next day.

Your textbooks are there for you to accumulate knowledge. Your attendings are there to help you apply that knowledge and to help you develop judgment. You have three years with your attendings, you have a lifetime with your books. Learn from them what you can’t learn from a book.

Use your faculty to get feedback about your practice. This is priceless information that you will never really be able to get again. Every day, find out what they think you could have done better. We are all learning and trying to get better, so take any suggestions as ways you can improve. You will learn multiple ways to accomplish the same task. We were all residents at one point and know it can be frustrating to be told to do something one way, just to be told the very next day to do it some other way. Take it all in stride and learn why different people do things different ways so you can establish your own style of practice.

The next three years will be some of the most challenging but rewarding years yet. You have a whole department of people here to help you through this process…never hesitate to ask for it. Because of how difficult this time can be, don’t forget to continue to live your life. Continue to exercise, get plenty of sleep and always remember why it is we do what we do. We are caring for patients. Despite how hard we may work or how hard our day may have been, our patients are suffering from cancer or are about to have open heart surgery. They are nervous and need a kind, caring, and knowledgeable anesthesiologist to help them through an incredibly stressful time. Be that person for them.

–Preetham
GOALS AND OBJECTIVES

1) Preoperative

- Understand and perform basic machine check and verify backup O2 supply
- Draw up appropriate medications for a case
  - i) Know indication, dosages, and side effects for routine medications
    1. Anxiolytics: Midazolam
    2. Induction agents: Propofol, etomidate
    3. Neuromuscular blockers: Succinylcholine, rocuronium/vecuronium
    4. Opiates: Fentanyl, morphine, hydromorphone
    5. Acetylcholinesterase inhibitors: Neostigmine
    6. Anticholinergics: Glycopyrrolate
    7. Vasoactive agents: Ephedrine, Phenylephrine, Esmolol, Labetolol
    8. Volatile agents: Isoflurane, Sevoflurane, Desflurane, Nitrous Oxide
  - ii) Know how and where to check out narcotics
- Preop H&P
  - i) Perform focused history
  - ii) Conduct focused physical exam
  - iii) Review the medical record (EPIC, Vista)
  - iv) Order, review and interpret relevant labs and tests
  - v) Complete preoperative chart
  - vi) Assess if your patient is optimized for surgery
  - vii) Select appropriate anesthetic plan considering patients comorbidities
  - viii) Discuss risks and benefits of proposed plan with patient
- Concisely present patient, relevant information and anesthetic plan to attending

2) Intraoperative

- Determine appropriate premedication for patient
- Transport patient from preoperative holding area to OR
- Transfer patient from locked gurney to locked OR table
- Position patient
  - i) recognize mechanisms for positioning injuries
  - ii) recognize ideal sniffing position
  - iii) know indications for ramp
- Select appropriate monitors
  - i) Be able to independently place all routine monitors on patient
  - ii) Understand how each monitor works, sources of error and management of perturbations
    1. SpO2
    2. EtCO2
    3. NIBP
    4. ECG
    5. Temp
- Perform effective preoxygenation
  - i) Recognize signs of adequate preoxygenation
- Perform patient specific induction
  - i) Know rationale behind drug selection and dose
- Perform effective mask ventilation
  - i) Recognize signs of effective mask ventilation
  - ii) Understand use of adjustable pressure limiting valve
- Perform successful laryngoscopy
Perform successful LMA placement

Recognize and manage basic intraoperative events
i) Hypoxemia
ii) High Peak Airway Pressure
iii) Hypercarbia
iv) Hypotension
v) Hypertension
vi) Bradycardia
vii) Tachycardia
viii) ST Depression
ix) Hypothermia
x) Low Urine Output
xi) Aspiration
xii) Failure to Awaken

Extubation
i) Prepare for and assess patient for extubation readiness
ii) Extubate patient
iii) Accurately assess adequacy of ventilation postextubation

Complete and accurate and legible OR record or Docusys chart

Procedural skills
i) PIV
   (1) Start kit
   (2) Hot line setup
   (3) Deair tubing
ii) Arterial Lines
   (1) Placement
   (2) Sterile kits
   (3) Transducer setup and zeroing
iii) Neuraxial (optional)
   (1) Patient selection
   (2) Prep
   (3) Kit selection
iv) Central line (optional)
   (1) Indications
   (2) Prepackaged kit
   (3) Sterile technique

3) Postoperative
   - Transport patient to PACU
   - Monitor and recognize adequacy of ventilation during transport
   - Provide complete but concise signout to PACU RN
   - Complete PACU orderset

4) Conduct Daily Feedback
   - CA-3 to CA-1 feedback
   - Faculty to CA-1 feedback
   - CA-1 to CA-3 feedback
   - CA-1 to faculty feedback
Some Anesthesia Do's:

1. Do assume vital sign changes are REAL until proven otherwise. Don't just assume artifact!

2. Do call your attending if someone from the surgical team asks you to do something you are uncomfortable with.
   
a. Example - Pulling an endotracheal tube when you have +ETCO2 and the patient is pink despite the O2 sat not picking up and the surgical attending telling you the tube is not in.

3. Do read your medication vials carefully (and double-check), including drug name, dosage, and expiration date.
   
a. Medication errors happen all the time!
   
b. Do not do anything else while drawing up your medications. If someone is trying to talk to you while you are drawing up your drugs either tell them to wait, or stop drawing up the drugs.

4. Do make sure the laryngoscope light is working before you use it to intubate. Always check all of your airway equipment prior to inducing anesthesia.

5. Do check (and double-check) your infusion settings when using drips in the OR.
   
a. Example - Programming phenylephrine infusion to mcg/kg/min instead of mcg/min will lead to gross overdosage.

6. Do make sure your patient is adequately reversed and spontaneously ventilating before extubation.

7. Do use caution when inserting a nasal trumpet into a patient who is on Asa, Plavix, or anticoagulant agents. The nose can bleed extensively!

8. Do know that insulin vials contain 100 units/ml and MUST be diluted. Always re-check the patient's blood glucose shortly after administering insulin.

9. Do know that epinephrine and vasopressin vials contain ACLS dosages and MUST be diluted before administering to a non-coding patient.

10. Do provide or obtain a thorough sign-out before the transfer of care of a patient to another provider. Errors due to transfer of care occur all the time!

11. Do not fail to notice that your patient is obstructing and not moving adequate air on the way to PACU

12. What appears to be a simple task can turn into an emergent cannot intubate/cannot ventilate situation! Do hope for the best but plan for the worst.
**Some Anesthesia Don'ts:**

1. Don't try to pre-oxygenate your spontaneously-breathing patient with the pop-off valve closed.

2. Don't forget to turn on the ventilator after intubating a patient.

3. Don't forget to provide anesthesia after paralyzing/intubating a patient.

4. Don't try to manage the airway alone with the bed turned away:
   a. Example: Trying to convert a nasal rae to an oral endotracheal tube with the bed turned 90 degrees away - when the nasal rae is pulled, the oral/nasal cavities fill with blood, no suction ready or within reach, unable to obtain a good larygoscopic view due to blood, difficult mask ventilation due to blood in upper airway.

5. Don't give hemabate or methergine IV (always IM) - this applies to OB anesthesia.

6. Don't push anything through an arterial line - especially drugs. It is also wise to avoid re-administering the "wasted" blood back through the arterial line to avoid inadvertent injection of air. You can give this blood back through a venous line.

7. Don't let yourself get behind when a patient is bleeding. Check hgb q30 min during any ongoing blood loss and keep in mind your patient’s estimated allowable blood loss.

8. Don’t hook up hotline tubing to a patient without flushing it first.
First things first: your initial response to low O$_2$ saturation, PaO$_2$, or blue patient

- Patient on 100% F$\text{O}_2$, look at all other vitals
- Check the airway
  - confirm ETT placement by verifying ETCO$_2$, listening to patient, bilateral chest rise, +/- FOB
- Hand ventilate (decrease machine factors)
  - feel compliance or leaks
  - recruitment maneuver, add PEEP
- Suction ETT
- Check surgical field, call for HELP if worsening or no clear cause, communicate to surgical team

Once you instinctively do the above, consider a systematic approach to diagnosing the problem.

One suggestion: start at the alveoli and work towards the machine

**Listen to lungs** (atelectasis, pulmonary edema, bronchoconstriction, mucus plug, secretion, mainstem intubation, pneumothorax, esophageal intubation)

**Check ETT** (cuff deflated, extubated, kinked ETT, biting on ETT)

**Check circuit** (disconnect at ETT or from machine)

**Check machine** (inspiratory and expiratory valves, bellows, pipeline and cylinder pressures, F$\text{O}_2$, MV)

**Check monitors to confirm** (pulse oximeter waveform, gas analyzer)

**Differential Diagnosis**

1) **Low F$\text{O}_2$**
   - Altitude
   - Hypoxic F$\text{O}_2$ gas mixture
   - In OR: if low F$\text{O}_2$ on “100% O2”, go to alternative O2 source i.e. TANKS on back of machine (open valve, disconnect O2 from wall to machine) or use separate tank with Mapleson circuit.

2) **Hypoventilation**
   - Drugs (opioids, BDZs, barbituates)
   - Neuromuscular diseases
   - Obstruction (OSA, upper airway compression)
   - In OR: check circuit leaks, low TV/RR or MV, residual NMB, high ETCO$_2$, high PIP, kinked/obstructed ETT, poor chest rise

3) **Ventilation-perfusion inequalities** (Dead Space ventilation: ventilated areas without perfusion)
   - COPD, ILD, Embolus (air, blood, fat, amniotic fluid)
   - In OR: remember things causing hypotension with poor perfusion (hypovolemia, MI, tamponade, sepsis)

4) **Shunt** (perfused areas that are not ventilated, V/Q = 0)
   - PNA, atelectasis, ARDS
   - Congenital (ASD, VSD, PDA), AVM
   - In OR: think about mainstem intubation, bronchospasm, anaphylaxis, mucus plug—LISTEN to patient

5) **Diffusion Impairment**
   - Increased diffusion pathway (pulmonary edema, fibrosis)
   - Decreased surface area (emphysema, pneumonectomy)
   - Usually chronic

6) **Artifact**
   - In OR: consider this LAST, if all else okay
   - Poor waveform: probe malposition, cold extremity, light interference, cautery, dyes (methylene blue, indigo carmine, blue nail polish), extremity movement (vibration, evoked potentials)
   - Poor perfusion: cold extremity, BP cuff inflation, tourniquet still from trying IV start
Alveolar Gas Equation
\[ P_AO_2 = F_iO_2(P_{atm} - P_{H2O}) - (PaCO_2 / 0.8) \]
\[ \approx 0.21(760-47) - (40/0.8) \]
\[ \approx 100 \text{ mmHg on RA} \]
\[ \approx 660 \text{ mmHg on 100}\% F_iO_2 \]

Alveolar-arterial (A-a) Gradient
\[ P_{(A-a)}O_2 = P_{A}O_2 - PaO_2 \]
Normal A-a gradient
\[ <10 \text{ mmHg (FiO2 =0.21)} \]
\[ < 60 \text{ mm Hg (FiO2 = 1.00)} \]
\[ <(age/4)+4 \]

Arterial O2 Content
\[ CaO_2 = O_2-Hb + Dissolved O_2 = (Hb x 1.36 x SaO_2/100) + (PaO_2 x 0.003) \]
\[ = (15 x 1.36 x 100\%) +100 x 0.003) \approx 20 \text{ cc O}_2/\text{dl} \]

Mixed Venous O2 Content
\[ CvO_2 = O_2-Hb + Dissolved O_2 = (Hb x 1.36 x SvO2/100) + (PvO_2 x 0.003) \]
\[ =(15x1.36x75\%) + (40x0.003) \approx 15 \text{ cc O}_2/\text{dl} \]

O2 Delivery
\[ DO_2 = CO x CaO_2 = 5 \text{ L/min x 20 cc O}_2/\text{dl} = 1 \text{ L O}_2/\text{min} \]

O2 Consumption (Fick Equation)
\[ VO_2 = CO x (CaO_2 - CvO_2) = 5 \text{ L/min x 5 cc O}_2/\text{dl} = 250 \text{ cc O}_2/\text{min} \]

O2 Extraction Ratio
\[ ERO_2 = (VO_2 / DO_2) x 100 = 250 / 1000 \]
\[ \approx 25\% \text{ (normal 22-30\%)} \]

Bohr Equation (Dead Space Fraction)
\[ V_D/V_T = (PaCO_2 - P_{ECO2})/PaCO_2 \]
\[ \approx \text{normal 33\%} \]

Oxygen-Hemoglobin Dissociation Curve

Rule of 30,60,90: PaO_2 of 30 is 60\% sat, 60 is 90\% sat, and PaO_2 of 90 is 100\% sat.
Venous: O_2sat of 75, PaO_2 of 75
P50 (the PaO_2 where hgb is 50\% saturated) is \approx 27 \text{ mmHg}
Elevated PIP – Thomas Griffiths, MD

**Peak Airway Pressure is made up from:**
1. Inspiratory flow resistance (resistive/dynamic pressure).
2. The elastic recoil of the lung and chest wall (elastic/static pressure).
3. The alveolar pressure present at the beginning of the breath (PEEP).

The approach – Preetham Suresh, MD

First address A, B, C’s
1. 100% FiO2
2. Switch to bag
3. Hand ventilate, verify BL BS and EtCO2

Address common or most likely diagnosis
1. Bronchospasm
2. Endobronchial Intubation
3. Secretions

Go through systematic differential of possible causes
Assess if Plateau pressure is elevated or just PIP

**Increased PIP**
**Normal Plateau**

**A. Mechanical**
1. Kinked circuit
2. Faulty inspiratory valve
3. Scavenging failure

**B. Endotracheal Tube**
1. Kinked
2. Secretions
3. Depth
4. Esophageal

**C. Conducting Airways**
1. Bronchospasm
2. External compression

**D. Alveolus**
1. Atelectasis
2. Edema
3. Aspiration
4. Restrictive lung disease
5. Gas trapping

**E. Pleural Space**
1. Tension pneumothorax
2. Hemothorax
3. Pleural Effusion

**F. Chest Wall**
1. Obesity
2. Paralytic wearing off
3. Surgeon leaning on chest
4. Narcotic induced rigidity

**G. Abdominal Compartment/ Diaphragm**
1. Respiratory effort
2. Coughing
3. Abdominal insufflation
4. Ascites
5. Trendelenberg
Hypercarbia

● Increased CO2 levels (measured by blood gas or etCO2)
● Caused by either inadequate ventilation of increased CO2 production.
  ○ Can lead to respiratory acidosis, increased pulmonary artery pressure and increased intracranial pressure

● Inadequate Ventilation
  ○ Central depression of medullary respiratory center
    ■ Meds - opioids, barbiturates, BDZ, volatile agents
    ■ CNS pathology - tumor, ischemia, edema
  ○ Neuromuscular depression
    ■ High spinal anesthesia
    ■ Phrenic nerve paralysis
    ■ Muscle relaxants
  ○ Inappropriate ventilator settings → Low minute ventilation
  ○ Increased airway resistance
    ■ Bronchospasm, upper airway obstruction, severe COPD, CHF, hemo/pneumothorax, ATX, pneumoperitoneum with CO2, surgical retractors preventing lung expansion
  ○ Increased dead space
    ■ ETT malfunction - kinked ETT, endobronchial intubation
  ○ Rebreathing of exhaled gases
    ■ Exhausted carbon dioxide absorber, inspiratory/expiratory valve failure, inadequate fresh gas flows in non-rebreathing systems
  ○ One lung ventilation - Especially in pt's with preexisting pulmonary pathology

● Increased CO2 production
  ○ Exogenous CO2 - Insufflation during laparoscopy
  ○ Reperfusion (release of tourniquet)
  ○ Hypermetabolic states - Malignant hyperthermia, sepsis thyrotoxicosis, fever/shivering, neuroleptic malignant syndrome

● Investigations/Treatments
  ○ Ensure appropriate ventilator settings
  ○ Ensure muscle relaxant reversal (if increased CO2 during emergence)
  ○ Assess for residual narcotic/anesthetic effects (if increased CO2 during emergence)
  ○ Examine CO2 absorber for exhaustion
  ○ Check ABG - electrolyte disturbances, hypoglycemia
  ○ If spontaneously breathing → Assist breathing, lighten anesthesia
  ○ If mechanically ventilated → Increase minute ventilation
  ○ Consider neurologic causes
BP = CO x SVR

HR x SV

- Rate
- Rhythm

- Afterload
- Preload
- Contractility

**Preload**
- Absolute hypovolemia;
  - Hemorrhage
  - Diuresis
  - Bowel prep, NPO status
- Relative hypovolemia (decreased venous return);
  - Increased intra-abdominal pressure- compartment syndrome, insufflation
  - Increased thoracic pressure- pneumothorax
  - Surgical IVC compression
  - Positional – Reverse trendelenberg
  - Cardiac tamponade

**Contractility**
- Ischemia
- Iatrogenic- beta-blockers, drug swap
- Cardiomyopathies, myocarditis

**Afterload**
- Vasodilation- sepsis, anaphylaxis, etc
- Drugs/drug swap
- Sympathectomy

**Management of Hypotension**
- Open IV fluids, place in Trendelenberg
- Administer vasopressors
- Room sweep
  - Confirm BP
  - Check EKG for rhythm/ST changes
  - Check ventilator for increased PIP, EtCO2
  - Check surgical field for hemorrhage, CO2 insufflation, retraction, position change
- Consider fluid status
Hypertension

Etiologies
1. Primary Hypertension
   - Long standing hypertension (aka primary hypertension)
   - Hypertension associated with specific disease process
     • Preeclampsia
     • Kidney Failure
2. Secondary Hypertension (i.e. sympathetic stimulation)
   - Hypoxemia
   - Hypercapnia
   - Pain (usually associated with tachycardia, unless beta blocked)
     • somatic (e.g. incision, fractured bone)
     • visceral (e.g. distended bladder)
     • sympathetic (e.g. tourniquet pain)
   - Unusual possibilities
     • medication error (e.g. inotropes running)
     • pheochromocytoma
     • carcinoid syndrome
   - Other
     • illicit drug use (e.g. cocaine, amphetamines)

Treatment - identify/treat the underlying cause
1. Improve oxygenation and ventilation (check SpO2, FiO2, ETCO2, ETT, ABG)
2. Increase the depth of anesthesia (check vaporizer, check IV esp w TIVA, tourniquet time, opioids)
3. Empty full bladder (check foley)
4. ETT (depth - carina ?)
5. Medications (last drug given ?, pressors)
6. Medicate
   - α/β adrenergic blocking agents (e.g. labetalol 5-10 mg IV)
   - β-adrenergic blocking agents (e.g. metoprolol 1 to 5 mg IV, esmolol 5-10 mg IV)
   - Vasodilators (e.g. hydralazine 2.5-5 mg IV, NTG gtt at 30-50 ug/min IV, Nitroprusside gtt at 30-50 ug/min IV)
   - Ca channel blockers (verapamil 2.5-5 mg IV, diltiazem 5-10 mg IV)
7. Other things to consider
   - drug contamination (e.g. epi soaked gauze)
   - autonomic hyperreflexia
   - elevated ICP (HTN, bradycardia, irregular respirations - unlikely if GA)
   - malignant hyperthermia
   - hypervolemia
Bradycardia

- Bradycardia
  - Defined as HR < 60bpm
  - May be Sinus Bradycardia (SB), or bradycardia due to problems with the heart’s conduction system (Heart Block)

- Sinus Bradycardia
  - In the absence of underlying heart Dz, SB is typically well tolerated until heart rates get very low (< 40bpm)
    - The exception is Peds as neonates’, infants’, and small childrens’ cardiac output is HR dependent due to fixed stroke volume and HR < 60 is poorly tolerated and warrants emergent aggressive Tx
  - when HR becomes very low, atrial or ventricular escape beats/rhythms may become evident
    - Etiologies for SB:
      - Hypoxia
      - Intrinsic Cardiac Dz: Sick Sinus Syndrome (SSS), Acute MI (especially inferior wall)
      - Drugs:
        - Succinylcholine (primarily in peds cases or w redosing)
        - Anticholinesterases i.e. reversal agents (Neostigmine)
        - B-blockers
        - Calcium channel blockers
        - Digoxin
        - Potent synthetic narcotics (fentanyl, remi, alfenta, sufenta)
        - Alpha-2 agonists (Dex)
        - Consider drug swap
  - Increased Vagal Tone/Reflexes:
    - Visceral traction (peritoneum, spermatic cord), laparascopic insufflation, brainstem manipulation, direct stimulation of vagus nerve or carotid body, vaso-vagal reaction, valsalva maneuver, oculo-cardiac reflex, Bezold-Jarisch reflex
  - Elevated ICP (Cushing Response)

- Treatment of SB:
  - Start with ABC’s:
    - A, B: ensure adequate oxygenation and ventilation
    - C: Determine whether pt is stable or unstable (cycle BP cuff, while waiting assess for drop in EtCO2 suggesting low CO). Unstable if MAP by >20%
      - Stable: Glyco 0.2 mg q 6 min or Ephedrine 5-10mg (1-2mL)
      - Unstable:
        - Atropine 0.5mg or Epi 50mcg
      - If unstable or not responsive to treatment, alert surgeon and consider removing the offending stimulus (ie desuflate abdomen, release ocular traction)
- If due to intrinsic cardiac Dz: atropine, + chronotropes (dopamine or dobutamine gtts), pacing

- **Heart Block**
  - 1\(^{st}\) Degree AVB: Prolonged PR interval >200ms with every p-wave conducted and followed by a QRS
  - 2\(^{nd}\) Degree AVB:
    - Mobitz 1 (Wenckebach): Conduction defect at the AV node with progressive PR prolongation until a p-wave fails to be conducted, typically a benign condition
    - Mobitz 2: Conduction defect distal to the AVN with a constant PR interval but random nonconducted P-waves, commonly progresses to 3\(^{rd}\) degree AVB
  - 3\(^{rd}\) Degree AVB: Conduction defect distal to the His Bundle with no A-V conduction, P-waves are regular but independent of a slow ventricular escape rhythm typically around 45bpm
  - Treatment of Heart Block
    - 1\(^{st}\) Degree: Does not require treatment
    - 2\(^{nd}\) Degree:
      - Mobitz 1: Treatment is via pacing and is only required in the presence of symptoms, CHF, or bundle branch block
      - Mobitz 2: Pacing
    - 3\(^{rd}\) Degree: Pacing

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**Figure 1**: Paulev-Zubieta. *Textbook in Medical Physiology And Pathophysiology Essentials and clinical problems*
NEW ONSET TACHYCARDIA
Intraoperatively or in PACU

Stable or Unstable?

Unstable

Cardioversion

Sinus Tachycardia
HR 100-160 bpm

Dysrhythmia

Polymorphic VT

Extensive differential

- Catecholamine excess
- Pain
- “Light” anesthesia
- Hypercapnea
- Hypoxemia
- Hypotension
- Hyponolemia
- Hypoglycemia

-Medications
  --- pancuronium, desflurane, atropine, ephedrine, dopamine, epinephrine, **etc**
- Fever, sepsis

-Myocardial Ischemia
- Malignant hyperthermia
- Pheochromocytoma
- Thyrotoxicosis
- Carcinoid Syndrome
- Tension Pneumothorax
- Pulmonary Embolism
- Cardiac Tamponade

IDENTIFY CAUSE

- Vital Signs, ST-segments, End-tidal CO2/agent,
- Ensure good oxygenation/ventilation
- Adjust depth of anesthesia
- Correct hyponolemia/low SVR
- Narcotics/Beta-Blockers may be useful, especially in patients with CAD.

Supraventricular Tachycardia

Paroxysmal SVT – HR 150-250

Diff Dx- WPW, Thyrotoxicosis, Stress, MVP, Caffeine, excess catecholamines.

Rx- Adenosine 6-18 mg

- Carotid massage
- Beta-Blockade

--- Metoprolol 2.5-5mg

--- Esmolol 5-10 mg

Can be unstable and require synchronized cardioversion

A-Fib/A-Flutter

Rate control

- Beta-blockade

--- Metoprolol 2.5-5mg

--- Esmolol 5-10 mg

--- Calcium Channel Blocker

--- Verapamil 2.5-5 mg

--- Diltiazem 10-20 mg

Rhythm Control

- Amiodarone – 150 mg

Example situation: The patient’s heart rate goes up.
After a check to assure adequate oxygenation and ventilation, and a tally of fluids in and out, I would check the EKG to make sure there weren’t any changes indicative of ischemia, then I would deepen anesthesia. If this maneuver dropped the blood pressure, then I would assume hyponolemia and administer a 500-cc fluid bolus

“Common things are common”
Light anesthesia, pain (surgical/tourniquet/bladder), hyponolemia or hypotension will be the problem the vast majority of the time.
ST Depression

ST segment changes are signs of myocardial ischemia, which in turn signals an imbalance of myocardial oxygen supply and demand. Classically, ST depression signals subendocardial ischemia, where the coronaries are patent but there is inadequate oxygen supply for the current demand. ST elevation signals transmural ischemia from complete coronary occlusion.

ST changes are measured relative to the “baseline” which is the T-P segment. This can be seen in the 1st and 3rd tracings at left. Depressions or elevations of at least 0.1mV or 1mm are considered significant.

General advice
- **Always** look at the EKG prior to induction as your baseline; if in any way abnormal, print a strip
- Leads II and V combined approach a 90% sensitivity for detecting ischemia; the precordial lead (V3, V4, V5) is the best single lead
- So, pay careful attention to appropriate placement of the precordial lead
- The ST segment is **not** useful for detecting ischemia in patients who are V-paced or have LBBB
- Positioning matters: the morphology of the entire EKG will change with lateral or prone position or when the leads are moved

• Many EKGs have baseline abnormalities, but only a few events can cause important changes in the EKG: change in rhythm, change in pacing, change in position, or ischemia

When ST depression happens:
- Is it significant, i.e. is it > 1mm (0.1mV), is it horizontal/downsloping? Look back at the ST trends that are stored in the monitor.
- Is it in contiguous leads?
- Is this the “usual” setup for subendocardial ischemia: is this an at-risk patient, is this a risky surgery, what are the recent surgical or anesthetic events, is there tachycardia+hypotension? (a similar concept applies for ST elevation)

<table>
<thead>
<tr>
<th>I Lateral</th>
<th>aVR</th>
<th>V1 Septal</th>
<th>V4 Anterior</th>
</tr>
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<tbody>
<tr>
<td>II Inferior</td>
<td>aVL Lateral</td>
<td>V2 Septal</td>
<td>V5 Lateral</td>
</tr>
<tr>
<td>III Inferior</td>
<td>aVF Inferior</td>
<td>V3 Anterior</td>
<td>V6 Lateral</td>
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Circulation
- **Decrease O₂ demand**
  - Treat tachycardia
  - esmolol vs metoprolol
  - Treat SBP, if extreme hypotension
  - NTG
- **Increase O₂ supply**
  - Increase time in diastole
  - O2 content (SpO₂, Hgb)
  - Increase DBP

Get more info
- Run 12 lead (from your machine)
- Arterial line
- ABG w/ Hgb
- TEE > PAC
- Cardiologist: L Heart cath, PTCA, IABP

Call for help
- Have circulator call attending
- Notify surgeons

Airway
- Verify etCO₂

Breathing
- 100% O₂
Hypothermia

Jackie Phan, MD

Definition: core body temperature <35°C

Mechanism:

- **redistribution**: core \(\rightarrow\) periphery 2/2 vasodilation + inhibition of central thermoregulation by volatile anesthetics
- **radiation**: heat loss from movement of atoms and molecules carrying energy away from exposed surfaces
- **convection**: transfer of heat from object to environment due to motion of fluids (i.e. high airflow rate in OR)
- **evaporation**: liquid vaporizes from mucosal and serosal surfaces, and skin—depends on exposed surface area and humidity of ambient gas
- **conduction**: heat transfer from warm to cool object in contact

Causes:
- cold OR
- body exposed to air
- cold fluids (skin prep, irrigation fluid, IVF, blood)
- ventilation with cool gas
- open abdomen/thorax (large evaporative losses)
- contact between patient and OR table

Tx:
- increase OR temp
- Bair hugger
- circulating H2O pad
- cover all exposed areas if possible, especially head
- warm IVF/blood
- circuit humidifier
- warming lights (especially for babies)

Benefits:
- metabolic rate decreases 8% per 1°C ↓ in body temp
- CNS protection/improved neurological outcome after cardiac arrest

Effects:

<table>
<thead>
<tr>
<th>Systems</th>
<th>Complications</th>
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<tr>
<td>Cardiovascular</td>
<td>Myocardial ischemia</td>
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<tr>
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<td>Hypertension</td>
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<td>Tachycardia</td>
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<td>Deep venous thrombosis</td>
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<td>Coagulation</td>
<td>Platelet activation</td>
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<td>Coagulopathy</td>
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<tr>
<td>Immune</td>
<td>Increased incidence of infection on surgical wound</td>
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<td>Hydroelectrolytic changes</td>
<td>Hypokalemia</td>
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<td></td>
<td>Hypomagnesemia</td>
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<td></td>
<td>Hypophosphatemia</td>
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<tr>
<td>Endocrine-metabolic changes</td>
<td>↓ Steroids</td>
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<td></td>
<td>↑ Insulin</td>
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<tr>
<td></td>
<td>↑ Peripheral resistance to insulin</td>
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<td></td>
<td>↑ TSH (\rightarrow) thyroxin</td>
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<td></td>
<td>Hyperglycemia</td>
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<td>Hypoglycemia</td>
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</tbody>
</table>

TSH = thyroid stimulating hormone
Hyperthermia

Definition: ↑ temp of 2°C/hr or 0.5°C/15 min

Causes: Hypermetabolic vs Other
• Infection/sepsis • excessive heating (rare)
• Thyroid Storm • allergic rxn (blood mismatch)
• Pheochromocytoma • Neuroleptic Malignant Syndrome
• Malignant Hyperthermia • anticholinergics (sweating inhibited)
• serotonin syndrome (MAOI, TCAs, amphetamines, cocaine)

Tx: cool exposed surfaces (ice, cooling blanket, reduce OR temp)
    internal lavage (stomach, bladder, bowel, peritoneum)
    alcohol to skin to promote evaporation
    conductive loss → vasodilate (nipride, NTG)
    meds: asa, Tylenol (NGT or rectal)

Effects: ↑ metabolic state = ↑ O2 consumption = ↑ cardiac work
Oliguria is defined as urine output less than 0.5mL/kg/hr. Etiologies can be divided into pre-renal, intrarenal, and post-renal causes.

- **Pre-renal**: due to decreased circulating blood volume, either actual (hypovolemia) or perceived (decreased cardiac output). Continued renal hypoperfusion may result in intrinsic renal damage.
- **Intrarenal**: In response to surgical stress, there is activation of the renin-angiotensin-aldosterone system and increased secretion of ADH, resulting in low urine output. Other causes include toxins or ischemia resulting in ATN.
- **Post-renal**: Obstruction that prevents emptying of urine. Causes include kinked, clogged, or disconnected foley, surgical manipulation of the kidneys, ureters, or bladder, renal calculi, neurogenic bladder, or prostatic disease.

Intraoperatively, try to maintain stable VS and UOP greater than 0.5ml/kg/hr. Steps to correct/treat low UOP include:

1) Rule out mechanical causes, such as a malpositioned or kinked foley.
2) Treat hypotension (colloids, crystalloids, or pRBCs) to ensure adequate renal perfusion.
3) Assess volume status (see below*). If hypovolemia is suspected, start with a fluid bolus. If oliguria persists, then acid/base status, a CVP measurement, systolic variation on an arterial line tracing (if present), or stroke volume variation measurement may help guide further fluid management.
4) If oliguria persists despite the above maneuvers and what appears to be an adequate volume status, urine output can be augmented with certain drugs. Keep in mind that they do not affect renal function or outcome.
   - Furosemide
   - Dopamine infusion, 1-3 mcg/kg/min
   - Mannitol
   - Fenoldapam, 0.1-0.4 mcg/kg/min
5) If a patient is on chronic diuretic therapy, they may require intraoperative diuretics.

*Remember to tailor your fluid management to the patient, surgery, and clinical evaluation. Ways to assess intravascular volume status include:
   - HR, BP, pulse oximetry waveform and changes with positive pressure ventilation
   - If an arterial line is present, serial ABGs can be sent, evaluating for hemo-concentration, rapidly decreasing Hct, acidosis
   - CVP, if present, with trends being more important than absolute values
   - PA catheter and TEE (usually used in patients where there is anticipated hemodynamic instability)

Urine and serum indices can also help distinguish prerenal, intrarenal, or postrenal causes. It is rare to send for these because most causes of oliguria can be fixed with the above maneuvers. Clearly, a concentrated urine osmolarity (>500) indicates a prerenal cause. However, if there is confusion, a FeNa may be helpful. FeNa<1% and urine Na<10mEq/L indicate prerenal etiologies while FeNa>2% and urine Na>20mEq/L indicate renal/postrenal etiologies.
ASPIRATION

Jacklynn Sztain, MD

General anesthesia causes depression of airway reflexes that predisposes patients to aspiration.

Airway Reflexes: Laryngospasm, Coughing, Expiration Reflex, and Spasmodic panting

Types:
1. Fecal material – high mortality in spite of treatment
2. Particulate material – chief features airway obstruction and atelectasis bronchial lavage can be helpful
3. Gastric acid – classically occurs with at least 25cc gastric contents
   a. pH ≤ 2.5 leads to destruction of surfactant producing cells and endothelium resulting in atelectasis, pneumonitis, and ARDS. Arterial hypoxemia most consistent manifestation
   b. Mendelson’s syndrome: pulmonary edema, pulmonary hypertension, cyanosis, and decreased pulmonary compliance. CXR mottled. Hypoxemia 2/2 right to left intrapulmonary shunt.

Risk Factors:
1. Delayed gastric emptying – diabetics, pain, bowel obstruction, and prior opioid administration.
2. Increased gastric volume – obesity, pregnancy, trauma, shock and recent food intake
3. GE sphincter disorders – hiatal hernia, acalasia, GERD and esophageal tumors
4. PACU patients - decreased airway reflexes

Severity: increased with pH ≤ 2.5 and/or volume > 0.4cc/kg

Signs and Symptoms:
1. severe bronchospasm, coughing, wheezing, tachypnea, dyspnea, and cor pulmonale 2/2 pulmonary hypertension.
2. Arterial hypoxemia not relieved by O2 therapy in severe aspiration.
3. Radiographic evidence most often seen in the right lower lobe and can be delayed 6-12 hours

Treatment (anesthetized patient unprotected airway):
1. place patient in Trendelenburg and turn head to side
2. suction upper airway
3. consider endotracheal intubation suction tube prior to placing on positive pressure to avoid pushing contents to distal airways
4. consider bronchoscopy to suction significant aspiration or removal of foreign body. Avoid normal saline irrigation because it can further aggravate damage.
5. don’t start antibiotics, or steroids, or obtain sputum cultures
6. monitor patient with pulse oximetry with ventilatory and supplemental oxygen as needed.

Prevention:
1. Minimize PO intake
   a. Clear liquids: 2 hours
   b. Breast milk: 4 hours
   c. Formula, Full liquids, or Light meal: 6 hours
   d. Heavy meal: 8 hours
2. Increase gastric emptying
   a. Prokinetics
      • Metocloramide: speeds emptying, increases LES pressure, and decreases pyloric pressure. Contraindicated in pheochromocytoma can cause catecholamine release.
3. Reduce gastric volume and acidity
   a. NG tube
   b. Nonparticulate antacid – sodium citrate
   c. H2-receptor blockers
      • Cimetidine: complications include bradycardia, heart block, increased airway resistance, confusion, seizure, retards metabolism and excretion of other drugs
      • Ranitidine: longer acting, more potent, fewer side effects, decrease dose in renal failure
   d. Proton pump inhibitors
   e. 5-HT3 receptor blockers: can prolong Q-T interval
4. Airway management and protection
   a. Cricoid pressure ???
   b. Cuffed endotracheal intubation
   c. Combitube
   d. ProSeal LMA
Delayed emergence is a serious event, but can be approached through a simple framework. Causes can be divided into 4 broad categories: pharmacologic, physiologic, metabolic, & neurologic. 2 useful algorithms to approaching delayed emergence are presented below, along with a differential diagnosis.

Algorithm 1 (adapted from Black et al, J Neurosurg Anesthesiol. 1998 Jan;10(1):10-5.)

1) Review administered drugs: pharmacologic causes
   a. residual anesthetics: volatile agent, propofol, ketamine, barbiturates, dexmedetomidine
      i. is the source of anesthetic agent off?
         if so, when did you turn off the agent?
      ii. account for half-life / context sensitive half-life, additive effects of polypharmacy, MAC modifiers of the specific pt, end-tidal volatile percentage – is the pt's level of consciousness appropriate?
   b. excess narcotic
      i. was the amount given appropriate for the anticipated level of post-op pain?
   c. excess benzodiazepines
      i. factor in amount of pre-sedation and duration of the procedure
   d. residual neuromuscular blockade
      i. when was the last time and dose of neuromuscular blocker?
      ii. how many twitches does the patient have, and what is the pattern of twitches and response to tetanic stimulation?
      iii. did the pt ever have return of twitches after the original dose, and is it possible they have an enzyme deficiency or medical condition precluding timely return of muscle strength (pseudo-cholinesterase deficiency, Guillain Barre)?
   e. acute alcohol or other illicit drugs, CNS depressants

2) Reverse if possible & prudent: reversal agents
   a. physostigmine 1.25mg IV can be considered for reversal of volatile agent (or scopolamine)
   b. naloxone IV can be given in 40 mcg (or smaller) boluses q 2 minutes (up to 0.2mg) to reverse opioids
      i. caution in chronic opioid users, can precipitate acute withdrawal, severe pain, autonomic disturbances, pulmonary edema
   c. flumazenil 0.2 mg IV q 1 minute (up to 1 mg) to reverse benzodiazepines
   d. neostigmine 70mcg/kg with glycopyrrolate 0.2 mg per 1 mg neostigmine to reverse neuromuscular blockade
      i. only reverse in the presence of at least 1 twitch
   e. continued observation: some scenarios (reversal is not possible or not advised, airway protection in doubt) warrant observation in the OR or taking the pt intubated to the PACU

3) Vital signs: physiologic causes
   a. hypotension
   b. hypoxia
   c. hypothermia or hyperthermia

4) Electrolytes, ABG: metabolic causes
   a. hypercarbia
   b. hypoxemia
   c. acidosis
   d. hypoglycemia / hyperglycemia
   e. hyponatremia
   f. underlying metabolic disorder
      i. does the pt have liver disease, uremia, severe thyroid derangements?

5) Neurologic exam, consultation, imaging: neurologic causes
Checklists for Management of Intraoperative Problems
Created by the UCSD Class of 2014
Edited by Geoffrey Langham and Joel Spencer
1. Hypercarbia
2. Hypoxemia
3. Elevated Peak Airway Pressure
4. Tachycardia
5. Bradycardia
6. Hypertension
7. Hypotension
8. Ectopy
9. Delayed Emergence
10. Hypothermia
11. Acidosis
12. Low Urine Output
1. Hypercarbia
Blake Fowler, MD

INITIAL STEPS:
- Basic survey of patient’s ABCs to rule out obvious problems
- Survey of the surgical field for unclamping or release of a tourniquet
- Quick check of muscle tone and vital signs to rule out MH

MANAGEMENT OF HYPERCARBIA
- Ensure adequate oxygenation and ventilation
  - Check airway (kinks in ETT, LMA seated well, etc.)
  - Check circuit (ventilate manually, any obstruction?)
  - Check minute ventilation (recheck ventilator settings or spirometry if available)
  - Consider deepening anesthesia +/- NMB which can help with difficult ventilation
- Check FiO2
  - Check valves (e.g. expiratory valve stuck open)
  - Check if CO2 absorber exhausted
  - Check if fresh gas flow inadequate
- Blood gases to confirm capnography
- Consider secondary causes, especially those requiring specific Tx (MH, thyrotoxicosis, etc.)
- Consider CXR or FOB to evaluate possible intrathoracic causes
- Treat complications of hypercapnia
  - Acidosis
  - Hypertension
  - Tachycardia/arrhythmias
  - Pulmonary hypertension
  - CO2 narcosis
  - Right shift of O2-Hb curve
- In some cases, the hypercarbia may prompt a change or cancellation of the procedure (e.g. patient with refractory hypercapnia during robotic prostate – completely valid to convert to open or abort the procedure)

CAUSES OF HYPERCAPNIA: remember PaCO2 = VCO2 / VA
- Decreased CO2 excretion (e.g. inadequate ventilation)
- Increased CO2 production
- Increased CO2 delivery to lungs

Differential Dx for DECREASED CO2 EXCRETION/INADEQUATE VENTILATION
- Inadequate ventilator settings (low minute ventilation)
- Neuromuscular hypoventilation (NMBs, high spinal, phrenic nerve paralysis)
- Respiratory depressant drugs (opioids, benzos, barbituates, volatile anesthetics)
- Central depression of medullary respiratory center (tumor, ischemia, edema)
- Altered respiratory mechanics (decreased compliance due to pneumoperitoneum, surgical retraction, obesity, Trendelenburg)
- Partial airway obstruction (kinked ETT, bronchospasm, COPD, pneumothorax)
- One lung ventilation (especially in pts with preexisting pulmonary pathology)

Differential Dx for INCREASED CO2 PRODUCTION
- Increased temperature (including MH, sepsis)
- Hyperthyroidism (including thyrotoxicosis)
- Exogenous (CO2 pneumoperitoneum during laparoscopy)
- Release of tourniquet
- NaHCO3 administration
- Shivering
- Convulsions
- Parenteral nutrition
- Compensation for metabolic alkalosis
Differential Dx for INCREASED CO2 DELIVERY TO LUNGS

- Rebreathing exhaled gases (exhausted CO2 absorber, increased circuit dead space, expiratory valve malfunction, inadequate fresh gas flows in non-rebreathing systems)
- Increased cardiac output
- Right → Left shunt
2. Hypoxemia  

Christopher Asher, MD

Initial Steps for acute/severe hypoxemia:
- Go on 100% Oxygen
- Check other vital signs: HR, BP, Rate+Rhythm, Capnograph, PIP
- Hand-ventilate to assess for lung compliance
- Auscultate lungs bilaterally (wheeze? No sounds?) Look for equal chest rise.
- Suction ETT
- Attempt recruitment maneuvers (sigh breath and PEEP)
- Call for HELP if etiology not easily discerned. Notify surgical team.

Further Work-Up: based on differential diagnosis
- Bronchoscopy for direct visualization of airway: use a large scope for good suction/irrigation
- Arterial blood gas sample
- Chest Xray (can also use C-arm/fluoro if in room)

Comprehensive differential diagnosis:
- Low FiO2:
  - Altitude
  - Hypoxic gas mixture delivery (wrong supply, defective mechanical component, leak downstream of control, inter gas used?)
  - O2 source exhausted
  - Nitrous diffusion hypoxia (at end of case)
- Hypoventilation:
  - Drugs (opioids, benzo, NMB, volatile anesthetics)
  - Neuromuscular disease
  - Obstruction (OSA, upper airway compression)
  - Inadequate ventilatory settings (rare)
- High V/Q Mismatch [dead space]:
  - COPD
  - Embolus (fat, air, clot, amniotic fluid)
  - Interstitial lung disease
  - Decreased CO
  - Anemia
- Shunt (Low V/Q mismatch):
  - Atelectasis (most common)
  - PNA
  - Mucus plugging
  - Mainstem intubation
  - Aspiration
  - Foreign object
  - Pulmonary edema
  - Anaphylaxis
  - ASD/VSD/PDA
- Diffusion Impairment: fibrosis, ephysema, pneumonectomy
- OTHERS/“artifacts”:
  - Poor waveform (probe malposition, cold extremity, light in room, cautery, extremity movement/evoked potentials)
  - Poor perfusion (decreased CO, anemia, cold extremity, BP cuff inflation, tourniquet still in place)
  - Dyes/pigments (indigo carmine, methylene blue, methemoglobinemia)
3. Elevated Peak Airway Pressure  Tom Griffiths, MD

Management of High Peak Airway Pressures:

- Initial Management:
  - Hand Ventilate on 100%.
  - ETCO2
  - Check Depth of tube
  - Auscultate bilaterally (wheezing/bronchospasm).
  - Assess depth of anesthesia, twitches, biting on tube.
  - Inspect surgical site.

- Unresolved from Initial Management:
  - Sweep from valves, circuit, ETT, Lungs, Chest wall
  - Attain plateau pressure to help guide differential
  - Suction ETT looking for secretions, blood, fluid.
  - Call for Bronch cart to physically look in tracheal tree.
  - OKR.
  - AutoPEEP?
  - Scavenging circuit occlusion

Never fear calling for help.

DDx and Management reminders:

Valves/Circuit/ETT—check valves are moving, circuit/ETT not kinked or obstructed.
Biting Tube—Neuromuscular blockade vs. Deepen anesthetic. Bite block.
Endobronchial Intubation—Lips to Carina (Male – 24-25cm, Women, 22-23cm head flexion can increase depth by 2cm)
Bronchosponis/Wheeze/Asthma/Smokers/Burns, Prolonged intubation—Deepen anesthetic, Albuterol, Epinephrine 5-10mcg pushes.
Parenchymal—Volume overloaded, new Heart failure—active ischemia?, baseline COPD, Copious secretions.
PTX—Central line placed, Diaphragmatic injury from surgery, trauma, popped bleb.
Chest wall—Restriction on chest wall excursion (med student/surgeon leaning). Burn, steep T-bug, obese, pregnant, autoPEEP.
4. Tachycardia

Seth Herway, MD

Initial steps:
- Confirm airway/breathing.
- Discern if it is a stable or unstable tachycardia.
- If unstable, follow appropriate ACLS guidelines.
- If stable, check EKG to rule out changes indicative of ischemia. Then ensure adequate anesthesia as you proceed along a differential diagnosis to determine the cause.

Differential Diagnosis
- Inadequate depth of anesthesia
  - Empty vaporizer, dislodged IV during TIVA, tourniquet time, opioids
- Inadequate analgesia
  - Somatic pain (incision, fractures, etc)
  - Visceral pain (distended bladder)
  - Sympathetic pain (tourniquet pain)
- Hypovolemia/hypotension
  - Check PPV, UOP, fluid responsiveness
- Hypoxemia/Hypercarbia
  - ABG, SpO2, FiO2, ETCO2, ABG, ETT depth
- Sepsis
  - Evaluate for SIRS criteria and potential source (drained abscess or release of infected surgical site)
- Hyperthermia
  - Determine patient temperature and assess for iatrogenic causes (warmer on too high, HIPEC, etc.)
- Drugs / Medications
  - What patient takes: missing BBlocker dose etc.
  - What you have given: pancuronium, desflurane, glycopyrolate, atropine, ephedrine, dopamine, epi etc.
- Myocardial ischemia
- Pacemaker-mediated tachycardia (pacer sensing wrong)
- Endocrine Causes
  - Thyroid storm or thyrotoxicosis
  - Pheochromocytoma
  - Carcinoid syndrome
- Hypermetabolic state
  - Post-trauma patient (weeks to days)
  - Burns
  - Neuroleptic malignant syndrome
  - Malignant hyperthermia
- Catastrophic events
  - Tension PTX, tamponade, embolism
    - Check breath sounds, EtCO2, physical exam, CXR, TEE

Treatments with medications
- Volatile and IV anesthetics to deepen anesthesia.
- Opioids for analgesia.
- Crystalloid, colloids, and blood products for hypovolemia
- β-adrenergic blocking agents (e.g. metoprolol 1-5 mg IV, esmolol 5-10 mg IV, labetalol 5-10 mg IV if accompanying HTN). Consider whether the pt needs the tachycardia to maintain hemodynamic stability before administering. Also consider whether or not the pt can tolerate the tachycardia.
- Be wary of treating medication induced tachycardia (due to epinephrine etc.) with a BBlocker as this will lead to unopposed alpha stimulation and possible circulatory collapse.
Further postoperative tests and work up to consider

- Workup for sepsis: vitals, CBC w diff, cultures. If sepsis seems likely, insures that pt has adequate access and monitoring (a-line) and institute broad spectrum antibiotics.
- Check for myocardial damage: EKG, serial cardiac enzymes, changes on ECHO, associated symptoms.
- Thyroid function tests
- Urine collection for catecholamine metabolites such norepinephrine, epinephrine, and dopamine
- Check/interrogate pacemaker
5. Bradycardia

Nathalie Hernandez, MD

Initial management
- Ensure adequate oxygenation and ventilation
- Determine whether patient is stable or unstable
  - Unstable if there is hypotension, weak or absent pulse
- If stable:
  - Give glyco 0.2 mg or ephedrine 5-10 mg
- If unstable:
  - Alert surgeons if the cause is surgical manipulation
  - Give atropine 0.5 mg or epi 50 mcg
  - Transcutaneous or transvenous pacing for severe or drug refractory

Further work-up
- ECG – determine whether SB or heart block
  - SB is less ominous and usually responds to medical treatment
  - Mobitz type II or 3rd degree heart block can be a sign intrinsic heart disease, will likely need transcutaneous or transvenous pacing
- TEE/TTE – structural or ischemic heart disease may lead to conduction abnormalities, therefore you may want to r/o common causes
  - MI – would see regional wall motion abnormalities, thickened myocardium in area of ischemia, diastolic dysfunction
  - Heart failure – enlarged RV or LV, low global ventricular function

Differential diagnosis for acute bradycardia
- Hypoxia
- Intrinsic cardiac dz:
  - Sick sinus syndrome/age-related sinus degeneration (most common)
  - Underlying parasympathetic state ± deep anesthesia (esp. athletes, young women)
  - Myocardial ischemia or infarction, esp. of RCA
  - Heart failure
- Drugs
  - Phenylephrine (reflex bradycardia)
  - Narcotics (esp. fentanyl derivatives)
  - β-blockers
  - Calcium channel blockers
  - α2 agonists (Dexmedetomidine)
  - Anticholinesterases
  - Succinylcholine (esp. in peds or redosing)
  - Digoxin
  - Always consider drug swap!
- Reflexes and increased vagal tone
  - Visceral traction
  - Laparoscopic insufflation
  - Brainstem manipulation
  - Direct stimulation of vagal nerve or carotid sinus
  - Valsalva maneuver
  - Vaso-vagal reaction
  - Oculo-cardiac reflex
  - Bezold-Jarisch reflex
- Sympathetic Blockade
  - Local anesthetic block with spinal or epidural (via block of cardiac accelerator fibers at T1-4 that occur with high spinal or epidural)
- Elevated ICP
6. Hypertension

David Bui, MD

Initial steps:
- Quickly confirm accurate measurement (re-cycle NIBP, correct arterial line transducer height)
- ABC. What is the overall clinical picture and vitals?
- Ensure adequate depth of anesthesia/analgesia
- Correct hypoxemia and hypercarbia (oxygenation and ventilation)
- Temporize with fast-on, fast-off drugs
- Diagnose the problem and ultimately treat the underlying cause

Differential Diagnosis

- Inadequate depth of anesthesia
  - Unexpectedly high MAC, preoperative anxiety, empty vaporizer, dislodged IV during TIVA, tourniquet time, opioids
- Inadequate analgesia (associated with tachycardia unless on beta blockers)
  - Somatic pain (incision, fractures, etc)
  - Visceral pain (distended bladder)
  - Sympathetic pain (tourniquet pain)
- Hypoxemia/Hypercarbia
  - ABC
  - SpO2, FiO2, ETCO2, ABG, ETT depth
- Primary hypertension
  - Essential hypertension is the most common cause of intraop hypertension
  - Patient not taking their BP meds
  - Rebound hypertension from discontinuing meds (clonidine, other BP meds)
- Drugs / Medications
  - What patient took: MAOIs, cocaine, methamphetamines
  - What you gave (wrong drug, wrong dilution): vaspressors, inotropes, ketamine, etc
- Measurement error (i.e. small BP cuff)
- Iatrogenic causes from surgeons
  - Aortic cross clamp with associated increased in SVR
  - Injection of local anesthetics with epinephrine intravascularly
  - Soaked gauze with epinephrine, cocaine, or phenylephrine
- Neurologic
  - Elevated ICP (Cushing’s triad: HTN, bradycardia, irregular respirations)
  - Autonomic dysreflexia (higher incidence with spinal lesions above T6)
- Endocrine
  - Family history of MEN syndrome?
  - Thyroid storm or thyrotoxicosis
  - Pheochromocytoma
  - Hyperaldosteronism (Conn’s syndrome)
  - Carcinoid syndrome
- Hypermetabolic state
  - Neuroleptic malignant syndrome
  - Malignant hyperthermia
- Pre-eclampsia
- Hypervolemia

Treatments with medications

- Volatile anesthetics
- Opioids
- Propofol
- α/β adrenergic blocking agents (e.g. labetalol 5-10 mg IV)
- β-adrenergic blocking agents (e.g. metoprolol 1-5 mg IV, esmolol 5-30 mg IV)
- Vasodilators (e.g. hydralazine 2.5-5 mg IV, NTG gtt at 30-50 ug/min IV, Nitroprusside gtt at 0.2-0.5 mcg/kg/min)
- Calcium channel blockers (verapamil 2.5-5 mg IV, diltiazem 5-10 mg IV)
Further postoperative tests and work up to consider

- Check for myocardial damage: EKG, serial cardiac enzymes, associated symptoms.
- Thyroid function tests
- Urine collection for catecholamine metabolites such as norepinephrine, epinephrine, and dopamine
- Associated ICP elevation: head CT, neurosurgery consult, maintain cerebral perfusion pressure, elevate head of bed, prevent hypoxemia/hypercarbia, promote venous drainage, etc
- If >20wk pregnant, check for proteinuria, platelet count, LFTs, etc for hypertensive disorders of pregnancy
7. Hypotension

Initial Steps

- Confirm hypotension
  - cycle BP cuff, flush arterial line, check a-line position
  - check surgical field for hemorrhage, insufflation pressures, retraction, position change
  - evaluate EKG on diagnostic mode for rhythm or ST changes
- Begin fluid resuscitation
- Place patient in Trendelenberg position
- Communicate with surgical team

Management

- Place additional monitors/access such as another IV, A-line, PAC/CVP
- Order blood, FFP, platelets as needed
- Check ECG, CXR as indicated
- Administer vasopressor agents appropriate for the setting
  - phenylephrine
  - ephedrine
  - vasopressin
  - epinephrine/norepinephrine
  - methylene blue
- Administer vagolytic or chronotropic agents as appropriate
  - atropine
  - glycopyrolate
  - beta-1 agonists
  - evaluate the patient’s pacemaker function, consider magnet
- TEE indicated for workup of hypotension that remains unexplained after above monitors/treatments done

Differential Diagnosis

- Preload Problems:
  - Hypovolemia due to hemorrhage, diuresis, bowel prep and NPO status (most common)
  - Relative hypovolemia due to: abdominal compartment syndrome or insufflation, increased intrathoracic pressure due to insufflation or pneumothorax, surgical IVC compression, reverse Trendelenberg positioning, cardiac tamponade, anaphylaxis
- Contractility Problems:
  - Cardiac ischemia
  - Beta-blockers, calcium channel blockers, high inhalation anesthetic concentration
  - Previously unrealized cardiomyopathy or myocarditis, obstructive cardiomyopathy
- Afterload Problems
  - Drug effects: high inhaled anesthetic concentration, propofol infusion (these two most common), pre-operative angiotensin I or II inhibitors or renin inhibitors, alpha-1 antagonists, protamine, anaphylaxis
  - Sympathectomy: due to neuraxial techniques, analgesics or surgical neurolysis
  - Reperfusion vasoplegia after tourniquet release, aortic crossclamp release or discontinuation of cardio-pulmonary bypass
- Rate and Rhythm Problems
  - New atrial fibrillation in a patient with valvular heart disease (stenotic lesions) or severe diastolic dysfunction
  - Symptomatic brady- or tachy-arrhythmias due to high dose analgesics, abdominal insufflation, or intrinsic pacemaker dysfunction
8. Ectopy

Lauren Knecht, MD

Arrhythmias during and after surgery are most common in patients with structural heart disease and most commonly associated with a transient insult such as central venous cannulation and wire/PA catheter placement, hypoxemia, ischemia, catecholamine excess, or electrolyte abnormality

**Initial Steps:**
- Identify if stable or unstable, if unstable, follow ACLS/ART guidelines
- Check rate/rhythm – slow/fast, irregular/regular
- Evaluate P wave
- Evaluate QRS complex
- Evaluate causes
- Decide on treatment, if needed

**Atrial Premature Beats (APBs/PACs)** - 10% intraop arrhythmias, ectopic beats from the atria, not SA node

<table>
<thead>
<tr>
<th>Rate: &lt;100 bpm, Rhythm: irregular</th>
<th>![Atrial Premature Beats Diagram]</th>
</tr>
</thead>
<tbody>
<tr>
<td>P wave may be lost in preceding T wave, diff morphology, QRS normal, T wave normal</td>
<td><strong>Causes:</strong> Atrial stretch, as in severe COPD, OSA, and CHF, ischemia, vagal stim, hypothyroid, meds (B-blockers, antiarrhythmics, digoxin)</td>
</tr>
<tr>
<td><strong>Treatment:</strong> usually not clinically relevant, but frequent APBs can lead to SVT (termed PAT)</td>
<td></td>
</tr>
</tbody>
</table>

**Junctional Rhythm** - (20% of intraop arrhythmias) AV junction has "automaticity" activity like SA node, but slower (40-60 bpm); ectopic beats from the AV junction typically arise 2/2 to SA node dysfunction (SA node bradycardia) as an "escape" mechanism, can cause a decrease in CO (15-30%)

<table>
<thead>
<tr>
<th>Rate: 40-180 bpm, Rhythm: regular</th>
<th>![Junctional Premature Beats Diagram]</th>
</tr>
</thead>
<tbody>
<tr>
<td>P/QRS: 1:1 but three varieties: P wave will be inverted if present</td>
<td><strong>a. high nodal</strong> - P wave precedes QRS, shortened PR interval (0.1s)</td>
</tr>
<tr>
<td><strong>b. mid nodal</strong> - P wave in QRS</td>
<td><strong>c. low nodal</strong> - P wave follows QRS</td>
</tr>
<tr>
<td>QRS is normal unless affected by P wave</td>
<td><strong>Causes:</strong> halogenated gases, SA node ischemia, SA node damage, high-degree AV block</td>
</tr>
<tr>
<td><strong>Treatment:</strong> usually harmless and reverts spontaneously; however, if pt unstable, tx is indicated. Atropine, ephedrine, isoproterenol can help increase activity of SA node as pacemaker; treat ischemia; <strong>pacing if high-degree AV block</strong></td>
<td></td>
</tr>
</tbody>
</table>

**Ventricular Premature Beats (VPBs or PVCs)** - common, 15% arrhythmias during anesthesia. Wide, bizarre looking QRS without P wave, typically with a compensatory pause following, and a normal sinus beat (P wave followed by normal QRS)

<table>
<thead>
<tr>
<th>Rate: usually &lt;100 bpm, Rhythm: irregular</th>
<th>![Ventricular Premature Beats Diagram]</th>
</tr>
</thead>
<tbody>
<tr>
<td>PR interval absent, retrograde P waves</td>
<td><strong>QRS</strong> is wide, &gt; 0.12s; T wave usually opposite direction of QRS with <strong>compensatory pause</strong> (note: if no pause, then the ectopy is likely APB with aberrant ventricular conduction)</td>
</tr>
<tr>
<td>Bigeminy - PVCs every other beat</td>
<td><strong>Trigeminy</strong> - PVC every 3rd beat</td>
</tr>
<tr>
<td>Couplets - PVCs in pairs</td>
<td><strong>Multifocal v unifocal</strong> - PVCs from 2 or more foci</td>
</tr>
</tbody>
</table>
as opposed to originating from one focus.
-MF and couplets have a higher risk of VF-req treatment.
**Non-sustained VT**: ≥3 consecutive PVCs, rate >120 bpm, lasting <30 secs- can be dangerous (>50% mortality in those with sig heart disease) to harmless in young, healthy people. Can be marker of sustained tachyarrhythmias and sudden cardiac death.

**Causes**: Stress, adrenaline, CAD (ischemia, MI), electrolyte (ie, low K, Mg, Phos) and blood gas abnormalities (hypoxemia, hypercapnia), drugs (digoxin), brainstem stim, trauma to heart, central venous cannulation/ PA catheterization. Serious until proven otherwise, R-on-T: when PVC falls on T wave, causing VT or VF

**Treatment**: if single, bigeminy or tri, and asymptomatic, usually do not need treatment. Treatment geared toward symptoms.

**First**: determine cause- ABG, electrolytes, ischemia? Treat any identified abnormalities. May consider treating PVCs (esp. MF and couplets) with **lidocaine** (suppresses ventricular function, bolus 1.5 mg/kg; if recurrent, a gtt can be started at 1-4 mg/min). Watch for bradycardia and hypotension with lidocaine.

**Additional medications**: esmolol, propranolol, procainamide, quinidine, atropine, verapamil, pacing.
9. Delayed Emergence

Kevin Smith, MD

Initial evaluation
- Are volatiles/drugs still present?
- Residual neuromuscular blockade
  - Check TOF
- Vital signs normal?
  - Hypoxemia, hypotension, hypo/hyperthermia
- Check labs
  - ABG, CMP, blood glucose
- Complete neurologic exam
- Specific type of surgery with known possible deficits
  - Craniotomy, aneurysm clipping/coiling, carotid, frontal lobe retraction
  - Beach chair or head-up position leading to cerebral hypotension

Additional steps
- Consider empiric reversal agents
  - Narcan, flumazenil
- Additional reversal of NMB with neostigmine
- More labs including ammonia, mag, phos, Thyroid
- CT head, EEG, neurology consult

Differential diagnosis
- Drug effects/overdose
  - Narcotics, benzos, volatiles, propofol, prolonged NMB, preoperative drug/EtOH use
  - Liver disease med interactions causing prolonged effect, Guillain-Barre or lambert Eaton syndrome leading to residual paralysis
- metabolic
  - Hyponatremia, hyperphosphatemia, hypercalcemia, hypoglycemia, hyperglycemia, acidemia, diabetic ketoacidosis, hyperosmolar coma
  - Uremia, hypothyroidism, hepatic encephalopathy
- Physiologic
  - Hypoxemia, hypotension, hypothermia, hyperthermia
- Neurologic
  - CVA, SAH, frontal lobe syndrome, herniation 2/2 increased ICP, damage to reticular activating system/midbrain, hypo-perfusion during CEA cross-clamp or secondary to head-up positioning (i.e. beach chair), ischemia 2/2 prolonged severe hypocarbia, tension pneumocephalous, status epilepticus, meningitis
10. Hypothermia

Definition: A core body temperature less than 35 degrees Celsius (95 degrees Fahrenheit). Mild hypothermia is defined as a core body temperature 1 to 2 degrees Celsius below normal core body temperature (normothermia = 36.5 to 37.5 degrees Celsius +/- 0.5 degrees Celsius). Moderate hypothermia is core body temperature equal to 35 degrees Celsius and severe hypothermia is below 35 degrees Celsius.

Sources of Heat Loss:

1) Radiation: Energy transmitted by waves transferred through a medium. Accounts for majority of heat loss in the OR. Mechanism involves vasodilation and cutaneous blood flow to body surfaces exposed to the cold OR environment. Small contribution to heat loss in the OR.

2) Evaporation: Physical process of converting liquid or solid into a vapor. Heat loss occurs from mucosal and serosal surfaces. Minor contribution to heat loss in the OR.

3) Conduction: Transfer of energy via sound, heat, nerve impulses or electricity. Heat transferred from a warm to a cool object in direct apposition. Small contribution to heat loss in the OR.

4) Convection: Transmission of heat in liquids and gases by circulation carried on by bulk movement of heated particles to a cooler area. Accounts for heat loss by conduction to a moving gas.

Monitoring:
Per the ASA standards of monitoring, “Every patient receiving anesthesia shall have temperature monitored when clinically significant changes in body temperature are intended, anticipated or suspected.”

Prevention:
- Placement of a forced air warmer (such as Bair Hugger System)
- Warm IV fluids
- Passive humidifier on the anesthesia circuit
- Increase temperature of the OR
- Warm CO₂ gases when insufflating the abdomen for laparoscopic cases
- Warm irrigation when irrigating large surfaces (such as the abdomen)

Checklist:
- Check temperature probe
- Place Bair hugger on any accessible body surface
- Place all IV fluids on a fluid warmer
- Ensure passive humidifier is on the anesthesia circuit
- Decrease fresh gas flow rate to lowest possible rate
- If temperature does not begin to improve, notify surgeons so they can adjust their methods:
  - Increase the temperature of the room (single most effective method)
  - Warm the irrigation fluid
  - If laparoscopic case, switch to warmed CO₂
- If does not improve:
  - Consider warming the anesthesia circuit

Differential Diagnosis:
- Radiation heat losses from exposed cutaneous surfaces is most likely cause (due to excessively cold OR temperature)
- Depending on case, especially in the abdomen, could be from cold irrigation fluids and/or cold insufflating gases
- High fresh gas flow rates
- If high IV fluid case, could be from large infusion of cold IV fluids
- Alternatively, blood products (pRBC’s and FFP) are refrigerated and can decrease core body temperature if large volumes are required
11. Acidosis

Erica Smith, MD

Initial Steps:
- Obtain ABG
- Acidosis = a pH <7.35; Life-threatening acidosis = a pH<7.1
- Categorize as respiratory or metabolic or mixed
- Metabolic: address underlying cause, consider increasing minute ventilation, consider bicarb administration if pH very low/pt unstable
- Respiratory: if intubated- increase minute ventilation (increase RR, Vt or both); if spont vent- assist ventilation, consider intubation/mech vent

Consequences of acidosis:
- pH<7.1 → decreased myocardial contractility, decreased myocardial responsiveness to catecholamines

Differential Diagnosis:
- Metabolic acidosis: elevated vs normal anion gap (consider Chem panel) recall anion gap= Na⁺-Cl⁻-HCO₃⁻
  - Elevated anion gap (>11mEq/L):
    - drugs- methanol, EtOH, salicylate
    - ketoacidosis- ie DKA
    - lactic acidosis (tissue hypoperfusion)
    - renal failure
    - liver failure/cirrhosis
  - Normal anion gap (3-11mEq/L)
    - iatrogenic- too much Cl⁻ admin (using NS)
    - bicarb loss - GI (diarrhea, ileostomy) vs renal (RTA)
    - drugs- ie acetazolamide
- Respiratory acidosis: low pH, elevated PaCO₂ (see Hypercarbia checklist)
  - Too much CO₂ production
    - Insufflation
    - tourniquet release
    - hypermetabolic syndrome- sepsis, MH
  - Too little CO₂ elimination (hypoventilation)
    - inadequate minute ventilation
    - resp depression- nars, sedatives, CVA
    - resp muscle weakness – spinal cord injury, Guillan-Barre, residual NMBD
    - chest wall disorder- flail chest, PTX
    - lung parenchyma disorder- ARDS, PNA, COPD, CHF, aspiration
    - abdominal distension- laparoscopic surgery, ascites, obesity

Recall
- Metabolic compensation for resp acidosis:
  - Acute- ΔpHa=.008 x ΔPaCO₂
  - Chronic- ΔpHa= 0.003x ΔPaCO₂
- Ventilatory compensation for metabolic acidosis:
  - Expected PaCO₂ = 1.5 x HCO₃⁻ + 8 (±2)

Guidelines for Bicarb administration:
- Sodium bicarb dose= Body weight (kg) x deviation of plasma bicarb from 24 mEq/L x Extracellular fluid volume as fraction of body mass (0.3). Administer half of calculated dose and repeat ABG to determine effect of tx.
**12. Low Urine Output**

Sara Meitzen, MD

(Oliguria - urine output less than 0.5mL/kg/hr x 6 hrs)

**Initial Steps**

- **ABCs** - Have adequate oxygenation and MAPs been consistently maintained? Vitals wnl?
- Fluid bolus if hypovolemia suspected
- Assess acid/base status, CVP measurement, systolic variation on an arterial line tracing (if present), or stroke volume variation
- Flush the foley
- Examine urine in foley for sediment, gross blood, abnormal color
- If oligura persists despite an adequate volume status, urine output can be augmented with Furosemide, Dopamine infusion, 1-3 mcg/kg/min, Mannitol, Fenoldapam, 0.1-0.4 mcg/kg/min (Keep in mind these have not been shown to alter outcome)
- Consider intraop diuretics for a patient on chronic diuresis at home

**DDx**

1. **Prerenal**

   - **Hypovolemia** - hemorrhage, over diuresis, dehydration, GI & insensible losses
   - **Decreased renal blood flow**
     - CHF or other low cardiac output states (abnormal rate, dysrhythmia, decreased contractility)
     - Excess renal vascular resistance - high SVR, dissection, stenosis, aortic or renal artery clamping (ARF possible even with infrarenal clamping, possibly 2/2 renal artery vasospasm), thromboembolic phenomena (i.e. aortic or renal artery clamping)
     - Pressor choice - alpha agonists generally decrease RBF
   - Sepsis
     - Abdominal compartment syndrome (see below)
   - Hyperventilation with positive pressure ventilation
   - Redistribution of blood flow with anesthesia

   - **Altered intrarenal hemodynamics**
     - Sepsis
     - Hypercalcemia
     - Cirrhosis/Hepatorenal syndrome
     - Abdominal compartment syndrome (with intraabdominal pressures > 15 mmHg, i.e. 2/2 massive fluid resuscitation, circumferential burns to abdominal area, ascites, laparoscopic, surgery, pneumoperitoneum, etc)

2. **Renal** (intrinsic pathology - ischemic, toxic, immune mediated)

   - ATN (follows renal hypoperfusion)
   - Drugs - Abx (aminoglycosides), chemo agents (cyclosporine, prograf), contrast dyes
   - Free hemoglobin/myoglobin (2/2 hemolysis, transfusion reaction, crush injury/ rhabdomyolysis/MH/hyperthermia/statins etc)
   - AIN (hypersensitivity rxn to certain meds)
   - Acute glomerulonephritis
   - Tumor lysis syndrome (uric acid nephropathy) - following chemo for lymphoma or leukemia but can also occur spontaneously
   - Release of ADH/SIADH - Multiple etiologies; a natural response to perioperative stressors, following head trauma, neurosurg patients, neuroendocrine neoplasms (bronchogenic CA)

3. **Postrenal**

   - Foley catheter dysfunction (kinked, clotted, or malpositioned)
   - Anatomic obstruction
     - Bladder outlet (BPH, pelvic tumor)
     - Ureteral (tumor, stone, sticture, edema, surgical ligation, blood clot)
   - Urinary retention with disruption of parasympathetic innervation bladder spinals /epidurals), anticholinergic meds, opioids
Further Workup and Labs

- Fluid challenge
- ABG - volume and acid/base status, Hct, lytes
- BMP
- CBC
- Urinalysis - Prerenal: Uosm > 500, UNa < 10, FeNa < 1%, Bun/Cr > 20
  - Intrinsic: Uosm < 350, UNa > 20, FeNa > 2%, Bun/Cr < 15
  - Postrenal: Uosm < 350, UNa > 40, FeNa > 4%, Bun/Cr > 15
- Consider further invasive monitoring (CVP, swan, TEE, Aline)
- Other labs to consider - Blood CK level, Urine myoglobin/hemoglobin, serum LDH, serum haptoglobin, urine microscopy, urine eosinophils
- Intraabdominal pressure - measured indirectly with intragastric, intracolonic, intravesical, or IVC catheters
- Consider bladder scan - Full? --> postrenal etiology. Empty? --> Prerenal vs possibility of bladder rupture?
- Renal US
a. new ischemic event
b. cerebral hemorrhage
c. seizures or post-ictal state
d. increased ICP or pre-existing obtundation

Algorithm 2 (adapted from Stanford Ether online resources)

- Confirm that all anesthetic agents (inhalational/intravenous) are off.
- Check for residual muscular paralysis with train of four monitor and reverse neuromuscular blockade as appropriate.
- Consider narcotic reversal
- Consider inhalational anesthetic reversal with physostigmine
- Consider benzodiazepine reversal with flumazenil
- Check blood glucose level and treat hypo or hyperglycemia.
- Check arterial blood gas and electrolytes
- Rule out CO2 narcosis from hypercarbia
- Rule out hypo or hypernatremia
- Check patient’s temperature and actively warm if less than 34 C.
- Perform neurological exam if possible: exam pupils, symmetric motor movement, presence or absence of gag/cough.
- Obtain stat head CT scan and consult neurology/neurosurgery to rule out possible cerebral vascular accident (CVA).
- If residual sedation/coma persists despite evaluating all the possible causes, monitor the patient in the ICU with neurology follow up and frequent neurological exams. Repeat the CT scan in 6-8 hours if no improvement.