UCSD Anesthesia
Manual of Intraoperative Events

July 2012
1st Edition

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Introduction:

1st Month Goals and Objectives:

Intraoperative Events:

1) Hypoxemia
2) High Peak Airway Pressure
3) Hypercarbia
4) Hypotension
5) Hypertension
6) Bradycardia
7) Tachycardia
8) ST Depression
9) Hypothermia
10) Low Urine Output
11) Aspiration
12) Failure to Awaken
INTRODUCTION

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 than 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 you 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
Hypoxemia

First things first: your *initial* response to low $O_2$ saturation, $\text{PaO}_2$, or blue patient

- Patient on 100% $F_\text{O}_2$, look at all other vitals
- Check the airway
  - Confirm ETT placement by verifying $\text{EtCO}_2$, 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 $F_\text{O}_2$
   a. Altitude
   b. Hypoxic $F_\text{O}_2$ gas mixture
   c. **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
   a. Drugs (opioids, BDZs, barbituates)
   b. Neuromuscular diseases
   c. Obstruction (OSA, upper airway compression)
   d. **In OR:** check circuit leaks, low TV/RR or MV, residual NMB, high $\text{ETCO}_2$, high PIP, kinked/obstructed ETT, poor chest rise

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

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

5) Diffusion Impairment
   a. Increased diffusion pathway (pulmonary edema, fibrosis)
   b. Decreased surface area (emphysema, pneumonectomy)
   c. Usually chronic

6) Artifact
   a. **In OR:** consider this LAST, if all else okay
   b. Poor waveform: probe malposition, cold extremity, light interference, cautery, dyes (methylene blue, indigo carmine, blue nail polish), extremity movement (vibration, evoked potentials)
   c. Poor perfusion: cold extremity, BP cuff inflation, tourniquet still from trying IV start
Alveolar Gas Equation
\[ P_{A\text{O}_2} = F_i\text{O}_2(P_{atm} - P_{H_2O}) - (P_{\text{aCO}_2} / 0.8) \]
\[ \approx 0.21(760-47) - (40/0.8) \]
\[ \approx 100 \text{ mmHg on RA} \]
\[ \approx 660 \text{ mmHg on 100% F}_i\text{O}_2 \]

Alveolar-arterial (A-a) Gradient
\[ P_{(A-a)\text{O}_2} = P_{A\text{O}_2} - P_{a\text{O}_2} \]
Normal A-a gradient
\[ <10\text{mmHg (Fio2 g=0.21)} \]
\[ < 60 \text{ mm Hg (FiO2 = 1.00)} \]
\[ <(age/4)+4 \]

Arterial O\(_2\) Content
\[ C_{a\text{O}_2} = O_2-Hb + \text{Dissolved O}_2 = (Hb x 1.36 x S\text{aO}_2/100) + (P_{a\text{O}_2} x 0.003) \]
\[ = (15 x 1.36 x 100\%) + 100 x 0.003) \approx 20 \text{ cc O}_2/\text{dl} \]

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

O\(_2\) Delivery
\[ D\text{O}_2 = CO x C_{a\text{O}_2} = 5 \text{ L/min x } 20 \text{ cc O}_2/\text{dl} = 1 \text{ L O}_2/\text{min} \]

O\(_2\) Consumption (Fick Equation)
\[ V\text{O}_2 = CO x (C_{a\text{O}_2} - C_{v\text{O}_2}) = 5 \text{ L/min x } 5 \text{ cc O}_2/\text{dl} = 250 \text{ cc O}_2/\text{min} \]

O\(_2\) Extraction Ratio
\[ E\text{RO}_2 = (V\text{O}_2 / D\text{O}_2) x 100 = 250 / 1000 \]
\[ \approx 25\% \text{ (normal 22-30\%)} \]

Bohr Equation (Dead Space Fraction)
\[ V_D/V_T = (P_{\text{aCO}_2} - P_{E\text{CO}_2})/P_{\text{aCO}_2} \]
\[ \approx \text{normal 33\%} \]

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\(_2\) sat of 75, PaO\(_2\) of 75
P50 (the PaO\(_2\) where hgb is 50\% saturated) is ~ 27 mmHg
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).

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 ventilaton
    - 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
Hypotension

\[ BP = CO \times SVR \]

HR \times 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
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), laparoscopic 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\textsuperscript{st} Degree AVB: Prolonged PR interval >200ms with every p-wave conducted and followed by a QRS
  - 2\textsuperscript{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\textsuperscript{rd} degree AVB
  - 3\textsuperscript{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\textsuperscript{st} Degree: Does not require treatment
    - 2\textsuperscript{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\textsuperscript{rd} Degree: Pacing

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

Stable or Unstable?

Sinus Tachycardia
HR 100-160 bpm

Dysrhythmia

Polymorphic VT

Unstable

Cardioversion

Sinus Tachycardia

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

Supraventricular Tachycardia

Junctional/Nodal
-frequently related to inhaled anesthetics
-reduce depth
-increase intra-vascular volume

Ventricular Tachycardia

Monomorphic VT
-treat with meds
-Amiodarone 150 mg
-Lidocaine 100 mg
-Procainamide 100mg

Extensive differential
-Catecholamine excess
-Pain
-“Light” anesthesia
-Hypercapnea
-Hypoxemia
-Hypotension
-Hypovolemia
-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

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 hypovolemia and administer a 500-cc fluid bolus

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

A-Fib/A-Flutter
-Rate control
-Beta-blockade
-Metoprolol 2.5-5mg
-Esmolol 5-10 mg
-Calculator Channel Blocker
-Verapamil 2.5-5 mg
-Diltiazem 10-20 mg
-Rhythm Control
-Amiodarone – 150 mg
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>
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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>
</tr>
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Call for help
- Have circulator call attending
- Notify surgeons

Airway
- Verify etCO₂

Breathing
- 100% O₂

Circulation
- Decrease O₂ demand
  - Treat tachycardia
  - esmolol vs metop
  - Treat SBP, if extreme é
  - 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
Hypothermia

**Definition:**
Core body temperature <35°C

**Mechanism:**

- **Redistribution:** core → 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)

**NMB:**
- Opioids (Demerol)
- Clonidine
- Chlorpromazine
- Warm via CPB

**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>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>Myocardial ischemia</td>
</tr>
<tr>
<td></td>
<td>Hypertension</td>
</tr>
<tr>
<td></td>
<td>Tachycardia</td>
</tr>
<tr>
<td></td>
<td>Deep venous thrombosis</td>
</tr>
<tr>
<td>Coagulation</td>
<td>Platelet activation</td>
</tr>
<tr>
<td></td>
<td>Coagulopathy</td>
</tr>
<tr>
<td>Immune</td>
<td>Increased incidence of infection on surgical wound</td>
</tr>
<tr>
<td>Hydroelectrolytic changes</td>
<td>Hypokalemia</td>
</tr>
<tr>
<td></td>
<td>Hypomagnesemia</td>
</tr>
<tr>
<td></td>
<td>Hypophosphatemia</td>
</tr>
<tr>
<td>Endocrine-metabolic changes</td>
<td>↓ Steroids</td>
</tr>
<tr>
<td></td>
<td>↑ Insulin</td>
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<td></td>
<td>↑ Peripheral resistance to insulin</td>
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<td></td>
<td>↑ TSH → ↑ thyroxin</td>
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<td></td>
<td>Hyperglycemia</td>
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<td>Hypoglycemia</td>
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</tbody>
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*TSH = thyroid stimulating hormone*
Hyperthermia

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

Causes: Hypermetabolic vs Other
- Infection/sepsis vs excessive heating (rare)
- Thyroid Storm vs allergic rxn (blood mismatch)
- Pheochromocytoma vs Neuroleptic Malignant Syndrome
- Malignant Hyperthermia vs anticholinergics (sweating inhibited)
- Serotonin syndrome (MAOIs, 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.
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

**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
      • Metoclopramide: 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
Failure to Awaken (Delayed Emergence)

Sameer Shah, MD

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?
      ii. was the pt opioid naïve or tolerant?
   c. excess benzodiazepines
      i. factor in amount of pre-sedation and duration of the procedure
      ii. was the pt benzo naïve or tolerant?
   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 (pseudocholinesterase 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
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.