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INTRODUCTION TO THE CA-1 TUTORIAL MONTH

We want to welcome you as the newest members of the Department of Anesthesia at Stanford! Your first weeks and months as an anesthesia resident are exciting, challenging, stressful, and rewarding. Regardless how much or how little experience you have in the field of anesthesiology, the learning curve for the next few months will be very steep. In addition to structured lectures and independent study, you will be primarily responsible for patients as they undergo anesthesia and surgery.

Several years ago, before the development of this mentoring and tutorial system, CA-1’s had little structure to their first month. While there were regular intra-operative and didactic lectures, the nuts and bolts of anesthesiology were taught with little continuity. CA-1’s worked with different attendings every day and spent as much time adjusting to their particular styles as they did learning the basics of anesthesia practice. Starting in 2007, the first month of residency was overhauled to include mentors: each CA-1 at Stanford was matched with an attending or senior resident for a week at a time. In addition, a tutorial curriculum was refined to give structure to the intra-operative teaching and avoid redundancy in lectures. By all accounts, the system has been a great success!

There is so much material to cover in your first couple months of residency that independent study is a must. Teaching in the OR is lost without a foundation of knowledge. Afternoon lectures are more meaningful if you have already read or discussed the material. This booklet serves as a launching point for independent study. While you review the tutorial with your mentor, use each lecture as a starting point for conversation or questions.

During your mentorship, we hope you can use your mentor as a role model for interacting with patients, surgeons, consultants, nurses and other OR personnel. This month, you will interact with most surgical specialties as well as nurses in the OR, PACU, and ICU. We suggest you introduce yourself to them and draw on their expertise as well.

Nobody expects you to be an independent anesthesia resident after just one month of training. You will spend the next three years at Stanford learning the finer points of anesthesia practice, subspecialty anesthesiology, ICU care, pre-operative and post-operative evaluation and management, etc. By the end of this month, we hope you attain a basic knowledge and skill-set that will allow you to understand your environment, know when to ask for help, and determine how to direct self-study. Sprinkled throughout this book, you’ll find some light-hearted resident anecdotes from all the good times you’ll soon have, too.

CA-1 Introduction to Anesthesia Lecture Series:

The Introduction to Anesthesia Lecture series, given by attendings, is designed to introduce you to the basic concepts of anesthesia. Topics covered include basic pharmacology of anesthetics, basic physiology, and various clinical skills and topics. This lecture series starts on July 7th at 4pm in the Anesthesia Conference room. You should receive a schedule of lectures separate from this book. The last lecture is July 30th. You will be relieved of all clinical duties to attend these lectures and it is best to attend them in person. The department has purchased Miller’s Basics of Anesthesia for use as a reference for these lectures.
ACKNOWLEDGEMENTS

Thanks to Janine Roberts and Melissa Cuen for their hard work and assistance in constructing the CA-1 Mentorship Textbook.

Thanks to Dr. Pearl for his support and assistance with this endeavor. His guidance is appreciated by all. If you ever feel like you’re staying too late, know that Dr. Pearl is probably still working in his office when you leave the OR.

Thanks to Dr. Macario, Residency Program Director, who will be one of the first attendings to know each of you by your first name.

Special thanks to Dr. Ryan Green, Class of 2008, founder of the CA-1 mentorship program, and principal editor of the first edition of the CA-1 Mentorship Textbook.

Lastly, thanks to all of the resident and faculty mentors at Stanford University Medical Center, Palo Alto VA, and Santa Clara Valley Medical Center for all of their time and effort spent teaching Stanford anesthesia residents.

As you start this July, don’t be too hard on yourself if you miss an IV or an intubation. If it were that easy, no one would need residency. Also, try to go with the flow if plans change on you suddenly. Flexibility is very important in this field. May your first month be a smooth transition to your anesthesia career.

Welcome to Stanford Anesthesia. We hope you love it as much as we do! Please do not hesitate to contact either one of us with questions or concerns.

Aileen Adriano, MD
Christopher Miller, MD

CONTRIBUTORS


Editors:
Christopher Miller, MD
Aileen Adriano, MD

Section Editors:
Lindsay Borg, MD
Sarah Clark, MD
Stephen Kelleher, MD
Amanda Kumar, MD
Josh Melvin, MD
Christopher Miller, MD
Ann Ng, MD
Lena Scotto, MD
Clair Secomb, MD
Jan Sliwa, MD
Shaina Sonobe, MD

Mentor Coordinator:
Christopher Miller, MD

Resident Mentors:
Jessica Ansari, MD
James Li, MD
Josh Melvin, MD
Christopher Miller, MD
Brita Mittal, MD
Kristen Noon, MD
Shelly Pecorella, MD
Lena Scotto, MD
Jan Sliwa, MD
Shaina Sonobe, MD
Lauren Steffel, MD
Anna Swenson, MD

Faculty Mentors:
Martin Angst, MD
Jennifer Basarab-Tung, MD
Melissa Berhow, MD, PhD
Divya Chander, MD, PhD
Marianne Chen, MD
Jeremy Collins, FRCA, MB ChB
Anna Crawford, MD, MS
Larry Chu, MD, MS
Alimorad Djalali, MD
Anthony Doufas, MD, PhD
Ruth Fanning, MD
Sara Goldhaber-Fiebert, MD
Natalya Hasan, MD
Erin Hennessy, MD
Maeve Hennessy, MD
Gilliam Hilton, FRCA, MB ChB
Bassam Kadry, MD
Jennifer Lee, MD
Meredith Kan, MD
Vivek Kulkarni, MD, PhD
Kevin Malott, MD
Ethan McKenzie, MD
Sesh Mudumbai, MD
Vladimir Nekhendzy
Jordan Newmark, MD
Sara Nikravan, MD
Periklis Panousis, MD
Andrew Patterson, MD, PhD
Catherine Reid, MD
Steven Shafer, MD
Pedro Tanaka, MD
Vivianne Tawfik, MD, PhD
Alexander Tzabazis, MD
Lindsay Vokach, MD
Tara Weineger, MD
Ahmed Zaafran, MD
Karl Zheng, MD


Editors:
Tammy Wang, MD
Aileen Adriano, MD

Section Editors:
Kevin Blaine, MD, MPH
James Flaherty, MD
Robert Groff, MD
Jason Johns, MD
Joseph Kwok, MD
Barrett Larson, MD
Ken Lau, MD
Eric Mehlberg, MD
Christopher Miller, MD
Krisen Noon, MD
Justin Pollock, MD
Christopher Press, MD
Alex Quick, MD
Lindsay Raleigh, MD
Jan Sliwa, MD
Shaina Sonobe, MD
Vicky Yin, MD

Mentor Coordinator:
Lindsay Raleigh, MD

Resident Mentors:
Nick Anast, MD
Kevin Blaine, MD MPH
Sarah Clark, MD
Rob Groff, MD
Leslie Hale, MD
John Johns, MD
Barrett Larson, MD
Ken Lau, MD
Josh Melvin, MD
Rafee Obaidi, MD
Shelly Pecorella, MD
Anil Pranigrahi, MD PhD
Alex Quick, MD

Amit Saxena, MD
Jan Sliwa, MD
Shana Sonobe, MD
Tanya Travkina, MD

Faculty Mentors:
Martin Angst, MD
Jennifer Basarab-Tung, MD
Melissa Berhow, MD, PhD
Divya Chander, MD, PhD
Marianne Chen, MD
Jeremy Collins, FRC, MA ChB
Anna Crawford, MD, MS
Larry Chu, MD, MS
Alimorad Djalali, MD
Anthony Doufas, MD, PhD
Ruth Fanning, MD
Sara Goldhaber-Fiebert, MD
Natalya Hasan, MD
Erin Hennessey, MD
Maeve Hennessey, MD
Ghislain Hilton, FRC, MA ChB
Bassam Kadry, MD
Jennifer Lee, MD
Meredith Kan, MD
Vivek Kulkarni, MD, PhD
Kevin Malott, MD
Ethan McKenzie, MD
Sesh Mudumbai, MD
Vladimir Nekhendzy
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Sara Nikravan, MD
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Vivianne Tawfik, MD, PhD
Alexander Tzabazis, MD
Lindsay Vokach, MD
Tara Weineger, MD
Ahmed Zaafran, MD
Karl Zheng, MD


Editors:
Natalya Hasan, MD
Aileen Adriano, MD

Faculty Mentors:
Aileen Adriano, MD
Martin Angst, MD, PhD
Alex Butwick, MB, FRC, MS
Divya Chander, MD, PhD
Fiona Clements, MD
Anna Crawford, MD, MS
Alimorad Djalali, MD
Anthony Doufas, MD, PhD
David Drover, MD
Ruth Fanning, MD
Rosario Garcia, MD
Sara Goldhaber-Fiebert, MD
Eric Gross, MD, MS, PhD
Maeve Hennessey, MD
Bassam Kadry, MD
Meredith Kan, MD
Vivek Kulkarni, MD
Neil Lawande, MD
Kevin Malott, MD
Diana McGregor, MB, BS, FRC
tFarheen Mirza, MD
Periklis Panousis, MD
Suma Ramzan, MD
Edward Riley, MD
Kimberly Valenta, MD
Karl Zheng, MD


Editors:
Becky Wong, MD
Natalya Hasan, M.D
Aileen Adriano, MD

Resident Mentors:
Catherine Reid, MD
Andrea Goodrich, MD
Roy Esaki, MD
Tatyana Travkina, MD
Calvin Lew, MD

Marianne Chen, MD
Marc Dobrow, MD, MBA
Morgan Dooley, MD, MPH
King Ganguly, MD
Estee Garazi, MD
Brice Gauldilliere, MD, PhD
Robert Groff, MD
Ashley Hawrylsny, MD
Boris Heifets, MD, PhD
Michael Marques, MD, PhD
Vanessa Moll, MD
Marie McHenry, MD, MS
Jared Pearson, MD
Catherine Reid, MD, MS
Dora Rodriguez, MD
James Tan, MD
Vivianne Tawfik, MD, PhD
Tanya Travkina, MD
Luis Verduzco, MD

Faculty Mentors:
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Michael Marques, MD, PhD
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Marie McHenry, MD, MS
Jared Pearson, MD
Catherine Reid, MD, MS
Dora Rodriguez, MD
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Vivianne Tawfik, MD, PhD
Tanya Travkina, MD
Luis Verduzco, MD

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Anthony Doufas, MD, PhD
David Drover, MD
Ruth Fanning, MD
Rosario Garcia, MD
Sara Goldhaber-Fiebert, MD
Eric Gross, MD, MS, PhD
Maeve Hennessey, MD
Bassam Kadry, MD
Meredith Kan, MD
Vivek Kulkarni, MD
Neil Lawande, MD
Kevin Malott, MD
Diana McGregor, MB, BS, FRC
Farheen Mirza, MD
Periklis Panousis, MD
Suma Ramzan, MD
Edward Riley, MD
Kimberly Valenta, MD
Karl Zheng, MD
Boris Heifets, MD, PhD
Ethan Mckenzie, MD
JJ Desai, MD
Laura Downey, MD
Christie Brown, MD
Josh Edwards, MD, MBA
Sam Chen, MD
Justin Workman, MD

Faculty Mentors
Tim Angelotti, MD, PhD
Martin Angst, MD, PhD
Aileen Adriano, MD
Divya Chander, MD, PhD
Fiona Clements, MD
Jeremy Collins, MD
Tara Conaby, MD
Ana Crawford, MD
Ali Djalali, MD
Anthony Doufas, MD, PhD
Ruth Fanning, MD
Sara Goldhaber-Fiebert, MD
Vivek Kulkarni, MD, PhD
Alex Macario, MD, MBA
Diana McGregor, MD
Periklis Panousis, MD
Andrew Patterson, MD, PhD
Lindsay Ralls, MD
Suma Ramzan, MD
Ed Riley, MD
Pedro Tanaka, MD, PhD
Karl Zheng, MD


Editors:
Becky Wong, MD
Kate Ellerbrock, M.D
Aileen Adriano, MD

Resident Mentors:
Sarah Bain, MD
Christie Brown, MD
Dora Castaneda, MD
Michael Charles, MD, PhD
Kate Ellerbrock, MD
Erin Hennessey, MD
Jody Leng, MD

Javier Lorenzo, MD
David Medina, MD
Brett Miller, MD
John Peterson, MD
Rohith Piyaratna, MD
Becky Wong, MD
Andrew Wall, MD
Romy Yun, MD

Faculty Mentors
Tim Angelotti, MD, PhD
Martin Angst, MD, PhD
Lindsay Atkinson, MD
Alex Butwick, MD
Divya Chander, MD, PhD
Larry Chu, MD, MS
Jeremy Collins, MD
Ana Crawford, MD
Ali Djalali, MD
Anthony Doufas, MD, PhD
Ruth Fanning, MD
Sara Goldhaber-Fiebert, MD
Lee Hanowell, MD
Gill Hilton, MD
Jerry Ingrande, MD, MS
Richard Jaffe, MD, PhD
Vivek Kulkarni, MD, PhD
Steve Lipman, MD
Alex Macario, MD, MBA
Kevin Malott, MD
Diana McGregor, MD
John Nguyen, MD
Periklis Panousis, MD
Suma Ramzan, MD
Ed Riley, MD
Vanila Singh, MD
Pedro Tanaka, MD, PhD
Ying Ting, MD
Kimberly Valenta, MD
Karl Zheng, MD


Editors:
Jessica Kentish, MD
William Hightower, M.D
Tara Conaby, MD

Resident Mentors and Contributing Authors:
Sarah Bain, MD
Marisa Brandt, MD
Erin Hennessey, MD
Billy Hightower, MD
Jesse Hill, MD
Meredith Kan, MD
Zoe Kaufenberg, MD
Jessica Kentish, MD
Zeest Khan, MD
Milo Lochbaum, MD
Nate Ponstein, MD
Tzevan Poon, MD

Faculty Mentors
Aileen Adriano, MD
Tim Angelotti, MD, PhD
Jeremy Collins, MD
Tara Conaby, MD
Anthony Doufas, MD, PhD
Ruth Fanning, MD
Sara Goldhaber-Fiebert, MD
Cosmin Guta, MD
Leland Hanowell, MD
Vivek Kulkarni, MD
Hendrikus Lemmens, MD, PhD.
Diana McGregor, MD
Alex Macario, MD, MBA
Kevin Malott, MD
Ed Riley, MD
Cliff Schmiesing, MD
Pedro Tanaka, MD
Alex Tzabazis, MD


Editors:
Jerry Ingrande, MD
Aileen Adriano, MD


Editors:
Ryan Green, MD, PhD
Aileen Adriano, MD
KEY POINTS AND EXPECTATIONS

Key Points:
- The program will last 4 weeks.
- Mentors will consist of faculty members and senior residents (CA-2s and CA-3s).
- CA-1s scheduled to start in the Stanford GOR will be assigned a different mentor each week (CA-1s scheduled to begin at the Palo Alto VAMC or Santa Clara Valley Medical Center will be mentored according to local program goals and objectives).
- Faculty will provide one-on-one mentoring while senior residents will provide one-on-one mentoring with oversight by a supervising faculty member.
- Mentors (both faculty and residents) and CA-1s will take weekday call together. CA-1s will take call with their mentor, but only in a shadowing capacity; both mentor and CA-1 take DAC (day-off after call) together.
- All CA-1s (including those starting at Stanford, VAMC, and SCVMC) will receive the syllabus of intra-operative mini-lecture topics to be covered with their mentors. These mini-lectures provide goal-directed intra-operative teaching during the first month. CA-1s will document the completion of each mini-lecture by obtaining their mentors’ initials on the “Checklist for CA-1 Mentorship Intra-operative Didactics.”
- CA-1s will receive verbal feedback from their mentors throughout the week, as appropriate, as well as at the end of each week. Mentors will communicate from week to week to improve longitudinal growth and mentorship of the CA-1.

Expectations of CA-1 Residents:
- Attend the afternoon CA-1 Introduction to Anesthesia Lecture Series.
- Participate in goal-directed learning by completing the CA-1 Mentorship Intra-operative Didactics with your mentors.
- Discuss cases with your mentor the night before.
- Take weekday call with your mentor. You will be expected to stay as long as the ongoing cases are of high learning value. You will take DAC day off with your mentor.
- CA-1s at SUH are not expected to take weekend call with your mentor (for those at the Valley and VA, discuss with your mentor).

Expectations of Senior Resident Mentors:
- Senior mentors will take primary responsibility for discussing the case, formulating a plan, and carrying out the anesthetic with their CA-1; if concerns arise, the senior mentor will discuss the case with the covering faculty member.
- Instruct CA-1s in the hands-on technical aspects of delivering an anesthetic.
- Participate in goal-directed learning by completing the CA-1 Mentorship Intra-operative Didactics with your CA-1.
- Take weekday call with your CA-1. When you go home, your CA-1 goes home. When you have a DAC, your CA-1 has a DAC.
- Provide timely feedback to your CA-1 every day and at the end of the week.
- Provide continuity of teaching by communicating with the CA-1’s other mentors.

Expectations of Faculty Mentors:
- Participate in goal-directed learning by completing the CA-1 Mentorship Intra-operative Didactics with your CA-1.
- Take weekday call with your CA-1. When you go home, your CA-1 goes home. When you have a DAC, your CA-1 has a DAC.
- Provide timely feedback to your CA-1 every day and at the end of the week.
- Provide continuity of teaching by communicating with the CA-1’s other mentors.
GOALS OF THE CA-1 TUTORIAL MONTH

Anesthesia is a “hands-on” specialty. Acquiring the fundamental knowledge, as well as cognitive and technical skills necessary to provide safe anesthesia, are essential early on in your training. The CA-1 Mentorship Program and the CA-1 Introduction to Anesthesia Lecture Series will provide you with the opportunity to achieve these goals. The following are essential cognitive and technical skills that each CA-1 resident should acquire by the end of their first month.

I. Preoperative Preparation:
   a. Perform a complete safety check of the anesthesia machine.
   b. Understand the basics of the anesthesia machine including the gas delivery systems, vaporizers, and CO₂ absorbers.
   c. Set up appropriate equipment and medications necessary for administration of anesthesia.
   d. Conduct a focused history with emphasis on co-existing diseases that are of importance to anesthesia.
   e. Perform a physical examination with special attention to the airway and cardiopulmonary systems.
   f. Understand the proper use of laboratory testing and how abnormalities could impact overall anesthetic management.
   g. Discuss appropriate anesthetic plan with patient and obtain an informed consent.
   h. Write a pre-operative History & Physical with Assessment & Plan in the chart.

II. Anesthetic Management
   a. Placement of intravenous cannulae. Central venous catheter and arterial catheter placement are optional.
   b. Understanding and proper use of appropriate monitoring systems (BP, EKG, capnography, temperature, and pulse oximeter).
   c. Demonstrate the knowledge and proper use of the following medications:
      i. Pre-medication: Midazolam
      ii. Induction agents: Propofol, Etomidate
      iii. Neuromuscular blocking agents: Succinylcholine and at least one non-depolarizing agent
      iv. Anticholinesterase and Anticholinergic reversal agents: Neostigmine and Glycopyrrolate
      v. Local anesthetics: Lidocaine
      vi. Opioids: Fentanyl and at least one other opioid
      vii. Inhalational anesthetics: Nitrous oxide and one other volatile anesthetic
      viii. Vasoactive agents: Ephedrine and Phenylephrine
   d. Position the patient properly on the operating table.
   e. Perform successful mask ventilation, endotracheal intubation, and LMA placement.
   f. Recognize and manage cardiopulmonary instability.
   g. Spinal and epidural anesthesia are optional.
   h. Record intra-operative note and anesthetic data accurately, punctually, and honestly.

III. Post-operative Evaluation
   a. Transport a stable patient to the Post Anesthesia Care Unit (PACU)
   b. Provide a succinct anesthesia report to the PACU resident and nurse.
   c. Complete the anesthesia record with proper note.
   d. Leave the patient in a stable condition.
   e. Make a prompt post-operative visit and leave a note in the chart (optional but strongly encouraged).
## SUGGESTED CHECKLIST FOR CA-1 MENTORSHIP
### INTRAOPERATIVE DIDACTICS

**Mentors initial** completed lectures

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Standard Monitors

Monitoring in the Past

Basic Anesthetic Monitoring

ASA Standards for Basic Anesthetic Monitoring

STANDARD I
“Qualified anesthesia personnel shall be present in the room throughout the conduct of all general anesthetics, regional anesthetics, and monitored anesthesia care.”

STANDARD II
“During all anesthetics, the patient’s oxygenation, ventilation, circulation, and temperature shall be continually evaluated.”

OXYGENATION
• F2O Analyzer
• Pulse Oximetry

VENTILATION
• Capnography
• Disconnected alarm

CIRCULATION
• EKG
• Blood Pressure
• Pulse Oximetry

TEMPERATURE
• Temperature Probe

Pulse Oximetry

Terminology
- $S_{O_2}$ (Fractional Oximetry) = $O_2Hb / (O_2Hb + Hb + MetHb + COHb)$
- $S_{pO_2}$ (Functional Oximetry/Pulse Oximetry) = $O_2Hb / (O_2Hb + Hb)$

Fundamentals
- The probe emits light at 660 nm (red, for Hb) and 940 nm (infrared, for $O_2Hb$); sensors detect the light absorbed at each wavelength.
- Photoplethysmography is used to identify arterial flow (alternating current = AC) and cancels out the absorption during non-pulsatile flow (direct current = DC); the patient is their own control!
- The $S$ value is used to derive the $S_{O_2}$ ($S = 1:1$ ratio = $S_{pO_2} = 85\%$).

\[
Pulse Oximetry
\]

Pulse Oximetry

Pearls
- Methemoglobin (MetHb) - Similar light absorption at 660 nm and 940 nm (1:1 ratio); at high levels, $S_{O_2}$ approaches 85%.
- Carboxyhemoglobin (COHb) - Similar absorbance to $O_2Hb$. At 50% COHb, $S_{O_2}$ = 50% on ABG, but $S_{O_2}$ may be 95%, thus producing a falsely HIGH $S_{O_2}$.
- Other factors producing a falsely LOW $S_{O_2}$ = dyes (methylene blue > indocyanine green > indigo carmine), blue nail polish, shivering, ambient light.
- Factors with NO EFFECT on $S_{O_2}$ = bilirubin, HbF, HbS, SuHb, acrylic nails, fluorescein dye.
- Cyanosis - clinically apparent with 3 g/dl desaturated Hb. At Hb = 15 g/dl, cyanosis occurs at $S_{O_2} = 80\%$; at Hb = 9 g/dl (i.e. anemia), cyanosis occurs at $S_{O_2} = 66\%$.

EKG

3-Electrode System
- Allows monitoring of Leads I, II, and III, but only one lead (i.e. electrode pair) can be examined at a time and the 3rd electrode serves as ground.
- Lead II is best for detecting P waves and sinus rhythm.

Modified 3-Electrode System
- If you have concerns for anterior wall ischemia, move L arm lead to V5 position, and monitor Lead I for ischemia.

5-Electrode System
- Four limb leads + V5 (left anterior axillary line, 5th ICS), allows monitoring of 7 leads simultaneously.
- V5 is 75% sensitive for detecting ischemic events; II + V5 is 80% sensitive; II + V4 + V5 together is 98% sensitive.
Noninvasive Blood Pressure

- Automated, microprocessor-assisted interpretation of oscillations in the NIBP cuff.
- MAP is primary measurement; SBP and DBP are derived from algorithms.
- Bladder should encircle ≥50% of extremity; width should be 20-50% greater than diameter of extremity.
- Cuff too small = falsely HIGH BP. Cuff too big = falsely LOW BP.

FYI:
MAP = SBP + 2DBP / 3

Arterial Blood Pressure

Indications
- Moment-to-moment BP changes anticipated and rapid detection is vital.
- Planned pharmacologic or mechanical manipulation.
- Repeated blood sampling.
- Failure of NIBP.
- Supplementary diagnostic information (e.g. perfusion of dysrhythmic activity, volume status, IABP).

Transducer Setup
- Zeroing = exposes the transducer to air-fluid interface at any stopcock, thus establishing P atm as the “zero” reference pressure.
- Leveling = assigns the zero reference point to a specific point on the patient; by convention, the transducer is “leveled” at the right atrium.

Effect of Patient & Transducer Position on BP Measurement

<table>
<thead>
<tr>
<th></th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABP</td>
<td>120/80</td>
<td>120/80</td>
</tr>
<tr>
<td>NIBP</td>
<td>120/80</td>
<td>120/80</td>
</tr>
<tr>
<td>ABP</td>
<td>120/80</td>
<td>120/80</td>
</tr>
<tr>
<td>NIBP</td>
<td>135/95</td>
<td>105/65</td>
</tr>
</tbody>
</table>

FYI:
10 cm H2O = 7.5 mm Hg

Capnography

- Gives you tons of information (both number and tracing):
  - Bronchospasm (upsloping trace)
  - Inadequate circulation resulting from hypotension indicating BP is too low for pt (number decreasing)
  - Pulmonary embolism (decreased number and increased different between ETCO2 and PaCO2)
  - Adequacy of CPR eliminating need for pulse checks and compression interruption (ETCO2>10; if sudden increase in ETCO2, then likely have ROSC)
  - Pt breathing spontaneously (more rounded trace)
  - Esophageal intubation, circuit disconnect (no ETCO2 tracing)
  - Exhausted CO2 absorbent (ETCO2 does not return to 0-5)

Capnogram Phases
I. Dead space gas exhaled
II. Transition between airway and alveolar gas
III. Alveolar plateau
IV. Inspiration

Capnography Example Traces
A. Spontaneous ventilation
B. Mechanical ventilation
C. Prolonged exhalation (spontaneous)
D. Emphysema
E. Sample line leak
F. Exhausted CO2 absorbent
G. Cardiogenic oscillations
H. Electrical noise
**Temperature**

Monitoring is now required (previously recommended)

**Sites**
- Pulmonary artery = “Core” temperature (gold standard)
- Tympanic membrane - correlates well with core; approximates brain/hypothalamic temperature
- Esophagus - correlates well with core
- Nasopharynx - correlates well with core and brain temperature
- Rectal - not accurate (temp affected by LE venous return, enteric organisms, and stool insulation)
- Bladder - approximates core when urine flow is high
- Axillary - inaccurate; varies by skin perfusion
- Skin - inaccurate; varies by site
- Oropharynx – good estimate of core temperature; recent studies show correlation with tympanic and esophageal temperatures

**Other Monitors/Adjuncts to Consider**
- Foley
- A-line
- OG tube
- CVC
- Esophageal stethoscope
- ICP
- Pulmonary Artery catheter
- BIS monitor/Sedline

---

**I just intubated, now what?!!**

- Remember your A's
- Adjust (vent settings, volatile)
- A temp probe
- Acid (OG tube)
- Antibiotics
- Air (Forced Air, aka Bair Hugger)
- Another IV
- A line

---

**Inhalational Agents**

**Pharmacokinetics**
- The pharmacokinetics of inhalational agents is divided into four phases
  - Absorption
  - Distribution (to the CNS/brain = site of action)
  - Metabolism (minimal)
  - Excretion (minimal)
- The ultimate goal is to establish a particular partial pressure of an agent in the alveoli ($P_A$)
  - This partial pressure will equilibrate with the CNS tissue to produce an anesthetized state.
- At equilibrium the following applies
  $$P_{CNS} = P_{arterial blood} = P_{alveoli}$$

---

**References**

Uptake and Distribution

• $P_A$ is determined by input (delivery) minus uptake (loss)
  – Input: inspired partial pressure, alveolar ventilation, breathing system
  – Uptake: solubility, cardiac output, alveolar-to-venous partial pressure difference

• Inhalational anesthetic uptake is commonly followed by the ratio of fractional concentration of alveolar anesthetic to inspired anesthetic ($FA/F_I$)
  – Inhalational anesthetic uptake is commonly followed by the ratio of fractional concentration of alveolar anesthetic to inspired anesthetic ($FA/F_I$)
  – Uptake into the bloodstream is the primary determinant of $FA$
  – The greater the uptake (in blood), the slower the rate of rise of $FA/F_I$
    – Uptake is proportional to tissue solubility
    – The gases with the lowest solubilities in blood (i.e. desflurane) will have the fastest rise in $FA/F_I$
    – They also have the fastest elimination
    – Rate of rise of $FA/F_I$ is proportional to clinical effect (i.e. the faster the rate of rise, the faster the induction and also elimination/emergence)

Factors That Increase or Decrease the Rate of Rise of $FA/F_I$

**INCREASE**                  **DECREASE**
Low $\lambda_B$          High $\lambda_B$ The lower the blood:gas solubility, the faster the rise in $FA/F_I$
Low $Q$                   High $Q$ The lower the cardiac output, the faster the rise in $FA/F_I$
High $\dot{V}_A$          Low $\dot{V}_A$ The higher the minute ventilation, the faster the rise in $FA/F_I$
High ($P_A - P_V$)       Low ($P_A - P_V$) At the beginning of induction, $P_V$ is zero but rises rapidly (thus $[P_A - P_V]$ falls rapidly and $FA/F_I$ increases rapidly. Later, during induction and maintenance, $P_V$ rises more slowly so $FA/F_I$ rises more slowly.

Parameters as described in Equation 15-16: $\lambda_B$, blood solubility; $Q$, cardiac output; $\dot{V}_A$, minute ventilation; $P_A$, $P_V$, pulmonary arterial and venous blood partial pressure. (Clinical Anesthesia 5th Edition; Barash, P.; Lippincott Williams and Wilkins; 2006)

Pharmacodynamics

• All inhalational agents decrease $CMO_2$ and increase CBF (via direct vasodilatation)
  – Increases in CBF can increase ICP
• All agents cause a dose-related decrease in blood pressure
• All agents produce muscle relaxation
• The older inhalational agents (halothane, enflurane) cause decreases in myocardial contractility
  – The newer agents have little to no effect
• All inhalational agents produce a dose-dependent depression of the ventilatory response to hypercarbia and hypoxia
  – Increase RR + decrease tidal volume = preserved minute ventilation

Nitrous Oxide

• Low potency (MAC 104% - can never reach 1 MAC!)
• Insoluble in blood
  – Facilitates rapid uptake and elimination
• Commonly administered as an anesthetic adjuvant
• Does not produce skeletal muscle relaxation
• Can potentially contribute to PONV
• Can diffuse into air filled cavities and cause expansion of air filled structures (pneumothorax, bowel, middle ear, ET tube balloons, pulmonary blebs, etc.)
  – Nitrous oxide can enter cavities faster than nitrous can leave
  – Often contraindicated in these settings
• Myocardial depression may be unmasked in CAD or severe hypotension

Isoflurane

• Highly pungent
• Second most potent of the clinically used inhalational agents (MAC 1.2%)
• Preserves flow-metabolism coupling in the brain
  – Highly popular for neuroanesthesia
• Has been implicated for causing "coronary steal"
  – Dilatation of "normal" coronary arteries causing blood to be diverted away from maximally dilated, stenotic vessels to vessels with more adequate perfusion
• Causes vasodilation
  – Decreases BP
  – Increases CBF (usually seen at 1.6 MAC)
  – Minimal compared to halothane
  – Increases ICP (usually at above 1 MAC; short lived)
  – Minimal compared to halothane
• At 2 MAC produces electrically silent EEG
Sevoflurane

- Half as potent as isoflurane (MAC 1.8%)
- Rapid uptake and elimination
- Sweet smelling, non-pungent
  - Quick uptake and sweet smell make this agent very popular for inhalational induction
- Potent bronchodilator
- Can form CO in desiccated CO₂ absorbent
  - Can cause fires
- Forms Compound A in CO₂ absorbent (nephrotoxic)
  - Recommended to keep fresh gas flows >2 L/min

Desflurane

- Lowest blood:gas solubility coefficient (lower than N₂O)
- Very fast uptake and elimination
- Low potency (MAC 6.6%)
- High vapor pressure
  - Must be stored in a heated, pressurized vaporizer
- Very pungent
  - Can cause breath-holding, bronchospasm, laryngospasm, coughing, salivation when administered to an awake patient via face mask
- Can form CO in desiccated CO₂ absorbent
- Can cause an increased sympathetic response (tachycardia, hypertension) when inspired concentration is increased rapidly

References

2. Miller’s Anesthesia 6th edition; Miller R.; Churchill Livingstone, 2005

MAC & Awareness

Alveolar concentration of a gas at which 50% of subjects do not respond to surgical incision

Important Points

- Remarkably consistent across species.
- MAC mirrors the brain partial pressure of an agent
- MAC is a population average; not a true predictor of an individual’s response.
- MAC is an ED₉⁰ concentration. The ED₉⁰ is ±25%, so at 1.3 MAC, 95% of patients will not respond to incision.
- MAC values are additive (e.g. 0.5 MAC isoflurane + 0.5 MAC N₂O = 1 MAC)
MAC of Inhaled Anesthetics

<table>
<thead>
<tr>
<th>Gas</th>
<th>Blood:Gas Partition Coefficient</th>
<th>MAC*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Halothane</td>
<td>2.4</td>
<td>0.75%</td>
</tr>
<tr>
<td>Enflurane</td>
<td>1.9</td>
<td>1.7%</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>1.4</td>
<td>1.2%</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>0.65</td>
<td>2.0%</td>
</tr>
<tr>
<td>N₂O</td>
<td>0.47</td>
<td>104%</td>
</tr>
<tr>
<td>Desflurane</td>
<td>0.42</td>
<td>6.0%</td>
</tr>
</tbody>
</table>

*MAC values for adults 36-49 years old

- MAC is an indicator of gas potency.
- The blood:gas partition coefficient is an indicator of solubility, which affects the rate of induction and emergence; it is NOT related to MAC.

More MAC Definitions

MAC-Awake (a.k.a. MAC-Aware)
- The MAC necessary to prevent response to verbal/tactile stimulation.
- Volatiles: ~0.4 MAC; N₂O: ~0.6 MAC

MAC-BAR
- The MAC necessary to “blunt the autonomic response” to a noxious stimulus
- ~1.6 MAC

MAC-EI
- The MAC necessary to prevent laryngeal response to “endotracheal intubation”
- ~1.3 MAC

Effect of Age on MAC

MAC is highest at 6 months, then begins to decline. After age 40, MAC declines ~6% per decade; MAC for an 80 year old is about 0.75 that of a 40 year old

Factors Increasing MAC

- Drugs increasing central catecholamines:
  - MAOIs, TCAs
  - Acute cocaine and amphetamine use
  - Ephedrine
  - Levodopa
- Hyperthermia (over 42C)
- Hypernatremia
- Chronic EtOH abuse
- Genetic factors
  - Redheaded females may have a 19% increased MAC requirement compared to brunettes.

Factors Decreasing MAC

- Drugs decreasing central catecholamines:
  - Reserpine, α-methyldopa
  - Chronic amphetamine abuse
- Other drugs:
  - Opioids, benzodiazepines, barbiturates, α-agonists (clonidine, dexmedetomidine), ketamine, lidocaine, lithium, verapamil, hydroxyzine.
- Acute EtOH intoxication
- Pregnancy (1/3 after 8-12 weeks, normal by 72h post-partum)
- Hypothermia (50% per 10°C)
- Hypotension (MAP<40 in adult)
- Hypoxemia (P_{O₂} < 38 mm Hg)
- Hypercarbia (P_{CO₂} > 95 mm Hg)
- Hyponatremia
- Metabolic acidosis
- Anemia (Hct < 10%)

Awareness

- Very rare
- Most common sensation is hearing voices
- Mostly occurs during induction or emergence
- More common in high-risk surgeries where deep anesthesia may be dangerous to an unstable patient (e.g. trauma, cardiac, cesarean section)
- Early counseling after an episode is very important
- Patient handout available at: www.asahq.org/patientEducation/Awarenessbrochure.pdf
Signs of Light Anesthesia

- Increase in HR or BP by 20% above baseline
- Tearing
- Dilated pupils
- Coughing or bucking
- Patient movement
- Signs of consciousness on EEG monitor (Bispectral Index or Sedline)

BIS & Sedline

- Both use EEG monitoring and algorithms to produce numbers (0-100) relating to depth of anesthesia.
  - 65-85 = sedation
  - 30-65 = general anesthesia
  - <30 = too deep
- Both have been shown to be fairly good predictors of loss and regaining consciousness
- Interpatient variability exists
- Both have a noticeable time lag (~2min)
- It is possible to display the raw EEG in real time on either device, and interpret on your own.

Management

If you suspect your patient may be aware:

- Immediately deepen the anesthetic with fast-acting agents (e.g. propofol).
- Talk to the patient, reassure them that everything is OK (hearing is the last sense to be lost).
- Consider a benzodiazepine for amnesia.
- Talk to the patient after the case to assess if they had any awareness.
- Set up counseling if necessary.
- Contact Patient Services and Risk Management (potential lawsuit?)

References


Mechanism of Action

- It is widely believed that most IV anesthetics exert their sedative and hypnotic effects via their interaction with GABA
  - GABA is the primary inhibitory neurotransmitter in the CNS
  - Activation of receptor causes increased chloride conductance, and therefore, hyperpolarization
  - Others work through NMDA receptors (Ketamine) or alpha-2 receptors (Dexmedetomidine)
- Propofol and the barbiturates decrease the rate of dissociation of GABA and its receptor
- Benzodiazepines increases the efficiency of GABA-receptor and chloride ion channel coupling

IV Anesthetic Agents
### Pharmacodynamics

- The principle pharmacologic effect of IV anesthetics is to produce increasing sedation and eventually hypnosis. They can be used to induce loss of consciousness at the beginning of an anesthetic or used as infusions to maintain general anesthesia.
- All hypnotics also effect other major organ systems:
  - They produce a dose-dependent respiratory depression (exception: Ketamine)
  - Profound hemodynamic effects can be seen with hypovolemia as a higher drug concentration is achieved at the central compartment
  - A large hemodynamic depressant effect can be seen in the elderly and those with pre-existing cardiovascular disease
- These patients often require a decreased dose requirement

### Induction Characteristics and Dosage Requirements for the Currently Available Sedative-Hypnotic Drugs

<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Induction Dose (mg/kg)</th>
<th>Onset (sec)</th>
<th>Duration (min)</th>
<th>Excitatory Activity*</th>
<th>Pain on Injection†</th>
<th>Heart Rate</th>
<th>Blood Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiopental</td>
<td>3-6</td>
<td>&gt;30</td>
<td>5-10</td>
<td>++</td>
<td>0++</td>
<td>++</td>
<td>1 ± 1</td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.2-0.4</td>
<td>30-90</td>
<td>10-30</td>
<td>0</td>
<td>0</td>
<td>++</td>
<td>1 ± 1</td>
</tr>
<tr>
<td>Methohexital</td>
<td>0.3-0.6</td>
<td>45-90</td>
<td>15-30</td>
<td>0</td>
<td>0</td>
<td>1 ± 1</td>
<td>1 ± 1</td>
</tr>
<tr>
<td>Propofol</td>
<td>1.0-2.0</td>
<td>15-45</td>
<td>5-10</td>
<td>++</td>
<td>0</td>
<td>1 ± 1</td>
<td>1 ± 1</td>
</tr>
<tr>
<td>Ketamine</td>
<td>1.2</td>
<td>45-60</td>
<td>10-20</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Etomidate</td>
<td>0.2-0.3</td>
<td>15-45</td>
<td>3-12</td>
<td>+++</td>
<td>0</td>
<td>1 ± 1</td>
<td>1 ± 1</td>
</tr>
<tr>
<td>Diazepam</td>
<td>0.03-0.06</td>
<td>60-120</td>
<td>60-120</td>
<td>0</td>
<td>0</td>
<td>1 ± 1</td>
<td>1 ± 1</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>1.7-3</td>
<td>75</td>
<td>0.8-1.3</td>
<td>0.8-1.8</td>
<td>11-22</td>
<td>1 ± 1</td>
<td>1 ± 1</td>
</tr>
<tr>
<td>Ketamine</td>
<td>1.1-1.6</td>
<td>12</td>
<td>2.5-3.5</td>
<td>2.5-3.5</td>
<td>12-17</td>
<td>2 ± 2</td>
<td>2 ± 2</td>
</tr>
</tbody>
</table>

* = decrease; † = increase; ++ = moderate; +++ = severe.

---

### Pharmacokinetic Values for the Currently Available Intravenous Sedative-Hypnotic Drugs

<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Distribution Half-Life (min)</th>
<th>Protein Binding (%)</th>
<th>Distribution Volume at Steady State (L/kg)</th>
<th>Clearance (ml/kg/min)</th>
<th>Elimination Half-Life (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiopental</td>
<td>2.4</td>
<td>85</td>
<td>2.5</td>
<td>1.4</td>
<td>11</td>
</tr>
<tr>
<td>Methohexital</td>
<td>5.6</td>
<td>85</td>
<td>2.2</td>
<td>11</td>
<td>4</td>
</tr>
<tr>
<td>Propofol</td>
<td>2.4</td>
<td>98</td>
<td>2.10</td>
<td>20-30</td>
<td>4-23</td>
</tr>
<tr>
<td>Etomidate</td>
<td>0.2-0.3</td>
<td>95</td>
<td>1.1-1.7</td>
<td>6.4-11</td>
<td>1.7-2.6</td>
</tr>
<tr>
<td>Diazepam</td>
<td>10-15</td>
<td>98</td>
<td>0.7-1.7</td>
<td>0.3-0.5</td>
<td>20-50</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>3-10</td>
<td>98</td>
<td>0.8-1.3</td>
<td>0.8-1.8</td>
<td>11-22</td>
</tr>
<tr>
<td>Ketamine</td>
<td>1.1-1.6</td>
<td>12</td>
<td>2.5-3.5</td>
<td>2.5-3.5</td>
<td>12-17</td>
</tr>
</tbody>
</table>

### Drug, Dose (mg/kg), Effects, Pearls

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose (mg/kg)</th>
<th>Effects</th>
<th>Pearls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propofol</td>
<td>1.5-2.5</td>
<td>Decreases cerebral metabolic O2, increases cerebral blood flow, intracranial pressure, decreases SVR, direct myocardial depressant in non-ischemic myocardium, decreases CPP, increases CBF</td>
<td>Pain on injection (32-47%) can be attenuated with lidocaine and injection into larger veins</td>
</tr>
<tr>
<td>Etomidate</td>
<td>0.2-0.3</td>
<td>Decreases cerebral metabolic O2, increases cerebral blood flow, decreases SVR, direct myocardial depressant in non-ischemic myocardium</td>
<td>Pain on injection, high incidence of PONV, ketamine can be attenuated with lidocaine and injection into larger veins</td>
</tr>
<tr>
<td>Thiopental</td>
<td>3-5</td>
<td>Decreases cerebral metabolic O2, decreases SVR, direct myocardial depressant in non-ischemic myocardium</td>
<td>Decreases cerebral metabolic O2, increases cerebral blood flow, decreases SVR, direct myocardial depressant in non-ischemic myocardium</td>
</tr>
<tr>
<td>Ketamine</td>
<td>1-2</td>
<td>Increases cerebral metabolic O2, reduces cerebral blood flow, decreases intracranial pressure, decreases SVR, decreases CPP</td>
<td>Pain on injection, high incidence of PONV, ketamine can be attenuated with lidocaine and injection into larger veins</td>
</tr>
</tbody>
</table>

### Propofol

- Produced in an egg lecithin emulsion because of its high lipid solubility
- Pain on injection occurs in 32-67% of subjects
- Can be attenuated with lidocaine or administering the drug in a larger vein
- Infusion doses ~100-200 mcg/kg/min for hypnosis and ~25-75 mcg/kg/min for sedation (depends on desired level of consciousness and infusion duration)
- Decreases CMRO₂, CBF, and CPP may decrease depending on effect on SBP
- Anticonvulsant properties
- Decreases SVR (arterial and venous), direct myocardial depressant
- Dose-dependent respiratory depression
- Has anti-emetic properties — often used for TIVA cases and as a background infusion for patients with PONV
- Formulations support growth of bacteria, good sterile technique and labeling of expiration times (typically 12 hours) is critical
- Propofol infusion syndrome: Risk in critically ill patients receiving high dose propofol infusions (>4mg/kg/hr) for prolonged periods of time. Causes severe metabolic acidosis, rhabdomyolysis, cardiac failure, renal failure, and possibly death
- High incidence of pain on injection
- Induction dose 0.2-0.3 mg/kg
- Rapid onset due to high lipid solubility and large non-ionized fraction at physiologic pH
- Myoclonus common upon injection
- Decreases CMRO₂, CBF, CPP maintained because less SBP decrease
- Anticonvulsant properties; but minimal effect on duration of ECT-induced seizure activity
- Maintains hemodynamic stability (even in the presence of pre-existing disease)
- Does not induce histamine release
- Inhibits adrenocortical synthentatic function
- Inhibition for 5-8 hours even after a single induction dose
- High incidence of PONV
Thiopental

- Highly alkaline (pH 9)
- Can precipitate in acidic solutions (DO NOT MIX with Rocuronium or LR)
- Intra-arterial injection can cause intense vasoconstriction, thrombosis and tissue necrosis; treat with papaverine and lidocaine or regional anesthesia-induced sympathectomy and heparinization
- Induction dose 3-5 mg/kg in adults, 5-6 mg/kg in children, 6-8 mg/kg in infants
- Rapidly redistributed into peripheral compartments (accounts for short duration of action)
- Larger doses can saturate the peripheral compartments resulting in a prolonged duration of action
- Decreases CMRO₂, CBF, ICP
  - Causes EEG burst suppression in larger doses (often used for neurosurgical procedures)
- Anticonvulsant activity
  - Exception: Methohexital
- Decreases SVR, direct myocardial depressant
- Dose-dependent respiratory depression
- Unlikely to use at Stanford but may use internationally

Ketamine

- Produces a dissociative anesthetic state
  - Profound analgesia and amnesia despite maintenance of consciousness
  - High incidence of psychomimetic reactions (attenuated by co-administration of midazolam)
- Induction dose 1-2 mg/kg
- NMDA antagonist
- Increases CMRO₂, CBF, ICP
  - Contraindicated in neurosurgical procedures
- Most likely to preserve airway reflexes among the IV anesthetics
- Minimal respiratory depression
- Cardiotonic effects secondary to direct sympathetic stimulation
  - Can be unmasked in patients with increased sympathetic outflow
  - Negatively affects myocardial oxygen supply-demand ratio
- Intrinsic myocardial depressant - may be significant in severely ill patients with depleted catecholamine reserves
- Can cause increases pulmonary artery pressure
- Causes bronchodilation
- Causes increased oral secretions
- Useful for chronic pain patients (common dose for intra-operative management is 0.5-1 mg/kg prior to incision (after intubation, unless using for induction) and then 0.25 mg/kg each hour (infusion or bolus)

Midazolam

- All benzodiazepines have anxiolytic, amnestic, sedative, hypnotic, anticonvulsant properties
- Premedication dose 0.04-0.08 mg/kg IV
- Induction dose 0.1-0.2 mg/kg IV
- Decreases CMRO₂, CBF, ICP
  - Does not produce EEG burst suppression
- Decrease SVR and BP when used as induction dose
- Causes dose-dependent respiratory depression
  - Exaggerated when combined with opioids and in patients with chronic respiratory disease
- Flumazenil is a specific antagonist
  - Very short acting
  - 45-90 minutes of action following 1-3 mg dose
  - May see re-sedation as benzodiazepine is eliminated more slowly compared to effects of flumazenil

Dexmedetomidine

- Selective α₂ adrenergic agonist
- Hypnotic and analgesic
- Opioid-sparing effect and does not significantly depress respiratory drive
- Usually an infusion at a concentration of 4 mcg/ml
- Loading dose 0.5-1mcg/kg over 10min
- Infusion rate 0.2-0.7 mcg/kg/hr (ask your attending)
- Rapid onset and terminal half-life of 2hr
- Decrease dosage for patients with renal insufficiency or hepatic impairment
- Main side effects are bradycardia, heart block, hypotension
- May cause nausea
- Can be utilized for sedation during awake FOB intubations

It was my first week of anesthesia residency and my mentor asked me to hang some blood to transfuse. I reached up and removed the spike from the bag of fluid that was already hanging...I was immediately soaked by the open IV fluid bag. My mentor later told me that he knew that would happen, but let me do it anyway so that I would always remember to bring the bag down first. I haven't forgotten.

I was in the preop area at the VA, and introduced myself to the patient as Dr. Taylor*. He quickly replied, "What was your name?", to which I said my first name, "Victoria". He looked at me amazed and said, "I can't believe it. I have your name tattooed on my ass". I asked if he was willing to show me. As he rolled over, the words "your name" appeared on his left butt cheek.

*Names have been changed
It was the 4th week of CA-1 year, and I knew I was going to need 2 PIVs for a relatively bloody case. That morning I prepared the fluid warmer with a blood pump, ready to go once I got the 2nd PIV inside the OR. In pre-op, I placed a PIV on the RIGHT side, then brought him in to the OR, connected the monitors and started giving fentanyl and propofol through the stop cocks on the LEFT blood pump. No change in the patient or vital signs—my attending and I were puzzled. I came to realize that I was basically feeding meds into the fluid warmer (which had the capacity to absorb the meds without causing significant resistance or dripping onto the floor). Yeah, I remember my attending giving me a smile, shaking his head and saying, “Give me the blood pump and connect it over here.” Regardless, the patient was induced and we played it off cool.

References

Rational IV Opioid Use

Basic Opioid Pharmacology

• Analgesia produced by mu (μ) opioid receptor agonism in the brain (periaqueductal gray matter) and spinal cord (substantia gelatinosa).
• Well-known side effect profile:
  – Sedation, respiratory depression
  – Itching, nausea, ileus, urinary retention
  – Bradycardia, hypotension
  – Miosis, chest wall rigidity
• Opioids are hemodynamically stable when given alone, but cause ↓CO, SV, and BP in combination with other anesthetics.
• Reduces MAC of volatile anesthetics.

Opioid Receptor Subtypes and Their Effects

• μ
  – Analgesia: Peripheral
  – GI: GI secretion, GI transit (supraspinal and peripheral), antidiarrheal
  – Other: Puritus, skeletal muscle rigidity, urinary retention, biliary spasm
• μ1
  – Analgesia: Supraspinal
  – Endocrine: Prolactin release
  – Other: Acetylcholine, catalepsy
• μ2
  – Analgesia: Spinal and supraspinal
  – Resp: Respiratory depression
  – GI: GI transit (supraspinal and spinal)
  – Other: Most cardiovascular effects
• μ3
  – Other: Anti-inflammatory

Opioid Receptor Subtypes and Their Effects

• κ
  – Analgesia: Peripheral
  – Endocrine: ADH release
  – Other: Sedation
• κ1
  – Analgesia: Spinal
  – Other: Antipruritic
• κ2
  – Pharmacology unknown
• κ3
  – Analgesia: Supraspinal
Opioid Receptor Subtypes and Their Effects

• δ
  – Analgesia: Peripheral
  – Resp: ?Respiratory depression
  – GI: ?GI transit (spinal), antidiarrheal (spinal and supraspinal)
  – Endo: ?Growth hormone release
  – Other: ?Urinary retention
• δ₁
  – Analgesia: spinal
  – Other: Dopamine turnover
• δ₂
  – Analgesia: Supraspinal
• Unknown (receptor type not identified)
  – Analgesia: Supraspinal
  – Other: Pupillary constriction, nausea and vomiting

Opioids

Morphine
– Slow peak time (~80% effect at 15 minutes, but peak analgesic effect is at ~90 minutes).
– Active metabolite, morphine-6-glucuronide, has analgesic properties and is renally excreted (not clinically relevant unless patient has renal failure)
– Can cause histamine release.

Hydromorphone (Dilaudid)
– "A rapid onset morphine" --> Peak effect in 5-10 minutes.
– About 8-fold more potent than morphine (i.e. 1 mg Dilaudid = 8 mg morphine)
– No active metabolites, no histamine release.
– Good choice for postop analgesia and PCA.

Fentanyl
– Fast onset & short duration of action (peak effect at 3-5 minutes; effect site half-life ~30 minutes.
– ~100-fold more potent than morphine.
– Very cheap.

Sufentanil
– Fast onset, but slightly slower than fentanyl
– 10-fold more potent than fentanyl (i.e. 5 mcg sufentanil ~ 50 mcg fentanyl).
– More rapid recovery than fentanyl.

Alfentanil
– Fastest onset time of all opioids (~90 seconds); pKa = 6.5, so it crosses the blood-brain barrier rapidly.
– Also causes more N/V, chest wall rigidity, and respiratory depression.
– Brief duration of action due to rapid redistribution.

Remifentanil
– Peak effect time ~90 seconds
– Unique pharmacokinetics - metabolized by plasma esterases.
– Short context-sensitive half-time after termination of infusion with predictable offset in ~5-10 minutes.

Meperidine (Demerol)
– Originally discovered as a local anesthetic ("pethidine")
– Peak effect in 15 minutes, lasts 2-4 hours.
– Active metabolite (normeperidine) lowers the seizure threshold; renally excreted.
– Useful for treating shivering.
– Anticholinergic side effects: tachycardia
– Avoid using with MAOIs; can cause CNS excitation (agitation, hyperpyrexia, rigidity) and/or CNS depression (hypotension, hypoventilation, coma)
– Causes histamine release.
– Has a euphoric effect with less respiratory depression than other opioids.

Comparison of Peak Effect Times

Onset and duration of action of each opioid depend on their lipid solubility and ionization.
Rational Opioid Use

Note: All anesthesiologists (attendings & residents alike) have different theories and opinions on the optimal choice and dose of opioids in different situations. The strategies presented here are simply suggestions, something to get you thinking rationally about how and when you use opioids for analgesia. Discuss the merits of these strategies with your attending before or during each case, but do not take these suggestions as firm guidelines for how all anesthetics should be done!

With that disclaimer in mind, continue reading…

Strategies for Opioid Use

• For a standard GETA induction, use fentanyl to blunt the stimulation caused by DL and intubation.
• For brief, intense stimulation (e.g. retrobulbar block, Mayfield head pins, rigid bronchoscopy), consider a bolus of short-acting opioid like remifentanil or alfentanil.
• For intraop analgesia:
  – Fentanyl is rapidly titratable, but requires frequent redosing; it may be more “forgiving” if overdosed.
  – Morphine has a long onset time to peak effect, but gives prolonged analgesia during the case and into the postop period.
  – Hydromorphone is rapidly titratable (like fentanyl) with prolonged analgesia (like morphine).

Strategies for Opioid Use

• For ENT cases, consider an opioid infusion (e.g. remifentanil, alfentanil, sufentanil, or fentanyl):
  – Stable level of analgesia
  – Induced hypotension
  – “Narcotic wakeup” reduces bucking on ETT
  – Smooth transition to postop analgesia
• For chronic opioid users (e.g. methadone, MS Contin, OxyContin, etc.), continue the patient’s chronic opioid dose intraoperatively PLUS expect higher opioid requirements for their acute pain.
• Use morphine and meperidine cautiously in renal patients (renal excretion of active metabolites).

Strategies for Opioid Use

• Meperidine is usually reserved for treatment/prevention of postoperative shivering.
• For postop pain control (i.e. PACU):
  – Consider fentanyl (rapid onset, easily titratable, cheap, and the nurses are familiar with its use).
  – Consider hydromorphone (rapid onset, easily titratable, prolonged effect, nurses are familiar with its use, and it is a good transition to PCA).
  – If surgery is ambulatory and/or patient is tolerating POs, give Vicodin or Percocet.

References


Intraoperative Hypotension & Hypertension
Determinants of Blood Pressure

Blood Pressure (BP)
- BP represents the force exerted by circulating blood on the walls of blood vessels.
- Determined by 1) cardiac output and 2) vascular tone

Cardiac Output (CO)
- \( CO = HR \times SV \)

Heart Rate (HR)
- Dependent on the interplay between the sympathetic and parasympathetic nervous systems.
- In infants, SV is fixed, so CO is dependent on HR.
- In adults, SV plays a much more important role, particularly when increasing HR is not favorable.

Stroke Volume (SV)
- Dependent on 1) preload, 2) afterload, and 3) myocardial contractility.

Preload
- Volume of blood in the ventricle at end-diastole (LVEDV)

Afterload
- Resistance to ejection of blood from the ventricle
- SVR accounts for 95% of the impedance to ejection
- \( SVR = 80 \times \frac{MAP - CVP}{CO} \)

Contractility
- The force and velocity of ventricular contraction when preload and afterload are held constant.
- Ejection fraction (EF) is one of the most clinically useful indices of contractility (normal EF is ~60%).

Components of Blood Pressure

Systolic Blood Pressure (SBP)
- Highest arterial pressure in the cardiac cycle.
- Dicrotic notch = a small notch in the invasive arterial pressure curve that represents closure of the aortic valve, producing a brief period of retrograde flow.

Diastolic Blood Pressure (DBP)
- Lowest arterial pressure in the cardiac cycle

Mean Arterial Pressure (MAP)
- \( MAP = \frac{2}{3} DBP + \frac{1}{3} SBP \), or \( (2 \times DBP + SBP) / 3 \)

Pulse Pressure
- \( PP = SBP - DBP \)
- Normal PP is ~40 mm Hg at rest, and up to ~100 mm Hg with strenuous exercise.
- Narrow PP (e.g. < 25 mm Hg) = may represent aortic stenosis, coarctation of the aorta, tension pneumothorax, myocardial failure, shock, or damping of the system.
- Wide PP (e.g. > 40 mm Hg) = aortic regurgitation, atherosclerotic vessels, PDA, high output state (e.g. thyrotoxicosis, AVM, pregnancy, anxiety)

Blood Pressure Measurement

Non-Invasive Blood Pressure (NIBP)
- Oscillometric BP determination: oscillations in pressure are detected through the cuff as it deflates.
- MAP is measured as the largest oscillation; it is the most accurate number produced by NIBP.
- SBP and DBP are calculated by proprietary algorithms in the machine.

Invasive Arterial Blood Pressure (IABP)
- Most accurate method of measuring BP.
- If system is zeroed, leveled, and properly dampened, SBP, DBP, and MAP are very accurate.

Intraoperative Hypertension

- “Light” anesthesia
- Pain
- Chronic hypertension
- Illicit drug use (e.g. cocaine, amphetamines)
- Hypermetabolic state (e.g. MH, thyrotoxicosis, NMS)
- Elevated ICP (Cushing’s triad: HTN, bradycardia, irregular respirations)
- Autonomic hyperreflexia (spinal cord lesion > T5 = severe; < T10 = mild)
- Endocrine disorders (e.g. pheochromocytoma, hyperaldosteronism)
- Hypervolemia
- Drug contamination - intentional (e.g. local anesthetic + Epi) or unintentional (e.g. “Roc-inephrine”)
Treatment of Hypertension

- **Temporize** with fast-onset, short-acting drugs, but ultimately diagnose and treat the underlying cause.

- Pharmacologic Interventions:
  - Volatile anesthetics (cause vasodilation while deepening anesthetic)
  - Opioids (treat pain and deepen the anesthetic, histamine release causes hypotension)
  - Propofol (quickly sedates and vasodilates)
  - Beta-blockers (e.g. esmolol, labetalol)
  - Vasodilators (e.g. hydralazine (takes 20min for peak), NTG, SNP)

Intraoperative Hypotension

- Hypovolemia: Blood loss, dehydration, diuresis, sepsis
  - Ensure: Adequate IV access, fluid replacement, cross match if necessary
- Drugs: Induction and volatile agents, opioids, anticholinesterases, local anesthetic toxicity, vancomycin, protamine, vasopressor/vasodilator infusion problem, syringe swap or drugs given by surgeon
- Regional/Neuraxial Anesthesia: Vasodilation, bradycardia, respiratory failure, local anesthetic toxicity, high spinal
  - Ensure: Volume loading, vaspressors, airway support, left lateral displacement during pregnancy
- Surgical Events: Vagal reflexes, obstructed venous return, pneumoperitoneum, retractor positioning
  - Ensure: Surgeon aware
- Cardiopulmonary Problems: Tension PTX, hemothorax, tamponade, embolism (gas, amniotic fluid, or thrombotic), sepsis, myocardial depression (from drugs, ischemia, electrolytes, trauma)

Treatment of Hypotension

- ```Temporize``` with fast-onset, short-acting drugs, but ultimately diagnose and treat the underlying cause.
  - Turn down (sometimes turn off) the anesthetic
  - Call for help. Inform surgeons
- **Volume**
  - Reevaluate EBL; replace with crystalloid, colloid, or blood, as needed
  - Consider art line, CVP, PAC, or TEE
- **Ventilation**
  - Reduce PEEP to improve venous return
  - Decrease I:E ratio to shorten inspiratory time
  - Rule out PTX
- **Metabolic**
  - Treat acidosis and/or hypocalcemia

Drugs (doses in parentheses are bolus starting doses)

- **Phenylephrine** (Neosynephrine) = $ \alpha_1 $ agonist (start at 100mcg)
  - Direct vasoconstrictor
  - Use in vasodilated state with tachycardia
  - Will cause reflex bradycardia
- **Ephedrine** = $ \alpha_1 $, $ \beta_1 $, and $ \beta_2 $ (less so) agonist (start at 5mg)
  - Direct and indirect adrenergic stimulation via NE release
  - Use in vasodilated, bradycardic, low CO states
- **Epinephrine** = $ \beta_1 $, $ \alpha_1 $, $ \alpha_2 $, and $ \beta_2 $ agonist (start at 5mcg)
  - Endogenous catecholamine
  - Causes vasoconstriction and increased CO
- **Inotropes** (in low CO states)
  - Epinephrine, Dopamine, Milrinone, Dobutamine (the last 2 vasodilate)
- **Stress-dose steroids** – consider 100mg hydrocortisone if steroids taken in past 6 months

References


Neuromuscular Blocking Agents
**Introduction**

- Neuromuscular blocking agents (N MBA) are used to facilitate tracheal intubation and mechanical ventilation, assure immobility, and improve operating conditions (e.g. laparotomy, orthopedic surgery).

- There are two categories of N MBA with distinct properties: depolarizing (succinylcholine) and nondepolarizing (e.g. rocuronium, vecuronium, cisatracurium).

- Postoperative residual paralysis occurs frequently. Monitoring of neuromuscular blockade and pharmacological reversal are the standard of care.

- N MBA have risks and there are a number of surgical and patient specific contraindications. N MBA should be used judiciously. Read your text book chapter on N MBA many times during residency!

**The Depolarizer: Succinylcholine**

- Structure = 2 adjoined ACh molecules
- Mechanism of action is by ACh receptor activation and prolonged muscle depolarization
- Intubating Dose = 1 to 1.5 mg/kg
- If you use a defasciculating dose of roc (0.03mg/kg), intubating dose of sux is higher (1.5-2mg/kg)
- Onset within 30-60 sec; duration ~10 min depending on dose
- Rapidly metabolized by pseudocholinesterases
- ~1:2000 individuals is homozygous for an abnormal plasma cholinesterase and paralysis can last 3-6 hours in such individuals. Consider checking twitches before giving nondepolarizing N MBA after sux.
- The test for abnormal plasma cholinesterase is the dibucaine test. Be sure to understand how this test works for the ITE.

**Contraindications to Sux**

- Hyperkalemia. Sux causes an increase in K+ of 0.5 mEq/L. Normokalemic renal failure is NOT an contraindication.
- Giving sux to patient with conditions that cause upregulation of nAChR on muscle cells may result in hyperkalemic arrest. This includes burn injury (after 24-48hrs), muscular dystrophy, myotonia, prolonged immobility, stroke, upper motor neuron disease
- Malignant Hyperthermia (sux is a trigger)
- Giving sux to patient with conditions that cause upregulation of nAChR on muscle cells may result in hyperkalemic arrest. This includes burn injury (after 24-48hrs), muscular dystrophy, myotonia, prolonged immobility, stroke, upper motor neuron disease
- Postoperative residual paralysis occurs frequently. Monitoring of neuromuscular blockade and pharmacological reversal are the standard of care.

**Additional Side Effects**

- Bradycardia, esp. in children. Often given with atropine.
- Tachycardia (catecholamine release)
- Anaphylaxis approx. 1:5000 – 1:10,000
- Fasciculation + myalgia. Largely preventable with defasciculating dose of roc.
- Trismus
- Increased ICP, IOP, and intragastric pressure. N.B. Benefits of securing the airway quickly often take precedent over small increases in ICP or IOP.

**Defasciculating Dose of Roc** (0.03mg/kg 3 minutes prior to sux)

- Can prevent myalgias and increased ICP
- Does NOT prevent hyperkalemia or increase IOP

**Non-Depolarizing NMBA**

- Mechanism of action is competitive inhibition of nicotinic ACh receptor (nAChR) at the NMJ.
- Fade with high-frequency nerve stimulation is characteristic. There are presynaptic nAChR which mobilize ACh containing vesicles. These presynaptic nAChR have a slightly different structure from post synaptic nAChR. Nondepolarizing agents block presynaptic nAChR and sux does not.
- Two structural classes:
  1. Benzylisoquinoliniums = “uriums”
     - Atracurium, Cisatracurium, Mivacurium, Doxacurium, d-Tubocurarine
  2. Aminosteroids = “oniums”
     - Pancuronium, Vecuronium, Rocuronium, Pipercuronium
     - No histamine release
     - Possible vagolytic effects (Pancuronium > Rocuronium > Vecuronium)
- There are many non-depolarizing agents and they are divided into classes based on duration of action.

**Agent** | **ED95 (mg/kg)** | **Intubating Dose (mg/kg)** | **Onset (min)** | **Duration to 25% recovery (min)** | **Intra-op Maintenance** | **Metabolism** | **Excretion**
--- | --- | --- | --- | --- | --- | --- | ---
**Succinylcholine** | 0.3 | 0.6 | 1.5-2 | 30-40 | Rarely done | plasma cholinesterase | Bile + Urine
**Rocuronium** | 0.3 | 0.1-0.2 | 3-4 | 35-45 | 0.1-0.2 mg/kg pm | Liver | Bile + Urine
**Vecuronium** | 0.05 | 0.15-0.2 | 5-7 | 35-45 | 0.3 mg/kg q20min pm | Hoffman elimination | Bile + Urine

Adopted from Table 20-2, Ch 20, Barash Clincial Anesthesia 6th edition

**Non-Depolarizing NMBA (cont.)**

- The most used non-depolarizing agents are the intermediate duration agents cisatracurium, rocuronium and vecuronium.
- Intubating doses are 2 x ED 95 (ED95, average dose required to induce 95% suppression of the twitch height in half of the population. I.E. if you give 0.3mg/kg of roc, 50% of the population with have 95% suppression of a monitored twitch.) A larger intubating dose speeds onset time but lengthens duration of block.
- In an effort to speed up onset, some anesthesiologist use a priming dose. 10% of the intubating dose is given 3 minutes prior to intubating dose (as with defasciculating doses prior to sux). Efficacy of a priming dose is debatable.
- Individuals can vary widely in their responses to non-depolarizing agents. Monitor twitches and adjust doses accordingly.
- Rocuronium can be used for rapid sequence inductions when sux cannot be used. However, the dose of roc necessary for RSI (1-1.2 mg/kg) causes prolonged relaxation.
- Cisatracurium is degraded by plasma esterases and Hoffman elimination. It is useful for patients with hepatic or renal dysfunction.
Monitoring NMBA 1

- It is recommended you read a detailed reference about NMBA monitoring.
- The train-of-four ratio is the common modality of monitoring nondepolarizing NMBA as no pre-NMBA control is necessary (see next slide). The number of twitches and the ratio between the 4th and 1st twitch are measured with the TOF. In the OR, we monitor twitch # and twitch height with sight or feel -- in studies, authors use mechanomyography or accelerometry.
- While number of twitches can be accurately assessed by feel/sight, the TOF ratio can NOT be accurately assessed. This means that a patient with “four strong twitches” can have significant weakness.
- A mechanomyographic TOF of 0.9 is considered fully strong.
- Surgical relaxation can be achieved when the patient has 2-3 twitches though this depends on where you monitor and the location of surgery.

Monitoring NMBA 2

Peripheral Nerve Stimulation Patterns

An aside about sux:

- Phase I block is typical for a single bolus of sux.
- Sux can develop a Phase II block at high doses or with with prolonged infusions.
- N.B. Neostigmine will potentiate a phase I block from sux but will reverse a phase II block.

Monitoring NMBA 3

- 5 seconds of sustained tetanus at 100hz indicates full recovery. 5 seconds of head lift does not.
- If placing electrodes on the face, do not deliver more than 20 – 30 mA or you will stimulate facial muscles directly. You would not be the first to be fooled into thinking your patient has twitches when he/she actually has none!
- Where you place the twitch monitor matters as different muscle groups respond differently to NMBA. See next page.
- N.B. pharyngeal muscles are one of the last muscle groups to recover and thus inadequate or lack of reversal leads to airway obstruction and aspiration. It also causes atelectasis and decreased pulmonary reserve.

Monitoring NMBA 4

- Variability in muscle blockade (most resistant ➔ most sensitive): vocal cords > diaphragm > corrugator supercilii (muscle controlling the eyebrow) > abdominal muscles > adductor pollicis > pharyngeal muscles > extraocular
- Pick one site to monitor (e.g. AP or eyebrow or posterior tibial nerve), but know how different muscles respond relative to that site.

Reversal of NMBA 1

- Anticholinesterase “reversal agents” indirectly increase the amount of Ach in the NMU by inhibiting acetylcholinesterase.
- Reversal should not be given until spontaneous recovery has started as anticholinesterases can paradoxically slow recovery if given too early. Many authors advocate waiting until 4 twitches are visible before giving reversal.
- Anticholinesterases cause yagal side effects (e.g. bradycardia, GI stimulation, bronchospasm) by increasing ACh activity at parasympathetic muscarinic receptors. Always administer with anticholinergics.
- Neostigmine with glycopyrrolate is most commonly used.
  - 40-50 mcg/kg of neostigmine is appropriate for most instances.
  - There is a ceiling effect. Do not give >70mcg/kg of neostigmine.
  - If recovery is seems complete (4 equal twitches), 15-20mcg/kg of neostigmine is probably OK
  - Dose of glycopyrrolate is 20% of the neostigmine dose (e.g. 3mg neostigmine with 0.6mg glyco)

Reversal of NMBA 2

- There are other Anticholinesterases besides neostigmine.
  - Neostigmine, Pyridostigmine, and Edrophonium do not cross the BBB.
  - Physostigmine crosses the BBB and can be used to treat central anticholinergic syndrome/atropine toxicity
- It is important to pair anticholinesterases and anticholinergics based on speeds of onset:
  - Edrophonium (rapid) w/ Atropine
  - Neostigmine (intermediate) w/ Glycopyrrolate
  - Pyridostigmine (slow) w/ Glycopyrrolate
Additional NMBA Facts

- Diseases RESISTANT to nondepolarizing NMBA:
  - Guillain-Barré, Burns, Spinal cord injury, CVA, Prolonged immobility, Multiple sclerosis
- Diseases SENSITIVE to nondepolarizing NMBA:
  - Myasthenia gravis (fewer AChR), Lambert-Eaton Syndrome (less ACh release), amytrophic lateral sclerosis
- Factors ENHANCING block by NMBA:
  - Volatile anesthetics, aminoglycosides, Mg, IV local anesthetics, CCBs, Lasix, Dantrolene, Lithium, anticonvulsants, sux, hypokalemia, hypothermia
- Common surgeries where you avoid NMBA
  - Axillary node dissection, ENT cases involving dissection near nerves, cases with neuromonitoring

Intra-op Discussion Topics

- How do you induce a patient with full stomach and open globe?
- Can you use sux with increased ICP?
- What degree of immobility can cause hyperkalemia with sux?
- Can you use rocuronium for a renal transplant?
- Does reversal cause PONV?
- You just gave reversal and there is a lap in the abdomen. How do you paralyze the patient?

References


For a while, one of the surgery residents referred to me as Superman. Not because of anything good, but because I woke his patient up and he emerged a little goofy. He insisted on keeping his arms stretched perfectly straight out in front him, and despite many attempts to get him to relax, he wouldn’t put them down. We sat the head of the bed up, thinking that might help, but it just made it more obvious to everyone we drove past on the way to the PACU, with this old guy holding his Superman pose.

I was giving report in the PACU and mistakenly reported that the patient was an otherwise healthy 64 year-old woman. She was awake, and corrected me, noting that she was in fact 44. She was indeed healthy, though.
A difficult airway is a clinical situation wherein a conventionally trained anesthesiologist has difficulty with face mask ventilation, tracheal intubation, or both.

A difficult airway arises from a complex interaction between patient specific factors, the clinical environment, and the skills of the anesthesiologist.

**STEP 1a**
Assess the likelihood of airway management problems: *Predictors of Difficult Face Mask Ventilation*

- History of prior difficulty
- Facial hair
- Obesity (BMI > 26 kg/m²)
- History of snoring
- OSA
- No teeth
- Age > 55 years
- Mallampati III or IV
- Limited mandibular protrusion
- Male gender
- Airway masses/tumors

**STEP 1b**
Assess the likelihood of airway management problems: *Predictors of Difficult Intubation*

- History of prior difficulty
- Underlying pathology (e.g. laryngeal/tracheal stenosis, epiglottitis, tumors)
- Neck range of motion (patient can’t touch chin to chest or extend neck)
- Thyromental distance (less than 3 finger breadths)
- Short, thick neck
- Long incisors
- Interincisor distance less than 3 cm
- Prominent “overbite”
- Highly arched or very narrow palate
- Decreased submandibular compliance (stiff, indurated, occupied by mass)
- Mallampati score (see next slide)

- Age
- Mental capacity
- Level of consciousness
- Obesity
- Facial hair
- Prior ENT surgery
- Prior radiation to neck

**STEP 1c**
Mallampati Score Assessment

**STEP 2**
Actively pursue opportunities to deliver supplemental O₂ throughout the process of difficult airway management:
- Face mask
- LMA
- FOB swivel adaptor ETT connector
- Patil-Syracuse mask (mask with fiberoptic port)
- FOB side port
- Rigid bronchoscope side port
- Nasal cannula (apneic oxygenation during intubation attempt)

**STEP 3**
Consider the relative merits and feasibility of basic management choices

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<td>Awake intubation vs. Intubation attempt after induction of GA</td>
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<td>B</td>
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<td>C</td>
<td>Preservation of spontaneous ventilation vs. Ablation of spontaneous ventilation</td>
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STEP 4

Develop primary and alternative strategies:

Algorithm A: Awake Techniques

- Awake FOI
- Awake DL
- Airway approached by non-invasive intubation
- Invasive airway access?
- Awake trach
- Cancel case
- Consider feasibility of other options?
- Awake trach
- Cricothyroidotomy
- Mask ventilation
- Local anesthetic
- Regional technique

Algorithm B: Intubation After Induction of GA

Non-Emergent Pathway
- CALL FOR HELP
- Mask ventilate with cricoid pressure
- Ensure optimal positioning
- Re-attempt DL with different blade
- Consider alternative techniques to secure airway
  • Gum elastic Bougie
  • LMA or intubating LMA
  • Video laryngoscope
  • Light wand
  • Fiberoptic intubation
  • Retrograde intubation

Emergent Pathway
- “Can’t intubate, can’t ventilate”
- CALL FOR HELP
- Emergency Non-Invasive Airway Ventilation
  • Rigid bronchoscopy
  • Combitube
  • Transtracheal Jet Ventilation
- Emergency Invasive Airway Ventilation
  • Cricothyroidotomy
  • Surgical tracheostomy

Algorithm B

Basics of Airway Management

Direct Laryngoscopy Views

Oral Airway  Nasal Airway
Airway Axis: “Sniffing” Position

Head elevation helps to align PA & LA before DL

Ramp obese patients until tragus is aligned with sternum

Pearls

- CALL FOR HELP
- Always pre-oxygenate (de-nitrogenate)
  - A pre-oxygenated patient can be apneic for 8-10 minutes until desaturation occurs
- The first attempt at DL is the best attempt
- Consider other airway options after 3 attempts at DL
  - Further attempts can cause airway edema and trauma
- Know airway anatomy
- Know pharmacology of anesthetic agents

References


The first time I had a patient with HIV, I was really nervous about putting in the IV. When I met him in preop, I was relieved that he had really great veins, and I knew he would be really easy. However, I kept missing IV after IV. After the third failed attempt, I finally paged my attending to come over. When he put on the tourniquet, I suddenly realized that that's what I had neglected to do in my previous attempts!

Fluid Management

5 minutes after manipulating an NGT that the surgeon insisted wasn't in the stomach (they always say this) when I knew it was because I was getting gastric contents (you always say this), the surgeon complains about a periodic whiff of a foul odor. We all started to notice it. I explained it was probably the gastric contents that leaked out when I was fiddling with the NGT. By the end of the 10 hour case, we pretty much all had some kind of pediatric face mask scent on our masks and everyone that came into our room complained of the smell out in the hall. Then off the came drapes and the horrible truth stared us in the face: The lower body bair hugger was making jerky out of a code brown so massive that it completely filled the void between the patient's legs.
**Evaluation of Intravascular Volume**

- **HPI**
  - Hypovolemia: vomiting, diarrhea, fever, sepsis, trauma
  - Hypervolemia: weight gain, edema, acute renal failure, liver disease (ascites)

- **Physical Exam (signs often unreliable)**
  - Hypovolemia: skin turgor, thready pulse, mucous membranes, tachycardia, orthostasis, axillary perspiration, decreased UOP
  - Hypervolemia: (in setting of CHF) pitting edema, rales, wheezing, cyanosis, elevated JVP

- **Labs**
  - Hypovolemia: rising Hct, contraction alkalosis then metabolic acidosis, Ur specific gravity > 1.010, Urine Na < 10, Urine Osm > 450, hypernatremia, BUN:Cr > 10:1
  - Hypervolemia: increased pulm vascular markings on CXR

**Intraoperative Intravascular Assessment**

- Monitor trends and compare multiple modalities to confirm clinical impressions

- **Vitals**
  - HR and BP (assess influence of positive pressure ventilation and anesthetics which may cause state of relative hypovolemia)
  - Pulse Oximetry: waveform wander from baseline (assuming patient normothermic and not in shock)

- **Foley Catheter**
  - UOP – consider that ADH levels may be increased 2/2 stress response to surgery (not reliable measure of volume status)

- **Arterial Line**
  - Serial ABGs to follow pH, Hct, electrolytes
  - PPV to assess for volume responsiveness, but has limitations
  - Commonly used when blood loss, fluid shifts, or prolonged OR time anticipated

**Fluid Compartments**

<table>
<thead>
<tr>
<th>Liquid</th>
<th>Fluid as % of TBW (%)</th>
<th>Fluid as % of body weight (%)</th>
<th>Volume, in 70 kg male (L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intracellular</td>
<td>67</td>
<td>40</td>
<td>28</td>
</tr>
<tr>
<td>Extracellular</td>
<td>33</td>
<td>20</td>
<td>26</td>
</tr>
<tr>
<td>TOTAL</td>
<td>100%</td>
<td>60%</td>
<td>42 L</td>
</tr>
</tbody>
</table>

**Q**: What is the intravascular volume of a 90 kg male?

**A**: 90 kg x 7% = 6.3 L

**Crystalloids**

<table>
<thead>
<tr>
<th></th>
<th>Osm (mOsm/L)</th>
<th>Na⁺ (mEq/L)</th>
<th>Cl⁻ (mEq/L)</th>
<th>K⁺ (mEq/L)</th>
<th>Ca²⁺ (mEq/L)</th>
<th>Buffer (mEq/L)</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NS</strong></td>
<td>308</td>
<td>154</td>
<td>154</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>5.0</td>
</tr>
<tr>
<td><strong>LR</strong></td>
<td>273</td>
<td>130</td>
<td>109</td>
<td>4</td>
<td>3</td>
<td>28 (lactate)</td>
<td>6.6</td>
</tr>
<tr>
<td><strong>plasmalyte</strong></td>
<td>294</td>
<td>148</td>
<td>98</td>
<td>5</td>
<td>0</td>
<td>27 (acetate)</td>
<td>7.4</td>
</tr>
</tbody>
</table>

**Advantages**

- Preferred for diluting pRBCs
- Preferred in brain injury

**Disadvantages**

- In large volumes produces hyperchloremic metabolic acidosis
- Hyperchloremia → low GFR

**Colloids**

**When to Consider Using Colloids**

- Initial intravascular volume resuscitation with crystalloid administration inadequate
- Concern that continued crystalloid may cause volume overload in certain clinical situations (ie. CHF, pulmonary edema, bowel edema)
- Patients with large protein losses and decreased oncotic pressure (burns), mostly benefit from albumin

**Mechanism**

- Intravascular volume expansion from increased oncotic pressure

**Dextran 40, 70**

- Not available in United States
Colloids

Hetastarch (6% hydroxyethyl starch, HES)
- Hespan (in NS) and Hextend (in LR) solutions
- Solution of highly branched glucose chains (average MW 450 kD)
- Degraded by amylase, eliminated by kidney
- Intravascular t1/2 = 25.5 hrs; tissue t1/2 = 10-15 days
- Maximum Dose: 15-20 ml/kg/day
- Side effects:
  - Can increase PTT (via factor VIII/vWF inhibition) and clotting times
  - Anaphylactoid reactions with wheezing and urticaria may occur
  - May interfere with platelet function
- Contraindications: coagulopathy, heart failure, renal failure

Albumin (5% and 25%)
- Derived from pooled donated blood after cold ethanol extraction and ultra-filtration; heat-treated (60 degree C x 10 hrs)
- Use 5% for hypovolemia; 25% for hypovolemia in patients with restricted fluid and Na intake
- Minimal risk for viral infection (hepatitis or HIV); theoretical risk of prion transmission
- Expensive, occasional shortages

Crystalloid or Colloid?

<table>
<thead>
<tr>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Crystalloid</strong></td>
<td><strong>Colloid</strong></td>
</tr>
<tr>
<td>Lower cost</td>
<td>Removes IV volume and HD with less volume, less time</td>
</tr>
<tr>
<td>Readily available</td>
<td>Longer IV t1/2</td>
</tr>
<tr>
<td>Dilutes plasma proteins</td>
<td>Maintains plasma oncotic pressure</td>
</tr>
<tr>
<td>Peripheral/pulmonary edema</td>
<td>Less cerebral edema (in healthy brain tissue)</td>
</tr>
<tr>
<td>May cause coagulopathy</td>
<td>Less intestinal edema</td>
</tr>
<tr>
<td>Expensive</td>
<td>Coagulopathy (dextran &gt; HES)</td>
</tr>
<tr>
<td>Type and Cross for pRBC and other blood products prior to surgery if anticipating significant blood loss</td>
<td></td>
</tr>
</tbody>
</table>

“Classical” Fluid Management

Maintenance
- "4-2-1 Rule" = 4 ml/kg/hr for the 1st 10 kg, 2 ml/kg/hr for the next 10-20 kg, and 1 ml/kg/hr for each additional kg above 20 kg.

Preexisting Fluid Deficits
- Multiply maintenance requirement by # of hours NPO.
- Give 1/2 over 1st hour, 1/4 over 2nd hour, and 1/4 over 3rd hour.
- Patients no longer undergo bowel preparation, so deficit decreased

Ongoing Losses
- Evaporative and Intestinal Losses (2/2 capillary leak)
  - Minimal tissue trauma (e.g. hernia repair) = 0-2 ml/kg/hr
  - Moderate tissue trauma (e.g. cholecystectomy) = 2-4 ml/kg/hr
  - Severe tissue trauma (e.g. bowel resection) = 4-8 ml/kg/hr
- Blood Loss
  - EBL = (suction canister - irrigation) + "laps" (100-150 ml each) + 4x4 sponges (10 ml each) + field estimate (very approximate estimation)
  - Replace with pRBCs, colloid, or crystalloid

Urine Output: Be aware of losses from increased urine output (diuretics, etc.)

Caveat: This is a general guide to help consider sources of volume loss and replacement, by no means the rule and not data driven as limited data exist

Suggestions for Fluid Management

- Tailor management to patient, surgery, and clinical scenario
- Use a balanced approach
  - Typically start with NS or LR
  - Switch to LR, except in neuro cases (because of decreased osmolality) or patients with hyperkalemia
  - Consider colloid for persistent hypotension despite adequate crystalloid administration.
- Type and Cross for pRBC and other blood products prior to surgery if anticipating significant blood loss (ie. trauma, coagulopathy); be aware that rapid volume resuscitation may worsen coagulopathy

Liberal vs. Restrictive Management

Consequences of Volume Overload
- Increased mortality and length of ICU/hospital stay
- Increased myocardial morbidity
- Increased pulmonary, periorbital, and gut edema
- Decreased hematocrit and albumin
- Worsened wound healing/ increased anastomosis dehiscence (2/2 edema)

Suggestions for Rational Fluid Management
- Use good clinical judgment.
- Tailor management to patient, surgery, and clinical picture.
- Maintain UOP > 0.5 ml/kg/hr, adequate CVP, and stable VS.
- Use balanced fluid therapy: use crystalloid for maintenance, consider use of colloid.
- Consider conservative replacement of interstitial losses or UOP unless VS unstable.

Burns

- Increased evaporative losses.
- H2O, electrolytes, and protein shift from normal to burned tissue, causing intravascular hypovolemia.
- Volume to infuse is calculated by the Parkland Formula

Parkland Formula
- Volume = %BSA x 4 ml/kg x kg
- Give 1/2 over the 1st 8 hours.
- Give 1/2 over the next 16 hours.
- Replace with LR.
- %BSA is determined by the “Rule of Nines”
**Intraoperative Oliguria**

1. **Pre-renal (decreased renal perfusion)**
   - Hypovolemia
   - Decreased CO (LV dysfunction, valvular disease)
   - Decreased MAP
   - Perfusion is compromised with increased intra-abdominal pressure (i.e. laparoscopy or abdominal compartment syndrome)

2. **Post-renal (post-renal obstruction)**
   - Foley kinked, clogged, displaced, or disconnected
   - Surgical manipulation of kidneys, ureters, bladder, or urethra

3. **Renal**
   - Neuroendocrine response to surgery (i.e. activation of renin-angiotensin-aldosterone system with increased ADH), is age dependent
   - Baroreceptor response to PPV also activates neuroendocrine response

**Treatments**
1. Relieve obstruction: check Foley; consider IV dyes (e.g. indigo carmine, methylene blue) to check for patency of ureters (i.e. Urology cases)
2. Increase renal perfusion: fluids (bolus vs increased maintenance rate), vasopressors/inotropes, or lasix

**References**

*The first time I emptied urine, it sprayed all over my scrubs. Apparently it’s better to aim the spout downwards into the empty bottle before you release the clamp, not up at yourself.*

**Transfusion Therapy**

**Type and Screen/Crossmatch**

**Type and Screen (takes 30-120 min, lasts 72 hr)**
- ABO-Rh typing and antibody screen
  - Recipient serum + type O RBCs for presence of A or B antibodies - no agglutination = negative screen
  - If antibody screen is positive: the serum is tested further
  - Recipient RBCs for presence of A or B antigens

**Type and Crossmatch (if T&S negative takes 30-60 min)**
- Immediate phase: recipient serum + donor cells test for recipient Ab to donor (5 minutes)
- Incubation phase: incubate products from first test to look for incomplete recipient Ab to donor (i.e. Rh system)
- Indirect Antiglobulin test: antiglobulin serum to products of first two tests to look for incomplete recipient Ab to Rh, Kell, Duffy, and Kidd

**Packed Red Blood Cells**

**Definition, Use, & Storage**
- Single donor; volume 250-300 ml with Hct ~70%.
- 1 unit pRBCs † adult Hgb ~1 g/dl or Hct ~3%.
- 10 ml/kg PRBC † Hct 10%
- Stored at 4°C in CPD (lasts 21 days), CPDA (lasts 35 days), or Adsol (lasts 42 days).
- **CPDA:**
  - Citrate (anticoagulant) - also binds iCa
  - Phosphate (buffer)
  - Dextrose (energy source)
  - Adenosine (precursor to ATP synthesis)
**Packed Red Blood Cells**

Indications (ASA Guidelines)
1. Hg < 6 in *young, healthy patients*
2. Usually unnecessary when Hg >10
3. At Hgb 6-10 g/dl, the decision to transfuse is based on:
   1. ongoing indications of organ ischemia
   2. potential or ongoing blood loss
   3. volume status
   4. risk factors for complications of inadequate O₂.

**Platelets**

Definition, Use, & Storage
- **Platelet Concentrate (PC)**
  - Platelets from one donated unit, vol = 50-70 ml; plt ~5000-10,000.
  - “6-pack” = 6 pooled PCs; rarely used anymore
- **Apheresis Unit**
  - Platelets from a single donor; vol = 200-400 ml; plt ~50,000.
  - Can give ABO-incompatible platelets, Rh tested only
  - Stored at room temperature for ≤5 days.
  - Hang separately – not through fluid warmer, level 1, or Belmont

Indications (ASA Guidelines)
1. Rarely when plt > 100,000
2. Usually when plt < 50,000 (spontaneous bleed at < 20K)
3. When plt 50-100,000, based on risk of bleeding
4. With platelet dysfunction (e.g. CPB, plt inhibitors)

**Fresh Frozen Plasma**

Definition, Use, & Storage
- Fluid portion from whole blood
- Contains all coagulation factors (except platelets)
- 1 unit increases clotting factors 2-3%
- Use ABO-compatible; Rh-incompatible is OK
- Stored frozen; takes 30 min to thaw; use within 24 hrs of thawing

Indications (ASA Guidelines)
1. Urgent reversal of Coumadin
2. Correction of known factor deficiency
3. Correction of 1) microvascular bleeding with INR > 1.5, 2) INR > 2, or 3) PTT > 2x normal
4. During massive transfusion (before lab results available)
5. Heparin resistance (i.e. antithrombin III deficiency) in patients requiring heparinization.

**Cryoprecipitate**

Definition
- Fraction of plasma that precipitates when FFP is thawed.
- Contains Factors VIII, XIII, I (fibrinogen), and fibroectin
- 1 unit contains ~6X more fibrinogen than 1 unit FFP.
- Use within 4-6 hours after thawed if you want to replace Factor VIII

Indications (ASA Guidelines)
1. Rarely when fibrinogen >150 mg/dl
2. When fibrinogen <100 mg/dl with microvascular bleeding
3. During massive transfusion when fibrinogen level not available
4. Bleeding patients with vWF disease
5. Congenital fibrinogen deficiency

**Equations**

**Arterial O₂ Content**

\[ C_{O_2} = O_{2-Hb} + Dissolved O_2 \]
\[ = (Hb \times 1.36 \times S_{O_2}/100) + (P_{O_2} \times 0.003) \]
\[ = (15 \times 1.36 \times 100\%) + (100 \times 0.003) \]
\[ = 20 cc O_2/dl \]

**Allowable Blood Loss**

\[ ABL = [ Hct (start) - Hct (allowed) ] \times EBV \]
\[ Hct (start) \]

**Volume to Transfuse**

\[ Volume = [ Hct (desired) - Hct (current) ] \times EBV \]
\[ Hct (transfused blood) \]

**Estimated Blood Volume (ml/kg)**

<table>
<thead>
<tr>
<th>Preemie</th>
<th>Term</th>
<th>&lt; 1 year</th>
<th>1-6 years</th>
<th>Male</th>
<th>Female</th>
<th>Obese</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>90</td>
<td>80</td>
<td>75</td>
<td>70</td>
<td>65</td>
<td>≤60</td>
</tr>
</tbody>
</table>

**Transfusion-Related Infections**

**Viral**
- CMV >1:100
- Hepatitis B 1 in 220,000
- Hepatitis C 1 in 1,600,000
- HIV 1 in 2,000,000
(Figures based on 2000-2001 estimated risk)

**Bacterial**
- Most common with *platelets* (1:2000) due to their storage in dextrose at room temperature.
- *pRBCs* not a major source (1:500,000) due to their storage at 4°C, but *Yersinia* is most likely organism.

Blood is screened for HCV, HBV core Ab, HIV-1, HIV-2, HTLV, syphilis
Transfusion Reactions

Febrile Non-Hemolytic Reaction
- Benign; occurs with 0.5-1% of transfusions
- R/O acute hemolytic reaction
- Treatment: Tylenol, Benadryl, supportive care

Allergic/Anaphylactic Reaction
- Occurs within minutes; life-threatening
- Signs/Symptoms: shock, angioedema, ARDS
- Treatment: D/C blood, fluids, Epi, antihistamines, ACLS

Acute Hemolytic Reaction
- Due to ABO incompatibility
- Symptoms (fever, chills, flank pain) masked by GA; watch for hypotension & brown urine; monitor for ARF and DIC.
- Treatment: D/C blood, maintain alkaline UOP (NaHCO₃-, mannitol, Lasix), supportive care.

Transfusion-Related Acute Lung Injury (TRALI)

TRALI
- An acute RDS that occurs ~4 hours after transfusion.
- Incidence: 1 in 1120 (but likely under-reported)
- Mortality 5-10% - Leading cause of transfusion-related mortality
- Due to plasma-containing products (platelets and FFP > pRBCs) - usually donor origin antibodies to leukocytes
- Signs & symptoms: Dyspnea, hypoxemia, hypotension, fever, pulmonary edema.
- Diagnosis of exclusion: first R/O sepsis, volume overload, and cardiogenic pulmonary edema
- Treatment: supportive care, similar to ARDS (O₂, mechanical ventilation, tidal volume 6-8 cc/kg). Diuretics are not indicated (etiology = microvascular leak, not fluid overload).
- TRALI is usually self-limited and resolves within 48 hours with supportive care.

Massive Transfusion

Definition
- Acute administration of greater than 1 blood volume (~10 units) in 24 hours.
- At Stanford, calling the blood bank for the Massive Transfusion Guideline (MTG) will get you 6 pRBCs, 4 FFP, and 1 unit of platelets.
- May take up to 30 minutes to have blood prepared and picked up for OR use.
- Plan ahead and use closed-loop communication with support staff.
- Typically will utilize Belmont, Level 1 or both for rapid infusion

Consequences
1. Hypothermia
   - Blood products are stored cold - use a fluid warmer! Connect tubing through Ranger Warmer.
2. Coagulopathy
   a. Dilutional thrombocytopenia
   b. Dilutional coagulopathies
      - Platelet count likely <100,000 after ~10 units pRBCs
      - Factors V & VIII (“labile factors”) in stored blood
      - Hypofibrinogenemia
3. Citrate Toxicity
   - Citrate is in CPDA storage solution as a Ca²⁺ chelator.
   - Massive transfusion can cause an acute hypocalcemia
   - Binds magnesium also causing hypomagnesemia
4. Acid-Base Abnormalities
   - At 21 days, stored blood has pH <7.0, due mostly to CO₂ production, which is rapidly blown off after transfusion.
5. Hyperkalemia
   - K⁺ moves out of pRBCs during storage.
   - If EKG changes occur, stop transfusion and treat hyperkalemia.
6. Impaired O₂-Carrying Capacity (?!?!)
   - 2,3-DPG decreases in stored blood, causing a left-shifted O₂-Hb dissociation curve.

References


Actual conversation in a case:
Nameless neurosurgeon (NN) “What’s the MAP”
Anesthesia Attending (AA) “65”
NN “Too high. Make it 55”
45 seconds later
AA “The MAP is now 55”
NN “That’s way too low. Make it 65 again”

Moral = sometimes you can just never win.
I was about to infiltrate a pt's arm with lidocaine for an IV, when both the patient and I both realized that he had an anaphylactic allergy to lidocaine! He recoiled in fear. I then proceeded to blow his IV without lidocaine.

### Hypoxemia

#### Causes of Hypoxemia

<table>
<thead>
<tr>
<th></th>
<th>P&lt;sub&gt;2&lt;/sub&gt;CO&lt;sub&gt;2&lt;/sub&gt;</th>
<th>A-a Gradient</th>
<th>DLCO</th>
<th>Corrects w/ 100% F&lt;sub&gt;O2&lt;/sub&gt;?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low F&lt;sub&gt;O2&lt;/sub&gt;</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Yes</td>
</tr>
<tr>
<td>Hypoventilation</td>
<td>↑</td>
<td>Normal</td>
<td>Normal</td>
<td>Yes</td>
</tr>
<tr>
<td>Diffusion Impairment</td>
<td>Normal</td>
<td>↑</td>
<td>↓</td>
<td>Yes</td>
</tr>
<tr>
<td>Shunt</td>
<td>Normal</td>
<td>↑</td>
<td>Normal</td>
<td>No</td>
</tr>
<tr>
<td>V/Q Mismatch</td>
<td>Normal / ↑</td>
<td>↑</td>
<td>Normal</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Shunt: perfusion without ventilation (V/Q=0), see ↑pCO<sub>2</sub>. No increase in pCO<sub>2</sub> (2/3 chemoreceptor mediated hypoventilation) until shunt fraction ≥ 30%.

Dead Space: ventilation without perfusion (V/Q=∞), see ↓pCO<sub>2</sub>

#### Equations

**Alveolar-arterial (A-a) Gradient**

\[
P_{(A-a)}O_2 = P_{A}O_2 - P_{a}O_2
\]

**Alveolar Gas Equation**

\[
P_{A}O_2 = F_{O2} (P_{atm} - P_{H2O}) - (P_{a}CO_2 / 0.8)
\]

\[
= 0.21 (760 - 47) - (40 / 0.8)
\]

\[
= 100 \text{ mm Hg}
\]

**Normal A-a Gradient:**

- ≤ 10 mm Hg (F<sub>O2</sub> = 0.21)
- ≤ 60 mm Hg (F<sub>O2</sub> = 1.00)
- ≤ (age / 4) + 4
- a/A ratio > 0.75

**Normal PaO<sub>2</sub>:**

- 103 - age/3

### Causes of Hypoxemia

1. **Low F<sub>O2</sub>**
   - Altitude (decreased barometric pressure)
   - Hypoxic F<sub>O2</sub> gas mixture (crossed gas lines, loss of pipeline pressure)

2. **Hypoventilation**
   - Drugs (opioids, benzodiazepines, barbiturates)
   - Chest wall damage
   - Neuromuscular diseases
   - Obstruction (e.g. OSA, upper airway compression)

3. **Diffusion Impairment**
   - Increased diffusion pathway (e.g. pulmonary edema, fibrosis)
   - Decreased surface area (e.g. emphysema, pneumonectomy)
   - Decreased rate of O<sub>2</sub>-Hb association (e.g. high CO, anemia, PE)

4. **Shunt** (i.e. perfusion w/o ventilation; V/Q = 0)
   - Congenital (e.g. ASD, VSD, PDA)
   - AVM (AVF, congenital)
   - Pulmonary fluid (pneumonia, CHF, ARDS, NPPV, TACO, TRALI)
   - Atelectasis (mucus plugging, bronchial intubation, GA)

5. **V/Q Mismatch**
   - Often multifactorial
   - COPD, ILD
   - Dead space (i.e. ventilation w/o perfusion; PE, surgical clamping)
   - Decreased CO (e.g. MI, CHF)

6. **Mixed Process**
   - Hypoxemia is often due to multiple causes.
   - Example: A tourist with COPD is visiting Denver, overdoses on heroin, now s/p MVA with chest wall trauma, pulmonary hemorrhage, Hct = 15%, and LV contusion. What is the cause of hypoxemia?
Hypoxemia in the OR
Take a systematic approach to the diagnosis and treatment of hypoxemia in the OR!

Suggestion: Alveoli → Machine

1. Listen to the lungs
   - Atelectasis (rales)
   - Pulmonary edema (rales, decreased BS)
   - Bronchoconstriction (wheezes, ↑PAP, shark-fin end-tidal CO2 tracing, ↑TV)
   - Mucus plug or secretions (↑PAP, ↑TV, mucus in ETT, rhonchi)
   - Right mainstem ETT (SpO2 ~90%, ↑PAP, ↑TV, unilateral BS. Repositioning, insufflation with laparoscopic procedures)
   - Pneumothorax (unilateral BS, ↑PAP, ↑TV, HD instability, tracheal deviation if tension physiology)
   - Esophageal intubation (no end-tidal CO2 tracing, BS in stomach & not lungs)

2. Check ET tube
   - Cuff deflation
   - Kinked/bitten or detached ET tube
   - Extubation (ENT/Neuro cases when bed turned 180, surgeons near head, leaning on ETTCircuit)

3. Check circuit
   - ETT disconnect
   - Circuit disconnect (check inspiratory/expiratory limbs at machine, connection near ETT, gas sampling line)

4. Check machine
   - Inspiratory & expiratory valves
   - Bellows
   - Minute ventilation
   - FiO2
   - Pipeline & cylinder pressures

5. Check monitors to confirm (you will probably do this 1st!)
   - Pulse oximeter waveform
   - Gas analyzer

O2-Hb Dissociation Curve

Useful “anchor” points:

<table>
<thead>
<tr>
<th>SO2</th>
<th>PO2 (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>50%</td>
<td>27</td>
</tr>
<tr>
<td>75%</td>
<td>40</td>
</tr>
<tr>
<td>97%</td>
<td>100</td>
</tr>
</tbody>
</table>

Note:
P50 ≈ 27 mm Hg

Factors Affecting Tissue Oxygenation

- Hb concentration
- O2 Saturation
- Cardiac Output
- O2 Consumption
- O2-Hb Affinity (P50)
- Dissolved O2 in plasma (little effect)

See “Equations” for a mathematical explanation of these factors.
Equations

**Arterial O₂ Content**
\[ \text{CaO}_2 = \text{O}_2-\text{Hb} + \text{Dissolved O}_2 \]
\[ = (\text{Hb} \times 1.36 \times \text{SaO}_2/100) + (\text{PaO}_2 \times 0.003) \]
\[ = (15 \times 1.36 \times 100\%) + (100 \times 0.003) \]
\[ \approx 20 \text{ cc O}_2/\text{dl} \]

**Mixed Venous O₂ Content**
\[ \text{CvO}_2 = \text{O}_2-\text{Hb} + \text{Dissolved O}_2 \]
\[ = (\text{Hb} \times 1.36 \times \text{SvO}_2/100) + (\text{PvO}_2 \times 0.003) \]
\[ = (15 \times 1.36 \times 75\%) + (40 \times 0.003) \]
\[ \approx 15 \text{ cc O}_2/\text{dl} \]

**O₂ Delivery**
\[ \text{DO}_2 = \text{CO} \times \text{CaO}_2 \]
\[ = 5 \text{ L/min} \times 20 \text{ cc O}_2/\text{dl} \]
\[ \approx 1 \text{ L O}_2/\text{min} \]

**O₂ Consumption (Fick Equation)**
\[ \text{VO}_2 = \text{CO} \times (\text{CaO}_2 - \text{CvO}_2) \]
\[ = 5 \text{ L/min} \times 5 \text{ cc O}_2/\text{dl} \]
\[ \approx 250 \text{ cc O}_2/\text{min} \]

**O₂ Extraction Ratio**
\[ \text{ER}_O_2 = (\text{VO}_2 / \text{DO}_2) \times 100 \]
\[ = 250 / 1000 \]
\[ \approx 25\% \text{ (normal 22-30\%) } \]

Other Concepts

**Diffusion Hypoxia** = low PaO₂ as a result of breathing air, in combination with the washout of N₂O into the alveoli, upon termination of an anesthetic.

**Absorption Atelectasis** = the tendency for airways to collapse if proximally obstructed; poorly soluble N₂ normally stents alveoli open, but patients on 100% O₂ have greater tendency toward atelectasis.

**Bohr Effect** = a property of Hb in which increasing CO₂, temperature, and acidosis promote decreased O₂-Hb affinity (i.e. right-shift of O₂-Hb curve).

**Haldane Effect** = a property of Hb in which O₂ promotes dissociation of CO₂ from Hb to the plasma (e.g. as when venous blood enters the lungs).

References


Electrolyte Abnormalities

In one of my first days of residency (I was at the Valley, where there are 5 or 6 different kinds of anesthesia machines), it took me about 10 minutes in the morning to find the power button for the ventilator. I felt pretty dumb. The problem ended up being that I had a towel draped over the tray and it was obscuring the otherwise direct view of the right button. But it's a humbling reminder that our job is a mix of complex physiology / pharmacology / etc. and very practical, mundane details. You can master all the ventilator physiology you want, but it won't do you much good if you can't turn the ventilator on.
Cardiac Action Potentials

<table>
<thead>
<tr>
<th>Phase</th>
<th>Phase Name</th>
<th>SA Node Fiber</th>
<th>Ventricular Muscle Fiber</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Rapid Upstroke</td>
<td>Slow inward $I_{Ca}$</td>
<td>Fast inward $I_{Na}$</td>
</tr>
<tr>
<td>1</td>
<td>Early Rapid Repolarization</td>
<td>–</td>
<td>Start outward $I_{K}$</td>
</tr>
<tr>
<td>2</td>
<td>Plateau</td>
<td>–</td>
<td>Slow inward $I_{Ca}$ = Outward $I_{K}$</td>
</tr>
<tr>
<td>3</td>
<td>Final Rapid Repolarization</td>
<td>Outward $I_{K}$</td>
<td>Inward $I_{Ca}$ &lt; Outward $I_{K}$</td>
</tr>
<tr>
<td>4</td>
<td>Diastolic Depolarization/Resting Potential</td>
<td>Slow inward $I_{Na}$</td>
<td>Outward $I_{K}$ (minimal)</td>
</tr>
</tbody>
</table>

Hyperkalemia

**Definition**
- Mild $K^+ = 5.5-6.5$ mEq/L
- Moderate $K^+ = 6.5-8$ mEq/L
- Severe $K^+ > 8$ mEq/L

**Contributing Factors**
- Renal disease
- Drugs (ACEI, NSAIDs, K-sparing diuretics, Digoxin, ß-blockers)
- Succinylcholine: acute increase of 0.5-1 mEq/L
- Acidosis
- Transfusions
- Hemolysis
- Rhabdomyolysis (tourniquet), trauma
- Administration of Dantrolene to patients on Verapamil or concurrent administration of both drugs
- Hyponatremia, hypocalcemia
- Old packed red blood cells

**Signs and Symptoms**
- Cardiac conducting system abnormalities including dysrhythmias, conduction abnormalities, and cardiac arrest.
  - Classically associated with administration of succinylcholine to paralyzed or burn patients.
  - If plasma $[K^+] < 6.0$ mEq/L, cardiac effects are generally negligible.
  - As the concentration increases, may see tall, peaked T waves, especially in the precordial leads.
  - With further increases, the PR interval becomes prolonged, followed by a decrease in the amplitude of the P wave.
  - Finally, the QRS complex widens into a pattern resembling a sine wave and eventually culminates in VF arrest and asystole
- At plasma $[K^+] > 7.0$ mEq/L, may have ascending paralysis that progresses to flaccid paralysis, inability to phonate, and respiratory arrest.
- Hyperkalemia may also accompany Malignant Hyperthermia.

**EKG Progression of Hyperkalemia**

<table>
<thead>
<tr>
<th>Serum Potassium</th>
<th>Typical ECG Appearance</th>
<th>Possible ECG Abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild (5.5-6.5 mEq/L)</td>
<td>Peaked T Waves</td>
<td>Prolonged PR Segment</td>
</tr>
<tr>
<td>Moderate (6.5-8.0 mEq/L)</td>
<td>Loss of P Wave</td>
<td>Prolonged QRS Complex</td>
</tr>
<tr>
<td>Severe (&gt;8.0 mEq/L)</td>
<td>Progressive Widening of QRS Complex</td>
<td></td>
</tr>
</tbody>
</table>

**Treatment**
- Reverse membrane effects
  - Ca gluconate (peripheral IV)
  - Ca chloride (central line)
- Transfer extracellular $[K^+]$ into cells
  - Bicarbonate (NaHCO$_3$) - 50-100 mEq over 5-10 minutes
  - Insulin (10-15 units) w/ Glucose (25 g)
  - Beta-2 agonists (Albuterol)
- Remove potassium from body
  - Kayexalate (PO/PR)
  - Diuretics (proximal or loop)
  - Dialysis

**Anesthetic Considerations**
- Consider cancelling elective cases if $K^+ > 5.5$
- Consider alternative to succinylcholine
- EKG monitoring
- Avoid hypoventilation (respiratory acidosis)
- Treat acidosis
- Use NS instead of LR
- Monitor for increased sensitivity to muscle relaxants
**Hypokalemia**

**Definition**
- Mild: $K^+ = 3.1-3.5$ mEq/L
- Moderate: $K^+ \leq 3$ mEq/L with PACs
- Severe: $K^+ < 3$ mEq/L with PVCs

**Contributing Factors**

- **Preoperative**
  - GI losses (NGT, N/V, Diarrhea)
  - Lasix, RTA
  - Magnesium deficiency

- **Intraoperative**
  - Alkalosis (both metabolic and respiratory)
  - Insulin therapy
  - Hypothermia

**Signs & Symptoms**
- Acute hypokalemia causes hyperpolarization of the cardiac cell and may lead to ventricular escape activity, re-entrant phenomena, ectopic tachycardias, and delayed conduction.
- Arrhythmias
  - PACs, PVCs
  - SVTs (esp. A Fib/A flutter)
- Metabolic alkalosis
- Autonomic lability
- Weakness, ↓DTRs
- Ileus
- Digoxin toxicity
- Enhanced response to muscle relaxants

**EKG Progression of Hypokalemia**

1. Flattened/inverted T wave
2. U waves
3. ST depression

**Treatment**

- Chronic hypokalemia = total body $K^+$ depletion ($1$ mEq/L decrease = $300-600$ mEq total body deficit)
  - Peripheral IV - $10$ mEq/hr
  - Central IV - $10-20$ mEq/hr
  - Life-threatening - $5-6$ mEq bolus
- Acute hypokalemia = likely a redistribution phenomenon
  - Reverse underlying cause (e.g. alkalemia secondary to mechanical hyperventilation)

**Hypokalemia Anesthetic Considerations**

- Consider cancelling elective cases if $K^+ < 3-3.5$ mEq/L (based on chronicity of deficit).
- EKG monitoring
- KCl replacement if arrhythmias develop
- Avoid hyperventilation (respiratory alkalosis)
- Consider reducing dose of muscle relaxant 25-50%

---

**Hypercalcemia**

**Contributing Factors**

- Hyperparathyroidism
- Malignancy (especially lung, ENT, GU, GYN, and multiple myeloma)
- Immobilization
- ARF
- Drugs (thiazide Ca²⁺ sparing diuretics, lithium)

**Signs & Symptoms**

- EKG changes (short QT)
- Hypertension
- Polyuria

**Treatment**

- Hydration (bolus crystalloid) + Lasix diuresis
- Dialysis
**Hypercalcemia**

**Anesthetic Considerations**
- Consider cancelling elective cases
- Avoid acidosis (reduces Ca²⁺-albumin binding)
- Check serial K⁺ and Mg²⁺

**Hypocalcemia**

**Contributing Factors**
- **Preoperative**
  - Hypoparathyroidism
  - Renal failure (decreased Vitamin D)
  - Sepsis
  - Magnesium deficiency (decreased end-organ response to PTH)
- **Intraoperative**
  - Alkalosis (increased Ca²⁺-albumin binding)
  - Massive pRBC transfusion (due to citrate binding)
  - Drugs (heparin, protamine, glucagon)

**Signs & Symptoms**
- EKG (prolonged QT, bradycardia)
- Hemodynamics (vasodilation, hypotension, decreased myocardial contractility, LV failure)
- Respiratory (laryngospasm, stridor, bronchospasm, respiratory arrest)
- Neuro (cramps, tetany, DTRs, perioral numbness, seizures, Chvostek’s sign, Trousseau’s sign)

**Treatment**
- Calcium gluconate - 1 g = 4.5 mEq elemental Ca²⁺ (give via peripheral or central IV)
- Calcium chloride - 1 g = 13.6 mEq elemental Ca²⁺ (give via central IV)
- Do NOT give Ca²⁺ and NaHCO₃ together in the same IV - it will precipitate!
- Replace magnesium

**Hypomagnesemia**

**Contributing Factors**
- GI/Renal losses
- β-agonists (cause intracellular shift)
- Drugs (diuretics, theophylline, aminoglycosides, amphi B, cyclosporin A)

**Signs & Symptoms**
- Usually asymptomatic alone, but symptomatic in combination with induced hypokalemia, hypocalcemia, and hypophosphatemia
- EKG (prolonged QT, PACs, PVCs, and A Fib)
- Neuro (neuromuscular excitability, AMS, seizures)

**Treatment**
- Replace with MgSO₄ to [Mg²⁺] > 2 mg/dl
- Watch for hypotension & arrhythmias with rapid administration!

**Anesthetic Considerations**
- EKG monitoring
- Check for coexistent electrolyte deficiencies.

**Hypermagnesemia**

**Contributing Factors**
- Renal failure
- Hypothyroidism
- Iatrogenic (tocolysis)

**Signs & Symptoms**
- EKG (widened QRS, prolonged PRI, bradycardia)
- Hemodynamics (vasodilation, hypotension, myocardial depression)
- Neuro (DTRs, sedation, weakness, enhanced neuromuscular blockade)

**Treatment**
- Hydration (bolus crystalloid) + Lasix diuresis
- Ca²⁺ administration
- Diuresis

**Anesthetic Considerations**
- EKG monitoring
- Consider reducing dose of muscle relaxants 25-50%

**References**
I was in the middle of a long, stable but tedious endometriosis case in the ASC. I tried to open my next vial of dilaudid and blam! It shattered in my hand and I had 2mg of dilaudid dripping down my fingers. Not wanting to be pegged as a CA-1 with a drug problem, I quietly called the pharmacy to ask them how to document the incident. The discussion took about a minute or so, and when I hung up, I realized the attending surgeon had stopped the case and was staring at me, as was everyone else in the room. He told me he gets “easily distracted” and so he was patiently waiting until I was off the phone!

During the middle of a straightforward case I was drawing up my drugs for the next case. I dropped the propofol vial but after inspection nothing was damaged. I proceeded to inject air into the vial making it easier to draw up. Needless to say it exploded on me......and the sterile operative field. Bummer.

CSI tip: In July, keep your eyes peeled for distinctive splatter patterns of white stuff on new residents’ scrubs, badges, or other paraphernalia. It is a sign that they, too, have been sprayed with either Propofol or Kefzol while trying to draw up a syringe. The needle tip has to stay inside the vial.

CSI tip: Don’t believe it if another CA1 has a BandAid on their finger or hand and they tell you they cut themself in the kitchen or have a paper cut. Odds are they stabbed themself with a needle drawing up drugs in the morning. Hope it was clean!

Hypothermia & Shivering

Definition and Measurement

- Hypothermia is defined as a core body temperature less than 36 degrees C
- Temperature is measured from:
  - Nasopharynx (accurately reflects core temp, but can cause epistaxis)
  - Tympanic Membrane (reflects brain temp, but can cause perforation of ear drum)
  - Esophagus
  - Bladder (lags behind core temperature with low urine flow)
  - Rectum (slow response to changes in core temp, inaccurate with stool in rectum, contraindicated in neutropenic pt, fistula, etc.)
  - Skin (variable accuracy depending on skin perfusion)
  - Thermistor of Pulmonary Artery Catheter

Thermoregulation

Afferent Thermal Sensing
- Thermal inputs travel along A-delta (cold) and C fibers (warm) via the spinothalamic tract.
- Input comes from the skin, deep abdominal & thoracic tissues, spinal cord, brain, and hypothalamus (roughly 20% each).

Central Control
- Thermal inputs are “preprocessed” at numerous levels within the spinal cord and brainstem.
- Modulated by NE, DA, 5-HT, ACh, PGE, and neuropeptides.
- The preoptic-anterior hypothalamus is the central autonomic thermoregulatory center.

Efferent Responses
- Behavioral responses (shelter, clothing, voluntary movement, etc) are most important and are determined by skin temperature.
- Autonomic responses (skin vasomotor activity, nonshivering thermogenesis, shivering, and sweating) are ~80% determined by core temperature.
Interthreshold Range

- **Interthreshold Range** = tight thermoregulatory range between cold-induced and warm-induced responses, usually ~0.2°C.
- **General anesthesia** inhibits thermoregulation and increases the interthreshold range ~20-fold, to ~4°C.
- **Regional anesthesia** inhibits thermoregulation to lower half of body, increasing the range ~4-fold, to ~0.8°C.

Development of Hypothermia

- Anesthetic-impaired thermoregulation
  1. Redistribution hypothermia
  2. Heat loss > heat production
  3. Heat loss = heat production (steady-state heat balance)

- Heat transfer to cold OR (in order of importance)
  1. Radiation
  2. Convection
  3. Evaporation
  4. Conduction

Benefits of Hypothermia

- Tissue metabolic rate decreases ~8% per 1°C decrease in body temperature.
- CNS protection from ischemic and traumatic injuries.
- Improves neurologic outcomes after cardiac arrest.
- Some protection against malignant hyperthermia.
- Cardiac protection as decreased metabolic and O2 requirement.

Consequences of Hypothermia

- Increased myocardial morbidity (3x)
- Impaired coagulation (especially platelets), increased blood loss, & increased transfusion rates
- Increased infection rate (3x)
- Prolonged duration of drug action, delayed emergence
- Left-shifts O2-Hb curve
- Increased SVR
- Difficulty monitoring patient (e.g. SpO2)
- Delays wound healing & jeopardizes grafts
- Altered mental status
- Increased sympathetic activity/stress response
- Increased postoperative shivering
- Prolonged PACU stay

Warming Strategies

**Prevention of hypothermia is more effective than treatment!**

**Active Warming**
- Forced air (Bar Hugger)
- Circulating warm H2O pad
- Radiant heat lamps
- IVF warmer
- Airway heating & humidification
- Warm the OR temperature

**Passive Insulation (not as effective)**
- Cotton blankets
- Surgical drapes
- Space blanket (silver plastic)

**Effect of Warming Strategies**

**Effect of IVF Warming**

Etiology of Postop Shivering

**Intraoperative hypothermia** (duh!)... however...
- Shivering does **NOT** always occur in hypothermic patients, and...
- Shivering **DOES** occur in normothermic patients

**Other possible etiologies:**
- Recovery from volatile anesthetics
- Pain may facilitate shivering-like tremor
- Fever increases the thermoregulatory set point causing shivering in normothermic patients.
Consequences of Shivering

- Increased O₂ consumption
  - Can be up to a 400-500% increase
- Increased CO₂ production and Vₐ
- Increased incidental trauma
- Increased intraocular and intracranial pressures
- Uncomfortable and/or painful
- Stresses wound edges
- Disrupts monitoring (e.g. NIBP, EKG, S₉O₂)

Rates of MI do NOT correlate with shivering!

Treatment of Shivering

1. Skin surface warming and passive insulation
2. Pharmacologic:
   - Meperidine 12.5-25 mg IV
   - Muscle relaxants (only in asleep, ventilated patients)

References


Postoperative Nausea & Vomiting (PONV)

Why do we care about PONV?

- Up to 1/3 of patients without prophylaxis will experience PONV (up to 80% among high-risk pts)
- Causes patient discomfort -- Patients report avoidance of PONV as a greater concern than post-op pain (willing to pay $56-100 out-of-pocket for effective PONV control)
- Prolonged PACU stay
- A leading cause of unanticipated hospital admission
- Possible aspiration risk and airway compromise
- Can lead to dehydration and electrolyte changes
- Can cause increased CVP, ICP, suture or mesh disruption, venous HTN and bleeding, or wound dehiscence

Evidence Based Risk Factors (Apfel et al., 2012)

- Christian Apfel (UCSF PONV guru) meta-analysis of 22 PONV studies (>95,000 pts)
- Highest risk factors:

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>OR (versus not having risk factor)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female Gender</td>
<td>2.57 (2.32-2.84)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>History of PONV/Motion Sickness</td>
<td>2.09 (1.90-2.29)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Non-smoking Status</td>
<td>1.82 (1.68-1.98)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Younger Age</td>
<td>0.88 per decade</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Use of Volatile Anesthetics</td>
<td>1.82 (1.56-2.13)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Post-op Opioids</td>
<td>1.39 (1.20-1.60)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
**Major Risk Factors**

**Patient-Related**
- Female > male
- History of PONV or motion sickness
- Young > old
- Non-smoker > Smoker

**Anesthetic-Related**
- Volatile anesthetics including N₂O
- Drugs (postoperative narcotics, neostigmine)
- Aggressive hydration (gut edema)

**Surgery-Related**
- Duration of surgery – higher risk if > 2 hours
- Type of surgery shown to have MINIMAL effect (once thought laparoscopic, ENT, neuro, breast, plastics, strabismus higher risk)

---

**Simplified Apfel Score**

**PONV Prophylaxis Based on Apfel Score**

<table>
<thead>
<tr>
<th>Risk Score</th>
<th>Prevalence PONV</th>
<th>Prophylaxis: No of Anti-emetics</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>9%</td>
<td>0-1</td>
<td>4 mg Ondansetron</td>
</tr>
<tr>
<td>1</td>
<td>20%</td>
<td>1</td>
<td>4 mg Ondansetron, 4mg Dexamethasone</td>
</tr>
<tr>
<td>2</td>
<td>39%</td>
<td>2</td>
<td>4 mg Ondansetron, 4mg Dexamethasone, 4mg Propofol infusion</td>
</tr>
<tr>
<td>3</td>
<td>50%</td>
<td>3</td>
<td>4 mg Ondansetron, 4mg Dexamethasone, 4mg Propofol infusion, 4 mg Scopolamine patch</td>
</tr>
<tr>
<td>4</td>
<td>78%</td>
<td>4</td>
<td>4 mg Ondansetron, 4mg Dexamethasone, 4mg Propofol infusion, 4 mg Scopolamine patch</td>
</tr>
</tbody>
</table>

- Combinations should be with drugs that have a different mechanism of action
- Try not to order agents for treatment in PACU that have already been used for ppx

---

**Antiemetic Classes**

**5-HT₃ Antagonists (e.g. Ondansetron, Granisetron)**
- Serotonin receptor antagonist
- More effective at preventing emesis than nausea
- All agents equally effective
- Zofran 4-8 mg IV or Kytril 0.1-1 mg IV before end of case (usually given ~30 minutes before emergence)

**Steroids**
- Cheap and effective
- Can be given anytime, for prolonged PONV relief
- Weigh risks/benefits in diabetics
- Decadron 4-10 mg IV anytime during case

**Gastrokinetic (e.g. Metoclopramide)**
- Dopamine antagonist, can cause extrapyramidal SEs
- Increases GI motility and LES tone, avoid in patients with bowel obstruction
- Reglan 20 mg IV before end of case
- Contraindicated in Parkinson’s patients

---

**Other Antiemetic Agents**

**Vasopressors**
- Ephedrine 50 mg IM
  - Prevents gut hypoperfusion

**Induction agents**
- Propofol 10-20 mg IV bolus in PACU vs low-dose infusion during case

**Antihistamines (H₂-blockers)**
- Cimetidine 300 mg IV
- Ranitidine 50 mg IV
**Chemoreceptor Trigger Zone**

**IMPACT Trial: Study Design** (Apfel et al., 2004)

- 5161 patients, 6 treatments ($2^6 = 64$ treatment groups)

**Randomization**
- Remifentanil gtt
- Fentanyl

**Induction & Intubation**
- $30\% \text{O}_2 + \text{N}_2$
- $80\% \text{O}_2 + \text{N}_2$
- $30\% \text{O}_2 + \text{N}_2\text{O}$

**Volatile Anesthetic**
- Propofol gtt
- +/− Dexamethasone 4 mg
- +/− Droperidol 1.25 mg
- +/− Ondansetron 4 mg

**Maintenance**
- 20 minutes after start
- +/- Dexamethasone 4 mg
- +/- Droperidol 1.25 mg
- +/- Ondansetron 4 mg

**Algorithm for PONV Treatment**

- Decrease risk of PONV in high-risk patient
- Consider regional anesthesia (RA)
- General anesthesia is used; reduce because of risk factors and consider using nonpharmacologic therapies (Y)
- Patients at moderate risk
- Consider antiemetic prophylaxis with monotherapy (adults) or combination therapy (children and adults) (Y)
- Patients at low risk

**IMPACT Trial: Results** (Apfel et al., 2004)

<table>
<thead>
<tr>
<th>Intervention</th>
<th>RR Reduction</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dexamethasone (vs. none)</td>
<td>26.4%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Ondansetron (vs. none)</td>
<td>26.0%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Droperidol (vs. none)</td>
<td>24.5%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Nitrogen carrier (vs. N$_2$O)</td>
<td>12.1%</td>
<td>0.003</td>
</tr>
<tr>
<td>Propofol gtt (vs. volatiles)</td>
<td>18.9%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Remifentanil gtt (vs. fentanyl)</td>
<td>-5.2%</td>
<td>0.21</td>
</tr>
</tbody>
</table>

- Interventions acted independently of each other; relative risk reduction (RRR) of combined therapy can be estimated by multiplying individual RRrs.
- Average PONV = 34% (59% with volatile + N$_2$O + remi + no antiemetics; 17% with propofol + N$_2$ + fentanyl + antiemetics x 3).
- Use the safest and cheapest antiemetic first; use combined therapy only in moderate or high-risk patients.

**Strategies to Reduce PONV**

- Use regional anesthesia vs. GA
- Use propofol for induction and maintenance of anesthesia
- Avoid N$_2$O and/or volatile anesthetics
- Minimize opioids (consider tyleanol, NSAIDs, etc.)
- Minimize (<2.5 mg) or eliminate neostigmine
- Maintain euvolemia; avoid hypervolemia (gut edema)
- Avoid hypotension and cerebral hypoxia
- Use a combination of antiemetics in different classes
- Consider acupuncture, acupressure, or transcutaneous electrical nerve stimulation (rarely used)

**References**

Extubation Criteria & Delayed Emergence

**Extubation Criteria - OR**

1. **Adequate Oxygenation**
   - $\text{SpO}_2 > 92\%$, $P_{O_2} > 60$ mm Hg

2. **Adequate Ventilation**
   - $V_t > 5$ ml/kg, spontaneous RR $> 7$ bpm, $ET_{CO_2} < 50$ mm Hg, $P_{CO_2} < 60$ mm Hg

3. **Hemodynamically Stable**

4. **Full Reversal of Muscle Relaxation**
   - Sustained tetany, TOF ratio $> 0.9$ (cannot be accurately assessed visually)
   - Sustained 5-second head lift or hand grasp

5. **Neurologically Intact**
   - Follows verbal commands
   - Intact cough/gag reflex

6. **Appropriate Acid-Base Status**
   - pH $> 7.25$

7. **Normal Metabolic Status**
   - Normal electrolytes
   - Normovolemic

8. **Normothermic**
   - Temp $> 35.5^\circ$C

9. **Other Considerations**
   - Aspiration risk
   - Airway edema
   - Awake vs. Deep (i.e. NOT in Stage II)

**Extubation Criteria - ICU**

**Subjective Criteria**
- Underlying disease process improving.

**Objective Criteria**
- Adequate mentation (GCS $> 13$, minimal sedation)
- Hemodynamically stable, on minimal pressors (e.g. dopamine $< 5$ mcg/kg/min)
- $\text{SpO}_2 > 90\%$, $P_{O_2} > 60$ mm Hg, $P_{O_2}/F_{O_2} > 150$ on PEEP $< 5-8$ cm H$_2$O and $F_{O_2} < 0.4-0.5$
- $P_{CO_2} < 60$ mm Hg, pH $> 7.25$

**Ventilator Criteria (during SBT)**
- RSBI ($RR/V_t$) $< 100$, NIF $> 20$ cm H$_2$O
- $V_t > 5$ ml/kg, VC $> 10$ ml/kg
- RR $< 30$ bpm

**Potential Difficult Extubation**
- History of difficult intubation
- OSA
- Maxillofacial trauma
- Generalized edema (e.g. prolonged surgery with significant fluid/blood resuscitation)
- Paradoxical vocal cord motion (preexisting)
- Post-procedural complications:
  - Thyroid surgery ($\sim 4\%$ risk of RLN injury, late hypocalcemia)
  - Diagnostic laryngoscopy +/- biopsy (laryngospasm, edema)
  - Uvulopalatoplasty (edema)
  - Carotid endarterectomy (hematoma, nerve palsies)
  - ENT surgeries (hematoma, jaw wires)
  - Cervical decompression (edema)

**Approach to Difficult Extubation**
- If intubation was technically difficult (e.g. multiple DLs, FOI), consider maintaining a “pathway” to the trachea (e.g. bougie, FOB, Airway Exchange Catheter).
- If airway edema is suspected due to fluids or traumatic intubation, consider performing a “Cuff-Leak Test”
  - Deflate cuff, occlude ETT, observe whether patient can breath around the tube.
  - A failed leak test does NOT always lead to failed extubation, but may warrant further patient observation; likewise, passing a leak test does NOT guarantee successful extubation.
**Stages of Anesthesia**

Historical terminology to describe depth of anesthesia upon gas induction. Today, more important for emergence.

**Stage 1**
- Sedated, intact lid reflex, follows commands

**Stage 2**
- Excited/disinhibited, unconscious, unable to follow commands or exhibit purposeful movement
- Irregular breathing & breath-holding, dilated & disconjugate pupils, conjunctival injection
- Increased incidence of laryngospasm, arrhythmias, and vomiting.

**Stage 3**
- Surgical anesthesia

**Stage 4**
- Medullary depression, cardiovascular/respiratory collapse

---

**Delayed Emergence**

**Definition**
- Failure to regain consciousness as expected within 20-30 minutes of the end of a surgical procedure – with all anesthetic off.

**Causes**
1. Residual drug effects
   - Absolute or relative overdose
   - Potentiation of agents by prior intoxication (e.g. EtOH, illicit drugs) or medications (e.g. clonidine, antihistamines)
   - Organ dysfunction (e.g. renal, liver) interfering with metabolism/excretion.
2. Hypercapnia and/or Hypoxemia
3. Hypothermia (<34˚C)
4. Hypo-/Hyperglycemia
5. Metabolic Disturbances
   - Acid-base, hyponatremia, hypo-hypercalcemia, hypomagnesemia
6. Organ Dysfunction
   - Renal failure, liver failure (e.g. hepatic encephalopathy)
7. Neurologic Insults
   - Seizure/post-ictal state
   - Increased ICP
8. Perioperative Stroke
   - Risk factors: AFib, hypercoagulable state, intracardiac shunt
   - Incidence: 0.1-0.4% in low-risk procedures; 2.5-5% in high-risk procedures

---

**Diagnosis and Treatment**

Ensure adequate oxygenation, ventilation, and hemodynamic stability first, then proceed with:

1. Administer “reversal agents”
   - Naloxone 0.40mg – 2mg IV Q 2-3 minutes. (Can dilute to give in 0.04mg increments)
   - If no response after 10 mg, reconsider narcotic overdose as cause of delayed emergence
   - Flumazenil 0.2 mg IV bolus Q 45-60 seconds over 15 seconds
     - May repeat doses. Maximum of 1 mg IV bolus. No more than 3 mg total in one hour.
   - Physostigmine 1-2 mg IV (for central cholinergic syndrome)
   - Neostigmine – maximum of 5 mg IV. Give with glycopyrrolate.
2. Ensure patient is normothermic
   - Use Bair Hugger, warm the room
3. Check ABG for PaO₂, PaCO₂, glucose, and electrolytes
4. Consider neurological insults – discuss with primary surgeon
   - Perform pertinent neurologic exam
   - Consider further workup (e.g. CT, MRI, EEG)
   - Consider Neuro consult

---

**References**

- MacIntyre NR et al. 2001. Evidence-based guidelines for weaning and discontinuing ventilatory support: a collective task force facilitated by the ACCP, AARC, and the ACCCM. *Chest*, **120**: 375S-95S.
- Rashad Net University (www.rashaduniversity.com/delem.html)

---

At the end of a general anesthesia case with a 60 yo male patient, I wheeled him into the PACU and he looked straight at me and very seriously said, "So, can I have your number?" His wife was in the waiting room, and I was 7 months pregnant. Classic VA.
Laryngospasm & Aspiration

Larynx Anatomy

Larynx Anatomy: Innervation

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Motor</th>
<th>Sensory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recurrent Laryngeal (from CN X)</td>
<td>Thyroarytenoid (tensor)</td>
<td>Subglottic mucosa</td>
</tr>
<tr>
<td></td>
<td>Lateral Cricoarytenoid (adductor)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Transverse Arytenoid (adductor)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Posterior Cricoarytenoid (abductor, tensor)</td>
<td></td>
</tr>
<tr>
<td>Superior Laryngeal (from CN X)</td>
<td>Internal branch</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>Epiglottis/Tongue Base</td>
<td>Supraglottic mucosa</td>
</tr>
<tr>
<td></td>
<td>External branch</td>
<td>Cricothyroid (adductor)</td>
</tr>
</tbody>
</table>

Does bilateral recurrent laryngeal nerve injury produce the same defect as succinylcholine?

What is laryngospasm?
- Closure of the true vocal cords (+/- the false vocal cords) from the action of laryngeal muscles → occlusion of the glottis/laryngeal inlet
- Consequences include hypoxia, hypercapnia, and negative pressure pulmonary edema

Predisposing Factors
- Stage 2 of anesthesia (excitement/delirium)
- Light anesthesia relative to surgical stimulation
- Mechanical irritants to the airway
  - Blood or secretions
  - Airway suctioning or instrumentation
- GERD
- Upper respiratory tract infection (0.85-5% incidence)

Prevention
- Ensure adequate anesthetic depth before manipulation or movement of patient
- Clear secretions before extubation
- Topicalize larynx with local anesthetic
- Muscle relaxants

Management - CALL FOR HELP EARLY!
1. Jaw thrust, head tilt, oral or nasal airway
2. Deepen anesthesia with IV agent (e.g. Propofol)
3. CPAP via bag-mask ventilation with 100% O₂
4. Suction oropharynx
5. Succinylcholine 10-20 mg IV, maintain airway with bag-mask or ETT until spontaneously breathing
6. Prepare for surgical airway
7. Monitor for post-obstructive negative pressure pulmonary edema (NPPE)
**Negative Pressure Pulmonary Edema**

**Causes**
- Laryngospasm
- Upper airway obstruction/ETT obstruction
- Incidence of 0.1% of anesthetics

**Risk Factors**
- Laryngospasm
- Young (20-40 years), healthy (ASA I-II), male (80%)

**Presentation**
- Laryngospasm, chest wall retraction
- Frothy, serosanguinous or bloody airway secretions
- ↓SpO₂, ↑ETCO₂, hypotension, large P(A-a) gradient
- CXR with pulmonary edema

**Pathogenesis**
- Negative intrathoracic pressure (up to 100 cmH₂O)
- RV preload ≥ pulmonary hydrostatic pressure
- RV preload → interventricular septum shift → LV diastolic dysfunction → ↑PCWP
- Hypoxia, hypercapnea, acidosis → HPV & ↑PVR
- Stress response → ↑SVR and ↑LV afterload
- Alveolar-capillary membrane leak → protein loss

**Treatment**
- Supportive care (O₂, IPPV, PEEP/CPAP)
- Conservative management until process reverses; consider volume and/or pressors PRN.
- Lasix is usually NOT helpful.

---

**Pulmonary Aspiration**

**Predisposing Conditions**
- Full stomach or unknown NPO status (e.g. trauma)
- Intra-abdominal process (bowel obstruction, ileus, inflammation)
- Gastroparesis (narcotics, DM, uremia, EtOH, infection)
- GE junction incompetence (GERD, hiatal hernia, scleroderma)
- Pregnancy, obesity
- Neuromuscular disease processes
- Difficult intubation and/or prolonged bag-mask ventilation

**Prevention**
- Follow NPO guidelines for routine elective cases
- Use metoclopramide, H₂-blockers, and antacids in high-risk patients
- Consider awake, regional anesthetic
- Consider awake, upright intubation and/or RSI
- If present, leave NGT to suction
- Apply cricoid pressure until ETT position confirmed
- Minimize bag-mask PPV and/or keep pressure <20 cmH₂O
- Extubate after recovery of protective reflexes

**Aspiration Pneumonitis**
- Sterile, chemical pneumonitis caused by aspiration of acidic and particulate material
- Highest risk in patients with gastric volume >25 ml and pH <2.5.
- Aspiration does NOT always cause pneumonia!

**Management**
- Place patient in head-down position
- Immediately suction pharynx and trachea before PPV
- 100% O₂, intubate, apply PEEP or CPAP
- Supportive care - monitor for chemical PNA/ARDS
- Possible bronchoscopy for removal of particulate matter, if suspected
- Antibiotics are not necessary unless subsequent infection develops (or, as happens more commonly in pediatrics, fecal matter is aspirated)
- Steroids are not indicated

---

**NPO Guidelines**

<table>
<thead>
<tr>
<th>Ingested Material</th>
<th>Minimum Fasting Period</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clear</td>
<td>2 hours</td>
</tr>
<tr>
<td>Breast Milk</td>
<td>4 hours</td>
</tr>
<tr>
<td>Formula</td>
<td>6 hours</td>
</tr>
<tr>
<td>Non-human Milk</td>
<td>6 hours</td>
</tr>
<tr>
<td>Light Meal</td>
<td>6 hours</td>
</tr>
<tr>
<td>Fatty Meal</td>
<td>6-8 hours</td>
</tr>
</tbody>
</table>

*There is no evidence for the routine use of metoclopramide, H₂-blockers, proton pump inhibitors, antiemetics, or anticholinergics in preventing aspiration or in reducing its morbidity/mortality.*

*If given preoperatively, only nonparticulate antacids (Sodium Citrate) should be used.*
Oxygen Failure in the OR

Etiology

Loss of Pipeline Oxygen
- Exhaustion of central O₂ supply.
- Obstruction of central O₂ supply line to OR.
- O₂ shutoff valve in OR is off.
- Obstruction or disconnection of O₂ hose in the OR.
- Failure of O₂ regulator in the anesthesia machine.

Faulty Oxygen Supply
- Crossing of pipelines during construction/repairs.
- Incorrect connection of gas hoses.
- Non-O₂ cylinder at the O₂ yoke.
- Wrong gas in the O₂ cylinder.
- Broken flowmeter.

Prevention

Pre-anesthesia Machine Check
- Check pipeline pressure ~50 psi.
- Check O₂ tanks >50% full.
- Calibrate O₂ analyzer.

Supply-Side Safety Features
- Color-coded gas tanks
- DISS, PISS, and Quick Connects

Anesthesia Machine Safety Features
- Flow-meter arrangement
- O₂:N₂O ratio controller
- Oxygen supply failure protection device ("fail-safe valve")

Gas Cylinders

<table>
<thead>
<tr>
<th>Gas</th>
<th>E-Cylinder Capacity (L)</th>
<th>Pressure (psi)</th>
<th>Color (USA)</th>
<th>Color (Int'l)</th>
<th>Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>O₂</td>
<td>660</td>
<td>1900</td>
<td>Green</td>
<td>White</td>
<td>Gas</td>
</tr>
<tr>
<td>Air</td>
<td>625</td>
<td>1900</td>
<td>Yellow</td>
<td>White &amp; Black</td>
<td>Gas</td>
</tr>
<tr>
<td>N₂O</td>
<td>1590</td>
<td>745</td>
<td>Blue</td>
<td>Blue</td>
<td>Liquid + Gas</td>
</tr>
<tr>
<td>N₂</td>
<td>650</td>
<td>1900</td>
<td>Black</td>
<td>Black</td>
<td>Gas</td>
</tr>
</tbody>
</table>

How long can you use an O₂ tank starting at 430 psi running at 5 L/min?

Diameter Index Safety System
Pin Index Safety System

Flowmeter Arrangement
- A leak in the upstream O₂ flowmeter (“Incorrect sequence”) results in a hypoxic gas mixture.
- A leak in the Datex-Ohmeda or Draeger flowmeter arrangements may deliver less Air or N₂O than expected, but the mixture will NOT be hypoxic because O₂ is closest to the FGF outlet.

O₂:N₂O Ratio Controller

Oxygen Failure Protection Device
If P₀₂ falls, N₂O cannot flow!

Detection
- Pressure gauges fall (pipeline, tanks)
- Low O₂ alarms (O₂ supply failure, F₁O₂ analyzer)
- Flowmeters fall (O₂ and other gases)
- O₂ flush inoperative
- Bellows inoperative
- Apnea alarms (spirometer, capnograph)
- Increasing O₂ flow makes the problem worse
- Hypoxemia, hypercarbia
- Arrhythmias, bradycardia, cardiac arrest

Management
- Notify surgeon, call for help.
- Verify problem (pressure gauges, flowmeters, O₂ flush, O₂ analyzer, capnograph).
- Switch to O₂ cylinder (calculate remaining time).
- Use manual ventilation to conserve O₂.
- Check valves, hoses, couplers.
- D/C supply lines if crossed pipelines suspected.
- Call for backup O₂ tanks.
- Close breathing circuit, manually ventilate.
- Switch to self-inflating bag (Ambu-Bag), Jackson-Reese with external tank, or mouth-to-ETT if necessary.
- Consider switching to TIVA until cause of failure is known.
References


Anaphylaxis

Overview

- Allergic reactions are an important cause of intraoperative morbidity and mortality (3.4% mortality)
- Account for approximately 10% of all anesthetic complications
- More than 90% of reactions occur within 3 minutes but can be delayed by hours with variable presentation
- Can be difficult to identify cause as multiple drugs are given early in anesthetic
- Usually the faster the reaction, the more severe the course
- Anaphylaxis involves a combination of systemic (pulmonary, CV, GI) and dermal signs & symptoms, all due to release of vasoactive mediators which:
  - Increase mucous membrane secretions
  - Increase bronchial smooth muscle tone
  - Decrease vascular smooth muscle tone and increase capillary permeability
- Anaphylactic and anaphylactoid reactions present similarly and are treated IDENTICALLY

Anaphylaxis vs. Anaphylactoid

Anaphylaxis

- IgE-mediated Type I hypersensitivity reaction
- Sensitization = prior exposure to an antigen which produces antigen-specific IgE antibodies that bind to Fc receptors on mast cells and basophils.
- Upon re-exposure to the antigen, IgE antibodies then cross-link Fc receptors causing degranulation and release of stored mediators (vasoactive)
- Reaction is *Dose Independent*!

Anaphylactoid

- Direct activation of mast cells and basophils by non-IgE mechanisms or activation of complement system
- May occur on 1st exposure to an antigen

Sequence of Events

![Sequence of Events Diagram]

Common Precipitants

<table>
<thead>
<tr>
<th>Substance</th>
<th>Incidence of perioperative anaphylaxis (%)</th>
<th>Most commonly associated with perioperative anaphylaxis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle relaxants</td>
<td>66.5</td>
<td>Vecuronium, atracurium, rocuronium (Roc &gt; Vec &gt; Cis &gt; Sux)</td>
</tr>
<tr>
<td>Natural rubber latex</td>
<td>12.1</td>
<td>Latex gloves, tourniquets, Foley catheters</td>
</tr>
<tr>
<td>Antibiotics</td>
<td>8</td>
<td>Penicillin and other β-lactams</td>
</tr>
<tr>
<td>Hypnotics</td>
<td>3.7</td>
<td>Propofol, hypnotics</td>
</tr>
<tr>
<td>Coagulants</td>
<td>3.7</td>
<td>Dextran, heparin</td>
</tr>
<tr>
<td>Opioids</td>
<td>1.4</td>
<td>Morphine, meperidine</td>
</tr>
<tr>
<td>Other substances</td>
<td>3.0</td>
<td>Propacetamol, aprotinin, chymopapain, protamine, bupivacaine</td>
</tr>
</tbody>
</table>

*Table 3. Drugs Involved in Perioperative Anaphylaxis*
### Latex Allergy

- Obtain a careful history:
  - Healthcare workers
  - Children with spina bifida
  - Urogenital abnormalities (history of multiple urogenital catheters)
  - Food allergies (mango, kiwi, avocado, passion fruit, bananas)
- Establish a latex-free environment:
  - Schedule patient as first case of the day
  - Most equipment & supplies are latex-free; if available, have a cart of latex-free alternatives available
  - Remove tops of multi-dose vials when drawing up drugs
- Prophylactic steroids and/or H1-blockers (uncertain benefit)
- Prepare for the worst, hope for the best

### Sign and Symptoms

<table>
<thead>
<tr>
<th>System</th>
<th>Symptoms (e.g. MAC/Regional)</th>
<th>Signs (e.g. General or Regional)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory</td>
<td>Dyspnea</td>
<td>Hypoxia Pulmonary edema</td>
</tr>
<tr>
<td></td>
<td>Chest tightness</td>
<td>Wheezing Laryngeal edema</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Dizziness</td>
<td>Hypotension Dysrhythmias Pulmonary HTN</td>
</tr>
<tr>
<td></td>
<td>LOC</td>
<td>Tachycardia Cardiac arrest</td>
</tr>
<tr>
<td>Cutaneous</td>
<td>Itching</td>
<td>Perioral edema Flushing</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Periorbital edema Hives</td>
</tr>
<tr>
<td>Renal</td>
<td></td>
<td>Decreased urine output</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Nausea, vomiting, diarrhea</td>
<td></td>
</tr>
<tr>
<td>Hematologic</td>
<td></td>
<td>DIC</td>
</tr>
</tbody>
</table>

Anaphylactic reactions may have variable presentations with some or all of these signs & symptoms.

### Management

**Acute Phase**
1. Stop administration of offending antigen
2. Notify surgeon AND call for help
3. Maintain airway; give 100% O2
4. In cases of severe cardiovascular collapse, consider discontinuation of all agents that may augment hypotension such as inhaled anesthetics (via vasodilation) & narcotic infusions (via suppressing sympathetic response).
5. Give other amnestic agents (e.g. scopolamine, midazolam)
6. Epinephrine = drug of choice due to alpha-1 supports BP; beta-2 bronchial smooth muscle relaxation
   1. Start 5-10 mcg IV boluses for hypotension; 0.1-0.5 mg IV PRN CV collapse. Escalate as needed.
   2. If no IV access, give 0.3-0.5 mg IM in anterolateral thigh, repeat q5-15 min
   3. ACLS doses (0.1-1 mg) for cardiovascular collapse

**Secondary Treatment**
- Intubation
- Invasive lines: large-bore IVs, arterial line, central venous catheter, Foley catheter
- Drugs
  - H1-blocker - diphenhydramine 0.5-1 mg/kg IV
  - Steroids – decrease airway swelling, prevent recurrent sx in biphasic anaphylaxis
    - Hydrocortisone 0.25-1 g IV, or methylprednisolone 1-2 g IV
    - Epinephrine gtt - start 50-100 ng/kg/min (4-8 mcg/min)
      - (Epi minidrip - 1 mg in 250 ml NS = 4 mcg/ml; run at 60 microdrips/min = 4 mcg/min; titrate to effect)
    - H2-blockers - not a first-line agent, but not harmful either!
    - Bicarbonate - 0.5-1 mEq/kg IV, as needed
    - Inhaled bronchodilator (Albuterol)

**Prevention**
- Obtain a careful history:
  - Previous allergic reactions?
  - Atopy or asthma?
  - Food allergies?
- Test dose drugs followed by slow administration
  - reduces anaphylactoid, but not anaphylactic reactions
- Judicious use of blood products
- Use prophylactic steroids and/or H1-blockers
  - H1-blockers: no clear benefit; may blunt early signs before presenting as full-blown episode.
- If no alternative agent, may pursue desensitization.
- Obtain consultation from an allergist if necessary.

**Testing for an Allergy**
- Testing may not be necessary if there is a clear temporal association between drug and reaction
- Measurement of serum mast cell tryptase levels can help establish the diagnosis in uncertain cases of anaphylaxis.
- Follow up with an allergist may be useful for establishing a diagnosis (e.g. skin testing)
Local Anesthetics

- Provide anesthesia and analgesia by disrupting the conduction of impulses along nerve fibers
- Block voltage-gated sodium channels
  - Reversibly bind the intracellular portion of these neuronal channels
  - Inhibit the influx of sodium, thus preventing an action potential from being reached
  - Resting membrane and threshold potentials are not affected

Local Anesthetic Structure

- Three Major Chemical Moieties:
  - Lipophilic aromatic benzene ring
  - Ester or amide linkage
  - Hydrophilic tertiary amine

- Local anesthetics are weak bases
  \[ \text{pK}_a > 7.4 \]

Physiochemical Properties

- At physiologic pH, local anesthetics are in equilibrium:
  \[ \text{Ionized (water-soluble)} \leftrightarrow \text{nonionized (lipid-soluble)} \]

- The ratio of the 2 forms depends on the pKa of the drug and the tissue pH

- Nonionized (base, lipid-soluble) form crosses the neuronal membrane
- Re-equilibration between the 2 forms occurs in the axoplasm
- Ionized (cationic, water-soluble) form binds to the Na channel
Physiochemical Properties

- Potency is related to lipid solubility
- Duration of action is related to protein binding
- Speed of onset is related to pKa (degree of ionization)
- Other factors involved: dosage, rate of systemic absorption, rate of elimination, et al.

Structure

- The type of linkage divides the local anesthetics into 2 categories:

<table>
<thead>
<tr>
<th>Esters</th>
<th>Amides (i before -caine)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cocaine</td>
<td>Lidocaine</td>
</tr>
<tr>
<td>2-Chloroprocaine</td>
<td>Bupivacaine</td>
</tr>
<tr>
<td>Procaine</td>
<td>Ropivacaine</td>
</tr>
<tr>
<td>Tetracaine</td>
<td>Mepivacaine</td>
</tr>
</tbody>
</table>

Amides vs. Esters

- Amides:
  - Metabolized by the liver
    - Aromatic hydroxylation, N-dealkylation, Amide hydrolysis
- Esters:
  - Relatively unstable in solution
  - Metabolized by plasma cholinesterases
    - Hydrolysis occurs at ester linkage
    - p-Aminobenzoic acid (PABA) metabolite can induce allergic-type reactions in a small percentage of patients

Clinical Usage

- Provide anesthesia and analgesia through several routes of delivery
  - Topical
  - Intravenous
  - Epidural
  - Intrathecal

<table>
<thead>
<tr>
<th>Drug</th>
<th>Onset</th>
<th>Max dose (mg/kg)</th>
<th>Max dose with Epi (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lidocaine</td>
<td>Rapid</td>
<td>4.5</td>
<td>7</td>
</tr>
<tr>
<td>Mepivacaine</td>
<td>Medium</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>Slow</td>
<td>2.5</td>
<td>3</td>
</tr>
<tr>
<td>Ropivacaine</td>
<td>Slow</td>
<td>4</td>
<td>N/A</td>
</tr>
<tr>
<td>Tetracaine</td>
<td>Slow</td>
<td>1.5</td>
<td>N/A</td>
</tr>
<tr>
<td>Chlorprocaine</td>
<td>Rapid</td>
<td>10</td>
<td>15</td>
</tr>
</tbody>
</table>

Toxicity

- Systemic absorption varies by site of injection (and is related to the vascularity of the tissue)
  - IV > tracheal > intercostal > caudal > epidural > brachial plexus > sciatic/femoral > subcutaneous
- Rate and extent of systemic absorption also depends on dose, the drug's intrinsic pharmacokinetic properties, and the addition of a vasoactive agent (i.e. epinephrine).
Toxicity

- CNS toxicity
  - Local anesthetics readily cross the blood brain barrier
  - Clinical manifestations: Lightheadedness, tinnitus, tongue numbness → CNS depression, seizure → coma

- Cardiovascular toxicity
  - Dose dependent blockade of Na channels → disruptions of cardiac conduction system → bradycardia, ventricular dysrhythmias, decreased contractility, cardiovascular collapse/circulatory arrest
  - In general, much greater doses of local anesthetics are required to produce cardiovascular toxicity than CNS toxicity

Treatment of LA toxicity

- Prevention is the 1st step
  - Avoid intravascular injection (aspirate prior to injection, small test doses), use appropriate monitoring
- Treatment if signs of toxicity
  - Stop local anesthetic
  - Begin supportive care – ACLS: CPR, meds (may need decreased dose of epi), airway management as appropriate
  - Initiate early intralipid (IL) therapy
    - Bolus IL 20% 1.5 ml/kg over 1 minute
    - Follow by infusion of 0.25 ml/kg
    - May repeat boluses q3-5 min
    - Total dose 12 ml/kg
    - Consider early initiation of cardiopulmonary bypass

References


Malignant Hyperthermia

Basics

Definition
- A hypermetabolic crisis that occurs when susceptible patients are exposed to a triggering anesthetic agent; underlying defect is abnormally increased Ca²⁺ levels in skeletal muscle causing acceleration of muscle metabolism

Genetics
- Genetic hypermetabolic muscle disease; autosomal dominant inheritance with variable penetrance and expression
- At least 6 chromosomal loci identified, but >80 genetic defects associated with MH
- Ryanodine receptor-1 (RYR-1), the skeletal muscle Ca²⁺ channel regulator, is best characterized

Incidence
- Rare, see in 1:15,000 pediatric vs. 1:40,000 adult patients
- May occur on a patient’s 2nd exposure to triggers (nearly 50% of MH episodes had at least one uneventful exposure to an anesthetic prior)
- May occur late in the anesthetic, possibly even in PACU
- Risk factors include personal/family history of MH, pediatric age, comorbid myopathies, caffeine intolerance, history of unexplained fevers/cramps/weakness, trismus on induction (precedes 15-30% of MH)

Excitation-Contraction Coupling

MH: Depolarization → mutant RYR-1 receptor remains open → unregulated calcium entry into cell from sarcoplasmic reticulum → sustained contraction → heat generation, CO₂ production, and cell damage
Sequence of Events

1. Triggers
   • All potent inhalational agents (except N₂O)
   • Succinylcholine

2. Increased Cytoplasmic Free Ca²⁺
   • Masseter muscle rigidity (trismus*)
   • Total body rigidity

3. Hypermetabolism
   • Increased CO₂ production (most sensitive and specific sign of MH!)
   • Increased O₂ consumption
   • Increased heat production
   *not all patients with trismus will go on to have MH, and not all MH cases will be heralded by trismus
   **Earliest recognized signs of MH= masseter muscle rigidity, tachycardia, and hypercarbia

4. Cell Damage
   – Leakage of K⁺, myoglobin, CK (may see dark-colored urine)

5. Compensatory Mechanisms
   – Increased catecholamines - tachycardia, hypertension, cutaneous vasoconstriction
   – Increased cardiac output - decreased Svo₂, decreased Pao₂, metabolic acidosis
   – Increased ventilation - increased ET_co₂, increased Ve
   – Heat loss - sweating, cutaneous vasodilation

6. Temperature Rise
   – A late and inconsistent sign of MH!
   – Temperature can rise 1-2°C every 5 minutes.

7. Secondary systemic manifestations
   – Arrhythmias
   – DIC
   – Hemorrhage
   – Cerebral Edema
   – Acute Renal Failure
   – Compartment Syndrome
   – Death (due to DIC and organ failure as result of delayed administration of dantrolene)

***The signs & symptoms of MH are seen often in the OR and are non-specific***
It’s important to be thinking of MH as missing it will have devastating consequences.
Clinically, you may first see trismus, but often hypercarbia will be your first sign.
Without another reasonable explanation for this (hypoventilation, pneumoperitoneum),
you should start looking for other signs. Look at your monitors – is there increased oxygen consumption? Tachycardia? Hypertension? Arrhythmias? Hyperthermia? Look at your patient – are they sweating? Rigid? Any combination of these findings should then make you want to rule out MH – consider an ABG (mixed metabolic and respiratory acidosis & hyperkalemia).

Differential Diagnosis

- Neuroleptic Malignant Syndrome (NMS) (in patients receiving antidopaminergic agents or in withdrawal from dopamine agents as in Parkinson’s)
- Thyroid Storm (would not see hyperkalemia or acidosis)
- Sepsis (b/c see fever, tachypnea, tachycardia, metabolic acidosis)
- Pheochromocytoma (∙HR, ∙BP, but normal EtCO₂ and Temp)
- Drug-induced (e.g. ecstasy, crack, amphetamines, PCP, LSD)
- Serotonin Syndrome (associated drugs interactions MAOIs + merperidine or MAOIs + SSRIs)
- Iatrogenic Hyperthermia
- Hypercarbia from CO₂ insufflation for laparoscopy (see EtCO₂ with tachycardia)

Treatment (Acute Phase)

1. Immediate reactions
   – Call for help; get MH cart (contains ALL the drugs you need)
   – D/C volatile agents and succinylcholine, switch to 100% O₂ and increase fresh gas flows
   – Notify surgeon; halt surgery ASAP, or continue with non-triggering agents (TIVA) if necessary.
   – Call the MH Hotline 1-800-MH-HYPER.
   – Check an ABG and place a foley catheter

2. Give Dantrolene, give more dantrolene
   – 2.5 mg/kg IV push.
   – Dissolve 20 mg in 60 ml sterile, preservative-free H₂O
   – Repeat until signs of MH are controlled – titrate to HR/CO₂
   – Sometimes, more than 10 mg/kg is necessary (= 35 vials of dantrolene! – consider dedicating an assistant to this).

Dantrolene
- A hydrophobic, hydantoin derivative with 12 hour t₁/₂
- Interferes with excitation-contraction coupling by binding the RYR-1 Ca²⁺ channel
- Relatively safe drug; causes generalized muscle weakness (including respiratory muscles).
- Formulation contains mannitol (hope you placed a foley!)
- Can also be used to treat NMS or thyroid storm.
Treatment (Acute Phase)

3. Treat acidosis
   - Hyperventilate patient.
   - Bicarbonate 1-2 mEq/kg until ABG available.
4. Treat hyperthermia
   - Cool if T > 39°C, but D/C if T < 38°C.
   - Apply ice to body surfaces; Cold NS via IV; Lavage stomach, bladder, or rectum PRN.
5. Treat hyperkalemia
   - Hyperventilate
   - Bicarbonate
   - Insulin & glucose (10 units in 50 ml D50)
   - Calcium (10 mg/kg CaCl₂, or 10-50 mg/kg Ca gluconate)
6. Treat dysrhythmias
   - Standard therapies, but avoid CCBs in the presence of dantrolene (may promote hyperkalemia).
   - May need antiarrhythmic if persists despite correction of hyperkalemia and acidosis
7. Maintain UOP/place foley
   - Lasix (1 mg/kg) (to establish diuresis and prevent ARF), and/or
   - Mannitol (0.25 g/kg) (dantrolene also contains mannitol)
8. Continue to monitor
   - ETCO₂, Temp, UOP & color, Electrolytes, ABG, CK, PT/PTT/INR

Treatment (Post Acute Phase)

1. Observe in ICU for at least 24 hours.
   - Recrudescence rate is 25%.
2. Continue Dantrolene
   - 1 mg/kg IV q4-6hrs for at least 24 hours.
3. Follow labs (watch for DIC, renal failure)
   - ABGs, CK, myoglobinuria, coags, electrolytes, UOP and color
4. Counsel patient and family
   - Future precautions.
   - Refer to MHAUS.
5. Refer patient and family to nearest Biopsy Center for follow-up.

Who is Susceptible to MH?

- Since autosomal dominant inheritance pattern, all closely related family members considered susceptible in absence of testing
  - This is even if have had previous uneventful anesthetics
- Several rare musculoskeletal disorders linked to MH
  - Core Disease
  - King Denborough Syndrome
  - Multiminicore myopathy
- Other disorders:
  - Muscular dystrophy and other neuromuscular diseases upon exposure to triggering agents have weak associations with MH-like events
  - Definitively avoid succinylcholine as can cause rhabdomyolysis, controversial whether to avoid volatile anesthetics; experts believe brief exposure should be small risk (i.e. inhalational induction in pediatric patients)
  - Should monitor capnography, minute ventilation, and core temperature; experts suggest that there be means to check serum electrolytes and urine screen for myoglobin if patient is signs of neuromuscular disorder so can document that individual has not suffered complication from anesthetic
  - History of exertional heat stroke—some suggestion that these people may harbor genetic changes found in MH susceptible individuals

Susceptibility Testing

Caffeine-Halothane Contracture Test (CHCT)
- Gold Standard
- Takes fresh muscle biopsy and exposes to triggers
- Sensitivity >97%, Specificity 80-93% (rule-out)
  - 10-20% false positive rate but zero false negative rate
- Available at 9 U.S. testing centers

Molecular Genetics
- RYR1 mutation screening
- Low sensitivity, but high specificity (rule-in)
  - Only screens for 20% of recognized mutations
- Typically reserved for patients with a positive CHCT, relatives of known MH susceptibility, or patients with highly suspicious MH episode.

Prevention in Susceptible Patients

Machine
- Change circuit and CO₂ absorbent
- Remove or disable vaporizers
- Flush machine at FGF of 10 L/min for ≥20 minutes and during case keep flows > 10L/min to avoid “rebound phenomenon” (see release of residual volatile anesthetic agent when FGF is reduced after a set period of flushing)

Monitors
- ASA monitors, especially temperature and \( \text{ETCO}_2 \)

Anesthetic
- Avoid succinylcholine and volatiles
- All other non-triggering agents are OK (including \( \text{N}_2\text{O} \))

Emergency
- Know where to find the MH cart
- Have dantrolene available
Why Antibiotics?

Because in 1984 a study including 51 acute care hospitals in New York State found that surgical site infection (SSI) was the most common adverse surgical event (and the second most common adverse event overall).


Timing of prophylaxis

• Antibiotic therapy should be given within 60 min prior to surgical incision for adequate serum drug tissue levels at incision.
• If a proximal tourniquet is used, the entire antibiotic dose should be administered before the tourniquet is inflated.
• Exceptions: Active ongoing antibiotic therapy (usually in-patients) or after a specimen is sent for culture.
• Epic tip: Click on “Patient Summary”, then the “Micro” tab. It will show you which antibiotics the patient is on and when they need to be redosed.


Administration and Common Dosages

• To be given via slow infusion (reconstitute in 100ml NS and give with microdripper)
  – Vancomycin (Red Man Syndrome) – over 30-60 mins
  – Gentamicin (ototoxicity/nephrotoxicity) - over 30-60 mins
  – Metronidazole (low pH) – over 60 mins
  – Cipro – over 30 mins
  – Clindamycin (QT prolongation) – over 10-15 mins
  – Ertapenem – over 30 mins
• Typical dosages for antibiotics commonly used in the OR: (these are frequently requested dosages here at Stanford – however this may change given new published guidelines)
  – Ampicillin 1gm
  – Cefazolin 1-2gm (2gm for patients > 80kg)
  – Cefoxitin 1-2gm
  – Clindamycin* 600-900mg
  – Gentamicin* 1.5mg/kg
  – Metronidazole 500mg
  – Zosyn 3.375gm
  – Ceftriaxone 1gm
  – Vancomycin 1gm
  – Cipro 400mg
  * can potentiate neuromuscular blockers
• Adjust for renal insufficiency (except for Clindamycin and Ceftriaxone)

Note: Ertapenem 1gm is favored by Drs. Shelton and Rhoades for their colorectal cases.

References

- Malignant Hyperthermia Association of the United States (MHAUS, http://www.mhaus.org)
- UCLA Department of Anesthesiology (http://www.anes.ucla.edu/dept/mh.html)
These are the most up to date guidelines from the 2013 IDSA, ASHP, and SIS.


Re-Dosing Guidelines

According to Stanford Pharmacy Guidelines

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Re-dosing Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cefazolin</td>
<td>4 hours</td>
</tr>
<tr>
<td>Cefoxitin</td>
<td>3 hours</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>6 hours</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>8 hours</td>
</tr>
<tr>
<td>Ertapenem</td>
<td>24 hours (n/a)</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>n/a</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>n/a</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>n/a</td>
</tr>
</tbody>
</table>

Types of Procedures

- Clean procedures (i.e. ortho, breast)
  - 1st generation cephalosporin (Cefazolin/Ancef) covers staphylococci and streptococci
- Procedures involving bowel anaerobes, Gram neg - bacilli, enterococci
  - 2nd generation cephalosporin (Cefotaxin or Cefotetan)
  - Bowel aerobic gram-neg bacilli (i.e. E. coli) can be resistant, so consider adding metronidazole.
- Craniotomies
  - 3rd generation cephalosporin for good CSF penetration (i.e. Ceftriaxone)
- Procedures involving groin incisions (i.e. vascular surgery, hysterectomy, colorectal surgery)
  - Consider adding gentamicin, ciprofloxacin, levofloxacin, or aztreonam to cover gram-neg bacteria.

Allergies and Interactions

- Penicillins and cephalosporins have similar β-lactam ring
- True incidence of allergy in patients with a history of PCN allergy is less than 10%. Only IgE-mediated reaction (type I, immediate hypersensitivity reactions) are true allergic reactions.
- The cross-reaction rate between PCN and cephalosporins is substantially less than 10%
- History of PCN allergy is a general risk factor for allergic manifestations to antibiotic administration that may not be specific to cephalosporins
- Cross-reaction rate between 3rd generation cephalosporins and PCN approaches 0%!
- For PCN-allergic patients, consider vancomycin or Clindamycin ± one of the following for Gram neg coverage (ciprofloxacin, levofloxacin, gentamicin, or aztreonam)

Allergies and Interactions

- If the allergic reaction to PCN is only “rash” or “hives,” many attendings would give a cephalosporin, but always ask your specific attending!
- However, hx of anaphylactic reaction to PCN is an absolute contraindication to cephalosporins.
- Test dose: Not always done. However, it may be prudent to give 1ml of the antibiotic first to see if the patient will have a reaction. This test dose only decreases the anaphylactoid reaction, not anaphylaxis.
- Allergic reactions are more likely from neuromuscular blockers than antibiotics.
Special considerations

• The American Heart Association guidelines recommend prophylaxis for those with conditions that place them at increased risk for infective endocarditis AND for those at highest risk for adverse outcomes when endocarditis does occur. These are patients with:
- Prosthetic cardiac valve
- Previous history of infective endocarditis
- Congenital heart disease and completely repaired congenital heart defect if it’s within the first 6 months.
- Cardiac transplant patients who develop cardiac valvulopathy

• Bacterial Endocarditis prophylaxis
- Ampicillin 1-2gm IV, 30min prior to surgery and
- Gentamicin 1.5mg/kg IV, 30min prior to surgery
- IF PCN allergic, use Cefazolin or ceftriaxone 1gm IV

• For mitral valve prolapse, do not need prophylaxis because, while there is increased risk for IE, the most serious adverse outcomes of IE do not usually occur in patients with this condition.

• Do not need prophylaxis for bronchoscopy without biopsy, vaginal delivery, hysterectomy, or GI/GU procedures, including colonoscopy.

Hall Question

Each of the following drugs can enhance the neuromuscular blockade produced by nondepolarizing muscle relaxants EXCEPT

A. Calcium
B. Aminoglycoside antibiotics
C. Magnesium
D. Dantrolene
E. Intravenous lidocaine

- See next slide for answer.

Hall Answer

• (A) Many drugs can enhance the neuromuscular block produced by nondepolarizing muscle relaxants. These include volatile anesthetics, aminoglycoside antibiotics, magnesium, intravenous local anesthetics, furosemide, dantrolene, calcium channel blockers, and lithium. Calcium does not enhance neuromuscular blockade and, in fact, actually antagonizes the effects of magnesium. In patients with hyperparathyroidism and hypercalcemia there is a decreased sensitivity to nondepolarizing muscle relaxants and shorter durations of action. (Miller: Anesthesia, ed 6, pp 514-518; Stoelting: Pharmacology and Physiology in Anesthetic Practice, ed 4, pp 224-226, 395).

References

• American Society of Anesthesiologists, ACE Program 2008. Pages 44-47.

I met my next patient in the VA preop area. I did my physical exam and was ready to place the IV. I had the lidocaine needle at his skin and announced, "Small prick!" He responded, "Honey, that’s what my ex-wife used to tell me, too."

It was time to bring the patient to the OR, and I was pushing him on a gurney down the ASC hallway. I got lost along the way and took a wrong turn leading to a dead end. I tried to play it off that we had taken this round about way just to get a patient hat for the OR. Unfortunately, despite the Versed, I think he saw right through the subterfuge.
Wheeled the patient into the room for a hip fracture repair. Nurse on the computer. Myself, anesthesia attending and ortho resident move the patient to the OR bed at which point the pt chuckles and smiles. I ask "what's so funny?" He responds, "I just had about a million dollars worth of education move me from one bed to another."

I anesthetized a trauma patient with multiple fractures. We did his hip while he was still intubated and I gave him a fair amount of ketamine for multimodal analgesia. The surgeons told me that when they rounded on him after he was extubated, the patient said, “Thanks for fixing my hip, but what are you going to do about my hind legs?” The patient then proceeded to explain that his hind legs needed to be fixed because he was a “centaur.” When I did his ankle fracture a few days later he told me that, “The last time I had anesthesia, I had a ‘bad trip.’”
Topics for Discussion

1. Your IV infiltrates during induction. What are your options?
2. You get stuck with a needle. How do you protect yourself and the patient?
3. You can't deliver positive pressure. What are your next steps?
4. You witness an unprofessional exchange between a surgeon and a nurse/med student/resident/etc. Who should you talk to?
5. You encounter an unanticipated difficult airway. You know you're supposed to CALL FOR HELP. Who do you call and what do you ask for?
6. You inadvertently administer the wrong medication. What should you do and who should you tell?
7. Your patient tells you that he wants only the attending to perform invasive procedures. How do you respond?
8. The surgeon insists that the patient is not relaxed enough, even though you just re-dosed a NDMB 5 minutes ago. What are your options?
9. You administer antibiotics after induction. An hour later, incision has still not been made. What should you do?
10. The surgeon appears to be struggling and the patient is rapidly losing blood. The surgeon insists that he does not need help. What should you do?